APPENDIX KK

INDEPENDENT CONSULTANT REPORT

BY

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Report to Senate Veterans Affairs Comm., 2/15/98
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THE REPORT
The Environmental Air Pollution Situation:

Background Environmental Conditions:

The Persian Gulf countries, including Kuwait and Saudi Arabia, are generally arid. They have very high levels of particulate matter (PM), sometimes reaching daily levels of 3 mg/m3 (related primarily to dust storms). The PM is mostly in the smaller size fractions: in urban Kuwait (in a Kuwait report for 1980-84), 42.5% of daily TSP was < 3.3 um (called respirable suspended particulate - RSP, for which there are no U.S. or Saudi Arabian standards) and averaged 438.2 ug/m^3; during sand disturbances ("rising sand" and dust storms, 32% of the days), 46.7% was RSP compared to < 41% on other days, and average RSP was 598-1437 ug/m^3; during rainy and clear days (also 32% of days) average RSP was 162-183 ug/m^3; the lowest was during days of "other phenomena" (only 4% of days) when the RSP average was only 112 and the TSP average was only 192ug/m^3. The average daily total suspended particulate (TSP, $\leq$ ~26um) was 192-2347.5 ug/m^3; 36% of days were conditions in which average daily TSP exceeded old U.S. standards (24hr=260ug/m^3). Saudia Arabian studies also indicate high levels of TSP and particulate matter of $\leq$ 10um (PM_{10})[1]. (U.S. National Ambient Air Quality Standards [NAAQS] for PM_{10}: 24 hour = 150 and annual = 50 ug/m^3.) These are extremely high concentrations in relation to any standards or guidelines (AQGs) and indicate that many days would probably exceed alert levels and be acutely dangerous. for health. Background levels for other pollutants can only be inferred from the last days of monitoring data available after the oil well fires were extinguished, as discussed below. Background pollen, mold, and bacterial (specifically bacillU.S. sp.) data indicate likely health-related problems in populations allergic to those aero-allergens as well[2-4]. Other allergenic substances (e.g., pigeon allergen) are in reports, but not substantiated. Background conditions could have contributed significantly to the total concentrations of air pollutants with the additional air pollution produced by activities before, during, and even after the Gulf War, related to the various activities accompanying deployment of personnel.

Air Pollution Related to the Oil Well Fires:

During the oil well fires, levels of several pollutants were very high not only in the plumes but near them. Stevens et al. (1993)[5] has provided the most concise, scientifically valid, and complete report. They indicate two types of plumes, black (75%) made of carbon-chain (C-chain) agglomerates, and white (25%) primarily with sodium and chloride compounds (NaCl and $CaCl_2$), indicating that they included large amounts of seawater extracts. They found high PM (10um from 22-4356ug/m^3), sulfur dioxide (SO_2) (12-130, with an outlier of 320 ug/m^3, = a few hundred ppb) and sulfate (SO_4) (3-95 ug/m^3, related to seawater), with a total sulfur-carbon (S/C) ratio of 1.02% (after backgrounds subtracted, and consistent with other study), metals at the level of typical urban levels (below AQG and standards), little NOx, and very little PAH (<1ng/m^3). Findings from other studies reviewed[6-12], and others quoted by all of these authors, are essentially in agreement with these. Most of the papers conclude that the NOx, SOx, carbon monoxide and dioxide (CO, CO_2) are not high compared to other regional and global sources (and were below U.S. NAAQS). Other carbon/hydrogen/sulfur compounds (i.e., CH_4 and H_2S) were slightly above ambient levels. Nonmethane hydrocarbons were in the clean to moderately polluted range (55-827ppbv; species included: 65-82% alkanes, 7-23% alkenes, 5-19% aromatics)[7]. Thus, the oil well fires produced significant amounts of air pollutants, but mostly around the fires themselves, and not to the extent that was initially thought likely (nor in the possible massive extensive regional dispersion originally assumed).

Regional Air Pollution Problems:
One study[8] found increased PM, SO_2, and nitrogen dioxide (NO_2) increased 120km downwind (and a slight increase above background in ozone (O_3) 2000 km away). Importantly, the evidence is that the smoke plumes generally head south and west, away from primary residential areas in Kuwait, but sometimes toward other countries[7]. Also, the plumes did not reach the stratosphere, although elemental analyses in Bahrain (SE of Kuwait), using nickel (Ni) and vanadium (V) and even in Hawaii (evaluating crustal and non-crustal Ni, V, Zn-zinc, Al-aluminum, lead-Pb, and sodium/chloride - Na/Cl) indicate some long-range transport of some of the PM[6,11], but none of the metals were at concentrations of concern. Several studies also reported a slight decrease in solar radiation locally and/or regionally. Ecological concern was regional and short-term[7-9,13,14].

Air Pollution Problems in Residential Areas:
Concentrations in residential areas were not necessarily excessive. Some early Interagency monitoring in two locations in Kuwait City found quite low SO_2 (38ppb max., < 140ppb U.S. NAAQS), but occasionally higher NOx (742 ppb, < 1.2ppm standard) and total hydrocarbons (18.1 ppm). During early May 1991 residential areas in Kuwait City had PM_{10} daily averages of between 141.7-367.3 ug/m^3, related primarily to type of PM (i.e., higher when filter was black > gray > other > white, with PM loads of 318 down to 124 ug/m^3), with some indication of the effect of northerly and northwesterly winds, both indicating the influence of the oil well fires (U.S. Interagency report, 1991). (U.S. PM_{10} NAAQS for 24 hr.= 150 ug/m^3.) Monitoring in July and August 1991[5] showed low NOx and SO_2, 7.4-90ug/m^3 of fine fraction particulate matter ($PM_{2.5}$ - ≤2.5um), though some of which were high levels, and essentially no polycyclic aromatic hydrocarbons (PAHs). Some indoor sampling indicated very low $PM_{2.5}$, SO_4 and Pb concentrations, but SO_2 at about the same level as outdoors (possibly related to indoor combustion). A short inversion (8/7) increased ambient values of PM, NOx, SOx, and also

increased Na and Cl concentrations indicating some contribution from the oil well fires. A British report[15] comparing levels in 1991 to 1989 found that the gaseous pollutants essentially exceeded the standards (if at all) more in the earlier year; noteworthy was that O_3 didn't exceed standards either year, confirmed by later studies. Nevertheless, some concern about general levels of PM during (and after) the war still is present, as stated in the Interagency Interim Report on the Kuwait Oil Fires (4/3/91). One study[16] tried to predict PAH levels from the data, and implied high levels, but the methodology and pollutant data basis for the predictions were quite incorrect. Generally, air pollution increased in residential areas during the period of PGW activities.

Other Major Air Pollution Problems Related to Gulf War Activities:

The primary focus of concern, and background for consideration of potential health effects of deployed personnel (and residents), is the air pollution produced by Gulf War-related activities and other similar activities in the region.

Extensive PM_{10} monitoring was conducted in Kuwait and Saudi Arabia by the U.S. Army (USAEHA-KRAT, Kuwait Oil Fire Health Risk Assessment, No. 39-26-L192-91); there were 22-117 valid days of data at four sites in Kuwait (Embassy-EM [39d], Camp Thunderbird-CT [113d], Military Hosp.-MH [117d], Ahmadi Hosp.-AH [22d], and 52-130 days in SA (Khobar Towers-KT [130d], 3 others), during May-Nov. 1991. PM_{10} values declined during the period, as the oil well fires were extinguished, and especially after the Shamal winds (that produce sandstorms, especially in Saudi Arabia) disappeared in September (There were strong correlations with wind speed in most locations.) Overall daily averages were 149-301ug/m^3 in Kuwait and 116-174ug/m^3 in SA; 95th percentiles were 237-871 and 206-312, respectively. CT exceeded 24 h NAAQS regularly thru September, MH thru October (even after the fires were out). MH exceeded "significant harm" levels (>600ug/m^3) 5 days, and CT did on at least three days (two of which were after the number of burning wells was < 250, with concentrations >1200ug/m^3). (In Kuwait, the wind rose shows wind emanating primarily from the northwest.) CT exceeded NAAQS some days in October, but both MH and CT <100ug/m^3 in November of 1991. KT also saw most days > NAAQS, until day 295; the daily averages also exceeded NAAQS thru Sept. KT had 4 days > 600ug/m^3. (In Saudi Arabia, the wind rose showed wind directions generally from the west through north.) As with other monitoring efforts, sulfate (SO_4) and nitrate (NO_3) were low. The major conclusions from these data appear different for the Army than for an EPA reviewer or this reviewer. The Army doesn't think PM was much of a risk (partly because it concluded that most of the PM was sand-based), which is most likely incorrect because of the very high levels from various sources (as discussed further below). The Army didn't do non-cancer risk assessments (HIs) for PM (or its metal content) but probably should have.

Among the trace metals, only lead (Pb) was > 1ng/m^3 in Kuwait City; the Pb was related to vehicular traffic (as indicated by the Pb/Br [bromide] ratio of about 3[5]). It was stated[5] that the other trace metals were essentially those of "local desert soils and comparable to ratios for Arizona soil", indicating wind-blown dust as their source. However, other monitoring by the Army (USAEHA-KRAT) would indicate some concern related at least to nickel (Ni), as discussed further. The 95th percentiles of the distributions of Ni from daily air sampling filters (May-Nov.'91) exceeded usual levels in Kuwait sites during July 1991 (U.S. Embassy),

September and October, 1991 (Camp Thunderbird-CT), and October 1991 (Military Hospital and Khobar Towers in Saudi Arabia). Also, three days' means at CT > 200 (still < WHO AQG). There is some judgment involved in determining if Ni stayed almost constant over the period (as stated by USAEHA); their data do indicate that mean Ni in November was higher than in June or August at CT (and that could be due to an accumulation). Ni and V were found in their soil/sand samples in Kuwait, and V in Saudi Arabia (at equivalent levels at KT to those at CT), consistent with Ni and V in fire plumes, and V found 200-250 km downwind (with plume elevations of 2K [5-21 and 9-27ng/m^3][17], and consistent with other long-range transport findings[6,11]; both Ni and V in soil/sand declined during the period of study. V and Cr only exceeded usual ambient levels one day at KT, but were < AQG; ambient V declined during the period. Other metals in ambient samples didn't exceed usual levels found elsewhere, though ambient Al, Ca, Fe [iron], and Na were high (related mostly to soil sources) and ambient Pb did increase with time in Kuwait (daily average at CT=191ng/m^3); ambient Pb stayed up in SA (KT) during the whole period (daily average=371ng/m^3); Pb findings are consistent with vehicular traffic temporary patterns. (All metals found in soil/sand also decreased with time in both Kuwait and Saudi Arabia.) [In spite of claims to the contrary by the Army, they showed some cancer risk was associated with metals, primarily attributed to Ni, as discussed below.]

One has to examine results of the monitoring studies[18-21] in Saudi Arabia prior to the Gulf War to put these results in perspective. Generally, these studies confirmed the high PM levels were typical of this region. Those studies characterized the nature of the PM more, and help provide information on levels that visitors would experience even if there wasn't an ongoing war. (In general, these studies appear to be of good quality.) One set of studies[20] evaluated PM (TSP and PM_{10}) in Riyadh (about 750 km SW of Kuwait oil fields) with decent monitoring methodology. They found high values of both sizes of PM indoors and out, with higher values found outdoors; PM_{10} (inhalable PM) averaged 137 ug/m^3 outdoors and 78 ug/m^3 indoors during the study (an I/O ratio of 0.59). This study was too short-term, but if these levels persist, they would be above U.S. standards. Interestingly, the PM_{10}/TSP ratio outdoors was about 0.25, indicating large amounts of larger particles outdoors; indoors the ratio was 0.5 indicating indoor sources. Another of the studies in Riyadh [20] evaluated NOx and CO outdoors and indoors; some of the nitrogen oxide (NO) levels were high indoors and out, but the NO_2 levels were below NAAQS (even though sampling methods might be considered suspect). In terms of NOx, it found that the major outdoor source was vehicular exhaust and the major indoor sources were unvented heating and environmental tobacco smoke (ETS). ETS also generates high levels of PM and some unvented heater types (specifically kerosene) generate both high levels of PM and gaseous pollutants (including SO_2 and acidic aerosols). Specifically, studies of unvented gas heaters, as shown in chamber studies[22], generate increased NOx, CO, and hydrocarbons. Kerosene space heaters emit excess amounts of PM, NOx, SO_2, CO, and hydrocarbons in chamber studies and houses[23] and in ongoing studies in tents placed in chambers (personal communication). The latter studies are being performed to simulate exposures that may have occurred among Gulf War troops. The levels of these pollutants are generally in excess of standards, sometimes very much so as in the case of kerosene space heaters. [All of these factors would have affected the U.S. personnel in the Gulf region (as discussed below).]

VOCs were not initially high during Army monitoring in Kuwait, but increased with time, which

they related primarily to increased industrial activity and vehicular traffic. Their monitoring in Saudi Arabia showed a bimodal distribution, with a general increase over time. Ambient benzene daily means did exceed World Health Organization (WHO) AQG, and ATSDR acute and intermediate recommended limits (MRLs). Army industrial hygiene studies around U.S. personnel did show that maximum coal tar measures exceeded threshold limit values (TLVs) more than once in one sight in Saudi Arabia, and maximum NO_2 exceeded TLVs more than once at MH. The GAO report (RCED-92-80BR) provides data of a similar nature derived from studies by the French, Norwegians, British, as well as by NIST, NSF, EPA/NASA, the Army, and the National Toxics Campaign Fund (in Saudi Arabia).

Biological Samples (Biomarkers): Limited studies of biological samples were performed in U.S. troops, apparently in two separate studies. VOCs in blood were evaluated in U.S. troops by CDC (reported in an interim report), and only tetrachloroethylene (PCE) was found higher than usual in a few troops, related to their de-greasing activities. The USAEHA-KRAT program also looked at biomarkers in some troops (28-61) pre-, during, and post- deployment from Germany to Kuwait. Generally, metals were found to either remain the same (i.e., Ni, V) or not be detected (i.e., arsenic-As, mercury-Hg). Only Pb increased in those deployed in Kuwait (though still within normal limits). There were no substantial changes in VOCs and most were within the range found by NCEH in the U.S.; 5 VOCs actually were significantly lower in Kuwait (ethylbenzene, xylenes, styrene, toluene); PCE was higher (0.048->0.2->0.067ppb) (as found by CDC) as was acetone (1.2->1.6->1.3 ppm); benzene went from 0.098 to 0.072 to 0.130 ppb, chlorobenzene decreased from 0.044 to 0.005 ppb and stayed at 0.005 ppb, and chloroform went from 0.017 to 0.02 to 0.022 ppb. PAH DNA adducts were higher pre- and post- implying low level exposures in Kuwait. (Sister chromatid exchanges [SCEs], which can indicate a large number of potential causes and effects, were higher during and post-deployment, implying to USAEHA that stress, etc. were major factors. It doesn't appear that there were such studies in reservists or civilian employees. There was also a study of 9 of the U.S. firefighters[24] with exposures, before deployment and within 3 weeks after return (having spent 6 weeks deployed), showed similar levels of DNA adducts; they were within the range of those reported by their lab for other, non-exposed groups. (The lab reports 2-3 fold increases for workers exposed to pyrolysis products, coke oven and iron foundry emissions.)

Attempts to set up a monitoring network and use it for preventive purposes was mentioned in the 1991 Interagency Interim Report (op cit.). It is not known if this was accomplished; if yes, results would be important to know. [The Congressional Report on Environmental Aftermath of the Gulf War (S.Prt. 102-84, 3/92) was the only one to significantly minimize potential effects, though the authors somehow thought that napthalene might be a problem; this wasn't discerned.]

In summary, sufficiently high levels of particulate matter, some metals, occasionally NOx and SO_2 indicate likely acute health hazards and potential for some chronic health hazards in those with pre-existing disease, those with other forms of susceptibility to respiratory problems, and/or those who stayed on in the Gulf (or who continued to have high exposures upon their return).

Potential and Likely Health Effects

This report will not attempt to repeat a review of a nature similar to the IOM (1996), or any other

commission or review body. Nor will it review all possible literature relevant to the topic, as the focus of one reviewer may be considered too narrow and the time restraints are too great. (For instance, this report will focus little on current DVA and DOD registries and their findings.) It will attempt to evaluate most of the literature available within the framework of the Work Scope and the deadline, and reach some conclusions as to possible acute and chronic health effects from Gulf War exposures of the U.S. personnel on duty and the civilian resident population, focusing on effects of air pollution. It will also attempt to estimate acute and chronic effects for potential high-risk populations. Some attempts will be made to evaluate potential confounders, effect modifiers, and various risk factors within the constraints of the data available.

The most difficult tasks, as indicated by the IOM report, are to overcome lack of good medical records (any surveillance system) and incomplete exposure information. There appears to be more of the latter than available to the IOM committee, but most of it is for examining potential aggregate exposures (i.e., potential exposures to groups of individuals rather than individuals per se). Also, some risk assessment has been performed for U.S. personnel by the Army EHA (op cit.), and some health studies were conducted before, during, and after the Gulf War. The report will depend on data available; it will ignore prior broad statements as to whether there were or could be such effects, as many of the statements were made at a time when little information was available, or were made without a broader view of possible adverse effects. An initial caveat: as previously stated, the most difficult task is to predict chronic effects given the nature of the broad range of exposures and unknown effects for many of them (alone or through interactions with other exposures or conditions).

Some Background on Health Effects from Air Pollutants:

The air pollutants of concern in the Persian Gulf, are the background pollutants (natural and man-made) and those produced by activities of the deployed personnel. The health effects associated with them have been reviewed extensively in U.S. documents, usually required by different Congressional Acts (such as the EPA Air Quality Criteria Documents [CDs], ATSDR Toxicological Profiles [TPs], NIOSH and OSHA reports), and by the World Health Organization (WHO) which includes U.S. agency and scientific involvement (such as the Environmental Health Criteria [EHC] reports, IARC reports, WHO/EURO reports, and books [e.g., Bertollini et al., Environmental Epidemiology, Lewis/CRC, 1995]); they have also been reviewed separately (cf. ref. 37).

Almost all can be considered irritants of the respiratory tract, eyes, and skin. Almost all of them can acutely reduce lung function, produce shortness of breath, coughing, nasal and throat soreness and dryness. Some predispose or precipitate acute respiratory infections and other illnesses (e.g., PM, NOx, SOx). The APs (especially the gaseous pollutants) have also been associated with increased reports of other acute problems, such as headaches and other minor complaints, such as dizziness and nausea. Oxidants (Ox), as found in photo-chemical smog are best known for producing eye irritation and headaches. Most of these acute health responses resolve quickly when exposures cease (or decrease to low levels). Some are considered asphyxiants (i.e., CO and NO thru reduction of the oxygen carrying-capacity of the blood), which in smaller concentrations can produce dizziness, headache and nausea; in high amounts they can cause unconsciousness and death. Some are considered poisons, such as hydrogen sulfide (H_2S),

which can also cause unconsciousness and even death. Most of the short-term ($\leq$ 24 hour) standards are derived to avoid these acute effects.

Some prolonged conditions can occur due to exposures even in normal individuals. A good example is the anemia produced by benzene. Benzene and other VOCs (and other chemicals) when inhaled in sufficient amounts can also have neurologic, immunologic and dermatologic consequences that may be prolonged (cf ATSDR TRs and WHO EHCs). More prolonged responses can occur if there is massive exposure, with or without subsequent low-level exposures, and/or the individual is susceptible (pre-disposed) to certain health effects (e.g., allergic respiratory or dermatological responses), and/or is sensitive due to pre-existing disease (e.g., asthma, chronic bronchitis). Massive exposures more often occur in occupational settings (cf NIOSH reports and ATSDR TPs), but can occur occasionally in environmental settings (cf EPA CDs, ATSDR TPs, WHO EHCs and Bertollini et al.). The concern is that massive exposures may precipitate respiratory or other disease, or can sometimes produce an inflammatory response in the airways that may persist (especially with continued more-normal exposures).

New or excessive exposures are thought to affect individuals who are more susceptible, such as those with pre-existing genetic traits, or other physiological, immunological, or biochemical deficits (that may, if ever tested, would indicate pre- or sub-clinical conditions); this has become a large research area. These susceptible individuals may with such exposures have a prolonged response and/or actually develop disease. It is conceivable that the inflammatory responses, such as pneumonitis (discussed below) or reactive airways syndromes may have been generated in some of these susceptible individuals. It is conceivable that even relatively short exposures (in months) to sensitizing pollutants, such as Ni, might produce an immunological change that would at least be prolonged (depending then on continued exposures in other settings).

The best known prolonged effects from acute exposures occur in those with pre-existing diseases, such as asthma or other respiratory conditions, cardiovascular conditions, contact dermatitis. The pre-existing diseases are exacerbated to a minor or major extent, depending on the nature and degree of exposure; they may on occasion be life-threatening. These effects can be observed with the appropriate testing and patient follow-up. (U.S. NAAQS are supposed to protect the sensitive populations, specifically those with pre-existing respiratory diseases.)

The chronic respiratory effects that may be produced from these air pollutants from long-term exposures include: chronic bronchitis (also possibly asthma and emphysema) from PM exposures (cf EPA CDs and WHO AQGs), and cancer from some PM-associated metals (and possibly particle-bound VOCs), from VOCs and from PAHs (cf ATSDR TPs, WHO EHCs and AQGs, IARC documents). SO_2 and NO_2 (and/or other SOx and/or NOx), Usually in combination with PM, may also help produce chronic bronchitis (op cit.; GAO report of 1/92). Long-term standards and AQGs are derived to protect against these chronic effects, but such standards do not cover all air pollutants.

Observed and Likely Acute Effects:
Reports of acute effects range from slight increases in dissatisfaction with the environment in

civilians[25] to significant pneumonitis, obstructive bronchitis, and bronchiolitis (with lung deposition of sand particles) in U.S. (and possibly British) deployed personnel[26-28] (discussed further). Between these extremes were various reports indicating increased respiratory illnesses and eye irritation from PM, other pollutants, and possibly allergens, in all deployed foreign personnel. (The various reports also indicate the occurrence of other general problems, such as GI problems (due mostly to common pathogens) and stress/psychiatric problems in deployed foreign personnel, that are probably not related to air pollution but may indirectly impact, as effect modifiers, on problems produced by air pollution.) Some of the increases in respiratory complaints in deployed American personnel were minor, but some lead to increased sick call (7% of sick call illnesses at U.S. bases compared to 19% in the PG)[23]. Respiratory distress was reported to be "common", but data aren't available to judge either the nature or extent of this problem.

A good survey of respiratory diseases was conducted in about 2600 military personnel in Saudi Arabia during operations there[29], even before the oil well fires started. This study found a significantly higher prevalence rate of respiratory complaints than normally seen, especially in those with pre-existing conditions (about 6% of troops had COPD) and those that smoked, as well as in those with a longer period of time in the region. Crowding, of course, was thought to help increase rates of illnesses. The investigators indicated that living conditions had a role: lower respiratory problems (cough and sore throat) were actually significantly higher in those living in air-conditioned structures while upper respiratory complaints of rhinorrhea were higher in those living in tents (and exposed to outside conditions), even after adjusting for the other significant explanatory variables. The rhinorrhea was assumed to be related to air pollutants and/or allergens. Only 1.8% were sick enough so as not to be able to perform their regular duties. Bacteria isolated were of the typical types found also in the U.S.

A British prospective study[15] evaluated 125 British troops pre- and post- deployment in Kuwait City using lung function and questionnaires. According to the study, there were no demonstrable changes in lung function, even stratified by smoking, but the standard deviations of the changes were large. (They don't show the actual lung function values.) Changes in flow ($FEF_{25-75\%}$ were larger in ex smokers and light smokers, but not significant; heavier smokers actually improved flows. Changes in flow by level of exposure to heavy smoke showed a slightly larger change in those not exposed and in those exposed once a week, with a positive change in those exposed less than once a week; these differences weren't significant either. Finally, changes in flows in those that had wheeze symptoms were greater than those without (mean diff.= 0.19 vs. 0.01), but again not significantly. (No symptom prevalence or incidence rates were shown.) According to the British study, these troops were exposed to high levels of pollutants, similar to that experienced in the London smog episodes of the 1950's. However, the small numbers of subjects and the large standard deviations of the changes were large, and so definitive statements can't be made about statistical significance. Nevertheless, one can infer that those who wheeze and/or do not smoke heavily are possibly at greater risk while the healthy non-smoker is at less risk from short-term air pollution exposures (similar to conclusions from the London smog episodes). [This may have implications for chronic effects, discussed below.]

The Army AEH-KRAT attempted to do the same kind of study, but the lung function results

were supposedly too poor to use. From the questionnaires (in < 75 troops), symptoms reported for the period of deployment in Kuwait were: noticeable increases in problems with the GI and GU tracts; some problems breathing, doing prolonged exercise, sleeping, and concentrating; slight problems in vision and hearing. Comparing post- vs. pre- deployment, these symptoms were reported as "worse" at a two-times greater rate, as were frequent colds, high blood pressure, and weakness/fatigue. Increased symptomatology related to the oil well fires specifically included increased phlegm; 52% said they were exposed to smoke (primarily from the oil well fires, 41% were within 1 mile of them) and 2/3 said it was worse than expected). In addition 44% said they were exposed to severe dust and 45% to gases/fumes. None of these or other troops, or oil well firefighters appear to have experienced the significant morbidity sometimes experienced by regular firefighters[31].

The report of pneumonitis[26] in Americans appeared, according to the authors, to affect an unspecified large number; they also claim that over 500 British were affected (though reports of such were not seen elsewhere). It is conceivable that some pneumonitis did occur related to exposures of deployed troops to high levels of inhalable sand particles, especially in conjunction with the other air pollutants (and maybe to allergens as well). Their SEM studies support the claim that the sand is very fine (as distinct from one other study[29] but not the Army report). Their sand cultures also support the presence of various fungi, as discussed previously. There is some evidence from occupational settings that this might occur in those exposed (e.g., hypersensitivity pneumonitis, sand-blasters pneumonitis). It is unclear what predisposing factors might be important. The important aspect of this report is in the nature of the host-defense changes, especially in immunologically active pulmonary alveolar macrophages (PAMs). The authors stated that it may not be found in civilian residents with adaptation of macraphage function to exposures starting early in life (though this may be difficult to prove).

When PAMs do lyse, and as are part of the primary defense against particles (biological or chemical) they release both chemoattractants to other active cells and some toxic chemicals. Thus, an immunological reaction can occur, which not only aids the development of a pneumonitis (or a lesser inflammatory response when the challenge is less) but can produce a more general inflammatory response. The latter may be similar to a reactive airways syndrome (RADS) or a bronchitis/bronchiolitis (if in the airways). The latter is supported by other findings[27,28]. It can also occur in more distal portions of the lung, a more diffUse disease which typically will produce infiltrates on chest x-rays. Under any of these pulmonary circumstances, opportunistic infections can also occur. (The authors speculate that the pneumonitis also effects the rest of the RE immunological system to predispose individuals to other agents.) The inflammatory changes may continue to smolder after the acute aspects of the disease disappear, in which case one would expect to see some of the individuals who had the disorder have major respiratory problems later. Also, if these inflammatory processes lead to allergic-type respiratory diseases (including IgG or IgE mediated diseases), one might expect to see higher prevalence rates of such diseases. The evidence for any of this happening long-term is not obvious from the reports on returning vets, but may be occurring in a minority of those with problems, reported or otherwise.

Acute Effects in Civilian Residents:

Two team members of the Interagency Team performed some limited interviews with some medical personnel, U.S. and Kuwaiti troops and reviewed some medical records (unknown number, location), and one visited hospitals and other medical facilities; indications were that civilians with existing respiratory problems reported experiencing elevated problems such as asthma and bronchitis (GAO, 2/92,p.18). Currently unsubstantiated studies reported elsewhere[16] indicate significantly increased rates of respiratory illness in residents of Kuwait City in 1991 (vs. 1987-89).

A CDC member of the team (RAE) evaluated civilian ER admissions in 2 hospitals (Mubarek and Sabeh) in Kuwait City during Jan-April 1991. She found asthma and acute respiratory illness admissions didn't increase during the oil well fires (2/23-4/30) compared to pre-fire (1/1-2/22) admissions (7.4% vs. 7.0%, and 29.2% vs. 19.0%, respectively. However, ER admissions for COPD (bronchitis, emphysema, bronchiectasis) did somewhat (0.004% vs. 0.5%). Hospital admissions overall also increased as well (from 44.8/day to 57.8/day), as did admissions for GI and psychiatric disorders and heart diseases. She noted that Kuwait has a fairly high rate of respiratory diseases: 5.9% of deaths (1988-89) are listed as respiratory; asthma was the third most common cause of all hospital admissions, excluding OB patients (2% in 1988), and a total of over 36% for cardiopulmonary conditions; there are over 20,000 asthma patients followed by the Kuwait Center for Allergic Diseases (which had 255 admissions for asthma out of 267 total admissions in 1988). It is clear that there are high-risk populations in Kuwait. She arranged to set up mortality analyses, ER and hospital surveillance, asthma patient surveillance, epidemiological studies, air pollution alert programs, and physician and population education; the statUs of those programs are unknown, as are any of the results of surveillance and analyses in 1991 and 1992. Kuwaiti children may be at higher risk of acute (and chronic) effects of total exposure to PM and gaseos pollutants (e.g., NOx). One group of children that may be at higher risk are those with indoor exposure to gas combustion (e.g., from gas stoves), as they have somewhat poorer lung function than those not so exposed[30]. (Passive smoking and socioeconomic status had neglible effects, so total exposures are more pertinent.) However, no such study appeared to have been conducted in 1991 or 1992.

Thus, little can be said.of the effects of the Gulf War on Kuwaiti civilians in general or in high-risk populations except the following: There was an increase in respiratory illnesses, especially related to bronchitis, and in ER visits measured for a short period at two hospitals for GI problems, heart disease, and psychiatric disorders. Based on risk associated with PM_{10}, $PM_{2.5}$, short-term SO_2 and NO_2 at levels experienced by Kuwait civilian general and high-risk populations, and likely also in some civilian populations in Saudi Arabia (op cit.), there should have been definite and significant acute respiratory effects, especially in the high-risk populations with existing respiratory diseases (EPA Air Quality Criteria Documents, 1995-96; WHO AQG, 1997/98; British Medical Effects of Air Pollution Documents, 1994-96).

Many other acute health effects could have been produced in deployed personnel by other exposures, some to hazardous air pollutants: exposures to unvented kerosene space heaters do increase the risk to acute respiratory illnesses (op cit.); vehicular exhaUst has well-known acute respiratory effects (EPA AQ documents on NOx, Ox, and PM, 1994-6); degreasing and sealants, jet fuel, and gasoline tank fumes produce VOCs that can produce a variety of effects (some

discussed elsewhere in this document, and in the IOM and PAC and DOD reports and specific ATSDR Toxicological Profiles).

Exposures of personnel to other chemicals (e.g. PB, and others Used personally) can produce some upper respiratory effects (due to direct or neurological influences) (ibid.). Up to half of PGW personnel seen in health care facilities were reported to complain of PB side effects[31], though not incapacitating (IOM, PAC, and DOD reports). Some of the combined exposures appeared to have affected respiration, as seen in one experimental animal study[31] (though the PAC did not think this study was relevant because dosage was too high). The impact of any exposures to chemical weapons' releases has been discussed by others (IOM 1996, 1997; PAC, 1997; SIU trip reports) and won't be discussed here. Hypothermia was not reported, though the combined effects of low temperatures and air pollutant exposures on morbidity are well known (op cit.). Various other possible exposures delineated elsewhere may have had acute effects (op cit.).

In addition, there are actual and some presumed exposures to vaccines, infectious agents and to allergens (op cit.) that have known acute effects. (In actuality, the risk of infections with some of the prevalent diseases in the region[32] did not appear to be as critical as expected, except for some visceral leishmaniasis); partly because personnel were there during the winter when some causes, such as sand flies, were quiescent.) Some of the deployed personnel were also at higher risk due to pre-existing conditions (such as asthma/COPD [6%]) (op cit.), or due to stress (IOM and PAC reports), and possibly due to other treatments. Most of these have not been fully explored systematically. Thus, it would have been surprising if some acute effects weren't observed.

Chronic effects:
It is conceivable that lung function could have been adversely affected in those at higher risk, as inferred from the British study (op cit.). It could also be that those with pre-clinical states or with sub-clinical disease (e.g., asthma) could have such diseases precipitated by the pollutant and allergenic exposures. (Some air pollutants, such as diesel fumes, do act as adjuvants in the development of allergic diseases [IOM, Indoor Allergens, 1993].) Further, inhalation injuries can be long lasting[33], especially pertinent for asthma and chronic bronchitis (cf also EPA AQC Documents, op cit., and ATSDR Tox. Profiles). These possibilities are pertinent for both deployed personnel and civilians. However, the long-term exposures to PM that usually lead to such chronic respiratory diseases and death[34-38] (as evaluated in EPA AQC Document, op cit.) were not experienced by deployed personnel for very long, though they have been experienced by civilians. Those returning to the States would have to continue experiencing significant exposures to PM to see those effects.

Pneumonitis and/or other conditions produced by fine sand[26-28] might, for reasons outlined above, be considered a precursor to other chronic conditions that might have occurred in personnel that were deployed. (In spite of speculations by the authors, it is not known if it occurs in resident civilians or armed forces.) There is good evidence that PM-cell interactions do produce cytokines that are involved in prolonged inflammation, effects on epithelial cells, and pulmonary fibrosis[39]. These possibilities will have to be explored further.

Other non-cancer chronic risk is suggested by the Army EHE (op cit.) related to VOCs, specifically benzene, with Hazard Indices up to 5; some exposures did exceed the ATSDR intermediate MLR (related primarily to immunological endpoints), and the WHO AQG (related also to neurological, hematological, dermal and respiratory endpoints). Other VOCs added little to the Hazard Indices. Ingested and Dermal exposures added essentially nothing. As indicated above, the U.S.AEHE incorrectly ignored the chronic risk of PM (cf EPA AQCD, WHO AQG, et al.) and of metals (especially Ni); these are of some concern because of their high levels and potential prolonged (and possible chronic) impacts (the basis for which are discussed above). For instance, the ATSDR MRL and WHO AQG for Ni is 200ng/m3; exceedances appear primarily to lead to respiratory problems (including ARDS, pneumonia, long-term macrophage effects, inflammation, and fibrosis), as well as some renal, immunological (including possible sensitization via an IgG mechanism). Some of these health problems may manifest themselves in deployed personnel in subsequent years (as discussed above). However, it does not appear that the other possible problems have been evaluated sufficiently in a systematic, longitudinal manner.

The Israelis appear to have evidence that PB taken under stressful conditions can produce long-lasting effects, as well as stress producing long-lasting impacts by itself (CDC trip report). Stess-induced changes in the immune system also occur (IOM 1997). However, the PAC did not consider most theories of long-term effects of stress (or of modified microorganisms) were of much good; possibly better studies are needed.

Most of the American panels evaluating potential causes of subsequent disease could not find reason to believe that the vaccinations produced long-term effects (op cit.); it would be easy enough to perform a good experimental animal test.

Of the other foreign troops participating in the Gulf War, some indicate no problems in returning servicemen (e.g. France), but others (e.g., Britain) are engaged in further studies. Studies of the subsequent complaints of U.S. personnel are under investigation (DOD, DVA, IOM, PAC reports), as are studies of guard units from which some were deployed and some weren't (ibid., MMWR 44:443-47, 1995). The latter indicate that a variety of non-specific illnesses (but not all symptoms or such illnesses) were higher in those deployed. Various panels (op cit.) appear to indicate they might be related to stress, but insufficient attention was given to possible long-term effects of some of the neuro-toxic exposures (e.g., pesticides). As these investigations were not sufficienty intensive, and as insufficient attention has been given to evaluating respiratory and immunologic parameters, they cannot provide evidence for this report.

Cancer risks were claimed by the Army EHE (op cit.) to be related very little to the oil well fires based on minimal differences in risks for deployed personnel in SA vs Kuwait, with both having maximal risks of 2×10^{-6}; though higher than the EPA acceptable 1×10^{-6} for 2 sites, it was less for the other sites. The primary carcinogenic substances evaluated were VOCs, primarily benzene. Some of the increased risk was probably related to natural and other (non-war) anthropogenic pollutants, but probably not to exposures related to the oil-well fires (as these were too small to increase such risks as would be expected in firefighters[41], and were, supposedly equivalent to PM exposures experienced by U.S. urban residents[42]). Further, most cancer risks

assume a lifetime exposure, and the increased exposures in the Gulf theatre of operations would also have to be followed by continued increased exposures; as indicated in many reports, VOC exposures at these levels do occur in urban U.S. settings.

It is somewhat difficult exploring the possibility that the potential chronic diseases discussed actually occurred, as many of them have lead times (latencies) of over 10 years. Nevertheless, if medical and other war records become better (as per PAC; IOM, 1996; others), then these possibilities could be explored over time. It will also be interesting to see the results on ongoing studies in the U.S. and UK. Certainly a more complete registry of all deployed personnel in the PGW seen in the VA system, with follow-up of 20 years, would be valuable.

Recommendations

1. Record searches in the Registries and in the VA system should be made to determine if deployed personnel are experiencing more respiratory problems. A systematic effort, with appropriate forms, will be needed.

2. It should be determined if there have been more respiratory diseases reported in civilians in Kuwait, and, if so, what kind.

3. An epidemiological study should be performed of respiratory and other toxic endpoints associated with PM, NOx, Ni and other metals, benzene, and other VOCs. It should be performed in all National Guard units in deployed and non-deployed personnel Using appropriate physiological, immunological and techniques, biomarkers of effects, and epidemiological questionnaires (including location of deployment and exposure information). This could start as a pilot study in the units studied in the 3 states in which some studies have been conducted. It is more feasibly performed on a larger scale if deployed personnel from various randomly selected Guard units are matched to either non-deployed personnel from the same states (but different units) for such studies. It would require a minimum of about 1000 deployed personnel and an equivalent number of non-deployed Guards.

4. Further studies of absorption, inhalation and ingestion of volatile organic and similar compounds Used in the PG theatre of operations should be performed in controlled human exposure studies, Using exposures at the maximum concentrations estimated for each. Physiological, immunological and neurological studies should be performed in these experiments. Once individual compounds have been evaluated, it can be determined if some combinations of exposures should be Used in further studies of this nature.

5. Further inhalation toxicological studies should be performed Using reasonable concentrations of mixtures of fine particles/diesel fumes with specific metals (e.g., Ni); these should be performed also with mixtures of PM/diesel and some of the VOCs seen in the Gulf. (Mixtures because such studies have already been performed on single pollutants.) It may also be worthwhile doing these kinds of studies with pre-exposures to PM/diesel/Ni/Cr (separately or as a mixture) followed by exposures to some of the ubiquitous PG regional fungi, and another set with pre-exposures to some mix of VOCs followed by fungal exposure. [One could also use some sets of experimental animals in each of the studies pre-treated with other chemical exposures experienced and with vaccines received (including anthrax vaccine and botulinin toxoid).

6. The DVA should start a more complete registry of all deployed personnel in the PGW seen in the VA system, with follow-up of 20 years, as a valuable determinant of long-term effects. Their rates of illness and death could be compared to similar aged U.S. residents. It would be of great benefit also if clinical work-ups of these personnel were standardized and included appropriate techniques for the various long-term effects expected (e.g., respiratory diseases, neurological diseases, cancer).

Of course, I agree also with the recommendations stemming from the IOM and PAC panels.

GLOSSARY

PG = Persian Gulf; PGW = Persian Gulf War

Air Pollution Terms, Abbreviations, U.S./SA Standards:

Standards/Guidelines:

NAAQS = National Ambient Air Quality Standards (U.S.)

AQG = Air Quality Guidelines - are not laws, but recommendations (e.g. WHO AQGs)

TLVs = Threshold Limit Values (NIOSH, ACGIH) = Time-Weighted Averages (TWAs)

MRLs = Minimal Risk Level (ATSDR)

LOAEL/NOAEL = Lowest/No Observable Adverse Effect Level (EPA, NIOSH, ATSDR, WHO)

RfC = Inhalation Reference Concentration (for Cancer) (EPA)

Natural sources = *those that occur in nature*

Anthropogenic sources = man-made

Particulate Matter (PM):

SPM/TSP = SU.S.pended Particulate Matter (SPM) and Total SU.S.pended Particulate (TSP); Measured primarily by high-volume (hi-vol) "impactors" (pump draws air thru specific size opening in enclosure at relatively high volumes and size desired lands on a filter, which is then weighed and possibly analyzed for metals, sulfate, nitrate, etc.).

The old U.S. 24 hour NAAQS for TSP was 260 ug/m^3; the WHO AQG is 120 ug/m^3.

PM_{10}/$_{2.5}$ = PM sizes $\leq$ 10 and 2.5 um (micro-meters mass median [aerodynamic] diameter, respectively; Measured U.S.ing high- (or low-) volume impactors.

PM_{10} also called tracheo-bronchial PM (since it's deposition is primarily in that region of the lower respiratory (pulmonary) airways); it is considered fine particulate (as vs. coarse particulate, which is > 15 or > 25 um), but includes both fine and coarse fractions, where $PM_{2.5}$ is considered the fine fraction.

The current U.S. 24 hour NAAQS for PM_{10}: 150 ug/m^3 [annual=50ug/m^3]; the WHO AQG is 70 for a 24-hour average.

$PM_{2.5}$ also called pulmonary/alveolar/parenchymal PM (since it's deposition is primarily in that region of the lower respiratory (pulmonary) tract).

RSP = Respirable PM = $PM_{3.3}$ or $PM_{3.5}$, an old terminology (measured U.S.ing impactors or light-scattering devices).

IP = Inhalable PM = PM_{15}; an old terminology (measured U.S.ing hi-vol impactors); SA has PM_{15} standards of 340 and 80 ug/m^3, for the 24-hour and annual averages, respectively.

BS = British/Black Smoke (a reflectance assessment), ~RSP; the WHO AQGs are 125 and 50 ug/m^3, for the 24-hour and annual averages, respectively.

Metals (trace/elemental metals):

Metals are emitted and monitored as particulates. They are analyzed from PM filters by one of several methods (e.g., ICP, AA, NA, XRF) and can be evaluated also by other electronic-microscopic methods (e.g., SEM). (Details are provided for the metals measured in the PG.)

Lead (Pb) is the most common metal found, and the only metal for which there is a

NAAQS (1.5ug/m^3 mean over 3 months). Its primary source (80-90%) is from alkyl lead additives in gasoline (thU.S., vehicular exhaU.S.t); it has also been U.S.ed in paints and as solder (e.g., in water pipes and cans), and is generated by lead mining and smelting, other ore smelting (e.g., copper smelting). coal and steel production, and coal combU.S.tion.

Aluminum (Al) is from natural and man-made sources; it often occurs in soil and can become wind-borne. There is no WHO AQG for Al.

Arsenic (As) has inorganic and organic forms. It is found naturally in rocks and appears in soil (and other media), ores, vegetation, pesticides, coal (and some oil) combU.S.tion, and in manufactured goods. No WHO AQG is provided as As is also carcinogenic (with 1 ug/m^3 is associated with a lifetime risk of $3x10^{-3}$).

Mercury (Hg) comes in three oxidation states; the one of importance is the mercuric form, which is either inorganic or organic (and the inorganic form can be transformed by methylation into the organic form). The mercuric form can come from emissions from soil and water, fossil fuel combU.S.tion (e.g., oil) and is derived from man-made products and U.S.e. The WHO annual average AQG is 1 ug/m^3.

Nickel (Ni) occurs naturally and is enriched in petroleum, the burning of which releases the Ni; over 75% of atmospheric Ni comes from petroleum combU.S.tion. There are multiple other man-made sources. No WHO AQG is provided as Ni is also carcinogenic (with 1 ug/m^3 is associated with a lifetime risk of $4x10^{-4}$).

Vanadium (V) is like Ni, mostly from petroleum and its combU.S.tion, and there are other natural and man-made sources. The WHO 24-hour AQG is 1 ug/m^3.

Iron (Fe) comes from both natural and man-made sources; there isn't a WHO AQG for Fe, but there other guidelines for soluble, insoluble, oxide, and chloride, Fe compounds.

Zinc (Zn) comes from natural sources but mostly occurs in air from man-made sources; there isn't a WHO AQG for Zn, but there other guidelines for oxide, chloride, and stearate Zn compounds.

Other Particle and Particle-bound Chemicals and Pollutants:

Sodium (Na), *Calcium (Ca)*, and *Chloride (Cl)* are primarily natural chemicals (e.g., NaCl = salt), and are U.S.ed to indicate sources of some of the particles (e.g., seawater, soil).

Carbon (C) is one of the most basic chemicals found in nature and in many man-made pollution; it is a basic part of organic compounds (mentioned below); it is plentiful in particle-form (and as a gas) from any kind of combU.S.tion. Particle- bound organic compounds (sometimes called P-bound VOCs [see below]) are important pollutants.

Hydrogen (H) is also one of the most basic chemicals found in nature (as a gas) and in many man-made pollution; it is a basic part of many compounds, including acids (see below); acidity of particles is often measured as H^+ ion from filters.

Sulfur (S) is also one of the most basic chemicals found in nature and in many man-made pollution; it is a basic part of many compounds, including those from fossil fuel combU.S.tion (see below).

Sulfur Oxides (SOx):

Sulfer Dioxide (SO_2) is a common product of fossil fuel combU.S.tion; its NAAQS are: 365 and 80 ug/m^3 (0.14 and 0.03 ppm) for 24 hour and annual averages, respectively. It is monitored by chemical and colorimetric methods (e.g., pararosaniline wet chemical process, UV

florescence); the latter methods allow it to be monitored continuoU.S.ly. (Sulfur trioxide [SO_3 is an unstable SOx that often occurs with SO_2, but is U.S.ually not measured per se.)

Sulfuric (H_2SO_4) and SulfuroU.S. (H_2SO_3) are aerosol acidic particles often found in the atmosphere from fossil fuel combU.S.tion (and indoors from kerosene or oil combU.S.tion).

Sulfate (SU.S.pended Sulfate - SO_4) is a common particle form of sulfur from combU.S.tion; there isn't a NAAQS, but some states have Sulfate standards (e.g.,, 25ug/m^3 annual).

Hydrogen Sulfide (H_2S) is a natural gaseoU.S. product (90%) and a product of human activities (10%) (mostly when sulfur-containing compounds react with organic materials at high temperatures), such as with fossil fuel combU.S.tion. It has a WHO AQG of 7 ug/m^3 with a 30-minute averaging time. SA has the following standards: 200 ug/m^3 (0.14 ppm) for 1 hour and 40 ug/m^3 (0.03 ppm) for 24 hours.

Nitrogen Oxides (NOx):

Nitrogen Dioxide (NO_2) is a common gaseoU.S. product of fossil fuel combU.S.tion (including from motor vehicles); its NAAQS is: 100 ug/m^3 (0.53 ppm) for an annual average (to protect against chronic effects). It is monitored by chemical methods (e.g., chemiluminescence), and it can be monitored continuoU.S.ly. It is photo-oxidized into ozone.

Nitrogen Oxide (NO) has the same sources as NO_2, but is less stable; it often converts in the atmosphere (though the process can be reversed in the body, and it can then either bound to red blood cells (producing methemoglobin) or can convert to other compounds such as nitrosamines). It is measured at the same time as NO_2 and the sum of the two is often called NOx. It does not have a separate NAAQS or AQG.

Nitric (H_2NO_3) is an aerosol particle often found in the atmosphere and indoors from fossil fuel combU.S.tion; it does not have a separate NAAQS or AQG.

Nitrate (SU.S.pended Nitrate - NO_3) is a common form of particulate NOx from combU.S.tion; it does not have a separate NAAQS or AQG.

Photo-Oxidants (Ox):

Ozone (O_3) is the primary gaseoU.S. product of photo-oxidation of NO_2 and volatile organic compounds (VOCs, mostly hydrocarbons, discU.S.sed below), and is mostly associated with photo-chemical conversions of vehicular exhaU.S.t (secondarily from conversions of the same gaseoU.S. pollutants from indU.S.trial or commercial sources). It has a current NAAQS of 235 ug/m^3 (0.12 ppm) for a daily one hour maximum; a longer-term moving average NAAQS is being considered (similar to the 8-hour WHO AQS of 120 ug/m^3 (0.06 ppm)).

Other Photo-Oxidants (Ox) are the other (20%) gaseoU.S. products of photo-oxidation, of which there are many; the old NAAQS for all photo-oxidants U.S.ed to be the same as the current O_3 standard.

Volatile Organic Compounds (VOCs):

VOCs actually represent a large class of organic carbon compounds that range from semi- to very- volatile. They are U.S.ually monitored actively and chemically assayed by GC, GC/MS, MS/MS; some can be monitored passively. They are emitted from many organic carbon compounds found in man-made products, from combU.S.tion, and also from natural sources.

Most have emission limits (under U.S. NESHAPS and other Acts) but not NAAQS or AQGs, as many are carcinogenic; some have TLVs, as they also produce other, non-carcinogenic health effects. They also are photo-oxidized into photo-oxidants (see above). They can also be bound to particles (e.g., formaldehyde, a primarily indoor VOC). Most of those of concern herein are hydrocarbons (aromatic and polynucleic aromatic).

Hydrocarbons:

Hydrocarbons as a group (of which there are many sub-categories) U.S.ed to have a NAAQS, but were considered more important as a primary VOC source of photo-oxidation, so they no longer have a separate NAAQS.

Major Aromatic Hydrocarbons:

Benzene is the prototypic VOC becaU.S.e it is so ubiquitoU.S. (associated with petroleum products, their processing and combU.S.tion, other organic combU.S.tion, and other man-made products) and becaU.S.e more is known about it.

Toluene is an aromatic hydrocarbon that was measured in the PG theater; it has a 24-hour WHO AQG of 8000 ug/m^3; the ratio of benzene to toluene is one way to determine how much of the benzene was from vehicular exhaU.S.t, as toluene is primarily from that source.

The others measured in the PG theater were: ethylbenzene, styrene, acetone, chlorobenzene, and chloroform (a not-atypical list of man-made VOCs). Only two have a lower value for non-carcinogenic effects:

Styrene is U.S.ed in plastics and is released in processing and combU.S.tion; it has a 24-hour WHO AQG of 800 ug/m^3.

Xylene is an aromatic hydrocarbon that was measured in the PG theater; it does have a WHO AQG.

Polynucleic (or Polycyclic) Aromatic Hydrocarbons (PAHs), which can also go under other abbreviations (e.g., POMs), are products of combU.S.tion; they are U.S.ually considered separately from other VOCs and measured differently (U.S.ing varioU.S. methods, including elution from filters and varioU.S. forms of gas chromatography and/or mass spectroscopy [GC/MS]); they do not have NAAQS or AQGs as they are carcinogenic.

Other VOCs of Concern:

Tetrachloroethylene (also known as tetrachloroethene and perchloroethylene [*PCE*]) is a degreaser; it has a 24-hour WHO AQG of 5000 ug/m^3.

The others measured in the PG theater include acetone, chlorobenzene, and chloroform; they do not have WHO AQGs (becaU.S.e of their carcinogenicity).

Carbon Monoxide (CO):

This gas is a product of natural gas and of incomplete combU.S.tion. It has NAAQS: 40 mg/m^3 (35 ppm) for 1 hour and 10 mg/m^3 (9 ppm) for 8 hours.

Carbon Dioxide (CO_2) is a natural and man-made gas; some countries have standards.

Health Terms:

Respiratory:

Respiratory is the term U.S.ed to denote both upper and lower anatomic parts of the breathing process; the upper part is often called the nasal-pharyngeal region (in the head) and also includes the mouth; the lower part includes the airways: trachea (and larynx), bronchi and bronchioles (including respiratory and terminal), and alveoli (air sacs) and the parenchyma (the

structure including the linings of the alveoli and the lung lining (pleura);

Pulmonary U.S.ually refers to the lungs and airways; however, the respiratory bronchioles and alveoli are often called the pulmonary breathing zone.

Chronic Obstructive Pulmonary Disease (COPD) is one form of chronic lung/respiratory disease, and refers to the diseases chronic bronchitis [a cellular/morphological airway disease] emphysema [an anatomic disease with central and/or peripheral destruction and fibrosis], and bronchiectasis [a chronic inflammatory disease of the bronchi/bronchioles]; it can also include bronchiolitis obliterans [obstruction/destruction of bronchioli].

Airway Obstructive Diseases (AOD) is the term referring to both COPD and asthma. (Asthma is a functional condition of the airways, U.S.ually associated with inflammation and marked by broncho-constriction; in its early stages, in may have a large reversible component; an irreversible component is more likely as the disease progresses or is more chronic.)

Pneumonitis is a term indicating inflammation in the parenchyma of the lungs. It often leads to some fibrosis in the lungs.

Acute Respiratory Illnesses is a term encompassing acute respiratory infections or other (non-microbiologically-induced) illnesses of the upper or lower respiratory tracts (URIs and LRIs, respectively).

Neurological:

Any disease of the *Central NervoU.S. System* (CNS-brain), or the *Autonomic (or peripheral) NervoU.S. System* (ANS). Neurotoxic effects can affect respiratory control (in the CNS or via the ANS), and can produce symptoms such as respiratory distress, shortness of breath.

Opthalmological pertains to symptoms/diseases of the eyes and can be produced by the same air pollutants that produce respiratory disease (as reported in Reference 31).