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ENVIRONMENTAL TOXICITY OF COMBAT ZONES

Health hazards in the deployed environment

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Contents

SUMMARY

This report has been produced at the request of the Royal New Zealand Returned and Services' Association (RNZRSA) who wanted to better understand the risk of environmental toxicity on the physical and mental health of defence force personnel deployed to modern combat zones.

Since the Gulf War there has been anecdotal and clinical evidence of physical health issues that have plagued veterans returning from combat zones. It appears the physically fit and healthy service men and women who were deployed to combat zones have returned with symptoms of physical illnesses, the causes of which are not easily identifiable, regardless of their role in these conflicts.

This proposes an important question: Is there something in the environment of combat zones which leads to an increase in adverse health responses? This report aims to answer this question by evaluating the range possible environmental exposures and their impact on human health.

As the first step of a larger study, this project conducted a literature review aimed to establish what is known in this area, as a basis for articulating the subsequent research steps. The key aims of the study were:

- to provide greater scientific certainty to what extent environmental toxicity of combat zones might present a health risk for deployed personnel; and
- if such risk exists, to establish what types of health responses could be reasonably expected to be observed in personnel after deployment.

The key findings of the study show that on a number of parameters it is possible to observe an increase in environmental toxicity of combat zones compared to non-combat environments. The most significant of those are:

- increased particulate matter content in air found in combat areas;
- increased prevalence of combustion products and other organic compounds in combat areas; and
- toxicity released from the use of ammunition.

For majority of these environmental hazards, the main exposure is through air and inhalation, and consequently an increase in prevalence of issues with respiratory healthy can be anticipated to be the most likely outcomes amongst veterans. Some of these could have very late onset, leading to delayed recognition or under-recognition of causal link between the exposure and health outcomes.

1. INTRODUCTION

The health effects of military deployment extend beyond the obvious. Physical symptoms which are obvious and commonly linked to combat and deployment range from physical musculo-skeletal injuries (i.e. sprained muscles, broken bones) and mild (i.e. concussion) to serious physical injury. Psychological impacts have also received research attention since the Vietnam War. However, the health effects of combat which are much harder to quantify and diagnose are the health effects related to common environmental exposures found in combat zones.

In order to start addressing this potential gap in knowledge, this report presents a review of current knowledge of the environmental health hazards found across various combat zones and generally environments in which military personnel are deployed. The specific environmental health hazards vary greatly, depending on the geographical location (e.g. climate and terrain – for example, dust exposure in the Middle East), or the particular conflict (e.g. defoliant exposure in Vietnam, and depleted uranium exposure in the Gulf and Bosnian Wars). Other environmental health hazards have been found across multiple deployed environments such as exposure to combustion smoke, vehicle exhaust and urban air pollution, and exposure to physically and psychologically stressful situations. In order to evaluate and describe the existing knowledge in this area, this report provides a summary of the key information reported on common exposures in combat zones.

1.1 METHOD

The literature review search was primarily undertaken using the NCBI database PubMed, which provides access to more than 29 million citations for biomedical literature. This was supplemented by google searches on specific topics, and by finding additional material in the references section of different papers. The initial search terms were:

1. ‘Health hazards in combat zones’
2. ‘(environmental hazards) AND (military) AND (health)’
3. ‘(combat) and (health)’
4. ‘((environmental exposures) AND veteran) AND health’
5. ‘(((environmental hazards) AND military) AND health) AND Bosnia’
6. ‘(((environmental exposures) AND war) AND health) AND Bosnia’
7. ‘(environmental health) AND Bosnia’
8. ‘((health) AND military) AND Bosnia’
9. ‘((military) AND health) AND New Zealand’

The initial searches mostly focused on the conflicts in Iraq, Afghanistan, Kuwait, and Vietnam. Other conflicts that are relevant to New Zealand forces are the 1991-1995 and 1999 conflicts in the former Yugoslavia states and Serbia respectively.

The results of the initial searches were articles and reports on generalised health hazards in the military environment (mountains, cities, and health hazards present across multiple environments), and specific health information for Vietnam and Korean Wars and the conflicts in South West Asia. This included studies looking at common exposures such as traumatic brain injury, stress, smoking, chemical exposures, and specific diseases associated with these exposures. Other studies looked at specific diseases such as amyotrophic lateral sclerosis, respiratory conditions or all-cause mortality, which are associated with multiple hazards in the deployed environment. These search terms bought up sufficient information for most of the recent conflicts, which are relevant to current War Veterans, but there was very little information on health hazards during the conflicts which took place in former Yugoslavia. To address this, some of the search terms specifically incorporated the word Bosnia. The results from these searches were primarily looking at general health effects in the region which resulted from significant environmental pollution to both soil and water, and health effects due to exposure to depleted uranium during and following conflict.

The abstracts of the search results were scanned for relevance to this report, and for subcategories of both hazards and health effects. Information from the relevant papers was summarised in this report. As a result of this process, the following key areas of focus have been developed: general health hazards, airborne hazards and respiratory health, airborne hazards and other health effects including, particulate matter, chemicals, and heavy metals.

2. GENERAL HEALTH HAZARDS

2.1 DISEASES AND CONDITIONS

Food and waterborne diseases are some of the most common health problems among military personnel. This generally comes about due to contaminated soil and water, incorrect purification methods, and poor sanitary and sewerage systems (Korzeniewski, 2011). The most common among deployed personnel are acute gastroenteritis however the aetiology of many of food and waterborne diseases are unknown, leaving some diseases undiagnosed (Korzeniewski, 2011). Overcrowding in urban environments can also lead to the exacerbation of certain diseases or increased risk of transmission and the emergence of drug-resistant strains of infection (Patterson, et al., 2017).

Vector borne diseases are also common. These are illnesses or diseases caused by pathogens (parasites, viruses and bacteria) which are transmitted from its disease vector. The best known disease vector is the mosquito which carries malaria. The incidence of malaria increases ‘significantly’ within combat zones due to the destruction of local infrastructure and a reduction in sanitary-hygienic standards. However, prophylaxis (preventative medication) is able to steadily reduce the incidence of malaria in the military (Korzeniewski, 2011). One should also be aware that many other disease vectors pose serious health

concerns in across many different areas, globally (e.g. sandflies, tsetse flies, ticks, fleas), and their prevalence could also be impacted by military or war activities.

The deployed environment has been linked to various respiratory symptoms, including acute effects, the development of chronic respiratory disease and the exacerbation of pre-existing respiratory disease (Morris, et al., 2016). Respiratory tract infections are widespread in the deployed environment (Korzeniewski, et al., 2015). They are caused by crowded living conditions, harsh environmental conditions, over-exertion, psychological stress, and exposure to pathogens (Korzeniewski, et al., 2015). However, more significant conditions have also been reported following exposures during deployment including chronic respiratory diseases (Morris, et al., 2016) and cancers. The causes of many of these respiratory conditions could be common exposures, while others could be specific to the combat environment. We note here that common exposures are likely to be contributing to respiratory symptoms. Later sections of this report will discuss specific respiratory risks found in combat zones.

Sexually transmitted infections (STIs) have always posed a health risk to the military population, and global increases of rates of STIs in the last decade have also affected military personnel (Aldous, et al., 2011). Recent reports on STI rates in the U.S. military have found generally higher rates than the general U.S. population (Aldous, et al., 2011; Stahlman, et al., 2014). Additionally, rates are often higher for women than men in the U.S. military (Stahlman, et al., 2014). Women are also more likely to be exposed to Military Sexual Trauma (MST) (Resnick, et al., 2012)

Urogenital health disorders which affect veterans and military personnel include; urinary tract infections (UTIs), pelvic organ prolapse (POP), urinary incontinence (UI) and bladder pain syndrome (BPS) (Resnick, et al., 2012). For women in the military the risk of these conditions is higher, due to the physical demands of the military and the conditions they work in. (Resnick, et al., 2012).

Clearly, most of the diseases and conditions discussed in this section are common in all populations. Simply, some higher rates have been observed in the military personnel.

2.2 TRAUMA AND STRESS

The jobs of military personnel (and civilian contractors) in combat zones often expose personnel to traumatic events and common combat zones stressors, such as threat of attack, experiencing an attack or witnessing traumatic events (Trupiano, 2016). These exposures can lead to Post-traumatic Stress Disorder (PTSD) and other comorbidities (e.g. depressive disorders), which increase the risk of suicide (Trupiano, 2016).

Because combat includes exposure to dead, dying and wounded people, studies of Vietnam War Veterans have found that combat exposure was associated with altered psychological functioning leading to antisocial behaviour (Barrett , et al., 1996), trait anger (Williams, et al., 2010) and mental health declines (Gade, et al., 2011). Additionally the act of killing both combatants and non-combatants were associated

with PTSD, dissociation, functional impairment and violent behaviours in Vietnam War Veterans (Maguen, et al., 2009).

Depression is the leading cause of disease-related disability globally (World Health Organisation, 2018). It causes an increased risk for many diseases, including osteoporosis, cardiovascular disease, metabolic syndrome, dementia, and 50% cardiovascular mortality in postmenopausal women (Resnick, et al., 2012). PTSD is defined by symptoms that last longer than 1 month and includes the re-experiencing of a traumatic event (Resnick, et al., 2012). PTSD in Veterans has been linked to higher systolic blood pressure variability, and an elevated percentage of heart rate and systolic blood pressure readings above baseline (Beckham, et al., 2003). PTSD is also a risk factor for Alzheimers in Veterans, along with depression, combat exposure, tobacco use, traumatic brain injury, obesity, physical fitness and potentially chemical and environmental exposures in Vietnam War and Gulf War (Vietch, et al., 2013).

Physical and emotional stress also leaves individuals more vulnerable to diseases and may explain various unexplainable health symptoms. It can be caused by sleep deprivation, fatigue, anxiety and depression (Kvietkauskaite, et al., 2014). Stress-associated hormones modify cellular and hormonal receptor functions and can change a wide range of defence mechanisms used by the body, such as the immune system. These stressor induced changes to the immune system have implications for disease susceptibility in otherwise health humans (Kvietkauskaite, et al., 2014). The hypothesis that stress leads to altered immune functioning has been supported by multiple studies. However, some have found stress impairs whilst others have found it activates the immune system (Kvietkauskaite, et al., 2014). A study looking at the salivary and sera immunoglobulin concentrations in association with military activities found that military missions activated mucosal immunity by enhance production of immunoglobulin-A (Kvietkauskaite, et al., 2014). Another study of 4225 Vietnam-era former US army personnel found that higher concentrations of immunoglobulin-G, A and M in serum are associated with an elevated risk of death from all-cause mortality. The authors noted that the high levels of immunoglobulins may also be associated with undiagnosed disease (Phillips, et al., 2015).

Because of the unique characteristics of elevated stress and potential for trauma in military setting, these present elevated potential risk for a range of health implications for the military personnel.

2.3 CLIMATE AND TERRAIN

Climate is the prevailing weather conditions of an area. It includes temperature, humidity, sunshine, cloudiness, precipitation, and winds. Climate can impact health through weather extremes: very hot or very cold weather (Korzeniewski, 2011).

Hot weather can be a hazard for military personnel on its own. However, the effects of hot weather can be exacerbated if combined with manual labour and individual thermoregulation ability (Korzeniewski, 2011). This can result in dehydration and the dysfunction of body organs and systems. This causes short term, moderately serious conditions, such as heat exhaustion, and more serious conditions, such as heat

stroke and rhabdomyolysis (Korzeniewski, 2011). Increased heat can also exacerbate some pre-existing chronic diseases such as cardiovascular and gastrointestinal diseases (Korzeniewski, 2011).

Cold weather causes an exacerbation in certain diseases such as the common cold and influenza (Korzeniewski, et al., 2015). Extremely cold weather can cause an increased risk of frostbite (Lechner, et al., 2018).

Climate can also cause health hazards in combination with the terrain (i.e. dusty terrain) or prevailing physical features (i.e. forests, cities). Suspended dust and sandstorms can cause acute minor health and more serious chronic health effects (Korzeniewski, 2011). The effects of suspended geological dust and other types of particle matter are discussed in greater depth in section 4.

Mountainous environments are difficult to navigate and increase the likelihood of injury and altitude related illnesses, such as hypoxia. Hypoxia can cause acute sickness, and in some cases lead to more serious conditions such as high altitude cerebral edema and high altitude pulmonary edema (Lechner, et al., 2018). The environment also limits access to medical resources, and altitude or cold can affect the performance of medical equipment (Lechner, et al., 2018).

Although impossible to directly influence and control, the climate and terrain will in some cases present an important health risk for the military personnel.

3. AIR POLLUTION AND RESPIRATORY HEALTH

Exposure to environmental pollution commonly occurs through respiration, ingestion or dermal contact. Of those, respiration is the most difficult to anticipate, adequately measure and eliminate as a hazard. The key variable for this is quality of local air, and recent studies which focused on indoor air quality show that in many cities, even in peacetime, pollution of outdoor air can directly adversely impact indoor air quality (Salthammer, 2013; Nielsen, et al., 2013). Therefore, one important focus for this report is on air pollution in military zones, and its impact on respiratory health.

3.1 HEALTH IMPACTS FROM AIR POLLUTION

The World Health Organisation recognizes air pollution as one of the significant factors which can adversely impact human health. They estimate that 4.2 million deaths per year can be attributed to exposure to outdoor air pollution, and that 91% of world population lives in places where air quality fails to meet the World Health Organisation guidelines (generally in low and middle income countries) (World Health Organisation, 2018). Similarly, respiratory issues are listed as three out of the top six causes of death in high income countries, and many cancers are in the top 20 causes of death (World Health Organisation, 2016). This suggest that there is a reliable international consensus that quality of air can impact human health quite tangibly.

Combustion and the products of combustion processes present one important subgroup within air pollution, and one of the important groups for this evaluation. Combustion of solid fuels (coal, firewood) accounted for 1.6 million deaths and 39 million DALYs (Disability-Adjusted Life Years, which considers years of life lost due to both disability and death) (World Health Organisation, 2002). Pollution from solid fuels is recognised as one of the ten most important worldwide threats to public health (World Health Organisation, 2007). This is a larger problem in less developed countries, however, even in Europe as many as 2 million DALYs per year, or 2 million years of healthy life are lost annually due to the total calculated burden of diseases attributed to indoor air quality (Jantunen et al., 2011). A number of other important factors contribute to indoor air quality issues, including combustion particles (either from indoor solid fuel combustion or tobacco smoke), building dampness, outdoor air pollution, and risks from building materials and the associated indoor volatile organic compounds (VOCs) (Jantunen et al., 2011). Because combustion can be elevated in combat zones, it stands to reason that the risks associated with combustion could be higher in such areas.

Concurrently, increases in related issues of asthma and allergic diseases have been clearly evident worldwide. The World Health Organization estimates that 15 million DALYs are lost annually due to asthma, representing 1% of the total global disease burden and leading to 250,000 annual deaths worldwide (Balachandran, et al., 2010). Similarly, it was estimated that by 1990, 20% of the population suffered from an allergic disease (Spengler, et al., 2000). Interestingly, the first documented case of hay fever was recorded mid 19th century by a British physician who had to collect data for another 10 years before he found 7 additional cases (Spengler, et al., 2000). Thus, hay fever was first recognised as a health condition around the time of industrialisation in the country where this process was most accelerated. In 2004, Sundell reported on multidisciplinary reviews of all scientific literature conducted in the Nordic countries and Europe, which established that the increasing incidence of asthma and allergy throughout the developed world in the past thirty years was probably due to environmental changes, as the period was not long enough for change through genetic evolution (Sundell, 2004). Sundell (2004) also pointed out that the available scientific means could still not provide sufficient explanation for this change, which might suggest a general decrease in health of general population. Similarly, globally, societies are witnessing an increase in the prevalence of modern diseases such as cancer, multiple allergy syndrome, allergies, asthma, autism, and attention deficit disorder (Thompson, 2004; Armstrong, 2007). While this is still to be fully explained, associations with the poor air quality are increasing.

Although the increase in recognition of asthma and allergies could be seen as correlating to an increase in combustion since industrialisation, it is also possible that the rise in the use of synthetic chemicals could have contributed. Since the mid-20th century, development and production of synthetic chemicals, made artificially by chemical reaction, has increased more than 10 fold: from less than 10 million metric tons in 1945 to over 110 million tons by the early 2000s (Baker-Laporte, 2008, p272). Some 15 years ago, it was estimated there were more than four million registered human-made chemicals in the world with 60-80,000 in common use, and 1,000 being added every year (Pearson, 1998, p61; Saunders, 2002, p9; Thompson, 2004, pp14-5). It is estimated that fewer than 2% of these synthetic chemicals have been tested for their effects on human health and more than 70% have not been tested at all (Snyder in Saunders, 2002, p9). It is also suggested that insufficient information exists for health assessments of 95%

of chemicals used in construction products (Pacheco-Torgal, 2012). However, development of new chemicals has been accelerating since (Petrović, 2018). In addition, there is a lack of information and research on the additive and synergistic effects of combinations of chemicals (Armstrong, 2007, p61).

Thus, in terms of impact on health, it can be said that air pollution presents a constant background 'noise' contributing to total burden, although with relatively moderate currently quantifiable direct impact on mortality. Recent data is starting to show that the general population experiences respiratory effects from air pollution at levels currently labelled as safe (Falvo, et al., 2015). Unfortunately, many chemicals found in contemporary polluted air are synthetic in origin, and to date the scientific knowledge on the health impacts of many of the synthetic chemicals is only partial (Petrović, 2018). If air pollution was to be elevated in combat zones, this would have an adverse health effect. Pollutants with the strongest evidence for public health concern, include particulate matter (PM), ozone (O3), nitrogen dioxide (NO2) and sulphur dioxide (SO2).

3.2 RESPIRATORY HEALTH

There are multiple reports of respiratory effects as a result of deployment. Some of these are common respiratory tract infections. Of those, some common upper respiratory tract infections (URIs) in the deployed environment are the common cold, sinusitis, tonsillitis and pharyngitis, while some common lower respiratory tract infections (LRIs) are pneumonia and acute bronchitis caused by viral infection (Korzeniewski, et al., 2015). Chronic respiratory diseases (CRDs) are asthma, chronic obstructive pulmonary disease (COPD), and acute respiratory distress syndrome. Other CRDs are allergic rhinitis and sinusitis, bronchiectasis, obstructive sleep apnoea syndrome and pulmonary hypertension.

Several existing studies have investigated evidence dealing with the links between deployment related exposures and respiratory effects. In 2016, Morris, et al. reviewed papers which discussed deployment-related respiratory health, and found that exposure to higher levels of airborne hazards gave rise to acute respiratory symptoms, and chronic effects such as lung disease, asthma and other lung diseases. The airborne hazards identified in the review were PM from dust, burn pit smoke, vehicle exhaust emissions, industrial air pollution and isolated exposure incidents (Morris, et al., 2016). Some other factors are smoking and undiagnosed pre-existing disease (Morris, et al., 2016), individual susceptibility, and factors that are specifically related to military service such as physical activity, stress and violence (Falvo, et al., 2015). Figure 1 shows a summary of the key exposures likely to adversely impact respiratory health (Falvo, et al. 2015).

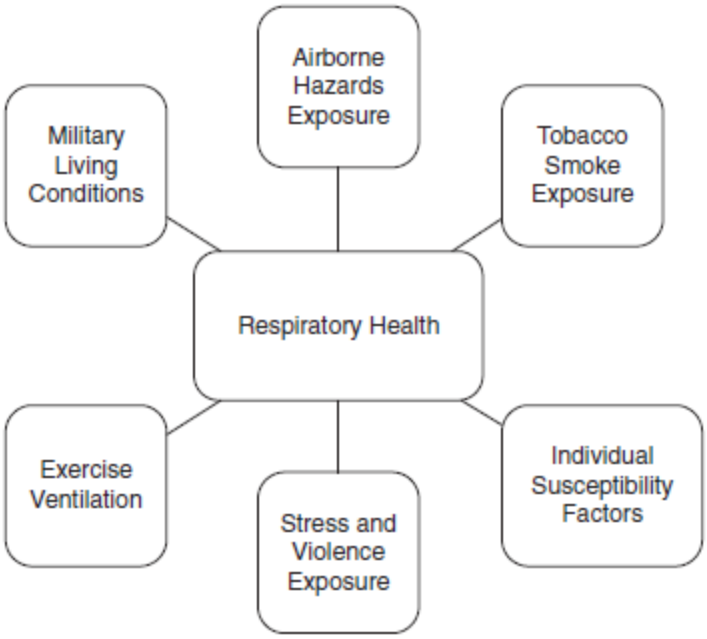

Figure 1: Summary of vulnerability and susceptibility factors affecting respiratory health of deployed military personal. (See Falvo, et al., 2015)

Papers which linked deployment to higher rates of 'new-onset' asthma in deployed personnel could not rule out undiagnosed pre-existing disease, although the results demonstrate that respiratory effects were exacerbated by the exposures in the deployed environment. In other papers, evidence of Acute Eosinophilic Pneumonia (AEP) was connected to deployment, however new-onset smoking was a risk factor as all were active smokers, and 78% reported recent onset of smoking (Morris, et al., 2016). A similar review by Falvo, et al. (2015) discussed how 'extreme' physical demands and poor hygienic settings during deployment may predispose military personnel to certain respiratory effects. However, Morris, et al (2016) concluded there were no clear causal relationships between any one exposure and respiratory symptom or condition, largely due to the fact that many of the studies were retrospective and/or did not have individualised exposure data (Morris, et al., 2016). Additionally the exposure measurement methods used in other workplaces may not suit the complex demands of day to day military life, which realistically limits the types of data that can be gathered (Magnusson, et al., 2012). Further to this, epidemiological studies which look at the types of exposure to airborne hazards in non-deployed settings are often for high intensity exposure over a short period, or low intensity exposure over a long period (Falvo, et al., 2015). The actual deployed environment is better characterised as high intensity over a moderate duration, which is the period of exposure most likely to induce cardiovascular events (Falvo, et al., 2015). Ciminera, et al. (2015) discussed some of these limiting factors in more depth, citing a lack of standard case definitions leading to variable interpretations of results, a lack of routine pulmonary testing for asymptomatic service members, limited individual exposure data, and a lack of consistent collection of both risk factors and confounders.

In recent years new developments in medicine are enabling better understanding of many harmful effects, at levels previously impossible to study. One of the key problems with exposure to even low levels of ambient pollution is the derangement of the normal biological pathways which starts to occur (Falvo, et al., 2015). It is believed that the airborne hazards start to impact the body through vascular, haematological, and atherosclerotic changes which lead to degradation of cardiovascular and cardiopulmonary function in additive and synergistic ways (Falvo, et al., 2015). More research is needed in this area, especially as it is becoming recognised that some individuals are more susceptible than others.

Despite the limited body of evidence linking exposure in the deployed environment to respiratory health concerns, research on airborne hazards and air pollution generally have established strong links between airborne pollution and health effects, including effects on respiratory health. What is especially relevant is that it is very possible that many common air pollution hazards are elevated in the combat zones and the wider deployed environment, suggesting a likelihood of very elevated health outcomes in veterans.

3.3 GENERAL AIRBORNE HAZARDS IN DEPLOYED ENVIRONMENT

In addition to general issues related to air pollution, military environments and especially conflict zones present their own set of unique risks. Poor air quality is a global concern, and subsequently is a common health concern in the deployed environment (Magnusson, et al., 2012). These include natural occurring and manmade forms of air pollution. Examples of naturally occurring airborne hazards are dust, geogenic emissions (volcanic emissions, natural fires), and biogenic emissions (gas emissions from forests and swamps) (Patterson, et al., 2017; Daly, et al., 2007). Manmade sources are domestic, industrial and agricultural gas and chemical emissions (Patterson, et al., 2017), and military activities (Ciminera, et al., 2015). These included sulphur oxides from coal and petroleum burning, nitrogen oxides from high temperature combustion, carbon monoxides from vehicular exhausts and incomplete combustion, carbon dioxide from combustion and cement production, along with multiple volatile organic compounds (VOCs) and aromatic compounds (benzene, toluene, and xylene)(Patterson, et al., 2017) from the burning of fuels.

As the number of people living in cities and megacities increases, so too does the likelihood of military operations within the urban environment increase (Patterson, et al., 2017). In dense urban environments military personnel are at heightened risk of exposure to toxic chemicals and materials from various industrial sources (Patterson, et al., 2017). In addition to these sources, risk of exposure to particulate emissions from construction and/or destruction activities is heightened in the urban environment.

The types of toxic substances in the deployed environment may be military specific or more general to the local area. Examples of military specific exposures would be heavy metal and radiation exposures from weaponry, chemical exposures due to the use of VOC emitting materials in temporary accommodation and coming into contact with common types of protective apparel, exposure to multiple health hazards from damage to buildings (asbestos, cement dust) and damage to other military targets (fuel storage facilities or factories using potentially harmful materials). Other exposures that may not be directly from military or combat activities are heavy metal emissions from industry and leaded-petrol, toxic chemical

emissions from the industrial activities. Chemical exposures have been found across multiple recent conflicts. The risk of exposure will continue to be an issue due to the worldwide prevalence of industrial production of multiple chemicals and chemical weapons.

Air pollution is made up of a diverse range of pollutants of different shapes, sizes and chemical compositions, and the health effects differ depending on these factors (Marzouni, et al., 2016). Particulate matter (PM) are considered a good surrogate for this 'complex mixture' of air pollutants which are emitted from various sources (Marzouni, et al., 2016), and as such, air pollution is generally quantified as the mass of particles (PM), within a given volume of air (μg/m3) (Falvo, et al., 2015). Types of airborne hazards include naturally occurring and manmade forms of air pollution (Ciminera, et al., 2015). Examples of naturally occurring airborne hazards are dust, geogenic emissions (volcanic emissions, natural fires), and biogenic emissions (gas emissions from forests and swamps) (Patterson, et al., 2017; Daly, et al., 2007). Manmade sources are domestic, industrial and agricultural gas and chemical emissions (Patterson, et al., 2017), and emissions from other military activities (Ciminera, et al., 2015). Some heavy metals occur naturally in the environment, but the majority of environmental heavy metal exposure is from manmade sources (Tchounwou, et al., 2012).

Urban environments tend to have higher levels of pollutants due to polluting human activities (industrial and vehicle emissions) and the density of these activities compared to other environments. As the number of people living in cities and Megacities (dense cities of over 10 million inhabitants) increases, so too does the likelihood of military operations within the urban environment increase (Patterson, et al., 2017). The components of manmade pollution found in urban environments include:

- sulphur oxides from coal and petroleum burning (Patterson, et al., 2017),
- nitrogen oxides from high temperature combustion (Patterson, et al., 2017),
- carbon monoxides from vehicular exhausts and incomplete combustion (Patterson, et al., 2017),
- carbon dioxide from combustion and cement production (Patterson, et al., 2017),
- multiple volatile organic compounds (VOCs) from combustion and consumer products (Patterson, et al., 2017),
- PAHs and oxy-PAHs from combustion (Magnusson, et al., 2012).
- heavy metals from industrial sources (Engelbrecht , et al., 2009; Tchounwou, et al., 2012), and
- dusts and smoke from construction and industrial activities.

Combat can also expose civilians and military personnel alike to new and increased levels of certain toxicants and particulate matter. This is due to specific military activities and related exposures, but it is also correlated with the incidence of military activities within unstable and unregulated environments. Examples of military specific environmental exposures include:

- depleted uranium from munitions,
- heavy metals from ammunition,
- exposure to PM and toxicants from damaged or unregulated industrial sources.

In order to better understand the specific risks in deployment environments, this report looks in more detail into a range of relevant, likely to be significant airborne hazards.

4. PARTICULATE MATTER (PM)

One of the significant group of airborne hazards are particulate matter (PM). The term 'particulate matter' or PM is the generic term for a broad class of extremely small, solid and/ or liquid particles of multiple sizes and chemical compositions (United States Environmental Protection Agency, 2009). These are extremely small inhalable particles suspended in air, which present a risk for human health because are inhalable (Weese, et al., 2009). As with air pollution generally, the particulates may be from natural or manmade sources, and may be directly emitted or formed in the atmosphere through transformations of gaseous emissions such as sulphursulphur oxides, nitrogen oxides, volatile organic compounds (United States Environmental Protection Agency, 2009). PM are generally measured according to their aerodynamic diameter (particle size), and are classified under either PM10 or PM2.5 – particles with an aerodynamic diameter of less than 10µm and 2.5µm respectively (Weese, et al., 2009). PM10 particles are able to reach the upper respiratory tract (upper airways and lung), whilst PM2.5 particles are able to reach deeper into the lung (Weese, et al., 2009). The larger PM10 particles often precipitate from the air within a few hours, whilst the smaller PM2.5 particles may remain suspended for days or weeks and can be transported by air over long distances. Smaller, ultrafine particles than PM1 or similar also commonly occur.

The World Health Organisation air quality guidelines set guideline values for the average concentrations of PM in air over 24-hour, and yearly time periods, measured as the mass of particles within a given volume of air (µg/m3) (Falvo, et al., 2015). The daily and yearly values for PM2.5 are 25 µg/m^3 and 10 µg/m^3 respectively. The daily and yearly values for PM10 are 50 µg/m^3 and 20 µg/m^3 respectively (World Health Organisation, 2005).

As with air pollution generally, the particulates may be from natural or manmade sources, and may be directly emitted or formed in the atmosphere through transformations of gaseous emissions such as sulphur oxides, nitrogen oxides and volatile organic compounds (United States Environmental Protection Agency, 2009). PM are generally measured according to their aerodynamic diameter (particle size), and are classified under either PM10 or PM2.5 – which are, particles with an aerodynamic diameter of less than 10µm and 2.5µm respectively (Weese, et al., 2009). The larger PM10 particles often precipitate from the air within a few hours, whilst the smaller PM2.5 particles may remain suspended for days or weeks and can be transported by air over long distances (Falvo, et al., 2015).

Coarse PM10 in urban environments are caused by mechanical processes such as construction activities, road dust re-suspension and wind (World Health Organisation, 2005). Fine PM2.5 primarily from combustion sources (World Health Organisation, 2005). Both particles are present in most urban

environments, but proportions of particles vary substantially between cities depending on local geography, meteorology and specific sources of PM (World Health Organisation, 2005).

PM is a 'ubiquitous hazard' in times of conflict (Baird, 2016). Sampling in Iraq and Afghanistan within the last two decades has demonstrated levels of air pollution and specific airborne hazards above the recommended exposure levels for human health (Falvo, et al., 2015). Sources of PM during the recent conflicts in Iraq (Operation Iraqi Freedom 2003-2011) and Afghanistan (Operation Enduring Freedom 2001-2014 and Operation New Dawn 2010 – Ongoing) include daily windblown dust, dust storms caused by Shamal winds, diesel exhaust, local pollution and open burning (Magnusson, et al., 2012; Baird, 2016). Other sources include general vehicle exhaust emissions, industrial air pollution, isolated exposure incidents (Morris, et al., 2016), and other occupational sources such as workshops (Magnusson, et al., 2012).

Studies of the PM in Middle Eastern conflict region have found PM2.5 levels which measured as twice the US National Ambient Air Quality Standard (Baird, 2016; Ciminera, et al., 2015). Another study of air pollutants at two urban military sites in Afghanistan (Kabul and Mazar-e Sharif) found outdoor PM10 and PM2.5 levels greatly exceeded daily and annual exposure guidelines (Magnusson, et al., 2012). The study also measured indoor levels of PM10 and PM2.5 in a workshop space and found that levels were lower indoors than outdoors, despite the activities which take place in the workshop being conducive to PM production (Magnusson, et al., 2012). A study of PM in 15 locations in the Middle East, including 6 in Iraq and 2 in Afghanistan, found that PM10 and PM2.5 levels exceeded occupational and military exposure guidelines at each location (Engelbrecht, et al., 2009). Levels of PM2.5 were approximately 10 times greater than observed at rural and urban monitoring sites in the US (Engelbrecht, et al., 2009). PM were identified as the main potential health hazard, with PM2.5 and PM10 exceeding several measures for safe levels outdoors (Magnusson, et al., 2012).

Primary sources of air pollution measured in Iraq and Afghanistan in one study were geological gust, smoke from burn pits and emissions from industrial processing facilities (Engelbrecht, et al., 2009). Dust and sand storms characteristic of the region were most responsible for elevated particulate matter levels, but trace metals (e.g., lead, arsenic, cadmium, and zinc) found within the PM2.5 fraction were the result of burn pits and industrial processing facilities (Engelbrecht, et al., 2009)

The guideline values set out by the WHO only represent the lower end of the range over which 'significant effects on survival' were observed. For example, 10µg is 'below the mean for most likely effects' according to the available scientific literature as at 2005 (World Health Organisation, 2005). Even the guidelines acknowledge that there is little evidence for a level below which no adverse health effects would be anticipated (World Health Organisation, 2005), and the risk for multiple health outcomes increased with increased exposure (World Health Organization, 1999). Additionally epidemiological evidence shows adverse effects from short and long-term exposures to PM (World Health Organization, 1999). 'Although adverse effects on health cannot be entirely ruled out below these levels, the annual average WHO AQG value represents that concentration of PM2.5 that has not only been shown to be achievable in large

urban areas in highly developed countries, but also the attainment of which is expected to significantly reduce the health risks' (World Health Organisation, 2005).

The background concentration of PM2.5 in Western Europe and the US at the time of the 2005 Air Quality Guidelines were 3-5 µg/m3 (World Health Organisation, 2005), whilst in developing countries, including Iraq and Afghanistan, the PM2.5 levels can routinely exceed 10,000µm/m³ for long periods of time (Falvo, et al., 2015). Increased levels of PM cause an increased risk of adverse health outcomes, including respiratory effects (Ciminera, et al., 2015; Marzouni, et al., 2016), cardiovascular effects (United States Environmental Protection Agency, 2009; Ciminera, et al., 2015; Marzouni, et al., 2016) and increased mortality (United States Environmental Protection Agency, 2009; Marzouni, et al., 2016).

The health effects depend on size, chemical composition and the duration or frequency of exposure (Ciminera, et al., 2015; Baird, 2016), and are difficult to measure due to the great variations in PM chemical and physical properties over time due to multiple factors (e.g. weather, time of day, source category) (United States Environmental Protection Agency, 2009). Both sizes are inhalable. PM10 particles are able to reach the upper respiratory tract (upper airways and lung), whilst PM2.5 particles are able to reach deeper into the lung (Weese, et al., 2009).

Minor effects occur for larger dusts (10-100µm) such as the irritation of the eyes, nose, mouth and upper respiratory tract, leading to nosebleeds, dry coughs and lip inflammation. It can also cause the irritation of the skin around the ears, armpits and groin (Korzeniewski, 2011). Smaller dust particles with diameters below 10 micrometres (10µm) are able to reach further into the respiratory tract (Weese, et al., 2009) and may become deposited in the alveolar region, where the inhaled particle can be absorbed into the blood (World Health Organization, 1999) Increased levels of fine particles are associated with heart disease, altered lung function and lung cancer (Patterson, et al., 2017).

Additionally, some toxicants and pollutants are particle bound meaning that proportion of the particles could contain additionally toxic chemicals attached to them. In fact, PM can be comprised of hundreds of different chemicals – each with their own toxicological profiles. These include PAHs, oxy-PAHs, metals/metalloids, nitrogen oxides, sulphur oxides, VOCs (aromatic compounds/hydrocarbons, n-alkanes) (Magnusson, et al., 2012).

Table 1: Summary of PM exposures reported by other sources in military deployment or combat zones.

	TSP µg/m^3	PM10 levels µg/m^3	PM5 levels µg/m^3	PM2.5 levels µg/m^3	
WHO annual		20		10	
WHO 24hrs		50		25	
US urban city		40		12	Engelbrecht , et al., 2009
US rural (desert)		13		5	
Kabul (outdoor geomean)		260		86	Magnusson, et al., 2012
MeS (outdoor geomean)		334		68	
Mes (workshop geomean)		153		41	
Djibouti	92	72		35	Engelbrecht , et al., 2009
Bagram, Afghanistan	174	108		38	
Khowst, Afghanistan	184	127		75	
Qatar	282	165		67	
UAE	196	140		52	
Balad, Iraq	242	184		56	
Badhdad, Iraq	371	250		103	
Tallil, Iraq	411	303		65	
Tikrit, Iraq	605	298		111	
Taji, Iraq	348	213		81	
Al Asad, Iraq	142	95		37	

Northern Kuwait	416	211		67	
Central Kuwait	352	298		87	
Coastal Kuwait	268	176		60	
Southern Kuwait	290	199		62	
Camp Victoria, Kosovo – summer	38 ± 4.6		25 ± 5.5		(Wingfors, et al., 2009)
Camp Victoria, Kosovo – winter	44 ± 12		36 ± 13		
Camp Victoria, Kosovo – summer	-		31		
Camp Victoria, Kosovo – winter	-		95 ± 69		
Camp Victoria, Kosovo – summer	-		96		
Camp Victoria, Kosovo – winter	-		56 ± 35		
Camp Victoria, Kosovo – summer	-		9		
Camp Victoria, Kosovo – winter	-		40 ± 24		

4.1 DUST STORM EVENTS

Until fairly recently there was a lack of information about the naturally occurring dust exposures commonly seen in Iraq and Afghanistan, instead with most studies focusing on other forms of air pollution, such as particulate matter, ozone, sulphur dioxide and carbon monoxide (Falvo, et al., 2015). A review by Falvo, et al. (2015) found that the evidence of associations between dust-storm events and cardiopulmonary morbidity and mortality is mixed, with studies finding both an increase and no association. The researchers concluded that the inconsistencies between the findings could be partly explained by regional variation in dust composition.

Two recently published studies in Iran have found an association between dust storm events and adverse health effects. Khaniabadi, et al. (2017) estimated the impact of Middle East Dust (MED) storms on

hospital admission for COPD and respiratory mortality (RM) in Ilam, Iran. They measured daily PM10 averages over a 1 year period (2015-2016) and found that increases in PM10 of as little as 10µg/m^3 were associated with an increase in COPD hospital admissions and RM, of 4.9% and 7.3% respectively. Additionally, hospital admissions increased when PM10 levels exceeded 200µg/m^3, as a result of MED events.

Another study of the effects of hourly PM10 on health in Kermanshah Iran found that 48% of all health effects in 2011 and 12% of all health effects in 2012 were attributable to MED storms. Of these health effects, in 2011, 13.5% of hospital admissions for respiratory disease and 15.1% of respiratory mortality were estimated to be associated with MED events. Of cardiovascular health effects, 7.6% of hospital admissions for cardiovascular disease and 11% of cardiovascular mortality were estimated to be associated with MED events (Marzouni, et al., 2016).

Additionally, a study of airborne hazards in Baghdad, Iraq, found that peak values for major soil-forming elements silicon, aluminium, calcium and manganese were associated with dust-blowing events (Engelbrecht, et al., 2009).

5. PARTICULATE MATTER (PM) IN DAMAGED BUILT ENVIRONMENT

In addition to pollution and naturally dustier environments, particulate matter can be elevated by a range of normal activities in the built environment. Therefore, any combat situations in built environment can present risks similar to many occupational exposures especially for those working on construction or demolition of buildings. The most relevant comparison presents work on explosion or implosion destruction of buildings.

Building demolition by implosion and the explosive destruction of buildings causes the release of airborne fibres and may additionally expose more fibre that must be handled with care in subsequent clearance or demolition works (Hoskins and Brown, 1994). In some cases, the very properties of the substances involved can lead to increased risks, and asbestos is a good example for this. Once liberated into the atmosphere following the building implosion or explosive destruction, the aerodynamic properties of asbestos ensure that respirable fibres can travel considerable distances, with the likelihood of localised high concentrations in and around damaged buildings (Hoskins and Brown, 1994). Airborne levels of fibre can remain high for months, with washout by rain or snow presenting the only cleaning mechanism for air (Hoskins and Brown, 1994). People at high risk from exposure to fibres are those in close proximity to the damaged buildings and those involved in clearance operations (Hoskins and Brown, 1994).

Following the implosion of a large inner-city hospital in Canada, airborne asbestos fibres were recorded at up to 0.362 f/mL at 50m from the site, which was considerably above the highest pre-implosion background level of 0.003 f/mL (Stefani et al., 2005). Before the implosion all friable and some non-friable asbestos was removed from the building to minimise asbestos exposure, however, the implosion was still

able to effectively aerosolize asbestos containing building materials, resulting in elevated levels of airborne asbestos recorded as far as 550m from the site (Stefani et al., 2005). Suspected asbestos containing materials that were not removed included plaster, mortar and floor tile (Stefani et al., 2005). Overall, public cancer risk was determined to be negligible, however, the impacts of high short term exposure could not be fully accounted for due to limited studies on the effects of short term exposure (Stefani et al., 2005). Additionally, surface deposition of airborne asbestos may create reservoirs for chronic exposure caused by tracking of outdoor contamination indoors, or posing a risk to children playing outdoors (Stefani et al., 2005).

Destruction of built environment in modern combat zones would share many features of explosions and implosions of buildings, presenting a risk to release of wide range of materials, fibres and particulate matter. In order to understand this better, this section overviews some of the most common particles released in construction and demolition of buildings.

5.1 CONTRIBUTION OF CONSTRUCTION AND DEMOLITION TO PARTICLE CONCENTRATIONS

Although numerous studies cite vehicular sources as a key source of particle emissions, comprising up to 80% of total PM10 and/or PM2.5 in urban areas, research also shows construction and demolition activities to have a significant impact on particle concentrations (Kumar, Pirjola, Ketzel, and Harrison, 2013). Many combat situations include exposure to demolition and therefore similar particle emissions.

Literature attempting to quantify the health impact from demolition report a wide range of results with little consistency in values, however, the general consensus in these findings is that construction and demolition activities result in a measurable increase in coarse and fine airborne particles (Table 2).

Table 2. Studies quantifying airborne particle emissions from construction and demolition activities

Study Overview	Country	Activity	Measure	Key Findings	Source
Analysis of how the PM10 fraction of road dust varies in load and chemical composition in different urban zones	Spain	Demolition	PM10 concentration	PM10 concentrations increased by up to ten times as a result of demolition work	(Amato et al., 2009)
Analysis of atmospheric particulate matter data from national Automatic Urban Air Quality Monitoring stations	UK	Demolition	PM10 concentration	Daily maximum exceedances of PM10 doubled in a period when demolition of a building had been carried out near a monitoring station	(Deacon et al., 1997)

Analysis of the indoor and outdoor air quality impact of imploding a 22-story building	USA	Implosion	PM10 concentration	Short term peak concentrations of PM10 during demolition over 1000 times higher than pre-implosion levels	(Beck et al., 2003)
Analysis of the impact of PM10 arising from a building and road works at over 80 monitoring sites	UK	Construction	PM10 concentration	Building and road works close to monitoring sites can cause daily mean PM10 to exceed the 50µgm-3 EU Limit Value	(Fuller and Green, 2004)
Analysis of particulate matter concentration at various distances during high-rise demolition	USA	Demolition	PM10 concentration	4 to 9 times increase of 6-h averaged PM10 concentrations against background levels at 42 m downwind of a demolition site	(Dorevitch et al., 2006)
Review of literature on nanoparticle emissions from 11 non-vehicle exhaust sources	Various	Construction /demolition	Total PNCs	PNCs close to construction and demolition activities comparable with and up to an order of magnitude larger than from road-tyre interaction	(Kumar et al., 2013)
Analysis of atmospheric particulate matter data from air quality monitoring stations near construction sites	UK	Construction	PM10 and PM2.5 concentration	Construction works close to monitoring sites can cause daily mean PM10 to exceed the 50µgm-3 EU Limit Value	(Azarmi et al., 2016)
Analysis of dust exposure among construction workers employed at different work posts	Poland	Construction	Dust concentration	Highest exposure in varnishing, renovation and welding for inhalable dust; and in welding, renovation and materials treatment for respirable dust	(Bujak-Pietrek and Szadkowska-Stanczyk, 2009)
Analysis of particulate matter exposure at and near building demolition sites	UK	Demolition	PM10, PM2.5 and PM1 concentration	Average respiratory deposited doses to coarse and fine particles inside the on-site temporary office increased by 13 and 2 times the background level	(Azarmi and Kumar, 2016)

Analysis of aerosol particles resulting from a skyscraper blasting	Germany	Blasting	PM10 concentrations	Maximum PM10 concentrations of 844.9 µgm-3 present for a short period but day mean of 32.6 µgm-3 did not exceed the 50 µgm-3 EU Limit Value	(Wagner et al., 2017)
Analysis of aerosol particles resulting from a hospital building implosion	Canada	Implosion	PM10 and PM2.5 concentrations	Elevated levels of airborne PM recorded as far as 20km from the building implosion site	(Stefani et al., 2005)
Analysis of building demolition contribution to the airborne particulate budget	UK	Demolition	PM10 concentrations	PM10, monitored 40 metres from the demolition site, indicated no abnormal concentrations during the demolition, possibly due to rainfall	(Brown et al., 2015)
Analysis of the differences in dust exposure between carpenters and demolition workers	Denmark	Construction /demolition	Dust concentrations	Exposure to respirable dust much higher for demolition workers (1.06 mg/m3) compared to carpenters (0.27 mg/m3)	(Kirkeskov et al., 2016)
Development of a probabilistic risk assessment model to explore the health effects of construction dust	China	Construction	Health risk	Workers in the template zone had the largest health risk compared to the steel, concrete and floor zones, while the office zone had the lowest risk	(Tong et al., 2018)
Development of a regional dust dynamical model to simulate surface dust concentrations	China	Construction	Dust concentrations	Urban construction activities contribute to 42% of dust emissions and are therefore a crucial factor in controlling urban dust pollution	(Li et al., 2016)

In order to understand health impact from demolition, it is important to know which materials are the main generators of dust, because dust from a range of typical building materials will have a range of different health impacts.

5.2 CONCRETE PARTICULATE MATTER

Literature reporting the negative health effects of concrete and dust from it cover three major themes: the respiratory effects of cement dust, the association of exposure to crystalline silica with various diseases, and the particle emissions produced during various construction and demolition activities involving concrete.

The cement industry is considered a major contributor to pollution due to the emission of dust and particulate matter at various stages of cement production (Pournourmohammadi et al., 2008). While cement is a key ingredient in the manufacture of concrete, widely used in the construction industry, literature reporting negative health impacts in relation to cement dust is predominantly limited to the assessment of worker exposure within cement factories.

The harmful effects of cement dust on living organisms are reportedly due to the irritating, sensitising and pneumoconiotic properties of its components (Maciejewska and Bielichowska-Cybula, 1991). This is most often associated in the literature with impaired lung function (Kakooei et al., 2012; Maciejewska and Bielichowska-Cybula, 1991; Meo, 2004; Meo, Al-Drees, Al Masri, Al Rouq, and Azeem, 2013; Moghadam, Abedi, Afshari, Abedini, and Moosazadeh, 2017; Oliver, Miracle-McMahill, Littman, Oakes, and Gaita, 2001; Pournourmohammadi et al., 2008).

Specific health impacts associated with long term exposure to cement dust for cement factory workers include:

- disorders of the upper respiratory airways, such as chronic rhinitis, laryngitis and pharynx catarrh (Maciejewska and Bielichowska-Cybula, 1991)
- chronic bronchitis (Maciejewska and Bielichowska-Cybula, 1991)
- cement pneumoconiosis (Maciejewska and Bielichowska-Cybula, 1991; Meo, 2004)
- inflammatory skin changes and in some cases chemical burns (Maciejewska and Bielichowska-Cybula, 1991)
- cough, sputum, wheezing and dyspnea (Kakooei et al., 2012)
- carcinoma of the lungs, stomach and colon (Meo, 2004)
- cement dust entering into the systemic circulation and thereby reaching all the organs of the body, affecting their micro-structure and physiological performance (Meo, 2004)

Occupational exposure to cement dust for workers in heavy and highway construction environments has also been assessed in one study, reporting increased risk for asthma and chronic bronchitis for labourers, tunnel workers and operating engineers (Oliver et al., 2001).

Table 3: PND, PNC, PM10, PM2.5 and PM1 values recorded as times higher than background levels and percentage of ultrafine particles recorded for various construction and demolition activities.

Activity	PND	PNC	PM10	PM2.5	PM1	UFP	Source
Mixing (GGBS)	3	4	32	58	86	Not recorded	(Azarmi et a., 2014)
Mixing (PFA)	12	15	32	50	89	Not recorded	(Azarmi et al., 2014)
Drilling	3.5	4	45	80	115	Not recorded	(Azarmi et al., 2014)
Cutting	8	14	50	80	112	Not recorded	(Azarmi et al., 2014)
Slab demolition		14				79% of total PNC	(Kumar et al., 2012)
Crushing of cubes		2				95% of total PNC	(Kumar et al., 2012)
Dry Recycling		17				73% of total PNC	(Kumar et al., 2012)
Wet Recycling		3				90% of total PNC	(Kumar et al., 2012)

5.2 CONCRETE PARTICULATE MATTER

The manufacture of fresh concrete, involving the mixing of coarse and fine aggregates with cement, water and admixtures in a rotating drum mixer, is an activity commonly associated with the generation of air-borne dust during construction work (Azarmi et al., 2014). Emissions and exposure to particles during the mixing of cement is reported to vary depending on the composition of the mixture, affected by factors such as the incorporation of Ground Granulated Blastfurnace Slag (GGBS), or Pulverised Fuel Ash (PFA) (Azarmi et al., 2014). Simulations of mixing activities undertaken with GGBS and PFA have resulted in peak PND values 3 and 12 times higher than the background peak, average PNC values 4 and 15 times higher than the background and PM10, PM2.5 and PM1 values 32, 58 and 86, and 32, 50 and 89 times the background (Azarmi et al., 2014). The mixing of concrete containing PFA resulted in a higher production of nucleation mode particles (those below 30µm) than GGBS, which is thought to reflect particle size, density and adhesion of the materials (Azarmi et al., 2014).

The cutting and drilling of hardened concrete are common activities associated with the production of coarse and fine particles (Azarmi et al., 2014). While drilling is generally associated with construction work, cutting is common during refurbishment, maintenance and demolition (Azarmi et al., 2014). Cutting and drilling activities have been found to release significantly more ultrafine particles than mixing activities, which is thought to reflect the higher rotational frequency, shear stresses and local energy density of drilling and cutting (Azarmi et al., 2014).

Simulations of drilling activities have resulted in peak PND values 3.5 times higher than the background peak, average PNC values 4 times higher than the background and PM10, PM2.5 and PM1 values 45, 80

and 115 times the background (Azarmi et al., 2014). Simulations of cutting activities have resulted in peak PND values 8 times higher than the background, average PNC values 14 times higher than the background and PM10, PM2.5 and PM1 values 50, 80 and 122 times the background (Azarmi et al., 2014). These values show that in comparison to drilling activities, cutting activities produce more particles, by number as well as mass (Azarmi et al., 2014). This is likely to reflect the larger surface area of concrete subjected to abrasion during cutting activities (Azarmi et al., 2014). Cutting activities are also reported to have a higher emission factor than drilling, representing a higher number and mass of particles that could be inhaled by an occupant during the activity (Azarmi et al., 2014).

While construction activities relating to concrete have been found to cause large quantities of coarse and fine particles, studies have found demolition and recycling activities to additionally produce ultrafine particles (below 100µm in diameter) (Jabbour et al., 2017; Kumar and Morawska, 2014; Kumar et al., 2012). These ultrafine particles are of increased concern due to their slower observed decay rate, reaching greater distances from the source, as well being hypothesised to pose a greater risk to human health than larger particles (Kumar and Morawska, 2014). The fracture of concrete has been reported by one study as the cause of the release of ultrafine particles from concrete, observing particles released with diameters of 27, 39 and 49 µm (Jabbour et al., 2017). The hypothesis for the cause of this ultrafine particle production is based on the production of secondary particles, resulting from the volatilisation of materials at the concrete fracture interface (Jabbour et al., 2017). As literature reports no known method for mechanical processes to create volatile secondary particles, such as those observed, the study concludesdthe mechanism for particle production is nucleation, occurring during the instant of the fracturing event due to sudden localised temperature increase (Jabbour et al., 2017).

Additional studies have further investigated and quantified the release of ultrafine particles from recycling and demolition activities involving the fracturing and crushing of concrete (Kumar and Morawska, 2014; Kumar et al., 2012). The total PNCs during concrete cube crushing and impact hammer demolition of a concrete slab are reported at 2 and 14 times the background PNCs respectively (Kumar et al., 2012). The majority of new particle emission for these activities was recorded in the ultrafine range, contributing to 95% and 79% of total PNCs for crushing and slab demolition respectively (Kumar et al., 2012). Although not conclusively demonstrated, the results of the study suggested that loading rate and concrete strength was likely to impact the size range of particles released during the activities (Kumar et al., 2012).

Due to the large proportions of ultrafine particles released during recycling and demolition activities, one study has reported respirators to have limited effectiveness, due to their design for the removal of particles over 560µm in size (Kumar and Morawska, 2014). Investigation of a dust respirator with a protection factor of 4, based on classification in standard EN149:2001, reported particles observed inside the mask at 2-times lowers that outside the mask, rather than 4-times lower as the classification suggests the mask should achieve (Kumar and Morawska, 2014). This limited effectiveness was due to a large majority of particles in the ultrafine range, with removal factor for these particles observed at only 1.78 (Kumar and Morawska, 2014).

Water spraying is cited as a common measure for suppressing airborne dust particles, reported to suppress PM10 particle concentrations during building demolition activities by up to 10 times (Kumar et al., 2012). Further evidence of water as an effective suppression method is shown by studies reporting a decrease in ultrafine particles with increasing relative humidity (de Hartog et al., 2005) and the fundamental phenomena of ultrafine particle scavenging (rainout) and aerosol–hydrometer coagulation (washout) (Kumar et al., 2012).

To determine the effectiveness of this technique at suppressing airborne particles produced by concrete recycling activities, one study has compared particle emissions for wet and dry recycling (Kumar et al., 2012). As was expected, the largest magnitudes of PNDs were found during dry recycling, demonstrating the effectiveness of particle suppression through water spraying (Kumar et al., 2012). Peak PNCs were also found to be higher for dry recycling, recorded at 17 times the background levels, compared to only 3 times the background levels for wet recycling (Kumar et al., 2012). During wet recycling, a higher relative proportion of ultrafine particles was recorded, suggesting water spray suppression may be more effective for larger sized particles compared to ultrafine particles (Kumar et al., 2012).

The incorporation of carbon nanotubes and plasticisers as admixtures within the concrete mix are reportedly becoming increasingly common, desired for their ability to enhance the workability and strength of concrete (Nazari and Riahi, 2011; Sanchez and Sobolev, 2010). This nano-modified concrete is considered a concern to human health due to the potential for the material to release in the ultrafine and nanosize range when fractured (Kumar and Morawska, 2014; Kumar et al., 2013).

5.3 SILICA PARTICULATE MATTER

As quartz is an abundant mineral, silica dust is largely emitted into the atmosphere from natural sources within wind generated soil dust (Gillette, 1997). However, industrial sources such as construction, demolition, foundries, glass manufacturing, abrasive blasting, industrial or commercial use of silica sand, mining and rock crushing also contribute significantly to respirable crystalline silica (RCS) emissions (Ehrlich et al., 2013). The quartz content of building materials such as concrete, mortar and bricks is 10-50%, therefore, the construction, renovation and demolition of buildings containing these materials can result in increased concentrations of silica dust (Brendstrup et al., 1990). Due to the well-known risk of occupational exposure to RCS, a number of occupational safety standards exist to limit exposures, utilised within the literature to evaluate measures of RCS for a range of building related activities and occupations.

RCS emissions resulting from a range of building related activities have been quantified by a number of studies aiming to determine which activities are likely to exceed occupational safety limits (Table 4). The most commonly noted building activities contributing to RCS include concrete related activities, due to the high quartz content of concrete, as well as abrasive blasting activities, due to the common use of silica sand as a blasting material. Although quantities vary greatly between studies, affected by distance of measurement from the source, the results of these studies show frequent exceedance of occupational safety limits and significant increases in RCS above ambient background levels.

Table 4. Respirable crystalline silica concentrations for various construction related activities, compared to baseline ambient measurement and recommended exposure limits.

Activity	**Country**	**Maximum RCS (mg/m3)**	**Average RCS (mg/m3)**	**Source**
Building sites	Denmark	1.8		(Brendstrup et al., 1990)
Abrasive blasting of concrete structures	US	14	2.8	(Linch, 2002)
Drilling concrete highway pavement	US	4.4	3.3	(Linch, 2002)
Concrete wall grinding (new building construction)	US	0.66	0.26	(Linch, 2002)
Concrete sawing	US	14	10	(Linch, 2002)
Milling of asphalt from concrete highway pavement	US	0.34	0.36	(Linch, 2002)
Pick and shovel work	Various		0.08	(Beaudry et al., 2013)
Abrasive blasting	Various		1.59	(Beaudry et al., 2013)
Masonry cutting	Various		0.7	(Beaudry et al., 2013)
Concrete grinding	Various		0.56	(Beaudry et al., 2013)
Breaking/Jack hammering concrete	Various		0.41	(Beaudry et al., 2013)
Cutting tunnels	Various		0.39	(Beaudry et al., 2013)
Sand and mineral processing	Canada	1.7	0.09	(Radnoff et al., 2014)
New commercial building construction	Canada	1	0.055	(Radnoff et al., 2014)
Aggregate mining and crushing	Canada	0.19	0.048	(Radnoff et al., 2014)
Abrasive blasting	Canada	0.12	0.027	(Radnoff et al., 2014)
Demolition	Canada	0.065	0.027	(Radnoff et al., 2014)
Earth Moving/ Road Building	Canada	0.068	0.013	(Radnoff et al., 2014)

Building demolition (downwind)	UK	0.012	0.001	(Stacey et al., 2018)
Brick cutting with grinder (downwind)	UK	0.012	0.006	(Stacey et al., 2018)
Road building (downwind)	UK	0.001	0.001	(Stacey et al., 2018)
Rural air ambient baseline	UK	0.0001	1.90E-05	(Stacey et al., 2018)
Urban air ambient baseline	UK	0.0003	1.00E-04	(Stacey et al., 2018)
Sahara Dust (Airborne dust episode)	UK		0.00034	(Stacey et al., 2018)
Quebec Occupational Exposure Limit	Canada	0.1		
NIOSH Recommended Exposure Limit	US	0.05		
ACGIH threshold limit	US	0.025		
Alberta Occupational Exposure Limit	Canada	0.025		

Another key theme within the literature is the quantification of exposure to RCS for different occupations, resulting from both direct exposure, while undertaking activities known to generate silica dust, and incidental exposure, resulting from proximity to such activities (Table 5). These studies aim to determine which occupations may be at a higher risk of exposure and where safety measures need to be undertaken. Results from these studies demonstrate exposures for the majority of trades to exceed occupational safety limits, highlighting the need for safety measures to be undertaken throughout work sites, rather than only for trades directly involved in RCS related activities.

Table 5. Respirable crystalline silica concentrations for various construction related occupations, compared to recommended exposure limits.

Occupation	**Country**	**Maximum RCS (mg/m3)**	**Average RCS (mg/m3)**	**Source**
Demolition workers	Iran	0.185		(Normohammadi et al., 2016)
Plumber/Steamfitter	Various		0.01	(Beaudry et al., 2013)
Tunnel construction skilled labour	Various		0.3	(Beaudry et al., 2013)

Cement mason/Concrete finisher	Various		0.28	(Beaudry et al., 2013)
Bricklayer/Stone Mason	Various		0.17	(Beaudry et al., 2013)
Ceramics plant workers	Israel	> 0.05		(Kolton et al., 1981)
Brick layer/mason/concrete cutting	Canada	1	0.105	(Radnoff et al., 2014)
Dispatcher/shipping, administration	Canada	0.084	0.008	(Radnoff et al., 2014)
Painter	Canada	0.12	0.036	(Radnoff et al., 2014)
Labourer	Canada	3.5	0.032	(Radnoff et al., 2014)
Carpenter	Canada	0.041	0.023	(Radnoff et al., 2014)
Supervisor/Foreman	Canada	0.28	0.016	(Radnoff et al., 2014)
Electrician	Canada	0.064		(Radnoff et al., 2014)
Welder	Canada	0.13		(Radnoff et al., 2014)
Truck Driver	Canada	0.11	0.013	(Radnoff et al., 2014)
NIOSH Recommended Exposure Limit	US	0.05		
Quebec Occupational Exposure Limit	Canada	0.1		
ACGIH threshold limit	US	0.025		
Alberta Occupational Exposure Limit	Canada	0.025		

Finally, the literature also explores the effectiveness of a number of control measures, aimed at reducing occupational exposure to RCS (Table 6). These studies generally conclude that while measures were significantly effective at reducing exposure, the resulting exposures still often exceeded occupational safety limits.

Table 6. Respirable crystalline silica concentrations for various construction related activities with and without control measure in place, compared to recommended exposure limits.

Activity	**Control Measure**	**RCS (mg/m3)**	**Source**
Indoor concrete grinding	Uncontrolled	61.7	(Akbar-Khanzadeh et al., 2007)
Indoor concrete grinding	Wet grinding	0.896	(Akbar-Khanzadeh et al., 2007)
Indoor concrete grinding	Local exhaust ventilation	0.155	(Akbar-Khanzadeh et al., 2007)
Concrete surface grinding	Uncontrolled	4.5	(Croteau et al., 2004)
Concrete surface grinding	Local exhaust ventilation	0.14	(Croteau et al., 2004)
Sandstone grinding	Uncontrolled	4.2	(Healy et al., 2014)
Sandstone grinding	On-tool shroud	0.03	(Healy et al., 2014)
Abraisive blasting of metal products	Sand blasting product	0.039	(Radnoff and Kutz, 2014)
Abraisive blasting of metal products	Non-sand blasting product	0.031	(Radnoff and Kutz, 2014)
Quebec Occupational Exposure Limit		0.1	
NIOSH Recommended Exposure Limit		0.05	
ACGIH threshold limit		0.025	
Alberta Occupational Exposure Limit		0.025	

The size of crystalline silica particles of most concern are those smaller than four microns (PM4), as these are considered respirable (Ehrlich et al., 2013). This Respirable Crystalline Silica (RCS) is classified by the International Agency for Research on Cancer (IARC) as a Group 1 carcinogen to humans (IARC, 1997), and is ranked among the most frequent occupational exposures to an established human carcinogen (Dahmann et al., 2008). Health effects associated with exposure to RCS include silicosis, associated with the development of lung cancer, various forms of chronic obstructive pulmonary disease (COPD) and a number of lesser documented effects such as pulmonary tuberculosis, rheumatoid arthritis, autoimmune disease, and renal disease.

Exposure to RCS has been associated within numerous research articles with the development of silicosis, as well as an increased probability of developing lung cancer (Ehrlich et al., 2013). The IARC has classified crystalline silica dust as a Group 1 carcinogen to humans based on epidemiological studies that reveal association between exposure to crystalline silica dust and an increased probability of developing lung cancer, as well as studies involving the administration of crystalline silica to rats by inhalation or intratracheal instillation that led to the development of lung tumours (IARC, 1997). Epidemiological studies show that the incidence of lung cancer is increased in workers with silicosis, therefore leading the IARC and The European Commission's Scientific Committee for Occupational Exposure Limits (SCOEL) to conclude that the first step in the reduction of cancer risk is to prevent silicosis through the reduction of exposure to RCS (IARC, 1997; SCOEL, 2003). It is reported that silicosis may occur in acute, accelerated and chronic forms with latency periods ranging from weeks to 40+ years (Mason and Thompson, 2010).

Occupational exposure is considered an important risk factor for chronic obstructive pulmonary disease (COPD), with silica dust considered one of the most important occupational respiratory toxins (Hnizdo and Vallyathan, 2003). Epidemiological and pathological studies suggest that even in the absence of radiological signs of silicosis, silica dust exposure can lead to the development of COPD, such as bronchitis, emphysema and/or small airways disease, and that the association between cumulative silica dust exposure and airflow obstruction is independent of silicosis (Hnizdo and Vallyathan, 2003). In addition, a review of studies on exposure to respirable quartz dust at the workplace and the development of COPD reported that most studies found a significant negative association of FEV1 and FEV1/FVC related to increasing exposure to crystalline quartz at the workplace, revealing airway obstruction consistent with COPD (Bruske, Thiering, Heinrich, Huster, and Nowak, 2014). Analysis of data from the US National Occupational Mortality Surveillance (NOMS) system also concluded that those postulated to have had detectable crystalline silica exposure had a significantly increased risk for COPD (Calvert, Rice, Boiano, Sheehy, and Sanderson, 2003). Longitudinal studies suggest that loss of lung function occurs with exposure to silica dust at concentrations of between 0.1 and 0.2 mg.m3, however, in the absence of silicosis, studies suggest this would not occur until between 30 and 40 years exposure (Rushton, 2007).

Literature reports consistently elevated risks of developing COPD associated with silica exposure for occupations within a range of industries, including construction, tunnelling, cement, brick manufacturing, pottery and ceramic, silica sand, granite and diatomaceous earth, gold mining and iron and steel founding (Rushton, 2007). Factors influencing variation in risk between industries includes; the presence of other minerals in the dust, particularly when associated with clay minerals; the size of the particles, the percentage of quartz and the physicochemical characteristics, such as whether the dust is freshly fractured (Rushton, 2007).

Although crystalline silica exposure is widely associated with silicosis, lung cancer and COPD, there is less, but still statistically significant support for an association with pulmonary tuberculosis, rheumatoid arthritis, autoimmune disease, and renal disease (Calvert et al., 2003; Mason and Thompson, 2010).

Because of the nature of activities and potential for destruction of buildings in combat zones, many of the health trends observed with the high levels of construction and demolition dust would also apply to military personnel.

5.4 MINERAL FIBRES (ASBESTOS)

Mineral fibres, including asbestos, are ubiquitous environmental contaminants, occurring partially due to natural sources such as the erosion of asbestos and asbestiform rocks or sand and the air lofting of dried clay or alluvial deposits (Hoskins and Brown, 1994). However, since the start of the industrial revolution, concentrations of these fibres have increased by approximately 10-fold due to human activities (Hoskins and Brown, 1994). Asbestos fibres are generally present at background levels below 1 fibre/L (0.001 f/mL) and up to 10 fibres/L (0.01 f/mL) in cities, which are assumed low enough not to present health risk (Hoskins and Brown, 1994).

Buildings may contain mineral fibres within thermal and acoustic insulation, fireproofing materials, the thermal lagging of pipes and boilers, structural reinforcing for cement products, cladding and roofing sheets or slates, soffit boards, flue pipes, rainwater pipes, gutters and large water mains (Hoskins and Brown, 1994). Additionally, some non-fibrous building materials may contain asbestos fibres as an impurity (Hoskins and Brown, 1994). Renovation, maintenance, weathering and demolition of buildings containing mineral fibres have therefore been found to increase airborne fibre concentrations (Table 7) due to the disturbance or damage of the building materials.

Table 7. Airborne fibre concentrations resulting from buildings.

Activity	**Airborne fibre concentration (f/mL)**	**Source**
Exposure of maintenance and repair workers in buildings with asbestos-containing materials	0.002 - 0.02	(Price et al., 1992)
Exposure of occupants not involved in maintenance and repair work in buildings with asbestos-containing materials	0.00003 - 0.0005	(Price et al., 1992)
Renovation of university building with asbestos-containing materials	0.0038 ± 0.0011	(Latif et al., 2011)
Roofers cutting corners of asbestos-cement sheets	100 (peak)	(Rodelsperger et al., 1980)
Sound absorbing mineral fibre boards installed as suspended ceilings in an office building	0.001 - 0.0035	(Thriene et al., 1996)
Production of ceramic fibres	0.07 - 0.27	(Wozniak and Wiecek, 1996)

Manufacture of ceramic fibre products	0.23 - 0.71	(Wozniak and Wiecek, 1996)
Application of ceramic fibre products	0.07 - 1.67	(Wozniak and Wiecek, 1996)
Water jet cleaning or painting of weathered asbestos cement roofing	0.1 - 0.2	(Brown, 1987)
Asbestos cement roofing replacement	0.1	(Brown, 1987)
Asbestos cement roofing demolition by removal of whole sheets	0.3 to 0.6	(Brown, 1987)
Asbestos cement wall demolition by removal of whole sheets	<0.1	(Brown, 1987)
The vicinity of buildings with weathered and corroded asbestos-cement products	0.00075	(Spurny et al., 1988)
Public housing demolition	None observed	(Dorevitch et al., 2006)
Skyscraper demolition by blasting	None observed	(Wagner et al., 2017)
3 hour average asbestos fibres immediately following implosion of a hospital building (measurements 50m from site)	<0.006 / 0.128 / 0.362	(Stefani et al., 2005)
21 hour average asbestos fibres post implosion of a hospital building (measurements 50m from site)	0.001 / 0.008 / 0.005	(Stefani et al., 2005)
Alberta indoor air clearance criterion following asbestos abatement	0.01	(Stefani et al., 2005)

Although asbestos is commonly used for its fire protective properties, in sheet form it does not offer fire resistance and is likely to crack or break apart in fires, especially if worn or impregnated with resin (Hoskins and Brown, 1994). Asbestos cement sheeting can also disintegrate explosively in fire, causing light fibrous material to loft and spread for several kilometres from the source, especially in the direction of the prevailing wind (Hoskins and Brown, 1994). Measurements taken after a factory fire in England found asbestos containing fallout within the surrounding urban area due to asbestos bitumen paper covering the factory roof (Bridgman, 2001). The lung cancer risk resulting from this was determined to be undetectably small due to the low level of public exposure, however, academically it is assumed there is no minimum exposure threshold for asbestos to cause cancer (Bridgman, 2001).

Asbestos miners and workers engaged in fabricating asbestos containing materials who were exposed to large quantities of airborne dust have long been known to suffer from the lung disease asbestosis, caused by the deposition of scar tissue in the lungs (Hoskins and Brown, 1994). Without significant occupational

exposure, such as handling, processing or removing asbestos, it is unlikely exposure levels are high enough to produce any demonstrable clinical effect to give cause for concern (Hoskins and Brown, 1994). However, paraoccupational exposure experienced in families of asbestos workers or exposure resulting from working or living near crocidolite asbestos mines or factories have been associated with the development of pleural mesothelioma and possibly lung cancer (Hoskins and Brown, 1994).

The risk of developing lung cancer increases linearly with cumulative exposure, while the risk of developing mesothelioma is proportional to fibre concentration and is exponentially related to time since first exposure (Hoskins and Brown, 1994). Lower exposures can also result in the formation of pleural plaques, however, this is not associated with any illness or possible progression into a more damaging condition (Hoskins and Brown, 1994). While most literature cites long term exposure as a health risk, single acute exposure to amphibole asbestos has also been associated with tumours appearing from 20 to 50 years later (Hoskins and Brown, 1994). Additionally, the risks of developing lung cancer from smoking and asbestos exposure are multiplicative (Hoskins and Brown, 1994).

The health effects of asbestos fibres are most clearly associated with inhalation, however, only a fraction of airborne fibres, those between 0.01pm and 3pm diameter, are of a size to be respirable and therefore a cause for concern (Hoskins and Brown, 1994). Contamination of food and water supplies are considered of less concern than airborne fibres as the ingestion of fibres is not as clearly associated with health effects, although elevated asbestos concentrations have been detected in colon cancers of asbestos workers (Hoskins and Brown, 1994). Some evidence suggests that short ingested fibres are able to penetrate the wall of the intestine and can then either be eliminated in the urine or accumulated in various organs (Hoskins and Brown, 1994).

5.5 MOULD AND FUNGAL SPORES

Demolition or destruction of buildings can lead to release of accumulations within the wall and other building cavities which could otherwise remain undisturbed for years. Mould and fungal spores can be contained in such placed. This would be especially common in wet combat conditions or where damaged buildings have been exposed to rain over a period of time, and it is one of the exposures that is not easy to fully predict or quantify.

The increased issue of fungal spores due to construction and demolition work is most commonly discussed in relation to the demolition or renovation of hospital buildings, due to the risk of exposure for immunocompromised hospital patients. Additionally, health risks are associated with the handling of mouldy building materials, especially of concern to disaster relief workers following hurricanes, typhoons, tropical storms and floods.

Construction and demolition works in or near hospitals are commonly cited as carrying a risk to patients, due to the health risk of fungal airborne infection, especially for immunocompromised patients (Barreiros, Akiti, Magalhaes, Nouer, and Nucci, 2015; Cheng and Streifel, 2001; Haiduven, 2009; Hansen, Blahout, Benner, and Popp, 2008; Streifel, Lauer, Vesley, Juni, and Rhame, 1983; Wirmann, Ross, Reimann,

Steinmann, and Rath, 2018). However, when sufficient preventive measures are in place, studies conclude there is no significant increased risk for invasive aspergillosis in immunocompromised patients (Barreiros et al., 2015; Hansen et al., 2008; Streifel et al., 1983; Wirmann et al., 2018). Key preventative measures include sealing the building windows and doors, or sealing occupied areas with impermeable plastic foil, as well as manipulating air-handling systems and continuously monitoring air quality to minimise the infiltration of airborne fungi (Barreiros et al., 2015; Hansen et al., 2008; Streifel et al., 1983).

The concentrations of airborne microbes during the repair of mouldy buildings have been found to increase during repair work, despite dust levels remaining low (Rautiala et al., 1996). Concentrations of viable total bacteria were also found to increase, however less significantly than airborne fungi (Rautiala et al., 1996). Study results concluded that workers involved in the repair of buildings with mouldy materials were exposed to high concentrations of microbes (Rautiala et al., 1996).

Therefore, due to the risk of fungal and bacterial infestation of damp building materials, natural disasters such as hurricanes, typhoons, tropical storms and floods present a health risk to unprotected workers, first responders, home owners and volunteers working in post disaster recovery and restoration (Johanning, Auger, Morey, Yang, and Olmsted, 2014). Remediation and demolition work of water damaged buildings post disaster can impact the airborne concentrations of microbes and their by-products significantly, leading to a higher exposure risk, especially of concern to unprotected workers and volunteers (Johanning et al., 2014).

Dampness-related fungi are associated with allergies, respiratory symptoms and diseases such as dermatitis, rhinosinusitis, bronchitis and asthma (Johanning et al., 2014). Additionally, immunological system, cognitive, endocrine, or rheumatological changes have been reported in association with disaster relief work (Johanning et al., 2014). Therefore, avoidance or minimisation of unnecessary fungal exposure is generally recommended in disaster response and recovery work, with the employment of trained remediation workers with medical clearance and proper personal protection equipment (PPE) recommended for larger scale projects (Johanning et al., 2014). Minimum PPE should consist of a facial dust mask, such as the National Institute for Occupational Safety and Health (NIOSH)-approved N95 respirator, with additional skin and eye protection recommended (Johanning et al., 2014).

Prevention of fungal infestation requires addressing moisture or water intrusion rapidly, as mould growth can occur within 48 hours (Johanning et al., 2014). If this is unsuccessful, systematic source removal is recommended, which can be achieved by cleaning with soap and water, or through the bulk removal of damp materials, followed by high-efficiency particulate air vacuuming (Johanning et al., 2014). The use of biocides is recommended to be avoided in occupied areas (Johanning et al., 2014).

5.6 LEAD AND HEAVY METALS FROM BUILT ENVIRONMENT

Any heavy metals used in the construction of buildings can also be released during their demolition or destruction. The most studied heavy metal in buildings is lead, because it has been commonly used in paint, plumbing and other building applications for a long time.

Two key themes are present within the literature discussing lead dust exposure in relation to the built environment. The first theme relates to exposure to lead through soil and house dust, most commonly discussed as resulting from long term deposition of lead from leaded paint and petrol. The second theme relates to the contribution of building demolition to the quantity of lead in ambient dust, leading to higher ambient exposure to lead in areas near demolition sites, typically of older buildings containing leaded paint. Lead dust is not discussed in the literature as an occupational health risk, but rather a public health risk, especially to children.

Historical use of leaded gasoline, lead in paint, smelting and mining activities, roof tiles, disposal of coal ashes and fertilization of land with city waste have contributed to lead pollution in urban soils (Laidlaw and Taylor, 2011; Walraven et al., 2016). Natural disasters are also discussed as an important contributor to lead in soil and dust, due to the destruction of buildings and deposition of sediment (Rabito Felicia, Iqbal, Perry, Arroyave, and Rice Janet, 2012). Comparison of residential soil and dust lead before and two years after Hurricane Katrina found significantly higher median soil lead levels, with 61% of households having lead above federal standards in soil and dust samples collected in and around the residence (Rabito Felicia et al., 2012).

Tracking in of lead dust and soil is discussed as a significant contributor to interior floor dust lead, with secondary contributors including the presence of lead based paint and smoking inside the home (Davies, Watt, and Thornton, 1987; Hunt, Johnson, Thornton, and Watt, 1993; Lucas et al., 2014). Common areas and entry spaces have been found to contain the highest levels of lead dust, suggesting the significance of exterior sources rather than interior sources of lead dust (Davies et al., 1987; Lucas et al., 2014).

Exposure to lead in soil and house dust is commonly cited as of most concern to children, and presents a greater risk factor for lead poisoning than exposure to lead based paint to children engaged in hand-to-mouth and pica behaviour (Mielke and Reagan, 1998). Therefore, abatement of soil lead is suggested to be more effective than abatement of lead based paint in reducing blood lead levels in children (Mielke and Reagan, 1998). Additonally, housing where soil lead hazard control activities had been performed was reported to have lower post intervention exterior entry, interior entry floor, windowsill, and other floor lead dust loading levels (Clark et al., 2004).

Short term air quality issues resulting from the implosion of a large inner-city hospital in Canada are reported to include a significant increase in settled and airborne lead dust against background levels (Stefani et al., 2005). At a measurement site 50m from the building site, average airborne lead increased from pre-implosion levels of 0.003μg/m^2, to 4.5μg/m^2 during the 3 hours of implosion and 0.04 μg/m^2 for the 21 hours after the implosion (Stefani et al., 2005). Additionally, lead in settled dust increased from <25 μg/ft^2 pre-implosion, to post implosion levels of 55, 239 and 1347μg/ft2 at three measurement sites 50m from the source, and 1347μg/ft2 at a measurement site 400m from the source (Stefani et al., 2005).

The implosion dust cloud travelled out to 20km from the demolition site, highlighting the risk of personal exposure and indoor migration of implosion dust at a large distance from the source (Stefani et al., 2005).

The implosion method of demolition was able to effectively aerosolize building materials, including lead containing painted surfaces, suggesting the need to remove these building components during pre-implosion preparatory work to limit the production and spread of lead containing dust (Stefani et al., 2005). Due to lead dust and other air quality issues resulting from building implosion, the study concludes that implosions in metropolitan areas should be prohibited (Stefani et al., 2005).

Numerous studies in the U.S. have reported increased lead dust fall and increased concentrations of lead in dust during the demolition of older, single family housing (Table 8 and 9). Additionally, one study found exposure to multiple housing demolitions to have significant effects on child blood lead levels (Rabito et al., 2007).

Lead dust is cited as a public health concern as it settles on surfaces, becoming a pathway to ambient lead exposure and interior residential lead exposure due to the tracking and blowing of dust indoors (Farfel et al., 2003). Settled dust may remain a concern for a long time period post demolition, with one study reporting lead loadings on streets, alleys, and sidewalks near demolition sites reduced by only 41–67% one month post demolition (Farfel et al., 2005).

Table 8. Dust fall rates during single family housing demolition in the U.S.

Activity	**Baseline µg/m2/h**	**During demolition µg/m2/h**	**Source**
Demolition without dust suppression		153	(Jacobs et al., 2013)
Demolition when buildings and debris were wetted		59	(Jacobs et al., 2013)
Demolition with dust suppression		3	(Jacobs et al., 2013)
Demolition	13	64	(Mucha et al., 2009)
Demolition	10	410	(Farfel et al., 2003)
Debris removal	10	61	(Farfel et al., 2003)

Table 9. Lead concentration in dust during single family housing demolition in the U.S.

Activity	**Baseline µg/m2/h**	**During demoliton µg/m2/h**	**Source**
Demolition	579	2,406	(Jacobs et al., 2013)
Demolition	950	2,600	(Farfel et al., 2003)

Debris removal	950	1,500	(Farfel et al., 2003)

Aside from discussion of lead, literature on the contribution of construction and demolition activities to changes in metal concentrations is limited (Brown et al., 2015). A few studies have recorded changes in the concentrations of some elements in dust at or near construction and demolition activities (Table 10).

Table 10. Heavy metals and other elements found in construction and demolition dust. (H=higher than background, L= lower than background)

Study Overview	Al	As	Ba	Ca	Cr	Cu	Fe	K	Ni	Mn	S	Sn	V	Zn	**Source**
Spatial and chemical patterns of PM10 in road dust deposited near construction and demolition activities	-	H	-		-	-	-	-	-	-	-	-	-	-	(Amato et al., 2009)
Metals in urban road dust attributed to construction and demolition activities	-	-	H	H	-	-	-	-	-	-	-	-	-	-	(Liu et al., 2014)
Risk assessment of exposure to particulate output of a demolition site	H	-	L	L	L	L	L	H	L	H	H	L	L	L	(Brown et al., 2015)
Heavy metals in dust fall from single-family housing demolition	-	H	-	-	-	H	H	-	-	H	-	-	-	-	(Jacobs et al., 2013)
Composition of heavy metals in the indoor environment of a building during renovation	-	-	-	-	-	-	-	-	-	-	-	-	-	H	(Latif et al., 2011)

Combat specific exposures to lead and heavy metals are discussed later in Section 7.3. However, it is relevant to note here that even just from the damage and destruction of buildings and built environment, any combat zone could have elevated concentrations to these toxicants. Table 11 summarises the health effects of a range of problematic elements commonly found in demolition dust.

Table 11. The health effects of elements found in demolition dust. Adapted from: (Brown et al., 2015)

Element	**Health concerns**
Aluminium (Al)	Neuropathological, neurophysical and neurochemical changes
Arsenic (As)	Carcinogenic
Barium (Ba)	Hypertension
Chromium (Cr)	Carcinogenic
Copper (Cu)	Free radical production
Iron (Fe)	Eye problems
Lead (Pb)	Affects brain and nervous system
Nickel (Ni)	Carcinogenic
Manganese (Mn)	Heart conditions
Tin (Sn)	Gastrointestinal conditions
Vanadium (V)	Affects nervous system
Zinc (Zn)	Pulmonary effects

5.7 TIMBER DUST

Existing literature does not discuss the impact of timber dust exposure during building demolition or destruction, although for timber structures this should also be considered.

Occupational exposures to timber dust discussed in association with health risks include; working in timber, plywood and wood chipping mills (Douwes, McLean, Slater, and Pearce, 2001; Douwes, McLean, van der Maarl, Heederik, and Pearce, 2000; Fransman et al., 2003; Liou, Cheng, Lai, and Yang, 1996; Mandryk, Alwis, and Hocking, 1999; Rosenberg et al., 2002) and joinery, furniture and carpentry work (Maciejewska et al., 1993; Mandryk et al., 1999). No literature has been found discussing the impact of timber exposure during building demolition.

The health effects of fresh wood dust are associated with the natural components of wood, such as resin acids or monoterpenes, as well as microorganisms such as moulds or bacteria (Demers, Teschke, and Kennedy, 1997; Douwes et al., 2000; Maciejewska et al., 1993; Rosenberg et al., 2002). Additionally, wood additives are associated with health effects, most notably formaldehyde and chromium (Fransman et al., 2003; Klein et al., 2001; Wolf et al., 1998). These health effects include respiratory symptoms, skin and eye irritations and adenocarcinoma of the sinonasal cavity.

Occupational exposure to both allergenic and non-allergenic wood dust, even at low concentrations, is associated with a number of respiratory symptoms, increasing in intensity with increased exposure (Carton, Goldberg, and Luce, 2002; Demers et al., 1997). The occurrence and severity of conditions vary with the type of wood, especially between hard and soft woods, climatic conditions, the use of fungicides and the manner in which the wood is handled (Enarson and Chan-Yeung, 1990). Symptoms associated with wood dust exposure include:

- extrinsic allergic alveolitis (Enarson and Chan-Yeung, 1990; Maciejewska et al., 1993)
- organic dust toxic syndrome (Enarson and Chan-Yeung, 1990; Maciejewska et al., 1993)
- occupational asthma (Demers et al., 1997; Douwes et al., 2001; Douwes et al., 2000; Enarson and Chan-Yeung, 1990; Fransman et al., 2003; Maciejewska et al., 1993)
- non-asthmatic chronic airflow obstruction (Demers et al., 1997; Enarson and Chan-Yeung, 1990; Shamssain, 1992)
- chronic bronchitis (Enarson and Chan-Yeung, 1990; Liou et al., 1996; Mandryk et al., 1999)
- upper and lower respiratory symptoms (Demers et al., 1997; Douwes et al., 2000)
- impairments of lung function (Carton et al., 2002; Goldsmith and Shy, 1988; Liou et al., 1996; Mandryk et al., 1999; Okwari, Antai, Owu, Peters, and Osim, 2005; Shamssain, 1992; Whitehead, 1982)
- symptoms of cough (Douwes et al., 2001; Mandryk et al., 1999; Okwari et al., 2005; Shamssain, 1992)
- chest pain (Okwari et al., 2005)
- rhinitis (Maciejewska et al., 1993)
- pneumonitis (Goldsmith and Shy, 1988)
- impaired mucociliary transport (Goldsmith and Shy, 1988; Whitehead, 1982)

Exposure to wood dusts is also associated with a number of non-respiratory irritations, mainly caused by hardwood dust and mainly due to Type IV allergy (Estlander, Jolanki, Alanko, and Kanerva, 2001). These include:

- allergic dermatitis (Estlander et al., 2001; Goldsmith and Shy, 1988; Maciejewska et al., 1993)
- conjunctivitis (Maciejewska et al., 1993)
- irritation of the eyes (Okwari et al., 2005; Rosenberg et al., 2002)
- irritation of the mucous membrane (Rosenberg et al., 2002)
- contact urticaria (Estlander et al., 2001)
- skin reactions (Rosenberg et al., 2002; Whitehead, 1982)

An increased risk of adenocarcinoma of the sinonasal cavity is associated with occupational exposure to wood dust, alone or chemically treated, constituting approximately half of the total number of cancers induced by wood dust (Carton et al., 2002; Demers et al., 1997; Goldsmith and Shy, 1988; Maciejewska et al., 1993; Nylander and Dement, 1993; Whitehead, 1982; Wolf et al., 1998). Additionally, an increased incidence of squamous cell cancers in association with wood dust exposure has been observed (Maciejewska et al., 1993). It is also assumed that exposure to wood dust can cause an increased incidence

of other cancers, especially lung cancer and Hodgkin's disease, however, evidence is less consistent (Maciejewska et al., 1993; Whitehead, 1982).

The strongest association of exposure to wood dust and development of adenocarcinoma of the sinonasal cavity is observed in exposures to hardwoods, most significantly, oak and beech (Demers et al., 1997; Maciejewska et al., 1993; Nylander and Dement, 1993; Wolf et al., 1998). However, the carcinogenic role of softwood dusts is not ruled out within the literature (Carton et al., 2002; Demers et al., 1997). The time between first occupational exposure to wood dust and the development of adenocarcinoma ranges from 7 to 70 years, averaging 40 years (Nylander and Dement, 1993).

Exposure to chromium and formaldehyde wood additives is associated with an increase in the number of squamous metaplasias (Wolf et al., 1998). Additionally, tumours in the respiratory tract are associated with chromate stain and oak wood dust and chromate is also theorised to play a significant role in the cause of tumours of the nasal cavity (Klein et al., 2001).

5.8 CONCLUSION ON PARTICULATE MATTER (PM) IN DAMAGED BUILT ENVIRONMENT

As this section shows, the process of demolition or destruction of buildings and built environments present a set of health risks. Generally this strongly relates to the elevated concentrations of particulate matter and health risks associated with such conditions. Because combat situations in urban environments are increasingly common, it appears likely that correspondingly deployed military personnel is exposed to increasingly to elevated particulate matter from the destruction of buildings and built environment.

6. VOLATILE ORGANIC COMPOUNDS (VOCS) AND OTHER CHEMICALS

In addition to particulate matter, volatile organic compounds (VOCs) and other chemicals might be present in the air in combat zones. VOCs are organic chemical compounds which have low boiling point, which means that they readily evaporate in normal indoor air conditions (EPA, 2016). Depending on boiling point and volatility, the World Health Organisation (WHO) categorises indoor organic pollutants as very volatile, volatile, and semi-volatile (Table 12). According to this definition, in normal indoor conditions, VOCs readily evaporate, VVOCs are gaseous, while SVOCs somewhat resist evaporation. The EPA acknowledges this classification as slightly arbitrary because all of these volatile compounds fall within the broad definition of the VOCs (EPA, 2016). Other non-organic chemicals could also be readily found in air.

Table 12: Classification of volatile organic pollutants. (Petrović, 2017).

Description	Abbreviation	Boiling Point Range (°C)	Example Compounds
Very volatile (gaseous) organic compounds	VVOC	<0 to 50-100	Propane, butane, methyl chloride
Volatile organic compounds	VOC	50-100 to 240-260	Formaldehyde, d-Limonene, toluene, acetone, ethanol (ethyl alcohol) 2-propanol (isopropyl alcohol), hexanal
Semi volatile organic compounds	SVOC	240-260 to 380-400	Pesticides (DDT, chlordane, plasticizers (phthalates), fire retardants (PCBs, PBB)

Although VOCs and generally indoor air quality has received more of recent research attention, many of the same chemicals are also commonly found outdoors, but then referred to as air pollution. VOCs, of all three classes shown in table 12, are commonly found in both urban areas and combat zones. Common sources of ambient VOCs outdoors are from the combustion of fossil fuels. Indoor sources include plastic products, composite timber products and furnishing materials. Elevated levels of VOCs in the environment have also been measured during or as a result of combat activities.

The VOCs and SVOCs and other compounds that are commonly found in air with established health effects are aldehydes (formaldehyde, acetaldehyde), aromatic hydrocarbons (benzene, toluene, ethylbenzene and xylenes), polycyclic aromatic hydrocarbons (benzo[a]pyrene, and benzo[a]pyrene equivalent measures), and polychlorinated compounds (polychlorinated biphenyls, polychlorinated dibenzo-p-dioxins and furans). Other airborne chemicals of concern in are sulphur and nitrogen oxides, ozone, and carbon oxides. Most of these are ‘primary pollutants’, which are pollutants emitted directly from identifiable sources. Others, such as ozone, nitric acid, sulphuric acid and carbonic acid are secondary pollutants, formed through chemical reactions in air between atmospheric constituents and primary pollutants.

6.1 VOC AND SIMILAR CHEMICAL EXPOSURES IN THE DEPLOYED ENVIRONMENT

Airstrikes by NATO on industrial targets in Serbia during the 1999 Kosovo Conflict resulted in significant releases of toxic chemicals to the environment, including high levels of polychlorinated biphenyls (PCBs) measured in soil and water (United Nations Environment Programme; United Nations Centre for Human Settlements, 1999).

The conflict in the Balkans throughout the 1990s led to destruction of buildings and infrastructure. In 2003, the United Nations Environment Programme (UNEP) produced a report which detailed the environmental consequences of the Kosovo conflict (United Nations Environment Programme; United

Nations Centre for Human Settlements, 1999). These consequences included widespread contamination of air, soil and water from damaged industrial facilities. Throughout 1999, NATO carried out air strikes on targets, which were described as 'tactical' or 'strategic' by a NATO spokesperson. Strategic targets included oil refineries and industrial facilities and factories, resulting in the leakages of multiple highly toxic chemicals to air and water. Leakages of 1-2,dichloroethane (EDC), and oil containing PCB were reported at least two instances. Burning of oil and petrochemicals such as the highly toxic vinyl chloride monomer (VCM) would have resulted in the release to air of dioxins, and noxious gases such as SO2, NO2, PAHs, lead and PM. Testing by the UNEP found very high levels of chemicals in soil and water, the result of both warfare and years of inadequate environmental protection within Yugoslavia. These included EDC, mercury, PCB, nickel, chromium, and VOCs (United Nations Environment Programme; United Nations Centre for Human Settlements, 1999).

In 2007, the UNEP released the report Iraq – Post-conflict Assessment, Clean-up and Reconstruction. The report detailed some of the environmental effects of the conflict, including environmental health hazards. Land contamination as a result of inadequate system of handling and disposal (i.e. hazardous waste in landfill) were reported for multiple industries prior to and as a result of the conflicts in the region. These industries included the minerals industry (e.g. sulphur, portash and phosphate), oil refineries and the arms industry. Additionally, the conflict left some secure materials and sites abandoned, which through subsequent looting or damage resulted in releases of substances to soil, air and water. Specific instances include the stockpiling of sulphur at a mining complex set alight during looting leading to regional human and environmental damage, a demolished site containing heavy metals and hazardous waste containing cyanide, looting of a warehouse complex which led to the spillage of pesticides including very toxic methyl mercury pesticides, looting of an oil refinery leading to fires and leakages (United Nations Envrironment Programme). The health risk of contaminated soil, uncontrolled disposal of hazardous waste in urban areas (i.e. dumped in the street) and releases of toxins to air pose a significant health concern to civilians and military personnel alike.

Chemical exposures in the Vietnam War have focused on the prevalence of exposure to Agent Orange, and other defoliants and pesticides. There have been numerous studies looking at disease incidence in Vietnamese, Korean Veteran and US Veteran populations who were exposed to defoliant, specifically Agent Orange during the war. Agent Orange was the most used of the many defoliants sprayed during the Vietnam War, and was a mixture of 2,4,5-trichlorophenoxyacetic acid and 2,4-dichlorophenoxyacetic acid, contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (dioxin) (Do, 2009). A study by the U.S. Institute of Medicine (IOM) on the health effects of exposure to herbicides during the Vietnam War found sufficient evidence for association with some soft tissue and blood cancers, and suggestive evidence of an association with respiratory cancers, prostate cancer and multiple myeloma (cancer of plasma cells (Institute of Medicine (U.S.). Committee to Review the Health Effects in Vietnam Veterans of Exposure to Herbicides, 1994).

Exposures during the Gulf War included oil well fires, smoke, petroleum products, pesticides, chemical warfare agents, biological warfare agents, immunisations, pyridostigmine bromide, infectious diseases, and depleted uranium. Despite an increase in symptoms in Gulf War veterans, a similar report from the

IOM was not able to find a causal link between the exposures above and symptoms reported, due to a lack of exposure data (Baird, 2016).

Table 13 shows that many organic compounds well recognised as problematic have been found in elevated levels in military camps. Likely chemical exposures during more recent conflicts in the Middle East, include vehicle exhaust, local pollution, emissions from open burning (Baird, 2016), and heavy metals in air (Engelbrecht et al., 2009).

Table 13: Summary of VOC exposures reported by other sources in military deployment or combat zones.

Measuring location or regulation		Sum VOCs (ng/m^3)	Formaldehyde (ng/m^3)	Acetaldehyde (ng/m^3)	BTEX (ng/m^3)	Benzene (ng/m^3)	Toluene (ng/m^3)	BAP (ng/m^3)	Source
WHO annual				50		0.17-17		0.012-1.3[3]	
EU standard						5		1	
Kabul	Outdoor	300	4.4	5.0	23	6	8.5	25 (geomean)	(Magnusson et al., 2012)
	Bedroom	390	13	7.6	26	6.0	9.5		
	Welfare room	500	13	6.5	25	5.7	9.3		
MeS	Outdoor	130	1.9	1.5	11	3.0	4.1	6.7 (geomean)	
	Bedroom	2300	8.1	5.6	38	0.25	8.5		
	Welfare room	300	7.3	2.6	12	2.8	5		
Camp Victoria, Kosovo	Summer	24 ± 2.6	2.3 ± 0.03	1.4 ± 0.32	3.0 ± 0.12			0.27 ± 0.04	(Wingfors et al., 2009)
	Winter	32 ± 5.0	4.7 ± 0.16	3.0 ± 0.084	16 ± 1.8			3.0 ± 1.4	
	Patrol /summer	50	7.6	5.2	8			4.1	
	Patrol /winter	91 ± 65	13 ± 7.0	6.0 ± 2.7	36 ± 12			8.8 ± 4.6	
	Workshop /summer	853	7.6	7.7	109			0.5	
	Workshop /winter	580 ± 550	12 ± 4.1	18 ± 15	209 ± 121			7.9 ± 4.3	

	Indoor /summer	913	46	22	20			0.09	
	Indoor /winter	210 ± 27	16 ± 0.65	22 ± 6.8	43 ± 15				

Some of the most concerning aromatic hydrocarbons for human health are benzene, toluene, ethylbenzene and xylenes. These are sometimes jointly called BTEX. Aromatic hydrocarbons can be monocyclic or polycyclic. Natural sources of BTEX compounds in the environment are crude oil, in the areas surrounding natural gas and petroleum deposits, and in gas emissions from volcanoes and forest fires.

Another very problematic class of organic compounds are polycyclic aromatic hydrocarbons (PAHs). The compounds often comprised of three or more fused benzene rings containing only carbon and hydrogen, and different configurations of the rings may result in different properties (Agency for Toxic Substances and Disease Registry, 2008). PAHs are solids with low volatility at room temperature (Agency for Toxic Substances and Disease Registry, 2008). Polycyclic aromatic hydrocarbons (PAHs) are produced by incomplete combustion and high-pressure processes (Agency for Toxic Substances and Disease Registry, 2008). Sources of PAHs are domestic fires, industrial combustion processes, vehicle exhausts.

The effects of PAHs are primarily in the pulmonary, gastrointestinal, renal and dermatologic systems and may also lead to cancer (Agency for Toxic Substances and Disease Registry, 2008). PAHs are often particle bound, which may play a part in the health effects observed (World Health Organization, 2013). Acute health effects are dependent on the mixture and concentration of PAHs. High concentrations of PAH mixtures in occupational settings have been shown to potentially cause eye irritation, nausea, vomiting, diarrhoea and confusion. Although, these symptoms may have been caused by other chemicals commonly found with PAHs (Abdel-Shafy and Mansour, 2016). Some mixtures of PAHs, and specific PAHs (anthracene, benzo[a]pyrene and naphthalene) have been shown to cause skin irritation and inflammation (Abdel-Shafy and Mansour, 2016). Chronic effects of exposure to PAHs are decreased immune function, cataracts, kidney and liver damage, breathing problems, asthma-like symptoms, and lung function abnormalities (Abdel-Shafy and Mansour, 2016). Some PAHs are potent carcinogens, through a genotoxic mode of action (World Health Organization, 2013; Agency for Toxic Substances and Disease Registry, 2008). Benzo[a]pyrene is a known human carcinogen (Group 1), whilst naphthalene, chrysene, benz[a]anthracene, benzo[k]fluoranthene and benzo[b]fluoranthene are probable human carcinogens (Group 2b) (Abdel-Shafy and Mansour, 2016). The most significant effect of inhalation exposure to PAHs is an increased risk of lung cancer (Abdel-Shafy and Mansour, 2016).
Sources of PAHs in the deployed environment include vehicle exhausts, emissions from burn pit and other fires, and local industrial combustion sources. PAH levels in military camps in Eastern Europe (Camp Victoria, Kosovo) and the Middle East (Afghanistan) were shown to have higher than recommended levels of the PAH benzo[a]pyrene (BaP) (Wingfors, et al., 2009; Magnusson, et al., 2012). The highest levels of BaP in Camp Victoria, Kosovo were measured in the workshop. (Wingfors, et al., 2009). In the Middle

Eastern study the highest levels were measured outdoors in Kabul and Mazar-e Sharif (MeS) in Afghanistan. The levels of BaP exceeded WHO and EU standards by 25 and 6.7 times respectively. The level of BaP measured in Kabul is similar to the levels found in other Asian megacities, and 100 times greater than the average levels measured in Western urban environments (Magnusson et al., 2012).

Ekstrand-Hammarström et al. (2013) compared the data from Kabul with samples from Umeå, in Sweden. Firstly, concentrations of PM10 were 5 times greater in Kabul than Umeå, and PM2.5 was 11 times greater than Umeå. Additionally, PAH and oxy-PAH levels in Kabul were 19 and 37 times greater respectively, in Kabul than Umeå. Based on other investigations which found higher concentrations of 'organic carbonaceous compounds' in ultrafine particles than coarse and fine particles, this indicates a higher proportion of ultrafine particles in Kabul than Umeå (Ekstrand-Hammarström et al., 2013).

Polychlorinated Biphenyls (PCBs) are a group of synthetic chemicals that are comprised of a mixture carbon, hydrogen and chlorine. PCBs do not have a known taste or smell (United States Environmental Protection Agency, 2018). PCBs are non-flammable, chemically stable, have a high boiling point and have electrical insulating properties (United States Environmental Protection Agency, 2018), which led them to be widely used as coolants and lubricants in transformers, capacitors and other electrical appliances and equipment (Agency for Toxic Substance and Disease Registry, 2014). PCB production and use in manufacturing was common in the early 20th century, but declined in the latter half of the century due to increasing evidence of negative health and environmental effects (Agency for Toxic Substance and Disease Registry, 2014).

PCBs have entered the environment through accidental releases (spills and leaks), and through their manufacture, use and disposal (Agency for Toxic Substance and Disease Registry, 2014). They persist in the environment, and can travel long distances (Agency for Toxic Substance and Disease Registry, 2014) by cycling through air, water and soil (United States Environmental Protection Agency, 2018). Additionally, PCBs accumulate in animals through the consumption of small aquatic organisms which take up the chemical (Agency for Toxic Substance and Disease Registry, 2014). In this way, PCBs progress through animal and human food chains.

The conflicts in Bosnia-Herzegovina and Kosovo, Serbia were both characterised by concerns about exposures to harmful chemicals, including petrochemicals (Teichman, 2012; Kirkpatrick, 2011). Leakage of PCBs and other chemicals to the natural environment present an additional concern. Some newer research suggests that certain chemical exposures can lead to multigenerational adverse health impacts, at times the impact increased in subsequent generations (Manikkam et al., 2013; Martinez-Arguelles et al., 2013).

6.2 SULPHUR (SO_2) AND NITROGEN OXIDES (NO_2)

In addition to air pollution discussion so far, combustion commonly releases high levels of sulphur dioxide (SO_2) and nitrogen dioxide (NO_2). The main health effects are on the respiratory system (WHO, 2018).

Sulphur dioxide is colourless gas in air, with a pungent odour. Anthropogenic sources of SO_2 in ambient air are industrial and domestic coal and oil burning, vehicle emissions, the smelting of mineral ores containing sulphur, paper manufacturing and petroleum refining (WHO 2005; WHO 2018; Agency for Toxic Substances and Disease Registry, 2014). Exposure to SO_2 is common in urban areas and around areas with heavy industrial activity. It can be absorbed through inhalation or dermal exposure (Agency for Toxic Substances and Disease Registry, 2014).

Acute exposure to low levels of SO_2 can cause irritation of the upper respiratory tract and eyes (Agency for Toxic Substances and Disease Registry, 2014). Acute exposure to very high levels of SO_2 causes respiratory effects such as bronchitis and bronchopneumonia, and may also be fatal (Agency for Toxic Substances and Disease Registry, 2014).

Chronic exposure to SO_2 is linked with effects on the respiratory system and altered lung function (Agency for Toxic Substances and Disease Registry, 2014). Additionally, irritation of the respiratory tract and subsequent inflammation may aggravate existing respiratory illnesses, such as asthma and bronchitis, and increase the risk of respiratory tract infections (WHO, 2018). The WHO daily guideline for SO_2 is 20 $\mu g/m^3$ (WHO, 2005)

Once in the air SO_2 may react with other constituents in the air to form other chemicals such as sulphuric acid (the main component of acid rain), sulphur trioxide and sulphates (Agency for Toxic Substances and Disease Registry, 2014).

The main sources of NO_2 in air are combustion processes such as vehicle emissions, power generation plants, and off-road equipment (United States Environmental Protection Agency, 2018). NO_2 is a common component of ambient air in urban and industrial areas, and around any combustion activities (WHO, 2018).NO_2 is corrosive and upon contact with water forms nitric acids and nitrous acids (Agency for Toxic Substances and Disease Registry, 2014), and in reaction with oxygen and water in the atmosphere may cause acid rain to form (United States Environmental Protection Agency, 2018).

NO_2 is used as the indicator for a larger group of nitrogen oxides, including nitric oxide (NO), nitrogen dioxide (NO_2), nitrous acid (HNO_2) and nitric acid (HNO_3) (Environmental Protection Agency, 2018; Agency for Toxic Substances and Disease Registry, 2014).

Both acute and chronic exposures to nitrogen oxides have respiratory effects. For acute exposures nitrogen dioxide is more toxic than nitric oxide – except for concentrations that are potentially lethal, where nitric oxide will kill 'more rapidly' (Agency for Toxic Substances and Disease Registry, 2014). Most of the higher nitrogen oxides are eye, skin and respiratory tract irritants, and may result in effects on the lung and respiratory system (Agency for Toxic Substances and Disease Registry, 2014). Exposure to nitrogen oxides are linked to an increased risk of pulmonary edema, pneumonitis, bronchitis, bronchiolitis, emphysema, coughs, hyperpnea (increased depth and rate of breathing), dyspnea (difficulty breathing)(Agency for Toxic Substances and Disease Registry, 2014), the aggravation of existing respiratory diseases such as asthma and COPD, and increased hospital admissions and emergency room

visits (United States Environmental Protection Agency, 2018; Agency for Toxic Substances and Disease Registry, 2014).

Nitrogen oxides can also cause cardiovascular, hematologic, dermal and ocular effects. Absorption of nitrogen oxides can cause a weak rapid pulse, dilated heart, chest congestion, and circulatory collapse. Exposure to nitric oxide can cause methemoglobinemia and impaired oxygen transport in blood. Exposure to high levels of NO_2 in air can cause skin and eye burns Agency for Toxic Substances and Disease Registry, 2014). Nitrogen oxides are not classified as having carcinogenic effects (Agency for Toxic Substances and Disease Registry, 2014).

The presence of SO_2 and NO_2 in air in the deployed environment is highly likely due to combustion process. Common exposure sources across multiple conflicts and locations are (military and other) vehicle emissions, burning of military targets and burning of military trash. This is supported by literature detailing exposures in the Middle East (Baird, 2016; Falvo et al., 2015; Teichman, 2012; Kirkpatrick, 2011), and the Balkans (Teichman, 2012; Kirkpatrick, 2011; United Nations Environment Programme, 1999). The Gulf War resulted in significant exposure to oil well fires and burn pit emissions (Baird, 2016; Teichman, 2012). Reports from the Balkans detailed exposure to burning petrochemicals (Teichman, 2012; United Nations Environment Programme, 1999) and burn pit emissions (Teichman, 2012). Exposures during 21st century conflicts in Iraq and Afghanistan are local pollution sources, diesel vehicle exhaust, burn pit emissions, (Baird, 2016).

In the U.S., improved monitoring techniques and technology were developed following the conflicts of the early 1990s (Kirkpatrick, 2011). Table 14 shows levels of ambient SO_2 and NO_2 as measured in Swedish military camps in Kosovo and Afghanistan (Wingfors, et al., 2009; Magnusson, et al., 2012). The data from Kosovo shows levels of SO_2 and NO_2 within WHO guideline values. The highest levels of NO_2 were measured in the workshop (Wingfors, et al., 2009). Data from sampling stations within Swedish military sites in Afghanistan show much higher levels of pollutants (Magnusson, et al., 2012). In Kabul, the highest levels of both pollutants were measured outdoors, indicating a high levels of background combustion. This is in keeping with the high levels of PAHs measured outdoors in Table 14. Additionally, the averages from the outdoor and indoor sampling stations in Kabul were all higher than WHO guideline values. In MeS, the highest levels of SO_2 and NO_2 were measured in the workshop due to the 'daily handling of fuels, grinding, welding, and exposure to engine exhaust'. The lower levels of pollutants in MeS, measured in outdoors and in a tent, were in keeping with the military camp in Kosovo (Magnusson, et al., 2012). This data demonstrates the contribution of specific activities (i.e. workshop activities) and local pollution to levels of SO_2 and NO_2 in the deployed environment.

Table 14: Summary of sulphur dioxide (SO_2) and nitrogen dioxide (NO_2) exposures reported by other sources in military deployment or combat zones.

		NO_2 µg/m^3	SO_2 µg/m^3	
WHO annual		40		
WHO 24hr			20	
Kabul	Outdoor	66	13	(Magnusson et al., 2012)
	Bedroom	69	4.5	
	Welfare room	58	4.8	
MeS	Outdoor	15	5.6	
	Tent	18	1.3	
	Workshop	32	20	
Camp Victoria, Kosovo	Base camp summer	9.2 ± 1.2	<0.3	(Wingfors et al., 2009)
	Base camp winter	7.8 ± 1.3	1.3 ± 0.32	
	Patrol /summer	7.9	<1.3	
	Patrol /winter	14 ± 8.1	0.73 ± 0.058	
	Workshop /summer	42	<1.3	
	Workshop /winter	21 ± 4.7	1.0 ± 0.15	
	Indoor /summer	16	<1.3	
	Indoor /winter	9.7 ± 3.0	0.90 ± 0.53	

7. HEAVY METALS AND RADITATION IN COMBAT ZONES

The primary purpose of this report is to investigate how much is known about possible but not well recognised environmental exposures in combat zones (Teichman, 2012; Martin et al., 2011; Di Lella et al., 2006). The most recognised problem area is the toxicity of ammunition itself, and therefore for the purpose of this review, the toxicity of ammunition is least unknown and consequently only briefly reviewed. Although ammunition is regulated by the relevant bodies, toxicity of ammunition leads to an increase of contamination of the environments in which the ammunition is deployed.

Although some metals, such as iron, are important for healthy functioning of human body, many, especially heavier metals have been shown to have toxic impact. However, higher atomic weight gives these materials excellent properties for use in ammunition, and therefore combat zones can have elevated heavy metal prevalence (Arnemo et al., 2016). Similarly, other harmful substances might be found in other parts of ammunition, such as depleted uranium. As discussed in section 5.6, combat zones could already have elevated heavy metals from the damage and destruction of built environment, and other local releases of contaminations can contribute to increased prevalence of heavy metals and radiation in combat zones (Zhivin et al., 2014).

In recent years, some reviews have started to establish higher potential of cancer incidences in the military personnel deployed in peacekeeping missions (Bogers et al., 2013; Strand et al., 2014; Peragallo at al., 2010). Other related reviews have started to demonstrate that the whole areas might be contaminated after modern military conflicts (Ghobarah et al., 2003). One study found that even prenatal exposure to war environment correlated to increase of lead content in children's teeth (Savabeisfahani et al., 2016). These examples of long term impact of modern warfare on the total toxicity of a particular area should receive more research attention.

7.1 HEAVY METAL CADMIUM (Cd) – SOURCES AND HEALTH EFFECTS

One of the most problematic heavy metals is cadmium. Cadmium (Cd) is associated with zinc, lead and copper ores and is found in the earth's crust. Most cadmium used in the U.S. is extracted a byproduct during the production of other metals such as zinc, lead or copper. Cadmium is also recovered from used batteries (Agency for Toxic Substances and Disease Registry, 2015). Negative health effects occur from both acute and chronic exposure to cadmium (National Center for Biotechnology Information, 2018).

Cadmium occurs in the environment (soil, water and air) as a byproduct of metal (iron, nickel, zinc and copper) mining and smelters, and its use in the manufacture of various products including nickel cadmium batteries, metal plating, electrical conductors, pigments, coatings and plastic stabilisers (National Center for Biotechnology Information, 2018). In the US the majority (approx. 80%) is used in batteries (Agency for Toxic Substances and Disease Registry, 2015). The main sources of cadmium in the air are through the burning of fossil fuels (e.g. oil and coal), waste incineration (National Center for Biotechnology Information, 2018). Cadmium is able to accumulate in the environment (e.g. in aquatic organisms and agricultural crops). In air, cadmium is a particle or vapour (if from high temp processes). It can travel long distances in the air and be deposited onto land or water. Cadmium and its compounds may travel through soil, however it generally binds strongly to organic matter which is more immobile. This enables plants to take up cadmium and there it may enter the food supply (Agency for Toxic Substances and Disease Registry, 2015). It may also enter the food supply through the use of contaminated water on agricultural crops (e.g. rice paddies) (National Center for Biotechnology Information, 2018). Smoking also exposes people to higher levels of cadmium, as tobacco leaves accumulate high levels of cadmium from the soil (Agency for Toxic Substances and Disease Registry, 2015).

It can be absorbed into the body by 'inhalation of its aerosol' and incidental ingestion (National Center for Biotechnology Information, 2018) (Agency for Toxic Substances and Disease Registry, 2015). The most common route of exposure is through inhalation of dust and fumes (Agency for Toxic Substances and Disease Registry, 2015). This happens in occupational settings, where industrial workers are exposed to cadmium through electroplating and smelting, among other sources (Agency for Toxic Substances and Disease Registry, 2015). This is known as 'cadmium fume fever' (National Center for Biotechnology Information, 2018).

The majority of cadmium that 'enters your body' goes to the kidney and liver, where it can remain for years. The body can excrete some cadmium in urine and feces, and most of it can be changed to a form which is not harmful to the body. Excess levels of cadmium, however, can overload the ability of the liver and kidney to change its form (Agency for Toxic Substances and Disease Registry, 2015).
Acute inhalation exposure to cadmium can result in a cough and sore throat. Exposure to high levels of cadmium may cause effects on the lung (e.g. bronchial and pulmonary irritation), and 'a single acute exposure' to very high levels of cadmium can also result in 'long-lasting impairment of lung function' (National Center for Biotechnology Information, 2018), and may cause death (Agency for Toxic Substances and Disease Registry, 2015). Acute exposure to cadmium can also cause eye redness and irritation/pain. Acute ingestion symptoms are abdominal pain, diarrhoea, headache, nausea and vomiting.

Chronic inhalation or oral/ ingestion exposure in humans can result in a build-up of cadmium in the kidneys, which in turn can lead to kidney disease (Agency for Toxic Substances and Disease Registry, 2015). The range of effects of the kidney include 'proteinuria, a decrease in glomerular filtration rate, and an increased frequency of kidney stone formation' (National Center for Biotechnology Information, 2018). Chronic exposure to low levels of cadmium may also cause bones to become fragile (Agency for Toxic Substances and Disease Registry, 2015). Chronic inhalation exposure to lead in the air also leads to effects on the lungs, including bronchiolitis, emphysema and pulmonary fibrosis (National Center for Biotechnology Information, 2018). It has also been linked to an increased risk of cancer and is defined as a 'probably human carcinogen' by the US EPA and is classified as 'carcinogenic to humans' by the IARC. The cancer sites are prostatic and lung cancer (National Center for Biotechnology Information, 2018).

7.2 HEAVY METAL LEAD (Pb) – SOURCES AND HEALTH EFFECTS

Lead (Pb) has been used by human society for at least 4-5,000 years (Brown and Margolis, 2012). Lead is a naturally occurring metal which, historically, has been used in architecture for its pliability in forming the glazing beads (cames) in lead windows, lead roofs, flashings and plumbing. During the 20th century it was mainly used in architecture as a stabiliser for lead-based paints.

As early as 370 BC, Hippocrates made some of the earliest recorded observations of health issues related to lead and other heavy metals (Philp, 2001, ch.6). Some sources have proposed that lead poisoning influenced the fall of the Roman Empire (Hayes, 2012). Better understanding of the risks eventually led to the passing of the first Food and Drugs Act by the British Parliament in 1875, but despite this cast lead solders and other lead toys were fairly common until the late 1940s and early 1950s (Philp, 2001, ch.6). Therefore, it is possible to observe that lead was already in the first stage of recognition of health risks before the start of the 20th century, but that the real action against it, indicative of transition to the second stage, did not start until mid 20th century.

Lead can negatively affect almost every organ, system and process in the human body, including the cardiovascular, gastrointestinal, hemolymphatic, urinary, immune, nervous, and reproductive systems, and can cause tumours in laboratory animals (Carlisle et al., 2009). Of all the systems listed, the main target for lead toxicity is the central nervous system (Sanders et al., 2009). For painters with high lead exposure peripheral neuropathy and cognitive impairment have been recorded, starting with problems with the upper limbs (Krishnan et al., 2012). However, lower levels of on-going exposure can lead to inhibition of several enzymes involved in heme synthesis, influencing functions of the peripheral and central nervous system and increasing blood pressure (Jakubowski, 2011).

Young and unborn children are more at risk from lead than adults because their brain and nervous systems are developing, and because their bodies absorb much higher proportions of ingested lead than adult bodies (Sanders et al., 2009). The blood-brain-barrier recognises one common form of lead in the body as calcium thus allowing its entry into the brain, where lead can take over the functional role of calcium, altering neurochemistry and behaviour, especially in the immature brain (Sanders et al., 2009). Acute lead poisoning in children starts with vertigo and irritability, progressing to delirium, vomiting and convulsions (Philp, 2001, ch.6). Studies have shown that lead exposure in children persists into adulthood, and has been associated with brain damage, mental retardation, behavioural problems, developmental delays, violence, and death at high levels of exposure (Sanders et al., 2009).

The half-life for lead in blood is generally 30-35 days, while in bones it is 25 years; bones, hair and teeth are where most of the free blood lead deposits (Philp, 2001, ch.6; Carlisle et al., 2009). Lead in the bones may be remobilised back into circulation at times of stress and tension, such as pregnancy, illness, traumatic life events, and aging (Zahran et al., 2009; Machida et al., 2009). Although the human body can eliminate lead, most of it stays in the body for a long time contributing to the total body load.

Lead can come in different forms, as elemental lead, in organic lead compounds, and inorganic lead compounds. It can also change forms, for example from organic to inorganic (American Cancer Society, 2014). In buildings the most likely form of lead is in inorganic lead compounds. Although, carcinogenicity of substances differs from their toxicity, lead is recognised as a risk in both areas, and all forms of lead are toxic. However, the International Agency for Research on Cancer (IARC) recognises different forms of lead as varying in their carcinogenicity: they classify inorganic lead compounds in Group 2A (probably carcinogenic to humans), elemental lead in Group 2B (possibly carcinogenic to humans), while organic lead compounds are classified as Group 3 (not classified for carcinogenicity to humans) (IARC, 2016).

While the negative impact of lead on memory, learning and IQ has often been studied, there is also evidence that lead influences other behaviours such as mood (depression), anxiety, schizophrenia and violence/aggression (Sanders et al., 2009). It has been noted that lead-exposed four to five year old children exhibit an increase in aggression, and from there lead exposure has been associated with juvenile delinquency and criminal behaviour (Sanders et al., 2009, Narag, Pizarro and Gibbs, 2009). Between 1979 and 1984, a Cincinnati-based study recruited pregnant women living in impoverished neighbourhoods with a high concentration of older, lead contaminated housing (Wright, 2009). It measured prenatal and early childhood exposure to lead, and subsequently compared that data with the local criminal justice records on arrests of the same children by 2005, then 19-24 years of age. Increased blood lead levels before birth and during early childhood were associated with higher rates of arrest than the control group. Also, average childhood blood lead was significantly associated with higher risk of arrest involving violent crime (Wright, 2009). In a similar New Zealand study, 1265 Christchurch children were studied from birth (in 1977) to age 21 (Fergusson, Boden and Horwood, 2008). Their dental lead levels at ages 6-9 were significantly associated with both officially recorded violence/property convictions and self-reported violence/property offending.

7.3 HEAVY METAL IN THE DEPLOYED ENVIRONMENT

When evaluating for impacts from heavy metals in deployed environments studies have found that commonly multiple metals are present and often in conjunction with other toxic substances. For example, in 2009, Englebrecht et al. found that high levels of particle-bound toxic metals were associated with certain ‘event days’. In Baghdad, the peak values for major soil-forming elements silicon, aluminium, calcium and manganese were associated with dust-blowing events (Engelbrecht, et al., 2009). However elevated levels of other metals, such as lead, zinc, arsenic and cadmium were not associated with any major regional dust storms but rather these metals were elevated on the same days, in association with other metals emitting events. The researchers suspected that the local lead battery and electronic-circuit-board smelters affected the sites they examined in Baghdad, Balad and Taji (Engelbrecht, et al., 2009). Additionally elevated levels of lead in Baghdad, Balad and Taji occurred on different days for the three sites, indicating that there are ‘local point sources’ with ‘fluctuating emission rates’ (Engelbrecht, et al., 2009). Depending on the analytical methods used the levels of aluminium, cadmium and lead were noted by the authors as possibly exceeding maximum guideline values from various agencies. The average/mean lead levels for all 15 sites were about three times exceed the WHO guideline of 0.5µm/m³. There is, however no level at which there is not a negative health effect from lead for humans.

The concentration of lead measured was almost the same for the three size fractions measured (TSP, PM10 and PM2.5), which indicated that the majority of all the lead occurs in the PM2.5 fraction (Engelbrecht, et al., 2009). The authors also noted that most combustion products (elemental carbon and particulate organic compounds) also occurred in PM2.5 and smaller fractions. Others have noted that fine lead and zinc, together with other associated metals, also are generated from condensed fumes emitted by metallurgical processes such as lead-zinc smelters or backyard electronic circuit board smelting operations (Carroll and Essik, 2008).

Research which evaluated for the impact of pollution of this kind on military camps found that many toxic elements were present in the studied military camps, as explained in the Table 15.

Table 15: Summary of elements found in military camps.

		Lead (Pb) ng/m³	Cadmium (Cd) ng/m³	Chromium (Cr) ng/m³	Nickel (Ni) ng/m³	Zinc (Zn) ng/m³	Arsenic (As) ng/m³	Mercury (Hg) ng/m³	Source
WHO annual		500	5	0.025-2.5	2.5-250		0.66-66'^		
EU		500	5		20		6		
Kabul	Outdoor (geomean)	34	0.63	7	14	63	<6		(Magnusson et al., 2012)
MeS	Outdoor (geomean)	33	1.43	13	15	400	<6		
	Workshop (geomean)	41	1.1	8.1	12	65	<6		
Camp Victoria, Kosovo	Summer	17 ± 6.8	0.19 ± 0.048	2.9 ± 1.3			<2.0		(Wingfors et al., 2009)
	Winter	14 ± 6.5	<0.83	62 ± 3.8			<11		
	Patrol /summer	8.3	<0.77	64			10		
	Patrol /winter	23 ± 10	<0.83	87 ± 15			<11		
	Workshop /summer	21	<0.82	91			11		

	Workshop /winter	19 ± 5.2	<0.83	79 ± 13			<11		
	Indoor /summer	<5.3	<0.79	66			<11		
	Indoor /winter	14 ± 2.4	<0.83	86 ± 7.7			<11		

8. CONCLUSION

This report shows that there is a series of possible problem exposures which can be part of deployment zones, presenting a risk for military personnel. Many of the most likely risks appear related to respiratory health and generally air pollution which tend to be elevated in military zones. Unfortunately careful studies of these trends can be difficult due to high level of prevalence of smoking which tends to be reported as common among the military personnel.

It is also possible to observe that the research in this area has greatly increased since the Vietnam War, but especially in the last twenty years. Nevertheless, the gaps in knowledge are substantial and the uniqueness of each different deployment location makes it difficult to generalize findings.

This review also shows that military personnel are not the only people at risk to exposures to environmental pollution from the modern combat zones. Local and future local populations are also exposed to elevated risks. This in itself suggests that potentially all combat zones should be considered as contaminated after any extensive conflicts. Considerations of this kind can be added to the long list of reasons against military conflicts.

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