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RESEARCH ARTICLES

Premature Mortality in the Kingdom of Saudi Arabia Associated with Particulate Matter Air Pollution from the 1991 Gulf War

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ABSTRACT

The State of Kuwait oil fires and military operations associated with the 1991 Gulf War resulted in substantially increased levels of airborne particulate matter (PM) in the Kingdom of Saudi Arabia (KSA) during 1991 and 1992. Using quantitative risk assessment methodology, this article estimates the increase in premature deaths in citizens of the KSA associated with the Gulf War–related increase in PM air pollution levels. Meta-analysis of daily time-series studies of non-accidental mortality associated with increased PM_{10} levels using two alternative methodologies yielded exposure-response relative risk functions of 2.7% and 3.5% per 50 $\mu g/m^3$ increase in PM_{10} concentration. Combining these exposure-response functions with estimates of the magnitude and duration of the increased PM_{10} exposure, the size of the exposed population and baseline mortality rates provided an estimate of approximately 1,080 to 1,370 excess non-accidental deaths of Saudi citizens during 1991–1992 associated with the Gulf War–related increase in PM levels.

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Key Words: particulate matter, Gulf War, risk assessment, mortality, Saudi Arabia.

INTRODUCTION

The August 1990 invasion of the State of Kuwait by Iraqi military forces resulted in a military response from a multinational coalition in January 1991. Although the duration of actual military combat was relatively short (on the order of several weeks), the potential public health impacts to non-combatants from the Gulf War extended for a period of time well beyond the actual military conflict. In addition to the direct impacts of the military conflict on public health in the State of Kuwait and in the Kingdom of Saudi Arabia (KSA), which served as the main staging area for the coalition forces, substantial environmental damage from air and water pollution occurred in the Gulf Region from the destruction of hundreds of oil wells and the operation of many thousands of military vehicles and heavy equipment. A number of events during the 1991 Gulf War had the potential to contribute to increased levels of air pollution, especially particulate matter (PM), and thus adversely affect the health of the population of the KSA. Most notable were: (1) burning of more than 600 oil wells, pools of spilled oil, and numerous oil-filled trenches in the State of Kuwait resulting in massive plumes of smoke, soot, and other combustion products that were carried by prevailing winds over large areas of the KSA; (2) disruption of large areas of the desert in the State of Kuwait, southern Republic of Iraq, and northeastern KSA affected by military operations that left the desert surface vulnerable to wind erosion and increased the already high concentrations of wind-blown PM that can occur in the KSA; and (3) increased mobile source emissions from an estimated 15,000 primarily diesel-powered military vehicles used to conduct and support the military campaign (Sadiq and McCain 1993).

Although the adverse public health consequences of environmental degradation due to military conflict have been previously recognized (Leaning 1993), there is a paucity of published assessments of health impacts to non-combatants. The potential scope of environmental damage from air and water pollution stemming from the 1991 Gulf War was recognized almost immediately upon cessation of hostilities (UNEP 1991). The United Nations Compensation Commission (UNCC) was established by the United Nations Security Council in 1991 to process claims and pay compensation for damages and losses resulting from the Republic of Iraq's invasion and occupation of the State of Kuwait (UNSC 1991). Widespread recognition of potential public health impacts from the war-related environmental degradation was the basis for including consideration of compensation claims made against the government of the Republic of Iraq for damages to the health of their citizens from environmental pollution exposures, as well as for remediation of natural resource damages, as part of this process (UNSC 2001). However, experience with the application of risk assessment techniques to the environmental consequences of military conflicts is quite limited, with a focus typically on combatant exposure to specific contaminants related to munitions (*e.g.*, depleted uranium [Bleise *et al.* 2003] and dioxins from Agent Orange [Institute of Medicine 2003]). This article addresses the health consequences of one prominent environmental pollutant exposure arising from the conflict—PM from the oil fires and disturbed desert surface dust that spread over eastern KSA, as well as from military vehicle exhaust.

The objective of this article is to estimate the increase in non-accidental premature deaths in citizens of the KSA associated with the increase in war-related PM air pollution levels using quantitative risk assessment methodology. This risk assessment was conducted as a component of the Public Health Monitoring and Assessment program initiated in 2002 by the KSA to document and assess the public health impacts of the 1991 Gulf War (KSA PME 2004) under the UNCC Gulf War compensation claims process. The program was authorized and funded by the UNCC.

DATA SOURCES AND METHODS

The risk assessment paradigm described in the National Research Council (NRC) document *Risk Assessment in the Federal Government: Managing the Process* (1983) was utilized for this analysis. The paradigm involves application of the following steps: hazard identification, dose-response assessment, exposure assessment, and risk characterization. This paradigm has been used in numerous air pollution health effects analyses, including the U.S. Environmental Protection Agency's (USEPA) recent risk assessment of health outcomes related to the current and alternative national ambient air quality standards for PM (USEPA 2005). A schematic diagram of the overall process used to calculate the PM-related excess deaths is provided in Figure 1. In order to estimate the number of Saudi deaths attributable to the war-related increase in airborne PM, information was needed on: (1) the magnitude and duration of the increase in PM concentrations, (2) the exposure-response relationship between increased PM levels and mortality, (3) the size of the exposed population, and (4) the number of deaths that would normally be expected in the exposed population in the absence of elevated levels of airborne PM (baseline mortality).

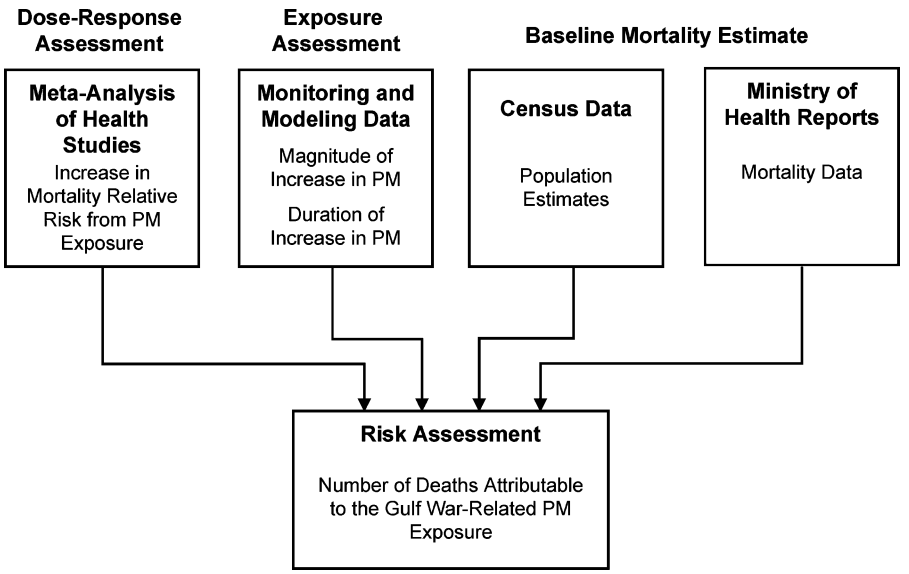

Figure 1. Schematic diagram of risk assessment process.

Hazard Identification

The Air Quality Criteria for PM (Criteria Document) released in October 2004 by the USEPA (USEPA 2004) provided the scientific basis for the hazard identification. This document contains an extensive compilation and assessment of the results of peer-reviewed clinical, epidemiological, and toxicological studies of the association between elevated PM_{10} concentrations with various adverse health outcomes, including premature mortality, published in scientific journals during the period from 1997 to 2003. The Criteria Document concludes that "the strength of evidence across such [human health] endpoints includes especially strong evidence for PM_{10} associations with total (nonaccidental) mortality" (USEPA 2004).

Dose-Response

Concentration-response functions for the risk of premature death from exposure to increased PM_{10} concentrations were developed from meta-analyses of epidemiological studies of PM_{10}-related mortality. All dose-response estimates used in the meta-analyses were from daily time-series studies of total non-accidental mortality and PM_{10} exposure published in the peer-reviewed literature and compiled from Table 8A-1 in the Criteria Document (USEPA 2004). To be included in the meta-analyses, study results had to be reported as the percentage increase in relative risk (%RR) with a 95th percentile confidence interval of non-accidental mortality corresponding to a 50 $\mu g/m^3$ increase in PM_{10} concentration. Because the duration of increased PM_{10} exposure for the Saudi population was limited to approximately a 14-month period, only results from short-term exposure studies reporting the association between daily ambient PM_{10} levels and all-cause mortality, excluding accidental deaths, were included in these meta-analyses.

Thirty-one study results listed in the Criteria Document reported results that met the eligibility requirements. These studies evaluate the PM_{10}-mortality association at numerous international locations using time-series analyses for multiple year periods. Because of a problem identified with the use of the original default settings for convergence criteria in the Generalized Additive Model (GAM) statistical software package (Dominici *et al.* 2002), the meta-analyses included results from 14 studies that were reanalyzed using revised GAM convergence criteria (Health Effects Institute 2003).

Two alternative meta-analytic methodologies were utilized to estimate the average percent relative risk (%RR) from the identified studies. First, a fixed effects approach multiplied each study's %RR increase per 50 $\mu g/m^3$ PM_{10} for total non-accidental mortality by a weight factor calculated as the inverse of the variance of the natural logarithm of the relative risk (Greenland 1987; Checkoway *et al.* 2004). The second approach evaluated the homogeneity among the study mortality risk estimates using the Q statistic, which is approximately distributed as a χ^2 (Dersimonian and Laird 1986). Following the homogeneity assessment, the random effects variance component was calculated and used as a revised weight factor for the summary %RR increase estimate under a random effects model (Berlin *et al.* 1993; Lipsey and Wilson 2001). Analyses were performed using Microsoft Excel Version 2003 (Microsoft Corporation, Redmond, WA, USA).

Table 1. Particulate monitoring conducted during the oil fire period.

Organization	Location	Parameters	Frequency	Period
Saudi Aramco	Dhahran	PM_{10}	Daily	Apr.–Nov. 1991
	Abqaiq	PM_{10}	Daily	Apr.–Nov. 1991
	Rahimah	PM_{10}	Daily	Apr.–Nov. 1991
	Tanajib	PM_{10}	Daily	Apr.–Nov. 1991
	E-W Pump #6	PM_{10}	Daily	May–Nov. 1991
KFUPM/RI	Dhahran	PM_{10}	Daily	Apr.–Nov. 1991
		PM_{15}	Daily	May–Nov. 1991
		Port. PM_{10}	Daily	Apr.–Aug. 1991
		TSP	Daily	Mar.–Nov. 1991
MEPA	Khafji	Port. PM_{10}	Alternate day	Apr.–Nov. 1991
		PM_{10}	Alternate day	Apr.–Nov. 1991
		TSP	Alternate day	Apr.–Nov. 1991
	Jubail	Port. PM_{10}	Alternate day	Apr.–Nov. 1991
	Riyadh	Port. PM_{10}	Alternate day	May–Nov. 1991
	Qaisumah	Port. PM_{10}	Alternate day	Apr.–Nov. 1991
Env. Prot. Dept.	Bahrain	PM_{10}	Daily	May 1991

Source: KFU PM/RI 1992, Table 7-1, page 129.

Exposure Assessment

Substantially increased concentrations of PM in the KSA were recorded during 1991 and 1992 by air pollution monitors. Key parameters for particulate matter air monitors operating in the eastern coastal and central regions of the KSA during the oil fire period are provided in Table 1. The war-related oil fires impacted a large portion of the eastern coastal and central portions of the KSA. Figure 2 indicates the area of the KSA impacted by 25 or more days of "moderate smoke" based on overhead daily afternoon satellite images from the NOAA-11 polar orbiting meteorological satellite. "Moderate smoke" corresponds to the visible boundaries of the smoke plume, excluding days where cloud cover obscured observation of the smoke plume (Johnson and Edmands 2006). The analysis period included the Gulf War oil well conflagration period from May 1991 until the oil fires were extinguished in November 1991, as well as the December 1991–July 1992 period when substantial military vehicle activity continued. The area directly affected by military activity is also indicated on the map, although the geographic region impacted by the disturbed dust was likely broader due to transport of the desert dust by prevailing winds.

The increase in the concentration of airborne PM_{10} that occurred in the KSA during 1991 and 1992 was estimated from a variety of sources in the KSA. Monthly mean PM_{10} concentrations measured at various locations in eastern KSA (Jubail, Rahimah, Dhahran, and Abqaiq air monitoring sites) were available to a varying extent for periods before, during, and after the conflict (1988–1995). In order to estimate the increased PM_{10} pollution due to the conflict and to allow for seasonal differences in PM_{10} concentrations that occur in the region, the increases in PM_{10} concentrations attributable to the conflict were estimated by comparing the mean monthly PM_{10} concentrations in eastern KSA during and immediately after the conflict (1991–1992)

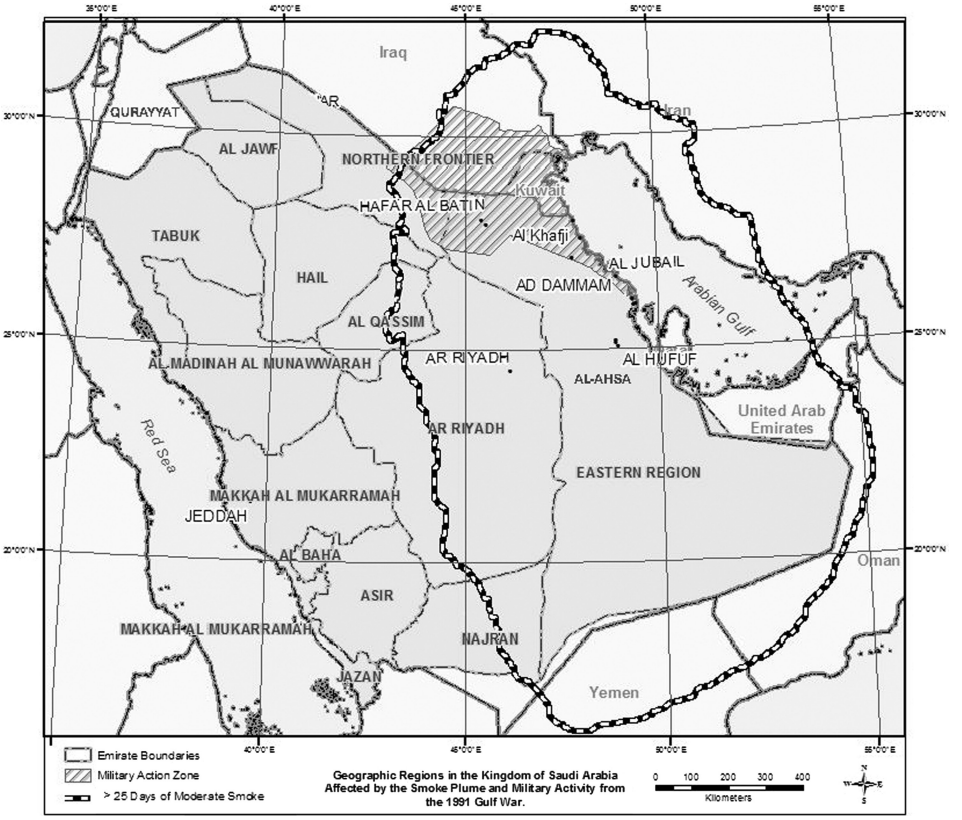

Figure 2. Geographic regions in the Kingdom of Saudi Arabia affected by the smoke plume and military activity from the 1991 Gulf War.

with PM_{10} levels measured during the same month for 3-year periods before and after the conflict (1988–1990 and 1993–1995).

Examination of PM_{10}concentrations recorded from monitors in the Eastern Region of KSA during the time of the Gulf War indicated a substantial degree of geographic similarity in the pattern of PM_{10} concentrations for the period 1991–1992 (Figure 3). Analysis of correlations of log-transformed monthly mean PM_{10} concentrations between monitoring sites across the Eastern Region of the KSA for both the war period (1991) and non-war period (1995) found generally high correlations between sites (Table 2), indicating that this region could be treated as a single air shed and that PM_{10} levels could be reasonably presumed to be uniformly distributed for areas where monitored data were not available. In addition, the monitored data were compared in terms of spatial distribution to modelled estimates of oil fire-related total suspended particulate matter (TSP) concentrations from the U.S. National Oceanic and Atmospheric Administration's HYSPLIT model (Draxler *et al.* 1994) obtained from the United States Army Center for Health Promotion and Preventive Medicine.

Table 2. Pearson correlation coefficient among sampling sites across eastern Saudi Arabia.

		1	2	3	4	5	6	7	8	9	10	11	12	13	14
1	Abqaiq 91	1													
2	Dhahran 91	0.98	1												
3	Rahimah 91	0.95	0.99	1											
4	Tanajib 91	0.96	0.99	0.98	1										
5	Dhahran 91h	0.95	0.94	0.95	0.94	1									
6	Jubail 91	0.92	0.70	0.68	0.73	0.73	1								
7	KKMC 91	0.95	0.94	0.88	0.98	0.58	0.80	1							
8	Kohbar 91	0.75	0.86	0.88	0.87	0.76	0.57	0.95	1						
9	Jubail 91	0.94	0.94	0.93	0.91	0.85	0.54	0.90	0.71	1					
10	Abqaiq 95										1				
11	Dhahran 95										0.89	1			
12	Rahimah 95										0.85	0.98	1		
13	Tanajib 95										0.90	0.95	0.95	1	
14	Jubail 95										0.86	0.92	0.93	0.86	1

Underline indicates statistical significance at $p = .05$.

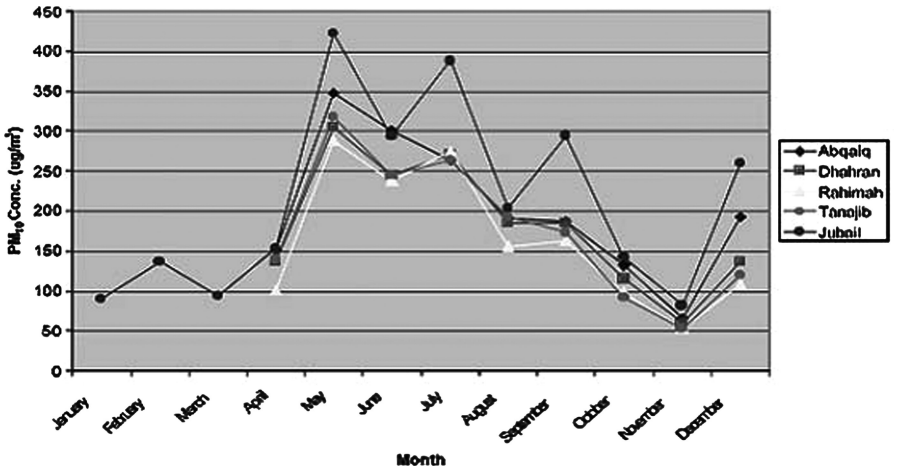

Figure 3. Monthly average PM_{10} concentration during 1991.

Risk Characterization

Baseline Mortality Estimation Process

The baseline non-accidental mortality rate for Saudi citizens living in the areas of the KSA affected by war-related increases in PM_{10} pollution during 1991 and 1992 was not directly available from the KSA Central Department of Statistics (CDS), the KSA Ministry of Health (MoH), or the World Heath Organization (WHO). Therefore the baseline mortality rate was estimated by the process outlined later and summarized in Table 3 as a numbered sequence of steps described next.

In Step 1, the number of Saudi deaths that occurred in MoH hospitals from April 2000 through March 2001,[1] derived from the MoH Annual Health Report for this time period, was used as the starting point (KSA MoH 2001). For Step 2, Eq. (1) was used to estimate the Total Saudi Deaths in 2000 (TSD_{2000}) by multiplying the number of Saudi deaths that occurred in MoH hospitals ($SD_{MoH2000}$) by the ratio of all deaths (male and female) in the KSA from the WHO Life Tables for KSA for 2000 (WHO 2006) ($TD_{WHO2000}$) to all deaths (Saudi and non-Saudi, male and female) in MoH hospitals in 2000 ($TD_{MoH2000}$).

$$TSD_{2000} = SD_{MoH2000} \times (TD_{WHO2000}/TD_{MoH2000}) \quad (1)$$

In Step 3, the number of deaths that occurred in a given year was assumed to be approximately proportional to the population of the Kingdom during that year. Population growth is generally assumed to be exponential, that is, a population increases by a similar percentage each year. Thus the population in the nth year after a base year can be estimated using Eq. (2).

$$P_n = P_0 x(1 + r)^n \quad (2)$$

where: P_n = the population in the nth year after the base year, P_o = the population in the base year, r = the fractional annual population growth, and n = the number of years elapsed after the base year.

The fractional yearly population change (r) in the KSA between 1992 and 2000, the years for which population data were available, was assumed to be constant and was estimated using Eq. (3).

$$r = [(KSAPop_{2000}/KSAPop_{1992})^{(1/n)}] - 1 \quad (3)$$

where (n) is the number of years elapsed between 1992 to 2000 (8 years).

In Step 4, the total Saudi Deaths in 1991 (TSD_{1991}) was calculated using Eq. (4) where n equals 9, the number of years elapsed between 2000 and 1991. For Total Saudi Deaths (TSD_{1992}) in 1992, n in Eq. (4) equals 8, rather than 9.

$$TSD_{1991} = TSD_{2000}/(1 + r)^n \quad (4)$$

The PM_{10} concentration increase in the Arabian Gulf coastal portion of the Eastern Region was estimated to be somewhat greater and more prolonged than that in the remainder of the exposed portion of the Kingdom because the impact of the smoke from the oil fires was greatest along the coast and because the coastal area was directly down wind from the portions of the Republic of Iraq, the State of Kuwait, and

[1]The equivalent Hijri calendar date is 1421 A.H.

Table 3. Estimation of baseline non-accidental Saudi deaths in exposed regions for war-related PM_{10} exposure period.

Step	Description	KSA	Eastern and Al-Ahsa Region	Other exposed regions	Data source
1	Saudi Deaths in MoH Hospitals 2000	24,669			KSA MoH, Annual Health Report, 2001
2	Total WHO Deaths in 2000	94,855			WHO Life Tables for Saudi Arabia - 2000
	Total MoH Hospital Deaths in 2000	35,833			KSA MoH, Annual Health Report, 2001
	Total WHO Deaths/MoH Hospital Deaths	2.647			
	Total Saudi Deaths in 2000	65,302			
3	Yearly Population Change 1992–2000	3.00%			KSA Central Department of Statistics[1]
			1991		
4	Total Saudi Deaths in 1991	50,049			
5	% Saudi Deaths in MoH Hospitals in Region	100.00%	10.97%	47.65%	KSA MoH, Annual Health Report, 1999[2]
	Saudi Deaths in Region in 1991	50,049	5,490	23,848	
6	% Non-Accidental Deaths	83.69%	83.69%	83.69%	KSA MoH, Annual Health Reports, 1998-2001[2]
	Non-Accidental Saudi Deaths in Region in 1991	41,866	4,595	19,959	

(Continued on next page)

Table 3. Estimation of baseline non-accidental Saudi deaths in exposed regions for war-related PM_{10} exposure period. *(Continued)*

Step	Description	KSA	Eastern and Al-Ahsa Region	Other exposed regions	Data source
7	% of Annual Expos. Per. (6 mos.: 5/91–10/91)	50.00%	50.00%	50.00%	
	Exposed Period Non-Accidental Saudi Deaths	20,943	2,297	9,979	
		1992			
4	Total Saudi Deaths in 1992	51,550			
5	% Saudi Deaths in MoH Hospitals in Region	100.00%	10.97%	47.65%	KSA MoH, Annual Health Report, 1999[2]
	Saudi Deaths in Region in 1992	51,550	5,655	24,564	
6	% Non-Accidental Deaths	83.69%	83.69%	83.69%	KSA MoH, Annual Health Reports, 1998-2001[2]
	Non-Accidental Saudi Deaths in Region in 1992	43,142	4,733	20,557	
7	% of Annual Expos. Per. (8 mos.: 12/91–7/92)	66.67%	66.67%	66.67%	
	Exposed Period Non-Accidental Saudi Deaths	28,763	3,155	13,706	

[1]2000 Demographic Survey 2 (by age, sex, nationality, and religion); 1992 Population and Housing Census 3 (by age, sex, and nationality); [2]Includes only deaths that occurred in Ministry of Health hospitals. Tables 1-17, 1-35, 1-34, and 1-33, respectively, from each yearly report summarize the data by cause of death, nationality, and gender. Tables 1-37, 1-35, and 1-34, available in the reports published in 1999 through 2001, provide data on mortality trends by disease group for the current and two preceding years. Table 1-36, which was only included in the 1999 report (for the period May 1998 to April 1999), provides data on the distribution of deaths by region and nationality.

KSA desert areas most severely affected by military activities associated with the war. Therefore the impact of increased PM_{10} levels on mortality was estimated separately for the areas near the Arabian Gulf coast and the remainder of the exposed region (see Figure 1). The Eastern Province, or administrative region, of the KSA is divided into three Ministry of Health regions, used for statistical reporting purposes in the MoH annual reports. The three MoH regions in the Eastern Province are the Eastern Region, centered on Ad-Dammam, the Al-Ahsa region, centered on Al-Hofuf, and the Hafr Al-Batin region, centered on Hafr Al-Batin. The Eastern (Ad-Dammam) and Al-Asha Regions are close to the coast, whereas Hafr Al-Batin is approximately 300 kilometers inland (west of the coast) and, along with the remainder of the exposed region, was less affected by the smoke plume.

Information on the distribution of deaths by MoH region was only available for deaths that occurred in MoH hospitals from May 1998 to April 1999.[2] Therefore, in Step 5 it was necessary to assume that the regional distribution of all Saudi deaths in 1991 and 1992 was similar to that of Saudi deaths in MoH hospitals during May 1998 to April 1999. The fraction of Saudi deaths that occurred in the Eastern and Al-Ahsa MoH Regions and the other exposed MoH regions (Hafr Al-Batin, Riyadh, Qaseem, Ha'il, Northern, Al-Jouf, and Qurayyat) were calculated from the MoH Annual Report for this period (KSA MoH 1999). The total Saudi deaths in these regions in 1991 were estimated by multiplying the total Saudi deaths throughout the Kingdom in 1991 (TSD_{1991}) by their respective fractions of MoH hospital deaths.

The exposure-response function derived from the meta-analysis of the epidemiological studies discussed earlier relates the relative risk of all-cause non-accidental deaths to an incremental increase in PM_{10} concentration. Therefore, for Step 6, deaths from injuries and other external causes need to be removed from the total number of Saudi deaths to obtain the number of Saudi deaths from all other (non-accidental) causes. This was done by multiplying the total Saudi deaths in each region by the average fraction of deaths from all causes except those in the Injuries, Poisoning and External Causes category for 1998 through 2001 from the MoH Annual Reports for this period (KSA MoH 1998; KSA MoH 1999; KSA MoH 2000; KSA MoH 2001). The average fraction for all of the available years of data was used, rather than a single year, to obtain a more statistically stable value.

The estimated number of deaths from all non-accidental causes expected in the two regions was for an entire year, whereas the increased PM_{10} levels in 1991 and 1992 were for 6- and 8-month periods, respectively. Therefore, as indicated in Step 7, the expected annual deaths were multiplied by the appropriate fractions to account for the portions of 1991 (May-October = 50%) and late 1991–1992 (December-July = 67%) during which PM_{10} levels were elevated.

Excess Mortality Calculation Process

The key variables used to estimate total war-related Saudi deaths in the regions exposed to war-related increases in PM_{10} are summarized in Eq. (5). The estimated War-Related Saudi Deaths (WRSD) in the exposed regions due to war-related increases

[2]The equivalent Hijri calendar date is 1419 A.H.

in PM_{10} was summed across both exposed regions and for 1991 and 1992.

$$WRSD = NASD \times \%RR/50 \times \Delta PM_{10} \times EF \quad (5)$$

where NASD = baseline number of non-accidental Saudi deaths in each exposure region, %RR / 50 = percent relative risk increase in non-accidental deaths per 50 $\mu g/m^3$ increase in PM_{10} derived from the meta-analyses divided by 50 to yield percent relative risk increase per 1 $\mu g/m^3$; ΔPM_{10} = war-related increase in PM_{10} ($\mu g/m^3$) for the two major exposed regions of KSA; EF = fraction of the year during which the estimated increase in PM_{10} was observed.

RESULTS

Dose-Response

Results of the meta-analyses of the studies selected for development of the exposure-response function are presented in Table 4. Results are presented in terms of an increased relative risk of total non-accidental mortality per 50 $\mu g/m^3$ increase in PM_{10} levels, and include the reported study estimates and weighted relative risks for both the fixed effects and random effects methodologies. Using the fixed effects approach, individual mortality studies with relatively narrow confidence intervals due to larger observation sizes from multiple cities contributed relatively more weight to the average % RR. The summary risk estimate was 2.74% (95% CI: 2.47%, 3.01%) for a 50 $\mu g/m^3$ increase in PM_{10} levels.

For the random effects approach, the Q statistic was calculated to be 102.48 (χ^2 with 30 degrees of freedom, $p < .0001$). This indicated significant heterogeneity among study estimates of mortality risks. Therefore, we calculated the meta %RR using the random effects variance component of 0.000144 added to the inverse standard error of each study. The distribution of weighted %RR estimates was relatively smaller under a random effects model. This contributed to a higher average %RR of 3.47% (95% CI: 2.81%,4.13%) per 50 $\mu g/m^3$ increase in PM_{10} levels. Additional information regarding the meta-analyses results is provided in the Supplement.[3]

Exposure Assessment

Based on an analysis of the available monitoring data, the estimated increase in PM_{10} levels in the Eastern (Arabian Gulf coastal) Region of the KSA for the 6-month period from May through October 1991 ranged from 77–110 $\mu g/m^3$, with an average estimated increase of 94 $\mu g/m^3$ (Table 5). The estimated increase in PM_{10} levels for the 8-month period from December 1991 through July 1992 was estimated to be approximately 65% of that for the May through October 1991 period, or approximately 60 $\mu g/m^3$. These estimates of increased PM_{10} concentrations during these exposure periods were derived from an assessment using four PM_{10} monitoring locations for which data were available to compare PM_{10} levels during the 1991 oil fire period with those in the similar periods during 1988–90 and 1993–95. The war-related increase in airborne PM_{10} in the remainder of the exposed region of the KSA

[3]The supplemental material is available at www.jhsph.edu/RiskSciences/Research or upon request from the corresponding author.

Table 4. Results of the meta-analyses of the studies selected for development of the exposure-response function.

Ref #	Study	Period of study	Population	RR %**	Lower CI**	Upper CI**	Fixed effects wRR (1)	Random Effects wRR (2)
1	Dominici *et al.* (Re)	1987–1994	90 U.S. cities	1.4	0.9	1.9	160206.067	6749.028
2	Dominici *et al.*	1987–1994	20 U.S. cities	1.8	−0.5	4.1	7658.646	3637.173
3	Klemm and Mason (Re)	1979–1987	6 U.S. cities	3.5	2.0	5.1	17742.889	5189.951
4	Braga *et al.*	1986–1993	5 U.S. cities	4.0	2.6	5.3	23685.257	5423.450
5	Schwartz (Re)	1986–1993	10 U.S. cities	3.3	2.6	4.1	75352.151	6622.639
6	Levy	1990–1994	King County, WA	5.6	−2.4	14.3	650.432	600.160
7	Moolgavkar (Re)	1987–1995	Cook County, IL	2.4	1.3	3.5	34087.349	5874.601
8	Moolgavkar (Re)	1987–1995	Los Angeles, CA	2.4	0.5	4.4	10855.802	4352.719
9	Ostro *et al.* (Re)	1989–1998	Coachella Valley, CA	5.5	1.6	9.5	2891.220	2055.409
10	Fairley (Re)	1989–1996	Santa Clara Cty, CA	7.8	2.8	13.1	1816.779	1474.539
11	Pope *et al.*	1985–1995	Ogden, UT	12.0	4.5	20.1	888.998	803.156
12	Pope *et al.*	1985–1995	Provo, UT	1.9	−2.1	6.0	2477.956	1815.155
13	Schwartz and Zanobetti	1988–1993	Chicago, IL	4.5	3.1	6.0	20868.191	5462.615
14	Ito (Re)	1992–1994	Detroit, MI	3.3	−2.0	8.9	1427.171	1192.742
15	Gamble	1990–1994	Dallas, TX	−3.6	−12.7	6.6	371.321	356.607
16	Gwynn *et al.*	1988–1990	Buffalo, NY	12.0	2.6	22.7	537.710	522.323
17	Mar *et al.* (Re)	1995–1997	Phoenix, AZ	9.7	1.7	18.3	737.387	670.329
18	Burnett and Goldberg (Re)	1986–1996	8 Canadian cities	2.7	−0.1	5.5	5305.082	2995.495
19	Burnett *et al.*	1980–1994	Toronto, Canada	3.5	1.8	5.3	13918.547	4806.265
20	Katsouyanni *et al.* (Re)	1990–1997	29 European cities	3.3	2.7	3.9	117625.326	6761.406
21	Zanobetti and Schwartz (Re)	1977–1992	6 European cities	3.4	2.0	4.8	21665.295	5376.451

(Continued on next page)

Table 4. Results of the meta-analyses of the studies selected for development of the exposure-response function. *(Continued)*

Ref #	Study	Period of study	Population	RR %**	Lower CI**	Upper CI**	Fixed effects wRR (1)	Random Effects wRR (2)
22	Wordley *et al.*	1992–1994	Birmingham, UK	5.6	0.5	11.0	1643.251	1350.759
23	Hoek (Re)	1986–1994	The Netherlands	1.4	0.3	2.6	30312.469	5797.105
24	Ponka *et al.*	1987–1993	Finland	18.8	5.6	33.2	338.602	316.365
25	Peters *et al.*	1982–1994	Czech Republic	4.8	0.7	9.0	2567.203	1876.372
26	Stolzel *et al.* (Re)	1995–1998	Erfurt, Germany	6.2	0.1	12.7	1160.973	1006.799
27	Gouveia and Fletcher	1991–1993	Sao Paulo, Brazil	3.3	0.6	6.0	5806.143	3163.453
28	Hong *et al.*	1995–1996	Inchon, South Korea	4.1	0.1	8.2	2642.003	1915.066
29	Ostro *et al.*	1992–1995	Bangkok, Thailand	5.1	2.1	8.3	4646.987	2901.274
30	Castillejos *et al.*	1992–1995	Mexico City	9.5	5.0	14.2	2385.181	1817.889
31	Bremner *et al.*	1992–1994	London, UK	1.3	−1.0	3.6	7546.325	3601.796
	Number of Studies	31	Crude RR % Average^	4.855	Weighted RR % Average†		2.742	3.469

(Re) indicates reanalysis of previous study using stringent GAM model convergence criteria.

**All RR percent estimates are for standardized change in percent of relative risk per 50 $\mu g/m^3$ increase in PM_{10}.

(1) Fixed effects weighted relative risk = $[\ 1/\{(\ln(UCL_i) - \ln(LCL_i))/3.92\}2\ ] * RR\%_i$

(2) Random effects weighted relative risk = $[\ 1/(SE_i^2 + \upsilon_\theta)\ ] *RR\%$, where υ_θ is the random effects variance component ($\upsilon_\theta = 0.000144$)

^Formula for crude relative risk % average = (Σ RR%)/(Number of Studies).

†Formula for weighted relative risk % average = (Σ weighted RR%/Σ weights)

Table 5. Summary of PM_{10} concentrations in the eastern region of Saudi Arabia near the time of the 1991 Gulf War.

	Average PM_{10} Concentrations ($\mu g/m^3$) during May through October								PM_{10} Increase: 1991 vs.
Location	1988	1989	1990	1991	1992	1993	1994	1995	(1988–90 + 1993–95)
Abqaiq*	*129*	*169*	*138*	238	—	139	166	88	100
Dhahran*	*123*	*154*	*130*	218	—	139	148	84	88
Rahimah*	*117*	*148*	*124*	200	—	125	140	87	77
Jubail	176	202	186	294	214	154	263	119	110
Average increase during May through October 1991 vs. same period in other years									94

*Estimated values in italics.

(inland west of the Arabian Gulf coastal region) where there were no air monitoring data for the period from May through October 1991 is estimated to have ranged from approximately 60–90 $\mu g/m^3$, with an average increase of approximately 75 $\mu g/m^3$. This PM exposure level was estimated based on the HYSPLIT model results indicating a 15 $\mu g/m^3$ average differential in oil fire-related TSP levels (assumed to be mostly $\leq PM_{10}$ in diameter) between the coastal areas and those areas further inland that were less directly impacted by the oil fire smoke. Results from analysis of the monitored data were consistent in terms of spatial distribution with those from the HYSPLIT model (data not presented). PM_{10} concentrations measured at the various monitoring stations were typically 2 to 3 times greater than the levels projected by the HYSPLIT model, as the monitored data measured PM_{10} from all sources (crustal, oil fires, and other anthropogenic sources) whereas the model provided calculated TSP concentrations in the region related solely to the State of Kuwait oil fires.

No increase in PM_{10} exposure was calculated for November 1991 as available monitoring data did not indicate that such an increase occurred in the exposed region. The lack of a measured PM_{10} increase in November 1991 may be explained by the oil fires being extinguished by early November and by the relatively low wind speeds for that month, which minimized wind erosion of the disrupted desert surface during that period.

Risk Characterization

In Table 6 are summarized the values of the variables included in Eq. (4) described earlier and provides the number of excess deaths associated with exposure to increased levels of PM_{10} based on results from the two alternative meta-analytic techniques used to calculate the exposure-response function. From this analysis we estimate that 1082 (95%CI: 974,1188) to 1369 (95%CI: 1109,1629) excess deaths of Saudi citizens can be attributed to increased PM_{10} levels in 1991–1992 resulting from the Gulf War activities.

Table 6. War-related excess Saudi deaths in exposed regions from PM_{10} exposure.

Fixed Effects Method Weighted Average RR % = 2.742 per 50 $\mu g/m^3$ increase in PM_{10}					
Time period	Location	Average PM_{10} exposure ($\mu g/m^3$)	Dose-response per 1 $\mu g/m^3$ Incr. in PM_{10}	Baseline mortality in KSA	Excess Deaths
May 1991 to October 1991	Eastern Province	94	0.0005484	2288	118
	Remainder of Exposed Region	75	0.0005484	9940	409
December 1991 to July 1992	Eastern Province	60	0. 0005484	3156	104
	Remainder of Exposed Region	60	0.0005484	13711	451
			TOTAL	29095	1082
Random Effects Method Weighted Average RR % = 3.469 per 50 $\mu g/m^3$ increase in PM_{10}					
Time period	Location	Average PM_{10} exposure ($\mu g/m^3$)	Dose-response per 1 $\mu g/m^3$ Incr. in PM_{10}	Baseline mortality in KSA	Calculated Excess Due to PM_{10}
May 1991 to October 1991	Eastern Province	94	0.0006938	2288	149
	Remainder of Exposed Region	75	0.0006938	9940	517
December 1991 to July 1992	Eastern Province	60	0.0006938	3156	131
	Remainder of Exposed Region	60	0.0006938	13711	571
			TOTAL	29095	1369

DISCUSSION

This retrospective assessment of the increased risk of total non-accidental mortality in the population of the KSA associated with increases in PM_{10} levels from the 1991 Gulf War was conducted more than 13 years after cessation of the War as a major component of the KSA's claim for compensation of war-related health damages under the UNCC process. This analysis provides new insight into an important component of the health consequences for non-military personnel of the environmental degradation associated with this military conflict. However, significant data

availability limitations required the use of several assumptions and this analysis is therefore subject to substantial uncertainty.

A key source of uncertainty in the mortality estimate is the selection of an exposure-response function for the risk of mortality from increased PM_{10} exposure. While there is a substantial scientific literature base on this topic providing generally consistent findings of an association between increases in PM_{10} levels and increased risk of mortality, there is a wide range in the relative risk estimates for this association (USEPA 2004). Additionally, the major sources contributing to the Saudi population war-related ambient PM_{10} exposure included uncontrolled oil fires, heavy-duty military vehicle diesel emissions and disturbed desert dust that may have been contaminated with combustion-related residues, which differ substantially from PM_{10} sources in the U.S. and European epidemiologic studies included in the meta-analysis. Thus differences in the PM_{10} composition due to the different PM sources in the studies used to derive the exposure-response relationship compared to those in the Gulf War contributes to additional uncertainty. Other potential sources of uncertainty include underlying differences in the population of the KSA and the populations in the epidemiological studies, and extrapolation to pollution ranges different from those in the original studies.

Single and multi-city daily time-series studies of the relationship between increased PM_{10} levels and risk of mortality were selected for inclusion in the meta-analyses so as to be relatively consistent with the scale of exposure duration (approximately 14 months total) for the war-related increases in PM levels. The daily time-series studies generally provide lower relative risk estimates for mortality associated with a standardized increase in PM levels compared to results from long-term prospective studies (USEPA 2004) and thus provide a more conservative exposure-response relative risk estimate. Additionally, although selection by study authors of the most statistically significant single-day lag increases the potential for overestimation of effect due to publication bias (Anderson *et al.* 2005), studies that also found a statistically significant effect for multiple days may have underestimated the multi-day effect when reporting only the most significant single-day result (Schwartz *et al.* 2003). Thus study biases in both positive and negative directions must be considered as contributing to uncertainty in selecting an exposure-response function from meta-analysis results that include results from single-city, single-lag analyses.

Due to demographic data limitations described earlier, several assumptions were required in developing an estimate of baseline mortality for the KSA regions exposed to increased PM_{10} levels, as these mortality data were not available from other sources. Assumptions included: (1) the ratio of Saudi male and female deaths in MoH hospitals to total Saudi deaths was assumed equivalent to the ratio of total deaths in MoH hospitals to total deaths in KSA; (2) the number of deaths that occurred in a given year was assumed to be approximately proportional to the KSA population during that year; and (3) the regional distribution of all Saudi deaths in 1991 and 1992 was assumed to be similar to that of Saudi deaths in MoH hospitals during 2000.

The assumption of an average uniform PM_{10} exposure increase across a wide region of the KSA, including areas where no monitoring data were available, is a potential source of exposure assessment uncertainty. The lack of a more extensive local air pollution monitoring network necessitated assuming geographically

uniform exposures. This assumption may have resulted in exposure measurement error because it is possible that actual exposure could have been either over- or under-estimated. However, the likely impact of this exposure measurement error would be to underestimate the pollution effects (Zeger *et al.* 2000); thus our risk estimates may be conservative. Available monitoring data clearly show a pattern of increased exposure across the entire Eastern Region with decreasing exposures in the more inland portions of eastern KSA. Analysis of variability of the exposure data indicated that the temporal variability component is substantially greater than the spatial component, suggesting that assuming an average exposure across the exposed region should not bias the results. In addition, the lack of air monitoring data from the more central regions of the country where PM_{10} exposures were lower required us to estimate exposure by adjusting measured concentrations from the eastern regions using HYSPLIT modelled results. Although we believe this is a reasonable approach, it likely produced an underestimation of exposure because the HYSPLIT model only accounted for the oil fire component of PM levels.

CONCLUSIONS

While acknowledging that this risk assessment is subject to the various sources of uncertainty discussed earlier, these results provide an estimated range for premature non-accidental deaths among Saudi citizens associated with exposure to elevated PM_{10} air pollution levels resulting from the 1991 Gulf War. The estimate of approximately 1,080 to 1,370 excess deaths from these air pollution exposures provides a strong indication that the population of KSA suffered a substantial adverse health impact from the environmental degradation associated with this conflict.

The limitations of the available air pollution exposure data and the need to extrapolate from several data sources to develop the baseline mortality rate highlight some key challenges in conducting military conflict–related environmental health risk assessments, particularly in areas where detailed environmental, health, and mortality data needed for this type of analysis are not readily available. The inherently disruptive nature of military conflict on normal societal operations such as environmental monitoring and public health recordkeeping also presents potentially significant barriers to assessing the public health impacts of war-related environmental degradation.

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