Final Report of the

Expert Panel to Review SAS Veterans' Health Concerns

December 2003

ACKNOWLEDGMENTS

The Expert Panel wishes to acknowledge the efforts of the numerous SAS veterans, the various doctors who were involved with the treatment of SAS members and the Counter Terrorist and Special Recovery Support Group, ASASA, in providing detailed information in relation to the activities, exposures and health concerns of current and former members of the SAS. This information contributed significantly to the Expert Panel's understanding of the issues faced by SAS veterans.

The Expert Panel acknowledges the provision of information and assistance when requested from the following: Brig David Lewis (National Chairman ASASA), the SASR, SAS veterans, the Repatriation Commission, and representatives of the Department of Defence and Department of Veterans' Affairs, especially Dr Ian Gardner and Dr Keith Horsley.

The Expert Panel would also like to thank Major Terry O'Farrell, Acting Commanding Officer, SASR for kindly hosting a tour of the Swanbourne Barracks and the Bindoon training area, thus allowing the Panel a better appreciation of the conditions under which the SAS regiment operates.

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GLOSSARY AND ABBREVIATIONS

ADF	Australian Defence Force
AGE	Arterial Gas Embolism
ASASA	Australian Special Air Services Association
ATSDR	Agency for Toxic Substances and Disease Registry
Carcinogen	Any cancer producing substance. Often a distinction is made between epigenetic and genotoxic carcinogens. An epigenetic carcinogen is one which does not itself damage DNA but causes alterations such as hormonal derangements, immunosuppression, or chronic tissue injury that predisposes to cancer. A genotoxic carcinogen is one that reacts directly with DNA or with macromolecules that then react with DNA.
CDC	Centers for Disease Control
CS Agent, CS "gas"	CS is o-chlorobenzylidene malononitrile. It is used as a "Tear Gas".
CI	Confidence interval. The 95% CI is the range in which one can be 95% confident that the true value lies, after allowing for the role of chance.
DoD	Department of Defence
DVA	Department of Veterans' Affairs
DCI	Decompression Illness
Genotoxic	Damaging to DNA
IARC	International Agency for Research on Cancer
Mutagen	A chemical or physical agent that induces or increases genetic mutations by causing changes in DNA.
NIOSH	National Institute for Occupational Safety and Health (US)
OSHA	Occupational Safety and Health Administration (US)
PTSD	Post traumatic stress disorder
RMA	Repatriation Medical Authority
RR	Relative risk
SAS	Special Air Services
SOPs	Statements of Principles
Teratogen	Any agent or factor that induces or increases in the incidence of a congenital abnormality in a developing embryo

EXECUTIVE SUMMARY

In December 2002 the Hon. Danna Vale, Minister for Veterans' Affairs, established the Expert Panel to identify and document exposures of concern arising through Special Air Services (SAS) operational skills enhancement and training and to examine their possible adverse health effects. The Expert Panel was also asked to consider the need for further research and whether any particular matters should be drawn to the attention of the Repatriation Medical Authority (RMA).

The Expert Panel held its first meeting in February 2003 in Perth, where the Special Air Service Regiment (SASR) is based, and a further eight meetings were held in 2003. During the visit to Perth, Panel members were able to observe a number of aspects of the current SAS training environment. In writing its report, the Expert Panel had regard to submissions from former members of the SASR, discussions with current and former members of the SASR, the medical-scientific literature in relation to the exposures of concern and its own expertise.

The Panel was able to document a range of potential exposures related to activities undertaken by the SAS. However, on the basis of the information provided to it, the Panel was not able to quantify the past or current extent of exposure to these factors.

Nevertheless, the Panel was able to determine that the SAS training environment involved a range of exposures that can lead to potential adverse health effects. In particular, development of the counter-terrorist capacity of the SAS in the late 1970s and early 1980s involved the development of new skills and expertise, which brought exposure to risks associated with experimentation and intense periods of enhanced hazard. While veterans were clearly proud of their contribution to the military's counter-terrorist preparedness, some reported that they believed that this contribution had come at personal physical and mental cost. The Expert Panel notes that the SAS is an internationally recognised elite force, which reflects to a large extent the input of the early developers who often willingly put the development of protocols before their own health and safety. Some veterans feel that this has not been fully understood.

Most of the possible adverse health effects of the exposures of concern are covered by the Repatriation Medical Authority's existing Statements of Principles. Recommendations have been made for the RMA to consider the few conditions which are not currently covered. The Panel was asked to give consideration to a number of particular exposures.

Lead is known to cause a number of forms of toxicity. Exposures may have occurred as a result of intensive training in indoor firing ranges. On the basis of limited information reported to the Panel, no SAS veterans have actually been identified by testing as having a high blood lead level or reported to have lead-related toxicity since the new indoor firing ranges became operational.

CS Agent ("tear gas") is well recognised as highly irritant to skin, mucous membranes and lungs and there is some evidence that it can cause long term lung damage and chronic lung disease, but no evidence that it is carcinogenic in humans. SAS veterans were exposed to very high levels of CS on occasions, sometimes without adequate protective equipment.

Coloured smoke and masking agents are a diverse group of substances, some of which may have adverse health impacts, particularly irritant effects, severe acute pneumonitis and chronic lung disease. There is equivocal evidence that hexachloroethane smoke, some of the chemical constituents of hexachloroethane and certain coloured smokes may be carcinogenic. SAS personnel commonly used these agents in their training. Records of the type and level of exposure appear to be very limited.

Asbestos exposure can cause mesothelioma, lung cancer and asbestosis. It is possible that SAS veterans were exposed to asbestos in some of the environments in which they trained. There is no indication from veterans' reports that this exposure was substantial.

Physical trauma and prolonged heavy physical activity are a common cause of injury and disability among serving and retired SAS and other service personnel. The Counter Terrorist Special Recovery Support Group stated in its submission that, in the period 1979 to 1998, around 1% of personnel who served in the SASR were killed and 32% were injured. The incidence of physical injury is likely to have been underestimated because of disincentives felt by serving members of the SASR to report injuries. There is evidence that repetitive or prolonged heavy physical activity may cause musculoskeletal injury without overt acute trauma. Such injuries include back pain, compartment syndromes and tendonitis as well as an increased risk of osteoarthritis of the lower limb in those with anatomical abnormalities or significant previous injury.

The psychological impact of physical disability is well recognised, although many people are resilient in such circumstances. SAS veterans reported particular problems when physical disability lead to early discharge or where compensation was perceived to be inadequate. The Expert Panel supports the implementation of the ADF's injury prevention and control program, including the improved surveillance of the incidence of injury associated with different activities or units.

Blast and overpressure exposure can cause both immediate injuries and long term disability, commonly including soft tissue, orthopaedic, head, ear and ocular injuries. Exposure to blast and overpressure occurred frequently in counter terrorist training.

Stress is defined for the purpose of this report as the adverse psychological and physical consequences of exposure to circumstances and situations which present threat or challenge to the individual (stressors). Stress and stressors have been variably associated with a range of ill health effects both physical and psychological. The response of individuals to stressors is modified both positively and negatively by other factors such as psychological preparedness, camaraderie, social support and context. Military service is commonly associated with exposure to many stressors and service training in part aims to prepare personnel for such exposures. SAS training and service may be associated with a higher level of stressor exposure but also a higher level of preparedness and skills and some compensations such as higher income and prestige.

Diving is a potentially hazardous activity even in controlled circumstances with adverse effects ranging from mild and self-limiting to fatal. Long term injuries can also occur as a result of diving, including damage to the middle and inner ear and possibly also long term neurological effects. There were many reports of diving accidents. The conditions of some of the diving exercises undertaken by SAS veterans suggest that the probability of injury may have been increased.

The synergistic effect of the above exposures is difficult to determine because of the lack of relevant evidence. The Expert Panel considers that further review would be uninformative on decision making and has made no recommendations in relation to this Term of Reference.

Genetic alterations to human cells, as measured by chromosomal aberrations, occur spontaneously and potentially by exposure to various environmental chemical and physical agents. SAS veterans were concerned that the various chemicals to which they had been exposed might have produced changes in their DNA which may in turn have had long term consequences to their health and that of their offspring. Based on the review of the evidence of the genotoxicity of the exposures reported by the SAS and the likely level of exposure, it is highly unlikely that these exposures would produce adverse health effects.

The interpersonal relationship, behaviour and lifestyle alterations that may be associated with the above exposures are difficult to establish because the issues are complex and the literature is still at a relatively primitive stage. In addition, most of the published studies are not specific to high intensity units such as the SAS and there are particular aspects of SAS life that are both potentially positive and negative compared to regular service life.

This review has identified a number of important gaps in the scientific literature concerning the health effects of the exposures of concern, and also in documentation of exposures among members of the ADF. The Expert Panel considers that baseline health surveillance for service personnel, from the time of entry into service and at regular intervals, should form the basis of shorter and longer term health studies of both positive and negative outcomes. This should include documentation of the exposures of personnel in training and operational service, and should also cover the post-service period. In this way it will become possible to answer accurately veterans' anxieties about the health consequences of exposures.

SUMMARY OF RECOMMENDATIONS

In making its recommendations, the Expert Panel has been conscious of the need to ensure that members of the SASR receive every opportunity to train and prepare for action in a manner that is as realistic as possible. Although there are guidelines that soldiers generally work within, there is also a need for them to have the freedom to make decisions that vary from those guidelines if the situation calls for it.

Having had regard to the likely levels of exposure reported by SAS veterans and the sound medical-scientific evidence as to the adverse health effects of the exposures of concern, the Expert Panel has made the following recommendations:

1. In relation to past exposure to lead, testing is unnecessary for SAS veterans, except where indicated in the context of clinical investigations.

2. Occupational health authorities within the Australian Defence Force (ADF) should determine whether current practice for measuring airborne lead concentration in training facilities and blood lead levels in SAS members in training is satisfactory in terms of occupational standards [National Standard for the Control of Inorganic Lead at Work, National Occupational Health and Safety Commission, 1994- see Appendix B).

3. For the purposes of the relevant Statements of Principles, the RMA should consider whether "irritant" definitions and other listed definitions should include CS exposure.

4. Respiratory function monitoring is not considered necessary for all those exposed to coloured smokes and masking agents, except where clinically indicated.

5. The RMA and occupational health authorities within the ADF should monitor literature on the potential human carcinogenicity of hexachloroethane smoke and its combustion products and also 2-aminoanthroquinone, solvent yellow 33 and disperse blue 180 (chemical constituents of certain coloured smokes). Within the limitations of training and operational requirements, it would be prudent to minimise exposure to coloured smoke and masking agents.

6. The ADF should maintain a central registry of the type and composition of coloured smokes and masking agents in order to facilitate the future risk assessment of veterans.

7. Potential exposure to asbestos in SAS veterans should be taken into account where indicated in the context of clinical investigation.

8. Statements of Principles for common overuse injuries should be developed.

9. The RMA should continue to monitor the medical-scientific evidence on the health effects of stress and stressors and modify Statements of Principles when appropriate.

10. The RMA should develop Statements of Principles for certain diving related medical conditions not currently covered, including decompression illness, pulmonary barotrauma and dysbaric osteonecrosis and should consider diving and pressure effects in relevant Statements of Principles.

11. There is no indication for or benefit from testing all SAS veterans or their offspring for chromosomal aberrations.

12. In view of the possible concerns arising in the context of the previous genetic testing performed on some SAS veterans, those veterans and their families should be provided with the opportunity to receive genetic counselling and, if appropriate, chromosome studies at an accredited laboratory.

13. The programs to facilitate transition to civilian life currently being piloted by the ADF and DVA should be further evaluated and, if shown to be effective, disseminated as per usual practice.

14. A systematic, prospective program for health surveillance of positive and negative outcomes should be established for serving personnel and veterans and should include documentation of relevant exposures.

INTRODUCTION

BACKGROUND

In December 2002, the Hon. Danna Vale, Minister for Veterans' Affairs, commissioned an independent Expert Panel to investigate certain health concerns and associated issues raised by the Australian Special Air Services Association (ASASA). The impetus for this came from some former members of the Special Air Services Regiment (SASR) who felt that there was a lack of appreciation of the stressful and hazardous nature of SAS service, due in part to a lack of understanding of the unique features of such service, particularly in relation to counter terrorist and special recovery duties. The SAS veterans felt that these duties were often dangerous and stressful and they reported that they experienced high rates of injury because of the need to maintain readiness for operational service. The veterans were concerned about exposures to CS Agent, coloured smoke and masking agents, lead, asbestos, explosives, blast effects and loud noise. There was additionally a concern about the adverse effects of stress and the potential interpersonal impacts of these exposures.

The Expert Panel was asked to identify and document exposures of concern, to review the medical-scientific literature on the adverse effects of these matters of concern and to recommend any further research considered desirable as a result of the investigation.

BRIEF HISTORY OF THE SASR

The following brief history of the SASR was obtained from SAS veterans during the Expert Panel's visit to Perth in February 2003.

The SASR is based at Swanbourne Barracks in Perth, Western Australia. The SAS was formed in 1957 with 120 members. It achieved regiment status in 1964 and now comprises 664 personnel. A counter terrorist role was added in the late 1970s following the Hilton Hotel bombing in 1978. Members of the SASR have participated in campaigns in Borneo and Vietnam and recent operations have included the Persian Gulf, Iraq, Kuwait, East Timor and Afghanistan.

The SASR is a highly flexible, specialised force, consisting of experienced, quality individuals chosen for their physical fitness, intelligence, mental toughness and teamwork and leadership skills. The regiment is held at the highest levels of readiness and training is therefore conducted under conditions which are as realistic as possible. The SAS conducts operations across the continuum of conflict, including surveillance and reconnaissance, combat search and rescue, limited offensive tasks, amphibious operations, airborne operations, training of guerrilla forces and Counter Terrorism (CT).

The SASR forms part of Special Operations Command and is made up of three sabre squadrons, a training squadron, an administrative/support squadron and a signals squadron. The sabre squadrons are comprised of two contingency squadrons and a recovery squadron. The contingency squadrons deal with incidents outside of Australia and are made up of water troops, air operations troops, mobility troops, and signals troops. The recovery squadron deals with the counter terrorist role within

Australia and overseas. It is comprised of water troops, land troops, sniper troops and signals troops. The SASR uses the latest equipment and training devices and conducts approximately 30 exercises and more than 60 courses per year.

TERMS OF REFERENCE

Minister Vale approved the following terms of reference (ToR), subsequent to discussions between the Government and the SAS Association:

1. *Identify and document exposures of concern in relation to SAS operational skills enhancement and training of former members, particularly in relation to counter terrorist and special recovery duties, including:*

    1.1 Lead and heavy metals exposure
    1.2 CS Gas Exposure
    1.3 Smoke and masking agent exposure
    1.4 Asbestos exposure
    1.5 Physical trauma and prolonged heavy physical activity
    1.6 Blast and overpressure exposure
    1.7 Stressor exposure
    1.8 Pressure effects associated with diving; and consider
    1.9 The synergistic effects of the above exposures
    1.10 The potential for genetic alteration associated with the above exposures, and
    1.11 The interpersonal relationship, behaviour and lifestyle alteration that may be associated with above exposures.

2. *Examine and report on whether there is any sound medical scientific evidence of adverse effects of the above to former members of the SAS and if so, the strength of that evidence and the nature of those effects.*

3. *Prepare a brief to the Minister for Veterans' Affairs and recommend:*
    - *any further research considered desirable as a result of the investigation; and*
    - *any particular matters that should be drawn to the attention of the RMA for its consideration.*

MEMBERSHIP OF THE EXPERT PANEL

The Expert Panel was made up of the five members of the Repatriation Medical Authority (RMA) and two experts in the fields of toxicology and genetics. Professor Ken Donald was appointed as Chairman of the Panel. The membership of the Expert Panel was as follows:

Professor Ken Donald MBBS, PhD, FRCPA, MRCPath, FRACMA, FRACS, Head of the School of Medicine, University of Queensland and formerly the Professor of Social & Preventive Medicine, and Head of Department of Social & Preventive Medicine, University of Queensland.

Professor Beverley Raphael AM MBBS, MD, FRANZCP, FACP, FRCPsych, FASSA, Director, Centre for Mental Health, NSW.

Professor Andrew Wilson B.Med Sci, MBBS (Hons), PhD, FRACP, FAFPHM, Professor in Public Health and Deputy Director of the School of Population Health at the University of Queensland and formerly Director of Clinical Policy and Practice, Chief Health Officer and Deputy Director-General, Public Health, in NSW Health.

Professor John Kearsley MBBS, PhD, FRACR, FRACP, Director, Division of Cancer Services, Cancer Care Centre, St George Hospital, Sydney and (conjoint) Professor of Radiology Oncology University of New South Wales.

Professor John Kaldor PhD, Professor of Epidemiology and Deputy Director of the National Centre in HIV Epidemiology and Clinical Research, University of New South Wales.

Professor Gillian Turner OA (for services to medical genetics) MBChB, DSc, FRCPE, MRCPE, Professor of Medical Genetics, University of Newcastle and formerly director of Hunter Genetics.

Professor Bill Webster BSc, PhD, Head of the Department of Anatomy and Histology, University of Sydney. Professor Webster is a member of the Australian Drug Evaluation Committee, Complementary Medicines Evaluation Committee and the Vietnam Veterans' Mortality Study Scientific Advisory Committee and past President of the Australian Birth Defects Society.

WORKING PROCEDURES

The Expert Panel's terms of reference required a consideration of submissions from former members of the SASR, discussions with current and former members of the SASR and a consideration of the primary and review literature, and other relevant materials.

The Expert Panel has relied on a number of published reviews for material relating to the chemical toxicity and carcinogenicity of lead and heavy metals, asbestos, CS, and coloured smoke and masking agents and stress. Primary literature relating to exposure to these agents and other exposures of concern was sought using the Medline

database, the Toxnet database and the reference lists of published articles. Expert Panel members had knowledge in the fields of toxicology, carcinogenesis, cytogenetics, pathology, epidemiology and mental health and were able to use their expertise, as well as the published literature, to report on the terms of reference.

The Expert Panel held its first meeting on 19th to 21st February 2003 in Perth, Western Australia. On this visit, the Panel visited the SASR to observe first hand the training facilities and activities at Swanbourne Barracks and Bindoon training area. On this visit the Panel also listened to presentations from former members of the SASR and received written submissions. Subsequent to this visit the Expert Panel held a further eight meetings over the course of 2003.

DATA COLLECTION ON ADVERSE EFFECTS

The Expert Panel received and reviewed submissions from the Australian Special Air Service Association Counter Terrorist and Special Recovery Support Group, individual former members of the SASR and persons associated with the SASR. In addition to this the Expert Panel examined published primary and review literature on the exposures of concern, and considered the background and substance of the reviews and the primary sources of published literature utilised in their production, including the following:

- Medline searches using MESH headings and textword searches for the listed exposures, adverse effects and epidemiology, in the general population, Special Forces, veterans and the Military. Specific searches and author searchers were undertaken for individual factors where information may not have been definitive in the primary search.
- Toxnet searches for chemical exposures.
- Where necessary, referenced texts, other publications, and reference lists were also used to identify primary source material, and to ensure that examination of reported associations was undertaken.
- Search of websites and databases including: Agency for Toxic Substances and Disease Registry, National Institute for Occupational Safety and Health, The Occupational Health and Safety Administration, The National MSDS Repository, National Institute of Health, International Agency for Research on Cancer and NATO as well as general Internet searches.
- Liaison with and data collection from members of the Australian Defence Force, Western Diagnostic Laboratories, and the Special Air Service Regiment.

MAJOR REFERENCES AND REVIEWS

In addition to the primary literature, the Expert Panel had the benefit of a number of significant contemporary reviews of the literature relating to exposures of concern and these included:

Agency for Toxic Substances and Disease Registry (2001). US Department of Health and Human Services. *Toxicological Profile for Asbestos.*

Agency for Toxic Substances and Disease Registry (1999). US Department of Health and Human Services. *Toxicological Profile for Lead.*

Agency for Toxic Substances and Disease Registry (1997). US Department of Health and Human Services. *Toxicological Profile for Hexachloroethane.*

Agency for Toxic Substances and Disease Registry (1997). US Department of Health and Human Services. *Toxicological Profile for White Phosphorus.*

Agency for Toxic Substances and Disease Registry (1994). US Department of Health and Human Services. *Toxicological Profile for Zinc.*

National Toxicology Program. *US Department of Health and Human Services. 10th Report on Carcinogens.*

International Agency for Research on Cancer (2001). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 79: Some Thyrotropic Agents.

International Agency for Research on Cancer (1999). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 73: Some Chemicals That Cause Tumours of the Kidney or Urinary Bladder in Rodents and Some Other Substances.

International Agency for Research on Cancer (1999). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 71: Re-evaluation of Some Organic Chemicals, Hydrazine and Hydrogen Peroxide.

International Agency for Research on Cancer (1995). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 63: Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals.

International Agency for Research on Cancer (1990). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 48: Some Flame Retardants and Textile Chemicals, and Exposures in the Textile Manufacturing Industry.

International Agency for Research on Cancer (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42. Supplement 7.

International Agency for Research on Cancer (1982). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking Water and Dental Preparations.

Committees on Toxicity, Mutagenicity and Carcinogenicity of Chemicals in Food, Consumer Products, and the Environment (1999). Statement on 2-chlorobenzylidene malononitrile (CS) and CS spray. *London: Department of Health.*

OMEGA Foundation (2000). Crowd Control Technologies. Working Document for the STOA Panel. *Published by European Parliament.*

National Research Council (1997). Toxicity of Military Smokes and Obscurants. Volume 1. Subcommittee on Military Smokes and Obscurants. *National Academy Press: Washington, DC.*

National Research Council (1999). Toxicity of Military Smokes and Obscurants. Volume 2. Subcommittee on Military Smokes and Obscurants. *National Academy Press: Washington, DC.*

National Research Council (2000). Toxicity of Military Smokes and Obscurants. Volume 3. Subcommittee on Military Smokes and Obscurants. *National Academy Press: Washington, DC.*

Centers for Disease Control (1997)[Second Printing]. Musculoskeletal disorders and workplace factors: a critical review of epidemiologic evidence for work-related musculoskeletal disorders of the neck, upper extremity, and low back. *National Institute for Occupational Safety and Health, US Department of Health and Human Services.*

1. LEAD AND HEAVY METALS

SUMMARY AND RECOMMENDATIONS

Lead is known to cause a number of forms of toxicity. Exposures may have occurred as a result of intensive training in indoor firing ranges. On the basis of limited information provided to the Panel, no SAS veterans have actually been identified by testing as having a high blood lead level or reported to have lead-related toxicity since the new indoor firing ranges became operational.

The Expert Panel recommends that:

1. In relation to past exposure to lead, testing is unnecessary for SAS veterans, except where indicated in the context of clinical investigations.

2. Occupational health authorities within the Australian Defence Force (ADF) should determine whether current practice for measuring airborne lead concentration in training facilities and blood lead levels in SAS members in training is satisfactory in terms of occupational standards [National Standard for the Control of Inorganic Lead at Work, National Occupational Health and Safety Commission, 1994- see Appendix B).

SOURCES OF REPORTED SAS EXPOSURE

Assessment of potential exposure to lead among SAS veterans and members was made on the basis of the following sources of information:

(i) Submissions from individual former members of the SASR.

(ii) Report from the SASR about the structure of facilities in which lead exposure potentially occurred and procedures for monitoring and recording lead exposure.

(iii) Published reports on blood lead levels and toxicity in people exposed in circumstances similar to those that occur in the context of the SAS training facilities.

SAS EXPOSURE TO LEAD

(i) Former members of the SASR

Many of the individual submissions made by former members of the SASR mention exposure to lead during counter terrorist training. Such lead exposure was reported to result from the regular firing of ammunition in outdoor and indoor shooting ranges and from cleaning firing ranges at the end of a shooting exercise. The exposure was exacerbated by a lack of adequate ventilation in indoor firing ranges and a lack of facilities to allow decontamination. The latter appears to refer to the original training facilities in place during the 1980s. One former SAS member reported having been diagnosed by medical authorities with an elevated blood lead level after approximately one year of service. The elevated blood lead level appeared to have occurred while serving in the counter terrorist role in the 1980s. Other former members of the SAS regiment mentioned that during their service they had not had

blood lead levels measured or this occurred rarely. The Expert Panel was unable to identify any systematic centralised records of blood lead levels for this group.

A serving soldier in charge of training facilities for the SAS regiment mentioned that the original facilities used during the 1980s had been hastily constructed due to operational requirements and little knowledge was available at the time to help in the design of such facilities. Hence, these facilities had not been engineered to minimise risk from various environmental hazards including lead.

(ii) Current members of the SASR

Following a request from the Expert Panel, the SAS regiment provided information concerning lead exposure. The current indoor firing ranges, which became operational between 1989 and 1991, were designed with the lead issue in mind. The actual ventilation flow rates were measured as part of the acceptance process of the current facilities. The actual firing rates have been less than the maximum allowed for in the design process. There was no mention of any environmental monitoring of lead in the current facilities eg airborne concentration of lead in firing ranges.

Serving SAS members reportedly have their blood lead levels measured before and after counter-terrorist rotation (ie every third year). According to army correspondence to the Expert Panel, no elevated blood lead levels have as yet been detected through this testing regime. This appears to refer to the last 13 years when the new indoor firing ranges have been operational. The reference range for blood lead used by a pathology laboratory involved in testing army personnel is outlined at Appendix C. The laboratory could only identify tests on 33 army personnel and were unable to distinguish whether any of the results related to SAS members. The Expert Panel noted an apparent discrepancy between the reported testing policy and the numbers reported by the laboratory, which it was unable to resolve within the timeframe of the Report.

No information was received to suggest that there had been exposure during SAS regiment training to any other heavy metals.

FIRING RANGES

Studies have demonstrated that the airborne lead concentration may exceed recommended safety levels in firing ranges. This is particularly so in indoor ranges with no or poor ventilation (Abudhaise et al 1996, ATSDR 1999, Tumpowsky et al 2000).

In one study, significantly higher blood lead levels were measured in military firing range trainees and instructors compared to controls (Abudhaise et al 1996). Elevated blood lead levels and levels above the recommended safety level (40-50 ug/dL) have been demonstrated in military and civilian users of firing ranges from case reports (White and Narula 1996, Shannon 1999, ATSDR 1999) or government occupational surveillance data (Tumpowsky et al 2000).

In some of these reports, users of firing ranges with abnormally high blood lead levels had clinical lead poisoning (Fischbein et al 1979, Landrigan et al 1975, Novotny et al 1987, all cited in Abudhaise et al 1996; White and Narula 1996).

The major routes of lead exposure are inhalation and ingestion of lead bearing dusts and fumes (ATSDR 1999).

ADVERSE HEALTH EFFECTS FROM LEAD

The toxicology of lead is well established and many reviews are available. The Agency for Toxic Substances and Disease Registry is an agency of the United States Health Department that produces peer-reviewed profiles on hazardous substances. It produced a thorough review of the toxicological profile for lead (1999) and this review was considered definitive for the purposes of the Expert Panel. Using this profile as a basis, adverse health effects from lead exposure appear to be several including:

- Death from severe lead encephalopathy.
- Gastrointestinal; abdominal pain, constipation, nausea, vomiting, anorexia and weight loss are early symptoms of lead poisoning in occupationally exposed subjects or with acute exposures to high levels.
- Haematological; profound effects on heme synthesis, decreased haemoglobin levels in adults seen at blood lead levels of 50 ug/dL.
- Musculoskeletal; case reports of high occupational exposure to lead and occurrence of muscle weakness, cramps, joint pain, and bluish-tinged line in the gums.
- Renal; nephropathy in some studies of lead-exposed workers at blood lead levels of approximately 60 to > 100 ug/dL. Acute nephropathy was seen in lead-intoxicated children, with primarily oral exposure and sometimes in lead workers. Chronic nephropathy was reported mainly in lead workers, with primarily inhalational exposure. Lead induced nephropathy can be a cause of gout.
- Neurological; encephalopathy can occur at blood lead levels of 100-120 ug/dL. This can lead to death or in permanent cognitive impairment. Neurological effects at low blood lead levels in adults is still unresolved and has not been demonstrated in lead-exposed workers at blood lead levels below 40 ug/dL. Peripheral neuropathy has been seen at blood lead levels as low as 30 ug/dL.
- Reproductive; lowered sperm counts and increases in the number of abnormal sperm may be associated with blood lead concentration below 40 ug/dL.
- Carcinogenic in animals at extremely high doses [renal tumours in rats and mice] but evidence for carcinogenicity in humans considered inadequate.

These effects all occur during exposure, when lead is detectable in blood. Subsequent to exposure, lead may be detectable in bone but is no longer biologically available to cause tissue injury, as long as it remains in bone. If lead is mobilised from bone for any reason, eg in osteoporosis, it can then again cause elevated blood lead and further tissue injury.

There is no evidence of birth defects in humans resulting from paternal exposure to lead. An association between blood lead and hypertension is still controversial.

For a more detailed discussion of the scientific articles relating to lead, see Appendix A.

COMMENT

Based on anecdotal reports from former members of the SASR, studies on firing ranges, and information from those responsible for the design of SAS training facilities, some former members of the SAS regiment may have had exposure to high levels of lead in the period prior to the installation of the current indoor firing ranges (1989-91) with the potential for an elevated blood lead level at the time of exposure.

It should be noted that there are limitations in both blood and bone lead measurements when the exposure has been in the past. As the half-life of lead in human blood is 28 to 36 days, levels in blood reflect relatively recent exposure (ATSDR 1999). Lead in bone as measured by noninvasive X-ray fluorescence techniques is considered as a biomarker of cumulative exposure to lead. This is because lead accumulates in bone over the lifetime of the individual and most of the lead body burden resides in bone (ATSDR 1999). Hence an elevated bone lead level reflects elevated cumulative exposure over a lifetime (ATSDR 1999) and cannot directly implicate one particular source of lead exposure.

The Expert Panel notes that facilities and lead monitoring in serving SAS members have improved. However, concern is expressed that no monitoring of lead concentration in air in the current indoor firing ranges occurs. The US Occupational Safety and Health Administration (OSHA) specifies 30 µg/m3 of air as the action level for employee exposure to airborne concentrations of lead (OSHA 1995). Under the requirements for medical surveillance and biological monitoring, the blood lead level of employees exposed to lead above the action level for more than 30 days per year must be determined at least every 6 months. Hence, current procedures of blood lead measurements after 12 months counter terrorist training may not be sufficient.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles are relevant to lead (see Appendix K for details of factors and definitions).

Table 1 Statements of Principles concerning lead

STATEMENTS OF PRINCIPLES	INSTRUMENT NO.
Gout	11 & 12 of 2000 amended by 43 & 44 of 2003
Peripheral Neuropathy	79 & 80 of 2001 amended by 13 & 14 of 2003

2. CS "gas"

SUMMARY AND RECOMMENDATIONS

CS Agent ("tear gas") is well recognised as highly irritant to skin, mucous membranes and lungs and there is some evidence that it can cause long term lung damage and chronic lung disease, but no evidence that it is carcinogenic in humans. SAS veterans were exposed to very high levels of CS on occasions, sometimes without adequate protective equipment.

The Expert Panel recommends that:

3. For the purposes of the relevant Statements of Principles, the RMA should consider whether "irritant" definitions and other listed definitions should include CS exposure.

INTRODUCTION

CS is o-chlorobenzylidene malononitrile. It is named after the people who first prepared it in 1928 - Corson and Stoughton - and is the active ingredient in so called CS "gas". Despite its name, CS "gas" is not a true gas. It has been described as a pyrotechnically-generated smoke, an aerosol, or a microparticulate spray. CS can be manufactured in powder formulations [CS, CS1, CS2] or in solution with a solvent. Several solvents have been used, including acetone, methylene chloride, mineral oil, methyl isobutyl ketone (MIBK). Each solvent has its own toxicological profile, which may act additively or synergistically with that of CS.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of potential exposure to CS among SAS veterans and members was made on the basis of the following sources of information:

(i) Submissions from individual former members of the SASR.

(ii) Report from the SASR concerning the use of CS in the Australian Defence Force.

SAS EXPOSURE TO CS

The SAS submission reports numerous instances of exposure to CS both as deliberate training exposure and due to ill-fitting gas masks.

In response to a request from the Expert Panel, the SAS regiment supplied information concerning the use of CS in the military. A variety of CS delivery systems are used by the Australian Defence Force. The SAS regiment confirmed use of CS grenades - M25A2, M7A3, L13A1 and L11A1. They also used CS cartridges M651, M674 and 38-mm penetrating anti-riot and canister hand held pressurised L1A1.

The formulations of CS used are not clearly identified. A Manual of Ammunition used by the Australian Defence Force includes three formulations of CS: CS, CS1 and

CS2. CS1 is a mixture of 95% micro-pulverised crystalline CS blended with a silicon compound. CS2 is a mixture of CS and another silicon compound (Cab-O-Sil). Some CS munitions contain solvents but details about solvents could not be ascertained from the information supplied by the SAS regiment.

During July the Expert Panel was still receiving information from the SASR concerning potential solvents in CS formulations. In order to avoid the possibility of indefinite delays, a decision was made to finalise the report on information to hand as at 14th July. It was further decided that any further follow up of chemicals in CS formulations should be referred to Occupational Health and Safety in the Australian Defence Force for appropriate consideration. See Appendix F for a copy of the letter to the Minister for Veterans' Affairs.

ADVERSE HEALTH EFFECTS FROM CS

CS produces dramatic, acute irritant effects on the eyes, respiratory tract and skin but few long-term sequelae are reported. The severity and duration of these acute effects are strongly dependent on the dose received. There are some case reports of uncommon longer-term health effects involving the eyes, respiratory tract and skin, which are discussed in more detail below. No large-scale prospective cohort studies of individuals exposed to CS were identified. For a more detailed discussion of the scientific articles relating to CS, see Appendix D.

Effects on the Eyes

There is instantaneous conjunctivitis, and burning and pain that lasts for 2-5 minutes. Concomitant spasm and closure of the eyelids is also common. The conjunctivitis lasts for about 25-30 minutes. Erythema of the eyelids may persist for an hour. Profuse tearing lasts for 12-15 minutes. Visual intolerance of light was marked in some volunteers and remained for up to an hour. Occasionally volunteers complained of "tired eyes" lasting about 24 hours.

There have been case reports of individuals exposed to CS aerosol or spray that have experienced persistent eye irritation, conjunctivitis or corneal erosion / keratitis (i.e corneal inflammation), however, no definite long-term ocular sequelae have been reported. Animal studies with CS indicate little potential for long-term ocular damage.

Effects on the Respiratory Tract

CS canisters mostly release a coarse spray with some particles less than 100 μm. The smallest droplets 28 to 50 μm could reach large and medium-sized airways of the lung. These are the airways that are affected in bronchial asthma.

An early report described controlled exposure to CS aerosol (Punte et al 1963). Volunteers reported that the first respiratory symptom was "burning" beginning in the throat and progressing down the respiratory tract. As exposure continued, burning became more painful and there was a constriction sensation throughout the chest, which was reported as incapacitating by these volunteers. The breathing pattern of exposed volunteers involved involuntary gasping when the aerosol was inhaled, then breath holding or slow shallow breathing, followed by paroxysms of coughing. An irregular respiratory rhythm was noted for several minutes after CS exposure was ceased. There have been many similar descriptions in subsequent literature.

In usual circumstances, affected individuals recover in non-polluted air and there are no sequelae. No effect on respiratory function tests from exposure to CS aerosol was apparent in human volunteers even after several exposures, as reported in one study (Punte et al 1963, Beswick et al 1972). Another study of human volunteers found a small reduction in exercise ventilation volume during the time of exposure to CS aerosol (Cole et al 1975).

There have been a few case reports of respiratory complications but available studies have not found long term effects. However, there are limited numbers of long term studies.

There have been four reported cases of onset of the asthma-like disorder, reactive airways dysfunction syndrome, in previously healthy individuals following exposure to CS aerosol in an enclosed space.

As CS is an irritant, concern has been expressed about aggravation in individuals with bronchial asthma, chronic bronchitis, or chronic obstructive airways disease and there are a few case reports of this (McClean 1969, Anderson et al 1996, Breakell and Bodiwala 1998, UK Department of Health Report 1999, Ballantyne 1977, Hu et al 1989, Sidell 1997). No data regarding permanent aggravation of asthma or new onset asthma was found (apart from reactive airways dysfunction syndrome).

Effects on the Skin

Irritant Contact Dermatitis

When CS comes into contact with the skin it causes stinging which is greatly accentuated by moisture and in rare cases it can cause an irritant contact dermatitis. Major factors associated with CS dermatitis are heat and humidity. In addition, occlusion and friction contribute as the fine particles are held in areas of hat bands, collars and intertriginous areas (i.e. areas where skin surfaces are opposed such as the groin) (Weigand 1969). The irritant contact dermatitis has been described as bullous in nature in some civilians and industrial workers exposed to CS.

Allergic Contact Dermatitis

A study of human volunteers showed it was possible to develop an allergic sensitisation to a CS solution applied to the skin (Maibach and Marzulli 1971). A few case reports of allergic contact dermatitis confirm this finding.

Burns

Patch tests of CS mixed with sodium hypochlorite bleach and exposure to CS aerosol produced first to third degree chemical burns (severe reaction with erythema, vesicles, sloughing, induration) in human volunteers. This is confirmed in human experiments and case reports but except in rare situations always resolved.

Carcinogenicity

There are no long-term studies of the carcinogenicity of CS in humans. CS is classed as an alkylating agent of the substitution nucleophilic second order type. Unlike the first order type, it can react directly with nucleophilic sites i.e. does not need to dissolve first. Covalent binding of a chemical to DNA is a potential first step in the induction of a tumour by a genotoxic agent. Since CS is known to react with the SH-groups of proteins and amino groups such as lysine it was considered possible that it would react with DNA. However, in a DNA binding study no CS binding to rat DNA was detected (von Daniken et al 1981).

In vitro studies show that CS may damage chromosomes or interfere with chromosomal segregation when cells divide. These results would have more significance if similar effects were seen in *in vivo* experiments, that is, in live animals

exposed to CS, as there is some evidence that chromosomal damage may be linked with an increased risk of developing cancer. However, *in vivo* studies in mice, which have looked for chromosome damage after CS exposure, have been negative (Wild et al 1983, Grawe et al 1997).

There are two carcinogenicity studies in which rats and mice were exposed to inhaled CS for 6 hours a day, 5 days a week for 2 years (US National Toxicology Program Technical Report No. 377). There was no evidence of any increase in tumours in the exposed animals, in particular there was no increase in tumours in the tissues of contact in the mouth and respiratory system.

The Expert Panel considers that the available evidence does not support the hypotheses that CS is a carcinogen or is a cause of transgenerational birth defects in humans.

MIBK and Genotoxicity

The Expert Panel has been unable to confirm that members of the SASR have been exposed to a CS formulation that also contains the solvent MIBK. This compound has also been examined for genotoxicity. The results were all negative in a series of *in vitro* tests and in bone marrow micronucleus assay.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles are relevant to CS (see Appendix K for details of factors and definitions).

Table 2 Statements of Principles concerning CS

STATEMENTS OF PRINCIPLES	INSTRUMENT NO.
Acute Blepharitis	115 & 116 of 95
Asthma	85 & 86 of 01
Chronic Blepharitis	117 & 118 of 95
Chronic Bronchitis And Emphysema	73 & 74 of 97
Conjunctivitis	111 & 112 of 96
Contact Dermatitis	65 & 66 of 97
External Burns	37 & 38 of 94 amended by 195 & 196of 95

3. COLOURED SMOKE AND MASKING AGENTS

SUMMARY AND RECOMMENDATIONS

Coloured smoke and masking agents are a diverse group of substances, some of which may have adverse health impacts, particularly irritant effects, severe acute pneumonitis and chronic lung disease. There is equivocal evidence that hexachloroethane smoke, some of the chemical constituents of hexachloroethane and certain coloured smokes may be carcinogenic. SAS personnel commonly used these agents in their training. Records of the type and level of exposure appear to be very limited.

The Expert Panel recommends that:

4. Respiratory function monitoring is not considered necessary for all those exposed to coloured smokes and masking agents, except where clinically indicated.

5. The RMA and occupational health authorities within the Australian Defence Force should monitor literature on the potential human carcinogenicity of hexachloroethane smoke and its combustion products and also 2-aminoanthroquinone, solvent yellow 33 and disperse blue 180 (chemical constituents of certain coloured smokes). Within the limitations of training and operational requirements, it would be prudent to minimise exposure to coloured smoke and masking agents.

6. The ADF should maintain a central registry of the type and composition of coloured smokes and masking agents in order to facilitate the future risk assessment of veterans.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of potential exposure to coloured smoke and masking agents among SAS veterans and members was made on the basis of the following sources of information:

i) Submissions from individual former members of the SASR.

ii) Report from the SASR concerning the use of coloured smoke and masking agents in the Australian Defence Force.

SAS EXPOSURE TO COLOURED SMOKE AND MASKING AGENTS

Based on information provided by the SASR, a large number of smoke-producing devices have been used in a range of training and operational contexts. Furthermore, specific agents used have changed over time. The information supplied about the smoking agents is attached at Appendix G. In this report, the Expert Panel restricts attention to those smokes for which it was able to find information about specific chemical ingredients. These smokes were hexachloroethane smoke, red phosphorus smoke, white phosphorus smoke, coloured smokes in M18 grenades, coloured smokes in "L" series grenades and coloured smokes in number 83 hand grenades.

In the SAS submission from a former soldier of the SAS regiment, he refers to a coloured smoke that he believes to be carcinogenic and the number 83 smoke grenades and the possibility that they may cause respiratory problems. Former members of the SASR also reported exposure to smokes during their training.

During July the Expert Panel was still receiving information from the SASR concerning coloured smoke and masking agents. In order to avoid the possibility of indefinite delays, a decision was made to finalise the report on information to hand as at 14th July 2003. It was further decided that any further follow up of chemicals in coloured smoke and masking devices should be referred to Occupational Health and Safety in the Australian Defence Force for appropriate consideration. See Appendix F for a copy of the letter to the Minister for Veterans' Affairs.

COMMENT ON COLOURED SMOKE AND MASKING AGENTS

The Expert Panel notes the difficulty in obtaining information on chemical exposures. For instance, the Expert Panel was only able to obtain information on the composition and exposures to a limited number of the products listed in Appendix G.

For a more detailed discussion of the scientific articles relating to coloured smokes, see Appendix E.

HEXACHLOROETHANE SMOKE

The grenades produce smoke by burning a mixture of hexachloroethane (HCE) and zinc oxide (ZnO), so called hexachloroethane smoke. Acute toxicity of hexachloroethane smoke is thought to arise from the $ZnCl_2$ which comprises about two-thirds of the mass of the smoke (zinc chloride 62.5%, zinc oxide 9.6%, iron oxide 10.7%, aluminium oxide 5.4%, lead oxide 1%, chlorinated vapours 10.8%). While zinc chloride is a major component of hexachloroethane smoke, chlorinated organic compounds are minor components of the smoke eg hexachloroethane, hexachlorobenzene, tetrachloromethane (ie carbon tetrachloride) and tetrachloroethylene. The Agency for Toxic Substances and Disease Registry (1997) noted that about 5% or less of the reagents in a hexachloroethane containing smoke device is released to air as hexachloroethane in the smoke.

ADVERSE HEALTH EFFECTS FROM HEXACHLOROETHANE SMOKE

The safety of hexachloroethane smoke was assessed in volume 1 of the report entitled, Toxicity of Military Smokes and Obscurants (1997). It was noted there that soldiers are required to wear protective masks before exposure to any concentration of smoke produced by M8 white-smoke grenades, smoke pots containing hexachloroethane smoke or metallic powder obscurants or when using smoke during urban terrain training in enclosed spaces.

Effects on the Respiratory Tract

At low concentration (< 160 mg~minute/cubic metre), $ZnCl_2$ fumes have no apparent adverse effect. As the concentration increases there is irritation of the nose, throat and chest, then marked irritation leading to hospitalisation. Death can occur due to oedema of the lungs or acute respiratory distress syndrome (ARDS) leading to respiratory insufficiency. In exceedingly high concentrations, such as have resulted from the spontaneous ignition of smoke generators in a tunnel, death may occur rapidly from asphyxia due to laryngeal oedema and spasm of the glottis (Cullumbine 1957).

Some exposure to zinc chloride fumes is permitted in industrial settings. The current U.S. Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) for zinc chloride fume is 1 milligram per cubic meter of air as an 8-hour time-weighted average (TWA) concentration and 2 mg per cubic meter as a 15-minute TWA short-term exposure limit (STEL). A STEL is the maximum 15-minute concentration to which workers may be exposed during any 15-minute period of the working day. The OSHA limits are based on the risk of respiratory irritation associated with exposure to zinc chloride fume.

Pneumonitis / Acute Respiratory Distress Syndrome (ARDS)

Unprotected exposure to high concentration of zinc chloride fumes can lead to severe acute lung damage known as ARDS. There have been numerous case reports and case series of acute chemical pneumonitis and ARDS following exposure to zinc chloride smoke from smoke bombs. ARDS usually develops rapidly, usually within five days of exposure and is characterised by increased permeability of the alveolar

capillary membrane and the development of pulmonary oedema. Death has occurred within minutes of exposure to 32 days after exposure. Respiratory function can be impaired for months following exposure and survivors may develop pulmonary fibrosis and emphysema.

Pulmonary Fibrosis

Pulmonary fibrosis refers to scarring that has occurred in the interstitium (tissue between the airsacs) or the alveoli (ie airsacs) of the lungs. Pathological findings in fatal human cases of ARDS included extensive interstitial and intra-alveolar pulmonary fibrosis, diffuse microvascular obliteration and widespread occlusion of the pulmonary arteries (Milliken et al 1963; Hjortso et al 1988 / Homma et al 1992).

Emphysema, Pneumothorax and Pneumomediastinum

In more severe cases of acute exposure, emphysematous changes and pneumothorax have occurred (Evans 1945; Matarese and Matthews 1986; Pettila et al 2000). Emphysematous changes have persisted in some cases. Pneumomediastinum with subcutaneous emphysema has also been described in several case reports.

Subglottic Stenosis

Subglottic stenosis was described in one case report and the individual required tracheostomy and ongoing periodic dilatation (Lumsden and Weir 1945).

Asthma

Zinc chloride is associated with asthma in solderers and zinc fume is a "known" sensitiser. Fumes containing zinc chloride have been identified as causing occupational asthma (Weir et al., 1989) and the asthma-like condition, reactive airways dysfunction syndrome (Demeter and Cordasco 1990).

Acute Irritant Effects

Exposure to high concentration of zinc chloride smoke from smoke bombs has been reported to be associated with acute irritant:

- Gastrointestinal effects (nausea, vomiting, epigastric pain).
- Ocular effects (acute conjunctivitis, corneal ulceration).
- Cutaneous effects (chemical burns). Contact of the skin with zinc chloride dust can cause primary dermatitis (OSHA 2003).

The US Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH) has established a permissible or recommended exposure limit for hexachloroethane itself, of 10 mg per cubic meter of air as an eight hour time-weighted average concentration. Exposure to hexachloroethane can occur through inhalation, ingestion, eye or skin contact and absorption through the skin, eyes and mucous membranes. Acute exposure to hexachloroethane by itself is moderately irritant to eyes, skin and mucous membranes in humans (OSHA, NIOSH). Excessive blinking, visual intolerance to light, tearing and reddened eyes have been reported in workers exposed to the vapours of hot

hexachloroethane but no permanent damage to eyes was noted (OSHA). In animals, eye and respiratory tract irritation, liver and kidney damage and central nervous system toxicity have been noted (OSHA, NIOSH).

Carcinogenicity

The Expert Panel could not identify any human studies on the carcinogenicity of hexachloroethane smoke. Some *in vitro* and *in vivo* animal studies have suggested that hexachloroethane smoke may be carcinogenic (Clode et al 1991, Marrs et al 1988).

NIOSH considers that the chemical hexachloroethane, is a potential occupational carcinogen while the US National Toxicology Program (10th Report on Carcinogens) considers that hexachloroethane is "reasonably anticipated to be a human carcinogen" based on sufficient evidence of carcinogenicity in experimental animals. The International Agency for Research on Cancer (IARC) has concluded that there is sufficient evidence in experimental animals for the carcinogenicity of hexachloroethane. High dose oral dosing in rodents has been associated with renal and hepatic tumours but the significance of these findings for the human, where exposure is usually by inhalation, is unknown. IARC (1999) considers that there is inadequate evidence in humans for the carcinogenicity of hexachloroethane.

Some other minor constituents of hexachloroethane smoke [eg hexachlorobenzene, tetrachloromethane (ie carbon tetrachloride), tetrachloroethylene], have been listed by the US National Toxicology Program (10th Report on Carcinogens) as "reasonably anticipated to be a human carcinogen". IARC (2001, 1999) considers that hexachlorobenzene and tetrachloromethane (ie carbon tetrachloride) are both possibly carcinogenic to humans (Group 2B), based on inadequate evidence in humans and sufficient evidence in experimental animals for carcinogenicity. IARC (1995) considers that tetrachloroethylene is probably carcinogenic to humans (Group 2A), based on limited evidence in humans and sufficient evidence in experimental animals for carcinogenicity.

The significance of these findings for the carcinogenicity of hexachloroethane smoke in humans is unclear.

An *in vitro* study found a significantly increased frequency of chromosomal aberrations per cell when zinc chloride salt was added to human leukocyte cultures compared to controls (Santra et al 2000). IARC, the National Toxicology Program, the US Occupational Safety and Health Administration, and the American Conference of Governmental Industrial Hygienists have all concluded that the evidence is insufficient to implicate zinc chloride as a carcinogen.

The Expert Panel considers that the available evidence does not support the hypothesis that hexachloroethane smoke is a cause of transgenerational birth defects in humans.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles are relevant to hexachloroethane smoke (see Appendix K for details of factors and definitions).

Table 3 Statements of Principles concerning hexachloroethane smoke

STATEMENTS OF PRINCIPLES	INSTRUMENT NO.
Asthma	85 & 86 of 2001
Chronic Bronchitis And Emphysema	73 & 74 of 97
Conjunctivitis	111 & 112 of 96
External Burns	37&38 of 94 amended by 195 & 196 of 95

RED PHOSPHORUS SMOKE

Red phosphorus formulation in grenades, as used by the United States Army, mostly contains a mixture of red phosphorus and butyl rubber (95:5), and upon combustion produces an aerosol of phosphoric acids in a complex mixture of polymeric forms (Toxicity of Military Smokes and Obscurants, Volume 1, 1997). The predominant component of red phosphorus-butyl rubber (RP-BR) smoke is phosphoric acid. Trace amounts of phosphine have been measured in some cases. In mortar shells, red phosphorus is combined with sodium nitrate and an epoxy binder in a ratio of 80:14:6 parts by weight, respectively.

Information about the precise formulation used by the Australian Defence Force was not available but is likely to be similar.

ADVERSE HEALTH EFFECTS FROM RED PHOSPHORUS SMOKE

Effects on the Eyes

In human volunteers, exposure to red phosphorus smoke at concentrations of 100-700 mg per cubic metre for 2 to 15 minutes, was associated with significant, but reversible, symptoms of eye irritation (cited in Ballantyne 1998).

Effects on the Respiratory Tract

Respiratory tract irritation and inflammation have been reported in humans and in animal studies following short-term exposure to RP-BR smoke (Toxicity of Military Smokes and Obscurants, Volume 1, 1997). The irritant effect was considered to be due to the high phosphoric acid content of RP-BR smokes. It was estimated that human exposure to RP-BR smoke at concentrations of about 2,000 mg/cubic metre for longer than 15 minutes might result in death due to respiratory tract injury and that masks must be worn when the concentration exceeds 700 mg/cubic metre. The American Conference of Governmental Industrial Hygienists (ACGIH) reported that in human workers, concentrations of RP-BR smoke exceeding 100 mg per cubic metre were unendurable except for the "hardened worker". In human volunteers, exposure to red phosphorus smoke at concentrations of 100-700 mg per cubic metre for 2 to 15 minutes, was associated with significant, but reversible, symptoms of respiratory distress (cited in Ballantyne 1998).

Of potential concern to humans, repeated inhalational exposure to RP-BR smoke in rats produced irreversible terminal bronchiolar fibrosis and the lowest concentration of smoke where this was observed was 180 mg per cubic metre (Toxicity of Military Smokes and Obscurants, Volume 1, 1997). No definite evidence of permanent respiratory sequelae in humans following exposure to red phosphorus smoke was identified in the literature but this does not appear to have been adequately studied. The permissible exposure guidance level for repeated exposure of military personnel during training exercises recommended for RP-BR smoke was based on the ACGIH's Threshold Limit Value time-weighted average for phosphoric acid [1.0 mg per cubic metre], since it was considered to be the combustion product of prime concern (Toxicity of Military Smokes and Obscurants, Volume 1, 1997).

Concern is expressed about the potential for acute bronchitis and laryngitis in humans after acute exposure to red phosphorus smoke, since the final combustion product for both red phosphorus smoke and white phosphorus smoke is expected to be phosphoric acid.

Carcinogenicity

There are no human studies concerning the carcinogenicity or genotoxicity of red phosphorus smoke.

Weak clastogenicity was observed in the micronucleus test in the bone marrow and red blood cells of rats after repeated inhalational exposure to RP-BR smoke over a two week period (Aranyi 1984). However, a 19-month study of mice, rats and guinea pigs, repeatedly exposed to red phosphorus smoke (one hour per day, five days per week, 36-40 weeks), observed no significant differences in the frequency of neoplasms between exposed and control animals (Marrs et al 1989).

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statement of Principles are relevant to red phosphorus smoke (see Appendix K for details of factors and definitions).

Table 4 Statements of Principles concerning red phosphorus smoke

STATEMENTS OF PRINCIPLES.	INSTRUMENT NO
Chronic Bronchitis And Emphysema	73 & 74 of 97
Conjunctivitis	111 & 112 of 96

WHITE PHOSPHORUS SMOKE

White phosphorus smoke is generated from a phosphorus-containing flammable matrix that burns to form solid particles of phosphorus pentoxide (P_2O_5) in air. P_2O_5 reacts with moisture to form an aerosol of phosphoric acids in a complex mixture of polymeric forms (Toxicity of Military Smokes and Obscurants, Volume 2, 1999). One of the main components of white phosphorus smoke is phosphoric acid. Small amounts of uncombusted white phosphorus might also be present in white phosphorus smoke.

ADVERSE HEALTH EFFECTS FROM WHITE PHOSPHORUS SMOKE

White phosphorus smoke was reviewed in volume 2 of the report entitled, Toxicity of Military Smokes and Obscurants (NRC 1999), and by the US Agency for Toxic Substances and Disease Registry (1997).

Effects on the Eyes

White phosphorus smoke irritates the eyes of humans in moderate concentrations (J R Army Med Corps 2002). However, no definite long-term ocular sequelae in humans following exposure to white phosphorus smoke have been reported.

Effects on the Respiratory Tract

The most sensitive toxic response to acute exposure (one exposure or multiple exposures occurring within a short time, usually 24 hours or less) to white phosphorus smoke is respiratory irritation and distress. Nasal and throat irritation, cough, tightness of chest and dyspnoea were observed in human volunteers after acute exposure to white phosphorus smoke (White and Armstrong 1935, Cullumbine 1944). There were also several case reports of acute bronchitis and laryngitis following acute exposure to white phosphorus smoke.

No definite evidence of permanent respiratory sequelae was identified in the literature but this does not appear to have been adequately studied.

The US Agency for Toxic Substances and Disease Registry (1997) commented that exposure to high concentrations of white phosphorus smoke would likely be fatal to humans, based on deaths in animals.

Although white phosphorus particles are very toxic, the US Occupational Safety and Health Administration has recommended a permissible exposure limit of 0.1 mg per cubic meter for white phosphorus particles. The National Institute for Occupational Safety and Health (NIOSH) considers a concentration of white phosphorus particles of 5 mg per cubic meter immediately dangerous to life or health. NIOSH notes that inhalation of the vapour of white phosphorus particles may cause lung oedema.

Carcinogenicity

There are no human studies concerning the carcinogenicity or genotoxicity of white phosphorus smoke.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles are relevant to white phosphorus smoke (see Appendix K for details of factors and definitions).

Table 5 Statements of Principles concerning white phosphorus smoke

STATEMENTS OF PRINCIPLES.	INSTRUMENT NO
Chronic Bronchitis And Emphysema	73 & 74 of 97
Conjunctivitis	111 & 112 of 96

COLOURED SMOKES

Coloured smokes reviewed here are those identified from information obtained from the SAS regiment and for which information on chemical composition was available to the Expert Panel:

A. Coloured smokes in M18 hand grenades.

B. Coloured smokes in "L" series smoke grenades.

A. COLOURED SMOKES in M18 HAND GRENADES

M18 smoke grenades contain a mixture of fuel and dye. The coloured smoke is produced by the heat from the fuel, which volatilises the dye, then condenses outside of the munition to form coloured smoke. US regulations required troops to wear protective masks if they enter the smoke plume.

Four colours, red, yellow, green and violet, were used in the original or "old smoke" formulations. Concern about potential health hazards was cited as the reason for the United States army developing four new formulations of the same colours (Toxicity of Military Smokes and Obscurants, Volume 3, NRC 2000). The new violet smoke formulation was subsequently removed because of its acute toxicity.

Based on available information, it appears that the coloured smokes to which SAS veterans were exposed are those referred to as the "old smoke" formulations (Toxicity of Military Smokes and Obscurants, Volume 3, NRC 2000).

These formulations contain coloured dyes mixed with sulfur, potassium chlorate and sodium bicarbonate, refined kerosene and tricalcium phosphate for control of dusting and caking respectively. In volume 3 of Toxicity of Military Smokes and Obscurants, the dye composition of the "old smoke" formulations is given as:

Yellow - benzanthrone (BZA) 54% and dibenzochrysenedione (DBC or vat yellow 4) 38%.

Green - benzanthrone (BZA) 24%, 1,4-di-p-toluidino-9,10-anthraquinone (PTA or solvent green 3) 62% and dibenzochrysenedione (DBC or vat yellow 4) (13%).

Red - 1-methylamino-anthraquinone (MAA or disperse red 9) 40%, anthraquinone 2-3%.

Violet - 1,4-diamino-2,3-dihydroanthraquinone (DDA) 80% and disperse red 9 20%.

The dye components in the old yellow and old green formulations remain essentially unchanged in the respective smoke following combustion. Disperse red 9 persists in the old red and old violet smoke after combustion but it is also partially converted to 1- and 2-aminoanthraquinones. 1,4-diamino-2,3-dihydroanthraquinone (DDA) is completely converted to 1,4-diaminoanthraquinone (DAA) in the old violet smoke on combustion.

ADVERSE HEALTH EFFECTS FROM COLOURED SMOKES IN M18 HAND GRENADES

The US National Research Council's report on the Toxicity of Military Smokes and Obscurants (volume 3) found no human studies on "old smoke" formulations (yellow smoke, green smoke, red smoke, violet smoke) or its combustion products. However, some concern was expressed over the safety of some of the individual dyes that were components of these smoke formulations. Namely, benzanthrone (component of old yellow and old green smoke) and disperse red 9 (component of old red and old violet smoke) have been shown to have some dermal toxicity in humans and concern about contact allergic dermatitis was expressed. Concern about the potential for pulmonary sensitisation was also expressed.

Effects on the Respiratory Tract

Standard reviews of occupational asthma noted that reactive chemicals such as anthraquinone dyes are causes of asthma and act via an IgE mechanism (Bernstein 1997, Brooks 1998). All "old smokes" (yellow, green, red and violet) contain anthraquinone dyes.

Concern was expressed about the poor solubility in the lung of 1,4-di-p-toluidino-9,10-anthraquinone (solvent green 3), hence it may accumulate with repeat exposures.

Effects on the Skin

Benzanthrone is a known photosensitiser – it causes photocontact dermatitis and eczema in industrial workers.

1-methylamino-anthraquinone (disperse red 9) is reported to be a skin irritant and sensitiser in humans but appears to have very low toxicity.

Carcinogenicity

Tests for the genotoxicity of benzanthrone were negative in the dominant lethal mouse assay and with Escherichia coli, but results have been inconsistent in the Ames test. Benzanthrone was weakly genotoxic in a forward mutation assay based on human B-lymphoblastoid cells (Durant et al 1996). Initial studies do not indicate it is carcinogenic in mice (unpublished report cited in vol 3 Toxicity of Military Smokes and Obscurants, 2000; Singh et al 1990). Recent concern about benzanthrone and carcinogenicity appears to be due to a report that a related compound, 3-nitrolbenzanthrone, is a powerful direct genotoxic as identified by the Ames test. The authors considered it to show the strongest activity of any chemical reported in the literature (Enya et al.,1998; Kawanishi et al., 2000). It has also been shown to be genotoxic *in vivo* and to form DNA adducts. 3-nitrolbenzanthrone may be formed by reactions between benzanthrone and lower oxides of nitrogen during the combustion process of fossil fuels. It is not known if it is formed during the combustion process involved in coloured smoke formation.

In vitro genotoxicity studies of dibenzochrysenedione (vat yellow 4) have been contradictory. Two dermal carcinogenicity studies in mice were reported as negative (cited in vol 3 of Toxicity of Military Smokes and Obscurants, 2000). When fed to rats and mice in the diet it was negative in rats and female mice but caused an increase in lymphomas in male mice at 50,000 ppm. It was concluded that the carcinogenic

potential was uncertain and there was low dermal and oral toxicity. IARC (1990) concluded that vat yellow 4 was not classifiable as to its carcinogenicity to humans (Group 3). This evaluation was based on limited evidence for carcinogenicity in experimental animals (lymphomas and hepatocellular tumours in male mice after oral administration) and no data were available from studies in humans.

In vitro genotoxicity studies of 1,4-di-p-toluidino-9,10-anthraquinone (solvent green 3) have been contradictory.

In vitro genotoxicity studies of 1-methylamino-anthraquinone (disperse red 9) have produced contradictory results. It was also positive in mouse lymphoma cells with and without activation and positive in unscheduled DNA synthesis assay using mouse liver S9. Dominant lethal and mitotic gene conversion tests were negative. In a nine-month carcinogenicity study, rats were dosed orally with 5g of disperse red 9 (total dose per rat). The results produced inadequate evidence of carcinogenicity (Griswold et al., 1968).

1,4-diamino-2,3-dihydroanthraquinone (DDA) was positive in one Ames genotoxicity test and marginal in another. Its combustion product, diaminoanthraquinone (DAA), was positive in the Ames test and more active than the parent compound.

The combustion products of 1-methylamino-anthraquinone (disperse red 9), the major component of old red smoke and a minor component of old violet smoke, were 1- and 2-aminoanthraquinones. The US Department of Health's Report on Carcinogens (Tenth Edition) considered that 2-aminoanthraquinone was reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals. When administered in the diet, 2-aminoanthraquinone increased the incidences of hepatocellular carcinomas and neoplastic nodules in male rats, hepatocellular carcinomas in mice of both sexes and lymphomas in female mice. There was no adequate data available in humans. An earlier evaluation by IARC (1987) assessed 2-aminoanthraquinone as group 3 - not classifiable as to its carcinogenicity to humans. There were no adequate data available in humans and only limited evidence for carcinogenicity in animals.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles are relevant to old coloured smokes in M18 grenades (see Appendix K for details of factors and definitions).

Table 6 Statements of Principles concerning old coloured smokes in M18 grenades

STATEMENTS OF PRINCIPLES &.	INSTRUMENT NO
Asthma	85 & 86 of 2001
Contact Dermatitis	65 & 66 of 97
Photocontact Dermatitis	63 and 64 of 97

B. COLOURED SMOKES in "L" SERIES SMOKE GRENADES

Another group of coloured smoke grenades in past and current usage come under the heading, "L" series. Colours are blue, green, red and yellow. The chemical constituents of the "L" series smoke grenades, from information supplied by the manufacturer to the SAS regiment, were stated as:

Red smoke (CC 147): dye Disperse Red 9, potassium chlorate 170, lactose monohydrate, zinc stearate, kaolin colloidal, gum acacia powder.

Green smoke (CC 204): dye Solvent Yellow 33, dye Solvent Green 3A, potassium chlorate 170, lactose monohydrate, zinc oxide, zinc stearate, gum acacia powder.

Blue smoke (CC 146): dye Disperse Blue 180, potassium chlorate 170, lactose monohydrate, zinc oxide, kaolin colloidal, zinc stearate, gum acacia powder.

Yellow smoke: No details were provided.

These chemical constituents appear to refer to the formulation in the grenade rather than the combusted smoke.

It was also reported that most members of the Australian Defence Force would have been exposed to the number 83 hand grenade when it was in-service. From information supplied by the manufacturer to the SAS regiment, the number 83 smoke grenade had the same properties as the current "L" series smoke grenades. The number 83 hand grenade is no longer used in the Australian Defence Force. Smoke colours for number 83 hand grenades were red, green, blue and yellow.

ADVERSE HEALTH EFFECTS FROM COLOURED SMOKES IN "L" SERIES SMOKE GRENADES

No studies in humans or animals exposed to the coloured formulations of "L" series grenades or the combusted smokes were found.

No studies in humans or animals exposed to the coloured formulations of number 83 hand grenades or the combusted smokes were found.

No human or animal studies involving solvent green 3A were identified. Whether solvent green 3A is related to solvent green 3 is not clear. Solvent green 3 (1,4-di-p-toluidino-9,10-anthraquinone) was discussed in the section on coloured smokes in M18 grenades. The main concern was that inhaled solvent green 3 accumulates in the lungs of animals and this accumulation results in an inflammatory response in the lungs (Toxicity of Military Smokes and Obscurants, Volume 3). Other dye components of these formulations are discussed below.

Effects on the Respiratory Tract

As mentioned previously, anthraquinone dyes have been implicated as causes of asthma (Bernstein 1997, Brooks 1998). The red and blue smokes contain anthraquinone dyes - disperse red 9 is an anthraquinone dye while disperse blue 180 is a mixture of anthraquinone dyes (Marrs et al 1989).

Concern was expressed over the respiratory sensitising potential of solvent yellow 33 but this has not been adequately investigated in animals or humans (Toxicity of Military Smokes and Obscurants, Volume 3, NRC 2000).

Effects on the Skin

Disperse red 9 is reported to be a skin irritant and sensitiser in humans but appears to have very low toxicity.

There have been numerous case reports of allergic contact dermatitis in humans associated with exposure to solvent yellow 33 in cosmetics, soap, and hair cream (Toxicity of Military Smokes and Obscurants, Volume 3, NRC 2000). There was also one case report of allergic contact dermatitis in a worker employed in a factory that manufactured coloured smokes for use in detonators. Several laboratory studies of human volunteers demonstrated cutaneous sensitisation from exposure to solvent yellow 33.

Carcinogenicity

Previous comments made about disperse red 9 and its combustion products (1- and 2-aminoanthraquinones) may also be relevant. This depends on whether disperse red 9 in the red smoke formulation of "L" series grenades, undergoes the same partial conversion to 1- and 2-aminoanthraquinones upon combustion, as it does in M18 hand grenades.

There are no human studies concerning the carcinogenicity or genotoxicity of solvent yellow 33. Animal cell studies that tested solvent yellow 33 for genotoxicity were inconclusive. No evidence of carcinogenicity was found in a mouse lung tumour study with solvent yellow 33 or a mixture of solvent yellow 33 and solvent green 3 (Stoner 1985). A 2-year carcinogenicity study in rats exposed to solvent yellow 33 in feed found some evidence of carcinogenicity (US National Toxicology Program 1997). This involved increased incidences of hepatocellular adenoma, renal tubule neoplasms (adenoma or carcinoma) and squamous cell neoplasms of the oral cavity (papilloma or carcinoma) in male rats and increased incidences of hepatocellular neoplasms (adenoma or carcinoma) in female rats. There was no consistent evidence of a dose response relationship and the study may have limited relevance since exposure to solvent yellow 33 began in-utero and was not inhalational.

There are no human studies concerning the carcinogenicity or genotoxicity of disperse blue 180. A 20-month animal study on the repeated inhalation toxicity of a smoke containing disperse blue 180 was found (Marrs et al 1989). The formulation used in this study was similar to, but the not the same as, the blue formulation, used in the "L" series grenade. It consisted of disperse blue 180 (48%), potassium chlorate (26%), lactose (23%) and zinc oxide (3%). This formulation was ignited to produce a blue smoke and female mice, rats and guinea pigs were exposed to the smoke for one hour

per day, five days per week, for 200 exposures (42 weeks) at three different concentrations (51.5, 156.2 and 500.4 mg per cubic metre). A significantly high frequency of alveologenic carcinoma in the high dose group compared to controls was observed in surviving mice. This study also found that the dye, disperse blue 180, was genotoxic for the strain TA 1537R+, but non-genotoxic for strains TA1535, TA1537, TA1538, TA98 and TA100, in the Ames Salmonella typhimurium assay, with and without metabolic activation by S9 mix.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles are relevant to coloured smokes in "L" series smoke grenades (see Appendix K for details of factors and definitions).

Table 7 Statements of Principles concerning coloured smokes in "L" series smoke grenades

STATEMENTS OF PRINCIPLES .	INSTRUMENT NO
Asthma	85 & 86 of 2001
Contact Dermatitis	65 & 66 of 97

4. ASBESTOS

SUMMARY AND RECOMMENDATIONS

Asbestos exposure can cause mesothelioma, lung cancer and asbestosis. It is possible that SAS veterans were exposed to asbestos in some of the environments in which they trained. There is no indication from veterans' reports that this exposure was substantial.

The Expert Panel recommends that:

7. Potential exposure to asbestos in SAS veterans should be taken into account where indicated in the context of clinical investigation.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of potential exposure to asbestos among SAS veterans and members was made on the basis of the following sources of information:

(i) Submission from individual former members of the SASR.

(ii) Presentation from a serving member of the Australian Defence Force in charge of current and past training facilities for the SASR.

(iii) Published literature on airborne asbestos levels in environments similar to those that occurred in the context of past SAS training.

SAS EXPOSURE TO ASBESTOS

Several of the individual submissions made by former members of the SASR mentioned exposure to asbestos during counter terrorist training. Exposure to asbestos was reported to have occurred from training exercises conducted near Wittenoom, and in old buildings such as power stations and in naval ships.

A serving soldier in charge of training facilities for the SASR confirmed that a number of sites with potential asbestos exposure had been used in training exercises. It was stated that such sites were no longer being used. However there may be unavoidable exposures to asbestos dust during operational deployments.

Studies have found that while small quantities of asbestos fibres are ubiquitous in air, in most cases, exposure of the general population to asbestos is very low (ATSDR 2001). Higher levels of airborne asbestos have been measured near asbestos mines and in buildings with deteriorating asbestos-containing material or when such material is disturbed (ATSDR 2001).

ADVERSE HEALTH EFFECTS FROM ASBESTOS

The toxicology of asbestos is well established and many reviews are available. The Agency for Toxic Substances and Disease Registry is an agency of the United States Health Department that produces peer-reviewed profiles on hazardous substances. It produced a thorough review of the toxicological profile for asbestos (2001) and this review was considered definitive for the purposes of the Expert Panel.

Effects on the Respiratory Tract

Asbestos exposure leads to non-malignant respiratory disease (ATSDR 2001). Chronic exposure to asbestos can result in asbestosis. This condition involves diffuse interstitial fibrosis of the lungs resulting in shortness of breath and cough. Loss of lung function and even death can occur in more severe cases. Chronic exposure to asbestos also affects the membrane surrounding the lungs, the pleura. Thickened fibrotic areas, called pleural plaques, can result from asbestos exposure, as does diffuse pleural fibrosis and pleural effusions. Laryngitis has also been reported in a few studies of workers with chronic exposure to asbestos (ATSDR 2001).

Carcinogenicity

IARC (1987) concluded that there was sufficient evidence for carcinogenicity to humans from exposure to asbestos (Group 1 carcinogen). Asbestos exposure can cause mesothelioma of the pleura and peritoneum and lung cancer. Laryngeal cancer is also considered to result from asbestos exposure, but the evidence is not as strong as that for lung cancer and mesothelioma (ATSDR 2001).

There is some evidence of an association between asbestos and cancer in other locations (eg oesophagus, stomach, colon, rectum, pancreas, kidneys and malignant mesothelioma of the tunica vaginalis testis) but these associations are less certain (ATSDR 2001). Some studies have implicated gastrointestinal cancer but the magnitude of the excess risk was small, there was inconsistency in results amongst studies and factors other than asbestos might be responsible for the observed association (eg misdiagnosis, exposure to other chemicals, diet, or alcohol) (ATSDR 2001).

Chromosomal aberration and sister chromatid exchange in blood lymphocytes, DNA double-strand breaks and DNA damage in blood leukocytes have been observed to be higher in asbestos workers. Sister chromatid exchange in blood lymphocytes was also elevated in Turkish residents whose homes were painted with an asbestos containing material (ATSDR 2001).

The Expert Panel considers that the available evidence does not support the hypothesis that asbestos is a cause of transgenerational birth defects in humans.

COMMENT

Based on anecdotal reports from former members of the SASR, information from those responsible for the design of SAS training facilities, and studies on airborne asbestos levels, some former members of the SASR may have had exposure to asbestos.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following factors and their respective Statements of Principles are relevant to asbestos (see Appendix K for details of factors and definitions).

Table 8 Statements of Principles concerning asbestos

STATEMENTS OF PRINCIPLES	INSTRUMENT NO.
Adenocarcinoma Of The Kidney	87 & 88 of 2001 (RH only)
Asbestosis	138 & 139 of 96
Malignant Neoplasm Of The Colorectum	58 & 59 of 2002 (RH only)
Malignant Neoplasm Of The Larynx	27 & 28 of 95 amended by 155 & 156 of 95, 151 & 152 of 96, 193 & 194 of 96
Malignant Neoplasm Of The Lung	35 & 36 of 2001
Mesothelioma	52 & 53 of 94 amended by 199 & 200 of 95

5. PHYSICAL TRAUMA AND PROLONGED HEAVY PHYSICAL ACTIVITY

SUMMARY AND RECOMMENDATIONS

Physical trauma and prolonged heavy physical activity are a common cause of injury and disability among serving and retired SAS and other service personnel. The Counter Terrorist Special Recovery Support Group stated in its submission that, in the period 1979 to 1998, around 1% of personnel who served in the SASR were killed and 32% were injured. The incidence of physical injury is likely to have been underestimated because of disincentives felt by serving members of the SASR to report injuries. There is evidence that repetitive or prolonged heavy physical activity may cause musculoskeletal injury without overt acute trauma. Such injuries include back pain, compartment syndromes and tendonitis as well as an increased risk of osteoarthritis of the lower limb in those with anatomical abnormalities or significant previous injury.

The psychological impact of physical disability is well recognised, although many people are resilient in such circumstances. SAS veterans reported particular problems when physical disability lead to early discharge or where compensation was perceived to be inadequate. The Expert Panel supports the implementation of the ADF's injury prevention and control program, including the improved surveillance of the incidence of injury associated with different activities of units.

The Expert Panel recommends that:

8. Statements of Principles for common overuse injuries should be developed.

INTRODUCTION

Physical trauma is a wound or injury, which may be either macroscopic or microscopic. Injury may be caused by a single incident or repeated exposure to heavy loads. Trauma may affect the musculoskeletal system, resulting in fractures, sprains, lacerations and contusions, or it may be a risk factor for diseases of other systems, such as aortic aneurysm, cerebrovascular accidents and glaucoma. This chapter will focus predominantly on the musculoskeletal aspects of unintentional injuries.

Musculoskeletal disorders can be difficult to classify because diagnoses are based on clinical symptoms and signs for some, and on structural and functional criteria for others. Some diagnoses can be supported by imaging information but many cannot. Some are grouped together by means of terms used to describe their presumed aetiology, such as repetitive strain injury and overuse injury, even though the aetiological and pathophysiological pathways are not well elucidated. In ICD 10, most musculoskeletal disorders are included in the chapter on diseases of the musculoskeletal system and connective tissue, which is broken down by tissue type (joints, soft tissue, bone). The section on soft tissue is further subdivided into disorders of muscle, disorders of synovium and tendon and other soft tissue disorders.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of potential exposure to stressors among SAS veterans and members was made on the basis of the following sources of information:

(i) Submissions from individual former members of the SASR.

(ii) Submission and minutes of consultations with the Australian Special Air Services Association Counter Terrorist and Special Recovery Support Group

(iii) Submission by Dr Peter Anderson, rehabilitation physician, about musculoskeletal disabilities in service and ex-service personnel.

SAS EXPOSURE TO PHYSICAL TRAUMA AND PROLONGED HEAVY PHYSICAL ACTIVITY

The preparation of SAS soldiers for military missions is intense and similar in many ways to elite athletes preparing for competition, with the additional requirement of expertise in certain specialised skills related to counter terrorist activities (for example, method of entry training using explosives, insertion techniques and diving). Training activities of selected members are intense and realistic. One submission stated that "close quarter battle was practiced almost on a daily basis" and "we worked continuously on possible terrorist scenarios so we would be ready for any possible threats". On assault training, soldiers would be required to jump from moving vehicles and fast rope to a target while carrying their own weight in equipment. Roping and climbing are frequently practised insertion techniques, and are often performed from great heights.

There are numerous examples in the submissions of activities which could result in physical trauma, including:

- High and low altitude parachuting
- Climbing rocks, buildings and oil platforms
- Rappelling or jumping from moving helicopters over sea or land
- Loading and unloading aircraft during deployments
- Weapons training, including daily close quarter battle practice with exposure to gas, grenades and explosives
- Closed circuit oxygen diving
- Submarine swimmer release
- Carrying very heavy loads (around own weight) over long distances
- High speed driving

One submission written by former medical officer to the SASR stated that creatinine kinase levels (a marker of muscle damage) had been measured at baseline and after completion of a two mile run in full battle order. It was reported that some members of the SASR showed very high levels of this enzyme. The exact amount was not specified but it was said to be equivalent to that seen in severely ill hospital patients.

There are several factors which increase the likelihood of trauma to SAS members from these activities:

- The frequency with which these activities are carried out
- The inherently dangerous nature of some of these activities
- The general conditions in which these activities occur; for example, operating at night, extreme weather conditions, steep terrain, sleep deprivation, tight deadlines, long working hours.
- Certain equipment related factors reported by SAS veterans, but not verified by the Expert Panel. The veterans reported equipment failures and that they sometimes used equipment contrary to safety regulations due to operational demands. Examples quoted included faulty rappelling devices, inadequate seals on respirators, unsafe distance from explosives and inability to use protective equipment.

In addition, if the trauma occurs in the field, the risk of complications from an injury may be increased by delayed medical care or delayed medical evacuation. One soldier stated in his submission that "it was very common practise in my day and I would say even now to finish the job or the course and then seek medical help".

These activities are consistent with those described in published studies on the effects of survival training, although such studies pertain only to extremely intensive four or five day survival training courses, not to routine training activities. The training courses described involved heavy and prolonged physical exercise, including calisthenics, timed runs, long swims, open-water paddling, obstacle courses, and simulated combat exercises (Opstad 1991, Smoak 1990, Morgan 2000). These physical activities took place in environmental and psychological conditions which would have been conducive to injury. Smoak (1990), describing the selection training for US Navy Sea, Air and Land (SEAL) trainees, stated that "in addition to the physical stress, trainees experience psychological stress in the form of activities with no-win situations, verbal confrontations, and performance anxiety". The survival training for the US Army involved an evasion phase in which soldiers had to hide during the day and conduct movements at night (Morgan 2000).

ADVERSE HEALTH EFFECTS FROM PHYSICAL TRAUMA AND PROLONGED HEAVY PHYSICAL ACTIVITY

INCIDENCE OF PHYSICAL TRAUMA

The submission by the Counter Terrorist and Special Recovery Support Group stated that during the period 1979 to 1998, 2346 personnel served in the SASR and of these, 25 were killed and 770 were injured (35 seriously). Individuals reported in the submissions that they knew of or had witnessed deaths among their colleagues from a burst lung, a gunshot wound to the head, helicopter crashes and being hit by a boat. Similarly, they knew of or had witnessed many severe injuries, including: brain damage, injuries to ankles, knees and backs; concussion; gassing; a fractured skull; burns; lacerations; perforated ear drums and body parts being blown off (hand, calf, finger). As well these acute injuries, many submissions from SAS veterans detailed chronic disabilities, including degenerative spinal, neck, shoulder and knee problems, deafness and tinnitus.

The Expert Panel requested data from the Department of Defence on injury rates in the SAS in comparison to other military corp groupings, but such data were not available. However, data were available on the numbers of casualties reported by individuals to the ADF injury surveillance system DEFCARE in the period from mid-1997 (when the database became operative) to January 2003. In this period there were 389 casualties documented in the SAS regiment, of which 102 (26.2%) were due to sport and fitness training and 285 (73.3%) were work related. The ADF acknowledges that injury numbers are likely to be higher than those documented on the database because compliance with the injury reporting system has been poor. Minor injuries in particular tend to go unreported (Draft ADF Health Status Report 2002).

The ADF is in the process of upgrading its software for injury data recording which will provide more reliable figures and link previously separate systems, including sick leave data and hospital data. Early results from this system show an incidence rate of 810 injuries per 1000 full time personnel over 12 months. While these rates are very high, they are comparable to the US army incidence of 785 injuries per 1000 personnel for the same period (Draft ADF Health Status Report 2002).

Of the injuries that have been reported to the DEFCARE system in the last three years, 24% were sprains and strains of joints and adjacent muscles, 19% were due to poisoning and toxic effects of substances, 7.6% were hearing injuries, 6% were fractures and 5.6% were disorders of muscle, tendons and other soft tissues (Draft ADF Health Status Report 2002).

Soldiers are very conscious of the risk of being discharged if deemed medically unfit and this is a disincentive for reporting injuries in the military in general and the SAS in particular. The SAS veterans' submissions describe an attitude of stoicism to injuries and a reluctance to report them. Soldiers are often unable to report injury, both in training and on active service due to operational requirements. A reluctance to report injuries has been noted in US soldiers undergoing combat training for the Rangers. Martinez-Lopez (1993) states that "it is common practice for Ranger students to conceal medical problems to all except the primary care medic". The draft 2002 ADF Health Status Report also states that there is a strong cultural pressure on ADF personnel not to complain about injury. This practice may increase the risk of exacerbating an injury and thereby increase the likelihood of long term disability. It also makes subsequent claims for compensation harder to substantiate.

Although data on injury rates in the SAS in comparison to other units were not available, the activities and traumas listed above suggest that, anecdotally at least, the injury rates in the SAS are disproportionately high. Tomlinson (1987), reporting on injury data in the US military collected from 1984 to 1985, found that Special Forces had the highest rate of new injuries compared to infantry, rangers and artillery/aviation. The rates in these groups were respectively 12.1, 11.2, 10.1 and 4.5 new injury events per 100 soldier-months.

In the US, more military personnel die of unintentional injuries each year than any other cause (Powell et al 2000) though it should be noted that injury is a leading cause of mortality in this age group in the general population. From 1980 to 1992, injuries (unintentional injuries, suicides and homicides combined) accounted for 81% of all nonhostile deaths among active duty personnel in the Armed Services. The overall

death rate due to unintentional injuries was 62.3 per 100,000 person-years, compared to 12.5 for suicide and 18.4 due to illness. In this period there was a downward trend of around 4% per year of fatal unintentional injuries, indicating that programs for injury prevention can be successful.

Jones and Knapik (1999) reviewed data from a systematic injury control program developed by the US Army. Data were collected from hospital records, computerised records of the Army Physical Disability Agency, military death records, and records of visits to outpatient facilities. The burden of injury was high and was the single biggest cause of hospital admissions. In 1994, musculoskeletal conditions and injuries accounted for 28% of hospital admissions, followed by digestive diseases at 12%. The table below summarises the injury data at each level of severity and shows what proportion are estimated to be training or athletics related. It is noteworthy that training causes a high proportion or injuries at every level, and that for every accidental death there are around 1900 outpatient visits. Most injuries treated in US Army outpatient clinics were lower extremity-related injuries.

Table 9 Frequency of injuries by level of care, US Army 1994

Patient category	**Injuries/year**		**Ratio of other injuries to accidental deaths**
	Total	Training or athletics related (% of total)	
Accidental death	230	60 (26)	1
Disability	4,500	2,400 (53)	20
Hospitalisation	23,000	6,000 (26)	100
Outpatient sick call	440,000	240,000 (54.5)	1900

Source: Jones and Knapik, 1999

For US Army trainees, risk of injury was higher in units whose members ran a greater total distance. The incidence of military parachuting injuries was reported to be 8 to 14/1000 aircraft exits, with ankle injuries accounting for 30 to 60% of the total (Jones and Knapik 1999).

The Expert Panel notes that the ADF has policies on sport and physical training and is in the process of addressing the problem of injury through the institution of the Defence Injury Prevention Program (Draft ADF Health Status Report 2002). Some preventive measures being introduced under this program are optimisation of work-to-rest ratios, attention to footwear, attention to proper progression of physical training, control of marching speeds and selection of appropriate routes.

The Expert Panel stresses the need for improved systematic monitoring of injury rates, both for planning purposes and for evaluation of prevention programs for serving personnel. More detailed data are needed to document the rates and elucidate the causes of injury within and between different operational units. Data would also be useful for looking at the longer term consequences for veterans.

ADVERSE EFFECTS OF PROLONGED HEAVY PHYSICAL ACTIVITY

PATHOPHYSIOLOGY OF EXPOSURE TO TRAUMA

Below is conceptual framework for pathophysiological pathways and factors that may lead to musculoskeletal disorder. Loads of various magnitudes are imposed on the body in various ways. The amount of load required to cause a response, and then possibly symptoms or disability, is affected by a number of outside factors, including work factors, organisational factors, social, individual and psychological factors and non-work related activities. A given load will lead to a response and, if the load exceeds the ability of the tissue to withstand the load, tissue damage may occur as part of the response. Symptoms may in turn affect the response, lead to adaptation, resolve or lead to impairment and disability. The model does not consider the effect of rest or interventions specifically.

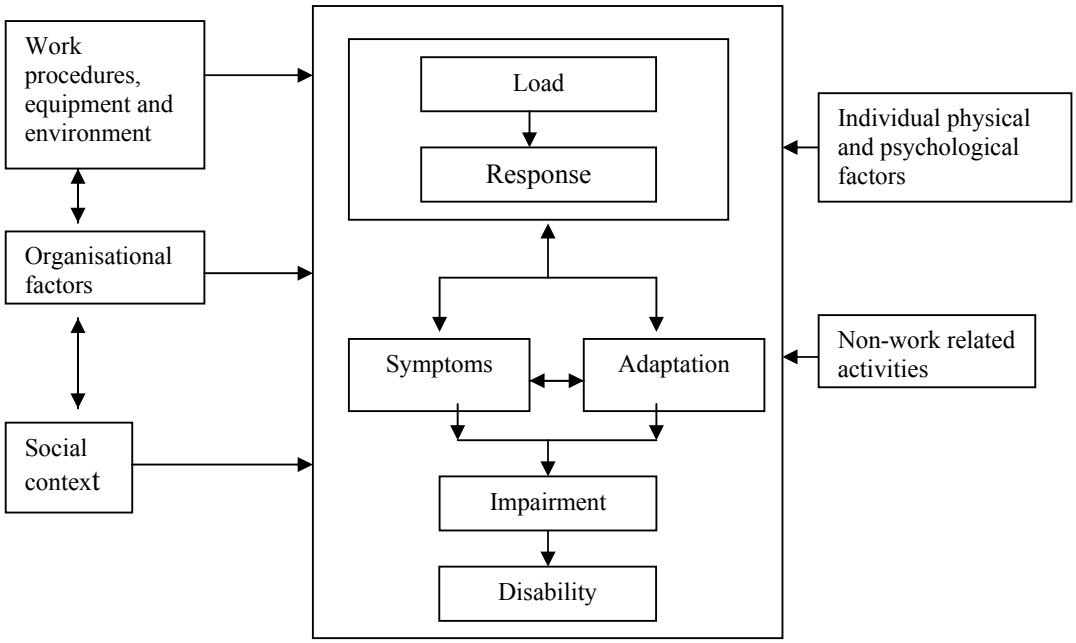

Figure 1 Conceptual framework of physiological pathways and factors that potentially contribute to musculoskeletal disorders (adapted from National Research Council 1999).

Given the numerous factors involved, it is not surprising that there is debate about the exact frequency, magnitude, duration or rate (ie dose) that renders mechanical forces harmful (Szabo and King 2000). The evidence for the dose which is required to cause harm varies according to the particular condition and body part. There is some evidence to support the association of certain injuries with certain activities and to show that ergonomic interventions are effective in reducing these injuries (NIOSH 1997, National Research Council 1999). Successful interventions require attention to individual, organisational and job characteristics as well as ergonomics.

The effect of the social environment on injury rates is illustrated by the finding in many studies of an association between military deployment and excess injury rates in

veterans (Hyams, Wignell and Rosell 1996). Non-battle injury remains the only documented cause of increased post-war mortality in US Gulf War veterans (Bell 2001). Bell describes five possible pathways by which this phenomenon may be mediated:

- A consequence of depression, PTSD and other psychiatric problems
- Adoption of behaviours which increase the risk of injury (eg heavy drinking)
- Indirect consequence of symptoms which are reported at a higher rate by veterans (dizziness, chronic fatigue, poor sleep, joint pains)
- Poorer health leading to reduced survival from injury
- Selection of risk takers into the military with continuation of risk taking behaviour

More research is needed to elucidate the cause of this association.

MUSCULOSKELETAL TRAUMA AND PROLONGED HEAVY PHYSICAL ACTIVITY

The RMA has varying definitions of prolonged heavy physical activity, based on the sound medical-scientific evidence for the disease in question. The activity may be defined as vigorous, strenuous, continuous, heavy, forceful, frequent, persistent or repetitive or a combination of the above. In some instances the actual load and/or length of exposure is specified. Particular postures such as twisting or bending may be important risk factors or cofactors for causing some conditions.

Heavy physical work has been defined as work that has high energy demands or requires some measure of physical strength (NIOSH 1997). Some biomechanical studies interpret heavy work as jobs that impose large compressive forces on the spine (Marras et al 1995, in NIOSH 1997). A problem with studies of this risk factor is that the judgement of what is heavy work is often subjective on the part of the subject or the investigator. There may also be confounding by other risk factors such as posture, twisting and lifting. Ideally studies should examine the effects of repetition, force, posture, vibration and heavy physical work separately, although it is not always possible and a combination of these effects may be necessary to cause injury.

A review of epidemiologic studies of musculoskeletal disorders in the workplace in the United States found an association with certain physical and psychosocial factors (Hales and Bernard 1996). Physical factors were intense, repeated, or sustained exertions, awkward, sustained or extreme postures of the body, insufficient recovery time, vibration and cold temperatures. Psychosocial factors included monotonous work, time pressure, high workload, lack of peer support and poor supervisor-employee relationships. Such factors may be both work and non-work related.

Repetitive strain injury, also called cumulative trauma injury, occupational overuse injury or work related musculoskeletal disorders (WRMSD), is a collective term for a range of conditions characterised by discomfort or persistent pain in muscles, tendons and other soft tissues in the back, neck, shoulder, elbows, wrists, hands or fingers (NOHSC). The term WRMSD is preferred because it does not introduce assumptions about the presumed cause (Grieco et al 1998). There is evidence that repetitious work related activities are related to certain disorders, in particular tendinitis of the shoulder, tendinitis of the hand/wrist and carpal tunnel syndrome (NIOSH 1997, Greico et al 1998).

In 1997 the US National Institute for Occupational Health and Safety published a critical review of the epidemiologic evidence for work-related musculoskeletal disorders of the neck, upper extremity and low back. The table below summarises this evidence. For all of the risk factors examined, the review was unable to find evidence of no effect.

Table 10 Evidence for causal relationship between physical work factors and musculoskeletal disorders

Body part	Risk factor	Strong evidence	Evidence	Insufficient evidence
Neck and neck/shoulder	Repetition		+	
	Force		+	
	Posture	+		
	Vibration			+
Shoulder	Repetition		+	
	Force			+
	Posture		+	
	Vibration			+
Elbow	Repetition			+
	Force		+	
	Posture			+
	Combination	+		
Hand/wrist				
Carpal tunnel syndrome	Repetition		+	
	Force		+	
	Posture			+
	Vibration		+	
	Combination	+		
Tendinitis	Repetition		+	
	Force		+	
	Posture		+	
	Combination	+		
Hand-arm vibration syndrome	Vibration	+		
Back	Lifting/forceful movement	+		
	Awkward posture		+	
	Heavy physical work		+	
	Whole body vibration	+		
	Static work posture			+

Source: US National Institute for Occupational Health and Safety, 1997.

The RMA has a factor for heavy physical activity in the SOPS for Achilles tendonitis or bursitis, sudden unexplained death, carpal tunnel syndrome, osteoarthrosis, fractures, lumbar spondylosis, thoracic spondylosis and cervical spondylosis. The RMA does not have a SOP for low back pain as it is considered to be a symptom and not a disease. The term low back pain encompasses aetiologies and pathologies which are not musculoskeletal in nature. However, there is evidence to suggest that low back disorders are associated with heavy physical work (NIOSH, 1997).

The term overuse injury is clinically often used to describe exertional pain and/or dysfunction when there is no evident acute trauma involved (Rolf 1995). The term "repetitive strain injury" tends to be used for work related injuries of the back and upper limbs. The term "overuse injuries" is more general, although it tends to be used

for injuries which are mostly sports or training related. In ICD10 there is a code for soft tissue disorders related to use, overuse and pressure but it mostly includes only diagnoses of bursitis. Overuse injuries are often described on the basis of symptoms and may include several different aetiologies and pathoanatomic pathways. The underlying cause of these injuries is still undefined and further research is needed to resolve the pathophysiological mechanisms of overuse injuries (Rolf 1995, Archambault et al 1995).

Overuse injuries may involve the following tissues: the muscles, producing compartment syndromes and muscle soreness; the bursae, producing bursitis; the tendons, producing tendonitis; the bones resulting in stress fractures, apophysitis and periostitis. Achilles peritendonitis and medial tibial syndrome are the most common specific overuse injuries among athletes in Finland, and they are especially a problem in endurance sports, such as long-distance running and jogging (Jarvinen 1993).

There is relatively little information on the incidence of overuse injuries, particularly for groups with the level of psychological and physical demand experienced by the SAS. Runners are a population group with some similarities to the SAS which has been studied better than most. Van Mechelen (1992) found that that the incidence of injury in runners reported in the literature varies between 37 and 56% or between 2.5 to 12.1 injuries per 1000 hours of running. Hoebring (1992) reviewed ten studies and found reported incidences of between 24 to 65%. Differences in the definition of runners or running and different periods of observation may account for the wide variation in rates.

Most running injuries are lower extremity injuries, with a predominance in the knee. About 50 to 75% of all running injuries appear to be overuse injuries due to the constant repetition of the same movement (Van Mechelen 1992). Several reviews concluded that higher weekly running distance and previous injury were risk factors for running injury (Van Mechelen 1992, Hoebrings 1992, Macera 1992, Hart 1994).

Footballers are another sporting group which conducts physical activities of an intensity similar to that experienced in the SAS. The frequency of football injuries varies in different studies depending on the code and how it is defined. Because of differing definitions, rates in different studies are not directly comparable. A review of studies of soccer injuries estimated that the injury rate in adult male players was approximately 10 to 35 per 1000 game hours (Dvorak and Junge 2000). In a season of rugby in New Zealand, the injury rate for adult males was 10.9 injuries per 100 player games, with injury being defined as injury events that caused a player to seek medical attention or to miss a game or practice session (Bird et al 1998). The majority of injuries occur in the lower extremities (Bird et al 1998, Dvorak and Junge 2000), particularly in the knees and ankles as well as the muscles and ligaments of the thigh and calf. Serious injuries of the brain and cervical spine also occur from participation in football. Case control studies have shown that footballers have a higher incidence of subsequent osteoarthritis of the lower extremity, particularly the hip (Dvorak and Junge 2000).

There is now increasing recognition of the importance of aggressive recognition and rehabilitation of acute football injuries, not only to maintain effectiveness of athletes but also to reduce the potential effects on the rest of their lives (Porter 1999). Previous

injuries and inadequate rehabilitation are the most important and well-established intrinsic risk factors for future injury (Dvorak and Junge 2000). A clear plan for handling return to play decisions needs to be in place before injuries occur. Managers of teams need to support the change in culture, because players are frequently willing to risk playing with injuries, especially at the elite level (Finch et al 2002).

As well as short term injuries, there is some evidence that heavy exercise may increase the risk of the development of osteoarthritis in weight-bearing joints. Panush and Lane (1994) reviewed the literature concerning the association of exercise and osteoarthritis of the lower extremities. They noted that normal joints in individuals of all ages may tolerate prolonged and vigorous exercise without adverse consequences or accelerated development of osteoarthritis. However, individuals who have underlying muscle weakness or imbalance, neurological abnormalities, anatomical variances, and who engage in significant amounts of exercise that stress the lower extremities, may accelerate the development of osteoarthritis. Individuals who have suffered injuries to supporting structures may also be susceptible to accelerated development of osteoarthritis in weight-bearing joints, even without increased stress to the joint from exercise.

Lahr (1996) also reviewed the literature on the relationship between running and osteoarthritis and came to a similar conclusion; moderate running in individuals without anatomical variances poses no increased risk for the acceleration of osteoarthritis. However, runners with abnormal anatomy and those with significant previous injury are at increased risk for the development and progression of lower extremity osteoarthritis.

NON-MUSCULOSKELETAL EFFECTS OF PHYSICAL TRAUMA AND PROLONGED HEAVY PHYSICAL ACTIVITY

Although most of the adverse effects of prolonged heavy physical activity are related to the musculoskeletal system, other systems may be affected. Miller, Barbarevech and Friedman (1994) reviewed causes of gastrointestinal bleeding and found running to be one of the less frequent causes. Kehat et al (2003) examined the long-term haematological effects of endurance training in Special Forces trainees. They reviewed the medical charts of 48 randomly selected naval Special Forces trainees and the same number of submarine trainees. The diet and characteristics of the two groups were similar. After two years of training, the haematocrit was significantly decreased in Special Forces trainees compared to the commencement of training, although the reduction was only slight (around 2%). There was no significant decrease in the haemoglobin, either before or after training or between the two groups at the end of training. This is similar to “sports anaemia” which has been observed athletes involved in endurance training. It is mild and essentially benign.

Many of the activities undertaken by the SAS involve prolonged exposure to extreme weather conditions or in bodies of very cold water (such as Bass Strait) which would place soldiers at risk of hypothermia, heat stress and heat stroke. Prolonged exposure to the sun causes sunburn in the short term and skin cancers in the longer term. Most civilians seldom spend more than 10% of the day away from a sheltered setting, whereas the mission requirements of a tactical fighting force may require a soldier to spend more than 30% of each day without shelter (Hanson and Goldman 1969, in Schissel and Barney 1998).

Some of the specialised activities conducted by SAS soldiers on training or operations have their own unique risks of physical trauma, and will be dealt with in more detail in the relevant chapters. Diving carries the risk of decompression sickness, cerebral arterial gas embolism, pulmonary barotrauma, carbon dioxide toxicity, oxygen toxicity, hypoxia, caustic inhalation, mask squeeze, drowning and death (Murrison 1991). High altitude activities expose soldiers to the risk of hypobaric decompression illness, hypoxia, barotrauma of the ear and sinuses. The risk of these effects can be reduced by provision of oxygen and pressurisation, but mission requirements for SAS members (such as high altitude air drop) do not always permit such measures (Molvaer 2000).

PHYSICAL TRAUMA AND DISABILITY

Anderson and Phillips (1997) reported on a clinical case series presenting to an orthopaedic surgeon. This series comprised 91 Australian ex-Vietnam combatants and 57 Army, Navy or Air Force veterans who were non-combatants. Subjects were a small clinical case sample and may not have been representative. Combatants had significantly more musculoskeletal disabilities than non-combatants. The highest prevalences of musculoskeletal disabilities (for both groups) were in the lumbar spine (86%), knees (58%), cervical spine (56%), shoulders (42%) and ankles (28%). Dr Anderson stated in his submission that the pattern of musculoskeletal disabilities in members and former members of the SASR was similar to that seen in Vietnam veterans as described in this paper.

In every year between 1996 through 2001, musculoskeletal disorders accounted for the overwhelming proportion of class A and B medical discharges from the Australian regular army (approximately 71% of all discharges in 2001), followed by mental disorders (20% in 2001). Among musculoskeletal disorders, the highest proportion was due to injuries to the spine, at 45% in 2001, followed by injuries to joints, at 43%in 2001 (Draft ADF Health Status Report 2002).

Under the Military Compensation and Rehabilitation Scheme (MCRS), knee injuries are the most common type of injury claimed for, followed by other leg injuries and injuries to arms and backs (Draft ADF Health Status Report 2002).

Fuerstein et al (1997) reviewed 41,750 cases on a US Army Physical Disability Agency Database. The cases occurred over a 5 year period between 1990 and 1994 and had been deemed temporarily or permanently unfit for duty. Musculoskeletal disability cases comprised approximately 51% of all disability case diagnoses. The five most prevalent musculoskeletal disorders were, in order of decreasing prevalence: musculoskeletal limitation of motion, degenerative arthritis or non-specific pain, lumbosacral strain, knee impairment and intervertebral disc syndrome. Occupations with heavy physical work requirements had the highest prevalence of musculoskeletal disability, including infantrymen and mortar crewmen.

Injuries which cause severe impairment often also cause major social and psychological repercussions. The injured person is usually unable to continue in their current occupation, and must consider modified duties, retraining or unemployment,

usually with associated loss of income. The individual has to live with severe physical disability, resulting in daily frustrations and the need to repeatedly come to terms with the disability. The injured person's family members also suffer a loss, yet are called upon to give emotional and practical support.

Depression due to pain, loss and frustration is a well known consequence of severe injury and disability, although many individuals manage to cope reasonably well. For example, Dr Trevor Anderson was blinded by a mine injury while working as a young medical officer in Vietnam. In his address to the 1998 RMA conference on Stress and Challenge, Health and Disease, he described feelings of fear and pervasive anxiety about being able to cope with his blindness. However, he retained a sense of hope through being able to exercise a considerable degree of personal control and he went on to become a psychiatrist. He stated that having compensation and therefore not having to worry about finances had been important in his recovery process.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles contain factors which are relevant to physical trauma and prolonged physical activity. The individual dose varies between different SoPs and readers are referred to the full SoP for doses and definitions.

Table 11 Statements of Principles concerning physical trauma and prolonged heavy physical activity

STATEMENTS OF PRINCIPLES	INSTRUMENT NO.
Achilles Tendonitis Or Bursitis	53 & 54 of 1996
Acquired Cataract	37 & 38 of 2001
Acute Pancreatitis	45 & 46 of 1997
Acute Sinusitis	209 & 210 of 1995
Acute Sprains & Acute Strains	50 & 51 of 1994
Adhesive Capsulitis Of The Shoulder	17 & 18 of 1999
Alkaptonuria	13 & 14 of 1995 worsening only
Alzheimer's Disease	17 & 18 of 2001
Anal Fissure	247 & 248 of 1995; 11 & 12 of 1997
Angle-Closure Glaucoma	15 & 16 of 1999
Aortic Aneurysm	66 & 67 of 1998
Atherosclerotic Peripheral Vascular Disease	65 & 66 of 2002 worsening only
Carotid Arterial Disease	346 & 347 of 1995
Carpal Tunnel Syndrome	89 & 90 of 2001
Cerebrovascular Accident	52 & 53 of 1999
Cervical Spondylosis	50 & 51 of 2002, 64 of 2002, 81 & 82 of 2002
Chondromalacia Patellae	33 & 34 of 2001
Cluster Headache	66 & 67 of 1999
Congenital Cataract	237 & 238 of 1995 worsening only
Cut, Stab, Abrasion Or Laceration	54 & 55 of 1994
Deep Vein Thrombosis	5 & 6 of 2001
Dementia Pugilistica	7 & 8 of 2000
Dental Pulp Disease	73 & 74 of 2002
Dislocation	290 & 291 of 1995
Epilepsy	79 & 80 of 1996)
External Bruise Or External Contusion	43 & 44 of 1994
Fracture	11 & 12 of 1994

Gingivitis	3 & 4 of 2002
Horseshoe Kidney	17 & 18 of 1995 worsening only
Impotence	97 & 98 of 1996
Internal Derangement Of The Knee	59 & 60 of 1997
Intervertebral Disc Prolapse	130 & 131 of 1996, 92 & 93 of 1997
Loss Of Teeth	374 & 374 of 1995
Lumber Spondylosis	46 & 47 of 2002, 77 & 78 of 2002
Open-Angle Glaucoma	69 & 70 of 2001
Osteoarthrosis	81 & 82 of 2001
Osteogenesis Imperfecta	11 & 12 of 1995 worsening only
Periodontitis	1 & 2 of 2002
Pes Planus	61 & 62 of 2001, 5 & 6 of 2002
Physical Injury Due To Munitions Discharge	9 & 10 of 2000
Plantar Fasciitis	3 & 4 of 2000; 47 & 48/2003
Polycystic Kidney Disease	55 & 56 of 1995 worsening only
Psoriasis	56 & 57 of 2002
Rotator Cuff Syndrome	83 & 84 of 1997
Secondary Parkinsonism	38 & 39 of 2002
Seizures	81 & 82 of 1996
Sensorineural Hearing Loss	29 &30 of 2001
Sensorineural Hearing Loss	29 &30 of 2001
Spina Bifida	59 & 60 of 1995 worsening only
Spondylolisthesis And Spondylolysis	15 & 16 of 1997
Sudden Unexplained Death	99 & 100 of 1996
Thoracic Spondylosis	48 & 49 of 2002, 79 & 80 of 2002
Tinnitus	25 & 26 of 2001
Tinnitus	25 & 26 of 2001
Trigeminal Neuropathy	81& 82 of 1995
Von Willebrand's Disease	61 & 62 of 1995 worsening only

6. BLAST AND OVERPRESSURE EXPOSURE

SUMMARY AND RECOMMENDATIONS

Blast and overpressure exposure is an occupational hazard of military employment and can cause both immediate injuries and long term disability. Evidence presented to the Expert Panel suggests that exposure to blast and overpressure occurred frequently in counter terrorist training.

The Expert Panel makes no specific recommendation in respect of blast and overpressure exposure.

INTRODUCTION

An explosion is caused by the rapid chemical conversion of a solid or liquid into a gas, with resultant energy release (Wightman and Gladish 2001). A blast is the wave of air pressure produced by the detonation of a high-explosive bomb shell or other explosion. A wave of high pressure velocity (shock wave, overpressure) is created and this is followed by one of negative decreased velocity, exerting a suction like action (Dorland's Medical Dictionary). The overpressure usually only lasts milliseconds or microseconds but it travels supersonically before decaying into an acoustic wave. In an enclosed space the blast wave can be complicated by reflections from walls and other structures (Argyros 1997). Following the blast wave there is a mass movement of air, referred to as a blast wind, which travels more slowly than the blast wave but which can propel objects and people considerable distances (Covey 2002, Elsayed 1997).

Blast injuries are categorised as primary, secondary, tertiary or miscellaneous (Covey 2002, Elsayed 1997). They may occur in isolation or in combination.

- *Primary* blast injuries are caused by the wave of overpressure which travels through the air or water and impacts on the body to cause internal damage with no visible external signs of injury. Gas-filled organs are the most commonly affected, with the lungs, ears and gastrointestinal tract being particularly vulnerable. Gas in disrupted tissues can be forced into vessels resulting in air emboli.
- *Secondary* blast injuries occur from objects that have been energised by the explosion to become projectiles. Most injuries among survivors of bombings have been shown to result from this mechanism (Mines et al 2000)
- *Tertiary* blast injuries result when a victim is thrown against the ground or an object or is injured by the collapse of a structure.
- *Miscellaneous* injuries include exposure to dust, toxic inhalations, thermal burns from the explosion or thermal burns from fires started by the blast and crush injuries from falling debris.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of the potential exposure to blast and overpressure among SAS veterans and members was made on the basis of the following sources of information:

i) Submissions from individual former members of the SASR.

ii) Submission by the Australian Special Air Services Association Counter Terrorist and Special Recovery Support Group

SAS EXPOSURE to BLAST AND OVERPRESSURE

Explosives training was practised as often as possible. One training activity described in the submissions was "method of entry" techniques, which involved the use of live ammunition, explosives, CS Agent, smoke generators, distraction grenades and stun grenades. It was reported that recommended safety distances from explosives were not adhered to because of the desire to make training as realistic as possible. This quote from one of the submissions gives an idea of the conditions of method of entry training, as practised by SAS veterans:

> *"Most times we had people in the building to test if hostages would survive. This was also done when shot guns were used to blow the locks and hinges off the doors to gain entry. Assaulters would stand as close as possible to the charges so they could enter the building as soon as a breach in the obstacle was made. Many close calls occurred when members were concussed, from shock waves or falling debris. [On] one occasion, while training a new team... the charge was placed at the wrong door. Luckily the team commander realised this and had it moved. ...Note, all CT actions occur in split seconds...not much if any time to realise and correct an error."*

Blast injuries reported in the submissions included concussion, a hand being blown off, a calf being blown off, being knocked unconscious from the shock wave associated with an explosion, perforated ear drums, burns and a lacerated eyeball from a hot shell casing. The submission by a former medical officer mentioned the possibility of brain injury from explosive blasts being misdiagnosed as a psychiatric condition.

ADVERSE HEALTH EFFECTS FROM BLAST AND OVERPRESSURE EXPOSURE

Pathophysiology of exposure to overpressure

The organs most vulnerable to the primary blast wave are the gas-filled organs, that is, the ear, the lungs and the gastrointestinal tract (Argyros 1997). Covey (2002) describes four putative mechanisms by which the blast wave damages living tissue.

(i) Spalling- particles from a more dense fluid are thrown into less dense fluid at the interface of two different media.
(ii) Implosion- there is a momentary contraction of gas pockets when the blast wave penetrates through tissues. As the blast wave passes and the pressure falls, these gas pockets re-expand causing injury from miniature internal explosions.
(iii) Acceleration-deceleration- the blast wave causes movement of body organs, but adjacent structures move at different speeds, causing shearing.
(iv) Pressure differentials- the impact of the blast wave on the body results in different pressures between the outer surface of the body and internal organs.

Exposure to a blast causes biochemical as well as physical effects on the body, which suggests that blast injury causes a major physiologic stress. Biochemical changes include elevations in the inflammatory mediators thromboxane A_2, prostacyclin and sulfidopeptide leukotrienes[1] (Cernak et al 1999). These mediators may contribute towards progression of pulmonary oedema (Guy et al 1998). Animal studies have also demonstrated depletion of antioxidants and lipid perioxidation (Elsayed 1997). These biochemical changes can continue for hours after the exposure.

Blast and fragment injuries

The severity of injury is directly related to the magnitude of the explosion, the distance from the blast, whether or not it occurred in an enclosed space and whether or not body armour was worn. Victims in a confined space are at greater risk of pulmonary or gastrointestinal primary blast injury. Blast waves that occur underwater are potentially more damaging than those that occur in air because of the higher density of water, greater transmission of pressure and enhanced intracorporeal energy transmission at the body wall (Abu-Zidan and Aman 2001, Guy et al 1998). Body armour protects against fragment injury but may increase the severity of primary blast injury, unless the armour incorporates materials of different densities (Argyros 1997, Wightman and Gladish 2001).

The table below characterises the expected injuries by distance from a high-explosive detonation in open air, assuming no victim protection. Secondary missiles have the longest range. In the 1998 Nairobi blast flying glass wounded people up to two kilometres away (Wightman and Gladish 2001).

Table 12 Injuries by distance from high explosives detonation in open air

	Closest							Furthest
Total body disruption	X							
Burns and inhalation injuries	X	X						
Toxic inhalations	X	X	X					
Traumatic amputations	X	X	X	X				
PBI* of the lung and bowel	X	X	X	X	X			
Tertiary blast injuries	X	X	X	X	X	X		
PBI of the ear	X	X	X	X	X	X	X	
Secondary blast injuries	X	X	X	X	X	X	X	X

Source: Wightman and Gladish 2001 *Primary Blast Injury

Most of the injuries seen after high-explosives detonations comprise conventional blunt, penetrating and thermal trauma. This type of injury is more common than primary blast injury because the victim does not have to be close to the bomb to be injured and even a small charge can lead to considerable injuries from flying objects. Soft tissue, orthopaedic and head injuries dominate in most series (Wightman and Gladish 2001). Estimates of the proportion of burns among survivors is variable, with some authors reporting that they are uncommon and others reporting that 31% of survivors suffered burn injuries (Stein and Hershberg 1999). Unless the victim is close to the detonation, the thermal flash usually causes only superficial burns.

[1] Thromboxane A_2 causes platelet aggregation and vasoconstriction and prostacyclin has the opposite effects. The sulfidopeptide leukotrienes cause vasoconstriction and increased vascular permeability.

Primary blast injury is less common and reports of proportions of this type of injury among blast victims vary from 1% to around 20% (Stein and Hershberg 1999). An analysis of patients injured by munitions by Cernak et al (1999) suggests that primary blast injury may be more common than was previously thought. These authors classified 1303 patients admitted to the Military Medical Academy in Belgrade with wounds caused by explosive munitions between 1991 and 1994. Inclusion criteria were explosive wounding of lower extremities without other penetrating injury, admission within 18 hours of injury and age under 50 years. 51% had symptoms and physical signs that were compatible with a diagnosis of primary blast injury. Injury grade was similar between the blast and non-blast groups. The authors speculated that this high number may in part be due to the many of the injuries having occurred within a confined space (armoured vehicles). Within the blast group, auditory injury was found in 72%, pulmonary blast injury was found in 28% and intestinal injuries in 16%. Mortality in the blast group was 5.26% and 2.16% in the non-blast group.

Kopchinski and Lein (2001) conducted a retrospective review of US Army non-combatant munitions injuries between August 1989 and September 1996. There were 742 incidents, resulting in 894 injured soldiers and 51 deaths. The most common types of injuries were thermal burns (26.7%), puncture wounds (23%) and lacerations (12.7%). The extremities were the anatomical areas most commonly involved. The most common activities associated with injuries were combat training exercises, munitions firing and rendering munitions safe. The use of newer weapons with larger killing radii and greater fragment projections increase the potential for injury and death. Exploding type munitions are particularly responsible for fatalities and are also responsible for hearing injuries.

Musculoskeletal Injuries

Primary blast injury
The primary blast injury can fracture bones and cause limb or head avulsions. Major limb avulsions appear to occur when the blast wave causes a fracture followed by avulsion through the fracture site by the blast wind (Covey 2002). Persons with limb avulsions have a grave prognosis.

Secondary blast injury
Fragments that strike the body are the most frequent cause of injury from blast exposure. Unlike bullets, fragments are not streamlined, so can tumble when they penetrate tissue, increasing tissue damage. Blast fragments can also increase tissue damage by crushing tissue and by introducing dirt, bacteria, clothing and casing fragments into the wound, increasing the likelihood of inflammation and secondary infection. The force of the blast may propel dirt into the wound far more proximally than is initially appreciated, necessitating more extensive and repeated debridement. The increasing use of modern body armour means that the limbs injuries are relatively more preponderant than thoracoabdominal injuries. Limb amputation may also occur from the impact of large flying fragments.

Tertiary blast injury
The blast wind can cause people to tumble along the ground or to be hurled through the air until they strike or are impaled upon objects. This can cause fractures, lacerations, contusions, amputations and concussion.

Lung injuries

Primary blast injury
Pulmonary manifestations of primary blast injury result from rupture of the alveoli and laceration of the lung parenchyma. They include haemorrhage, contusion, pneumothorax, haemothorax, pneumomediastinum, interstitial emphysema, subcutaneous emphysema and arterial air embolism (Argyros 1997). In open air pulmonary contusions occur or are worse on the side of the approach of the blast waves, but if the victim is located in a confined space they are more likely to be bilateral or diffuse.

Arterial air emboli to the brain or heart are believed to be responsible for most of the sudden deaths that occur within the first hour after blast exposure (Argyros 1997). Blast loads directed towards the chest can cause a unique, vagal nerve mediated form of cardiogenic shock (Wightman and Gladish 2001).

Some patients may present acutely, while others appear unharmed and develop respiratory failure 12 to 24 hours later (Guy et al 1998). Outcomes for victims of primary blast injury may be worsened by strenuous physical activity post exposure (Wightman and Gladish 2001). Positive pressure ventilation and excessive use of fluid resuscitation may also worsen outcomes by increasing the risk of tension pneumothorax, air embolism and pulmonary oedema (Guy et al 1998).

Secondary and tertiary blast injury
Rib fractures and damage to the chest wall are usually due to secondary or tertiary explosion injuries rather than primary blast injury.

Abdominal injuries

Primary blast injury
Abdominal manifestations are mainly in the form of intestinal contusion, intestinal haemorrhages and hollow organ rupture. Pneumoperitoneum may result from direct injury to a hollow viscus or indirectly from pulmonary barotrauma (Oppenheim et al 1998). The colon and ileo-caecal region, where gas tends to accumulate in most human beings, is the most common site of both haemorrhage and perforation. Perforations may be delayed, occurring 24 to 48 hours after the blast. Mesenteric, retroperitoneal and scrotal haemorrhages may occur (Wightman and Gladish 2001).

Solid organs are injured less commonly by primary blast injury than gas filled organs because of the more homogenous liquid densities. Non-bowel intra-abdominal injury is more likely to be caused blunt or penetrating trauma. However, shear waves can cause subcapsular haematomas, lacerations and rupture of the liver, spleen and kidney (Wightman and Gladish 2001). Injuries to the urinary bladder, renal pelvis and gall bladder are rarely reported, probably because of their high fluid content (Cripps et al 1999).

Ear injuries

Primary blast injury
Blast may damage both the middle and inner ear, causing hearing loss with or without tympanic membrane rupture. The ossicles can be dislocated or fractured. Fragments of keratinising epithelium may be forced throughout the middle ear and mastoid system and can later develop into cholesteatomas (masses of destructive keratinous debris). Other auditory manifestations include tinnitus and vertigo. Many blast survivors experience a profound, short lived sensorineural hearing loss with tinnitus. This may last a few hours, but a proportion of individuals will have a prolonged or permanent deficit (Cripps et al 1999). Surgical repair and medical management contributes to the restoration of hearing, but estimates of the prevalence of permanent sensorineural loss range from 30 to 55% of cases (Chandler and Edmond 1997). Hearing protection can attenuate the pressure reaching the tympanic membrane (Wightman and Gladish 2001).

Neurologic injuries

Severe head injury is a leading cause of death in victims of blasts. Subarachnoid and subdural haemorrhage are the most common findings in fatalities (Wightman and Gladish 2001). Open head injuries may result from fragments. Closed head injuries may be harder to detect and patients with closed head injury may have more subtle physical, affective, behavioural and memory problems (Cernak et al 1999).

Closed head injuries may result from blows to the head but there is debate about whether the blast wave is a direct cause of closed head injury. Wightman and Gladish (2001) state that, because of the brain's fairly homogenous density, primary blast injury is not likely to be a cause of cerebral concussion syndrome. They suggest that there could be other explanations for impairment of consciousness, such as arterial air embolism or cardiopulmonary events. Cernak et al (1999) speculated that kinetic energy from a blast wave could propagate throughout the body and be transferred to the central nervous system, causing an increase in intracerebral pressure. They also cited recent experimental data which demonstrated that pulmonary blast injury could cause brain oedema, electrolyte impairment and altered sodium-pump activity in brain tissue.

In the study by Cernak et al (1999), there were differences in the electroencephalogram (EEG) patterns of patients with symptoms of blast injury and non-blast patients. Of patients with neurological symptoms, 36% of the blast group had EEG alterations, compared to 12% of non-blast patients. In patients with positive EEG findings, a further examination occurred within a year. Long term alterations were shown in 30% of the blast group and in 4% of those without blast injury. Symptoms reflecting persistent CNS dysfunction in the group with blast injury included headache, vertigo, amnesia, mental blockage, apathy, lethargy, psychomotor agitation and anxiety. Patients complained about mental slowing and memory deficit.

Persistent EEG changes and attentional deficits suggestive of mild traumatic brain injury have also been observed in combat veterans with a history of blast concussion (Trudeau 1998). This was a small study (n=43), with self reported exposure to concussion and several uncontrolled confounders but it does raise some possibilities. Functional complaints without apparent structural changes may be diagnosed as

PTSD when they may in fact be due in part or in whole to chronic post-concussive syndrome, there being some overlap between the signs and symptoms of these disorders.

Sylvia et al (2001) reported a case of a soldier who was accidentally exposed to a back blast from an assault weapon. One month after the injury he reported headache, dizziness, irritability and insomnia and neuropsychological testing revealed some cognitive impairments. He also manifested vestibular balance abnormalities. The authors suggested that individuals who sustain relatively mild traumatic brain injury by blast or blunt trauma, but who appear objectively normal on physical examination, may have subtle disabilities that limit performance or compromise safety.

Ocular injuries

As many as 10% of survivors of terrorist blasts have ocular injuries and blast fragments are the cause of the majority of ocular injuries (Mines et al 2000). Wightman and Gladish (2001) only identified one case of ocular primary blast injury (a hyphaema) and it is likely that the eye's nearly homogenous density protects it from this mechanism of injury.

Eye injuries are often bilateral (between 50- 79% in different series) and the proportion of ocular injuries resulting in blindness can be as high as 75% (Muzaffar et al 2000). Temporary decreases in visual acuity can be due to oedema of the corneal epithelium, hyphaema and oedema of the macular region of the retina from concussion (commotio retinae).

An analysis of ocular injuries from 684 survivors of the Oklahoma City bombing found a range of eye injuries, namely lid/brow lacerations, open globe injuries, orbital fractures, retinal detachment, corneal burn, traumatic cataract, cranial nerve injury, vitreous haemorrhage, subconjunctival haematoma, retained intraocular foreign bodies, lacrimal system injury and optic nerve injury (Mines et al 2000). Glass accounted for nearly two-thirds of these injuries. Spectacles had a protective effect and the use of laminated glass, toughened window glazing and Mylar curtains was recommended as means of reducing glass projectiles in the blast vicinity.

An analysis of ocular and adnexal injuries of survivors of the Bali bombing on 12th October 2002 showed that of the 18 cases identified, six were due to the blast, seven were due to burns and five were due to a combination of the two (Crompton 2003). Injuries included corneal abrasions, foreign bodies, vitreous haemorrhage, eyelid burns, palsy of ocular nerves, choroidal rupture and orbital floor fracture.

Other injuries

Severe blast loads may occasionally cause cardiac contusions or oesophageal rupture (Wightman and Gladish 2001). Fractures of the mid-face can occur, though it is not clear whether they can be caused by primary blast injury or are due to blunt trauma.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles are relevant to blast and overpressure exposure (see Appendix K for details of factors).

Table 13 Statements of Principles concerning blast and overpressure exposure

STATEMENTS OF PRINCIPLES.	**INSTRUMENT NO**
Intervertebral disc prolapse	92 & 93 of 97
Otitis media	1 &2 of 2003
Physical injury due to munitions discharge	9&10 of 2000
Sensorineural hearing loss	29 & 30 of 2001
Tinnitus	25 & 26 of 2001

7. STRESSOR EXPOSURE

SUMMARY AND RECOMMENDATIONS

Stress is defined for the purpose of this report as the adverse psychological and physical consequences of exposure to circumstances and situations which present threat or challenge to the individual (stressors). Stress and stressors have been variably associated with a range of ill health effects both physical and psychological. The response of individuals to stressors is modified both positively and negatively by other factors such as psychological preparedness, camaraderie, social support and context. Military service is commonly associated with exposure to many stressors and service training in part aims to prepare personnel for such exposures. SAS training and service may be associated with a higher level of stressor exposure but also a higher level of preparedness and skills and some compensations such higher income and prestige.

The Expert Panel recommends that:

9. The RMA should continue to monitor the medical-scientific evidence on the health effects of stress and stressors and modify Statements of Principles when appropriate.

INTRODUCTION

Stress is difficult to define and the term is often used imprecisely and with different meanings or emphases by the public and by researchers. The term has been used to encompass such diverse concepts as stressful external events or situations (stressors), personality traits, negative emotions such as anger and hostility, and psychiatric conditions such as depression, anxiety and panic disorder.

It is useful to differentiate between stress and stressors. The term "stressors" is preferred to stress to denote a cause rather than an effect. Stress is the set of psychological and physiological responses to experiencing a stressor. For the purposes of this report, the Expert Panel interprets stress to mean stressors. In the following section of the report there has been an attempt to differentiate between stress and stressors, but the confusion between these terms in the literature meant that this was not always possible.

Stressors may be acute or chronic or both. Acute stressors are discrete and specific experiences or events which may be isolated, concurrent or recurrent and which present a threat or challenge to the individual. Examples include combat, loss and accident. Chronic stress can arise when stressful episodes occur too often for the individual to recover or when a single situation occurs indefinitely. It could arise out of occupational requirements, environmental impacts, social situations or family separation. Acute events may be superimposed on a background of chronic events.

The National Heart Foundation defined stress in its 1988 review according to three linked components:

- External stressors

- The way that the stressors are interpreted by the individual- there can be great variations in individual appraisals of a stressor which may in part be genetically determined or depend on past experience, personality and the social context in which the stressors occur.
- The individual's response- individuals who perceive a threat respond with some degree of emotional or physical disturbance or both.

Having considered the different definitions of stress and stressors, the Expert Panel concurs with the definition of severe stressors formulated by the RMA in consultation with ex-service organisations at its 1998 conference entitled "Stress and Challenge, Health and Disease":

> ***"****experiencing a severe stressor****"*** *means, the person experienced, witnessed or was confronted with, an event or events that involved actual or threat of death or serious injury, or a threat to the person's or other people's physical integrity, which event or events might evoke intense fear, helplessness or horror.*
>
> *In the setting of service in the Defence Forces, or other service where the Veterans' Entitlements Act applies, events that qualify as severe stressors include:*
> *(i) threat of serious injury or death; or*
> *(ii) engagement with the enemy; or*
> *(iii) witnessing casualties or participation in or observation of casualty clearance, atrocities or abusive violence;*

The Expert Panel notes that there are a wide range of other factors which have been thought to be potential stressors, and these have also been considered in this chapter.

The range of definitions is one problem with the measurement of stress and stressors and one which makes it difficult to compare studies. Another is the different methods for the assessment of stressors. Some studies measure self-reported stressors whereas others attempt to make an objective assessment.

Cross-sectional studies and case-control studies which use self-reported measures of stress are prone to bias due to "effort after meaning", that is, those with an illness would be more likely to attribute their illness to stressors than those without an illness. There is some evidence, for example, that individuals who are aware of their hypertension status may have an increased reporting of life stressors (Tennant 2001). Another problem with cross-sectional studies is that measurement of stressors at one point in time is not necessarily indicative of usual levels of perceived stress.

Assessment of multiple factors is important in studies of stress and stressors but is often limited. Evidence suggests that virtually no stressful event or set of stressful circumstances produces health problems in every exposed individual. Stress might best be viewed as a co-factor interacting with various other host vulnerability and resistance factors, including prior life experiences, social and cultural environments, genetic or biologic predispositions, personality factors, social support and coping resources, to increase the likelihood of illness and disease (Marshall et al 2000). The complex interaction of these factors makes it difficult to determine a clear relationship between stressors and subsequent illness.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of potential exposure to stressors among SAS veterans and members was made on the basis of the following sources of information:

i) Submission from individual former members of the SASR.

ii) Submission and minutes of consultations with the Australian Special Air Services Association Counter Terrorist and Special Recovery Support Group

iii) Submission entitled Report of the Risks associated with the Australian Army Special Air Service Counter Terrorist Offshore Assault Team 1982 by Claudio Gino Ferreri.

iv) Letter from Dr Oleh Kay, psychiatrist

v) Submission from Dr ACJ Maclean

SAS EXPOSURE TO STRESSORS

A summary of stressful activities outlined in the above submissions is made in the table below. Many of these activities and exposures are often only experienced occasionally as part of general military training or as part of active war service, but the submissions report that they are a frequent part of SAS training, especially when SAS members are required to practice terrorist neutralisation and hostage recovery techniques.

A former medical officer to the SASR stated that SAS training, and in particular counter terrorist training and operations, were stressful and dangerous in medical aspects, even in peacetime, and the risks were far in excess of those encountered in routine military training in conventional units.

Table 14 Examples of stressors reported by SAS members and former members

Stressors	**Examples**
Being placed in frightening and physically demanding situations.	Closed circuit oxygen diving; high and low altitude parachuting; climbing rocks, buildings and oil platforms; rappelling or jumping from moving helicopters over sea or land; daily close quarter battle practice with exposure to gas and stun grenades, submarine swimmer release; carrying very heavy loads (in excess of 50kg) over long distances, high speed driving.
Loud noise	Explosives, rifle fire, anti-armour weapons fire, aircraft noise.
Fear of injury to self and witnessing death or severe injury of fellow members	Death: burst lung, gunshot wound to the head, helicopter crashes, being hit by a boat. Injuries: hand blown off, falls resulting in brain damage and multiple injuries, concussion, being gassed, calf blown away, finger blown away, fractured skull. Minor accidents and near misses daily. Potential risk of exposure to biological weapons.
Stressful general work environment	Sleep deprivation, having to make rapid decisions with limited information, tight deadlines, inadequate rest periods, inadequate support staff, faulty equipment, operating in extreme environments (eg Bass Strait, steep terrain, thick vegetation) and in all weathers, day and night.
Stressful work requirements	Constant operational readiness, 24 hours a day for up to 2 years (called out once/week to once/month), not knowing if a call out is for a training exercise or a real incident, long periods of separation from families without adequate separation process (up to 10 or 11 months each year), long working hours.

STRESSORS AND EXPERIENCING STRESS

Weisath (1998) classified three forms of war stress:

- Shock traumas of brief duration.
- Repetitive or serial trauma.
- Prolonged exposure to danger characterised by varying degrees of predictability and control.

Marshall et al (2000) summarised the literature on war zone stressors, which produced a slightly different classification:

- Low intensity events, such as lack of privacy, long work hours, limited opportunity for recreation, difficult climatic conditions, separation from loved ones or "events which foster a sense of personal disheartenment, discomfort or demoralisation".
- Exposure to high-magnitude events, such as involvement in combat or its aftermath.
- Exposure to conditions in which individuals perceive either themselves or others to be at risk of serious injury of loss of life.

Military Survival Training

The military survival training experience particularly pertains to the SAS and incorporates a number of potential physical and psychological stressors. It is difficult to determine which one or more of these multiple stressors is responsible for outcomes. There could be additive or synergistic effects of combinations of stressors but studies of military survival training generally measure the effect of the total experience. Studies can be broadly grouped into those which examine the neuroendocrine or biochemical effects of survival training and those which examine the psychological effects, although there is some overlap.

Studies measuring neuroendocrine responses to military survival training studies consistently showed elevations in cortisol and catecholamine levels and decreases in steroid hormone levels, but they also showed that levels quickly returned to baseline levels once intense training had ceased (Morgan 2000, Opstad 1991, Opstad, 1994). Thus, these studies were not suggestive of long term adverse health effects, though they were not designed to show this. Conversely, positive effects of training on lipoprotein levels (Smoak 1990) may not necessarily produce lasting beneficial effects.

Two studies involving psychometric testing were identified, one after survival training and one after underwater training. Morgan et al (2001) found that 96% of subjects reported dissociative states (temporary segregations of normally integrated memories or subpersonalities from the dominant identity of the individual) after acute stress, but Special Forces soldiers dissociated less than general infantry soldiers. The fact that dissociation was common provides evidence that realistic military stress produces dissociation in psychologically healthy individuals.

A study of Special Forces soldiers doing underwater training (McDonald 1990) showed variable effects on different aspects of personality scales. Negative effects were significant decreases in likeability, service orientation and reliability and

significant increases in anger and fatigue. Variables in which there was no significant changes or changes in only one of the groups studied were: attraction, depression, vigour, confusion, total self-concept, adjustment, prudence, sociability, resiliency, clerical aptitude, sales aptitude, managerial aptitude. Some of these personality changes may potentially affect work capacity and/or spill over into interpersonal relationships, but there was no assessment of whether or not these changes were persistent.

Being On-Call

Members of the SASR are required to be available for duty within two hours of notice. In a submission from a psychiatrist to the Expert Panel, it was stated that that the extended periods on call were anxiety provoking and a veteran is quoted as saying that carrying a pager was "like walking around with a time bomb in my pocket".

There were only three studies of relevance identified from a literature search of stress and being on-call or on alert, and the subjects in two of these were US trainees in surgery and obstetrics (Sawyer et al 1999, Chatterton 1999). Both groups of trainees reported increased stress in periods of on-call. In the group of obstetrics trainees, stress was related to fatigue, sleep deprivation and errors made while on call. The authors pointed out that stress was related closely to the level of fatigue while off call, implying that the inability to engage in meaningful recreational activities outside of work may have caused more distress than actual workplace phenomena. This is consistent with the reports in submissions by the SAS of stress impinging on family life.

HEALTH IMPACTS OF EXPOSURE TO STRESS AND STRESSORS

Psychosocial effects reported in the SAS veterans' submissions included: depression, headaches, post-traumatic stress disorder, anxiety, adjustment difficulties, rage attacks, difficulty sleeping, difficulty socialising, alcohol abuse, stress on families, divorce and suicide. While on counter-terrorist training, soldiers were said to become aggressive and insensitive to the suffering of self and others. The difficulty of getting disability claims accepted was reported to add to the stress. The literature on the longer term consequences of exposure to stressors, both adverse and favourable, is summarised below.

Resilience and adaptation

The response to stressors, whether experienced in civilian or military life, is commonly held to lead to various adverse effects, both mental and physical. While there is no doubt that exposure to stressors can lead to adverse effects, it should be noted that such exposure can also lead to various positive effects, including mastery and growth, stress inoculation and improvement in ability to deal with future stressors.

Aldwin et al (1994) surveyed a group of 1,287 veterans and found that the men reported more desirable effects of military service than undesirable ones. All of the desirable effects and most of the undesirable effects showed linear trends with combat exposure, that is, greater effects were observed with heavier combat experience. The desirable effects included learning cooperation and teamwork, broadening of

perspective, coping skills, independence and self-discipline. It is possible that the balance of positive and negative symptoms might have been different if the sample in this study was biased due to non-response from those who were more bitter or in poorer health and those who were deceased. Ursano et al (1986, cited in Aldwin 1994) reported that Vietnam era prisoners of war also perceived benefits to their experience which were positively correlated with length of imprisonment.

Weisath (1998) points out that "the prevalence of some psychoses may decrease due to increased social cohesion as in a tight knit combat group: strong leadership, sense of purposes, sense of control, greater meaning of suffering, greater sense of importance and control". It would appear from the information presented that the SASR is a tightly knit organisation with a high level of morale. Studies of responses to stressful incidents have shown that tightly knit occupational groups which are frequently exposed to stressors have higher levels of resilience compared to the general population. For example, North (2002) found that there were significantly lower rates of PTSD in rescue firefighters following the Oklahoma City bombing compared with direct bomb blast survivors (13% compared to 23%).

Eid and Johnson (2002) explored the factors associated with acute stress reactions in three Norwegian submarine crews who had been exposed to major peacetime accidents. Submariner units have features which are similar to the SAS, that is, they are a highly reliable military unit characterised by careful selection and training and a unique organisational culture that reflects operational demands. Submarine duty is acknowledged to be highly stressful. However, compared to survivors from a Norwegian navy shipwreck, submarine crew members exposed to underwater accidents showed significantly less symptom reporting. Unit cohesion and a problem focused coping style were resilience factors in exposed crew members.

Chronic controlled exposure to stressors may lead to increased resilience (Seedat et al 2003). Biochemical studies in military aviators have suggested that adaptation to stress through experience can occur, at least in short term (Miller 1968, Ellis 1976).

Personality factors can affect whether or not high stress situations result in actual illness. Kobasa (1979) compared personality factors in executives who were exposed to high stress situations, as measured by the Holmes and Rahe Schedule of Recent Life Events. By comparison with high stress/high illness executives, the high stress/low illness executives were more "hardy". They had personality traits which led them to see stress a challenge, to evaluate stressful events in terms of a life plan and a willingness to act rather than be acted upon.

Given that SAS members are subject to rigorous psychological testing and are selected for abilities in the areas of self-discipline, leadership and teamwork, it can be postulated that they are likely to be a relatively stress hardy group. Two studies comparing SAS forces with other military trainees found the SAS soldiers to be more stress hardy (Morgan et al, 2001; Morgan et al, 2000). Whether this is an effect of training or due to the selection process or both is uncertain. The combination of social cohesiveness, job mastery and preparedness may serve to mitigate the adverse effects of exposure to stressors to some extent.

Military studies

The SAS submissions report that much SAS training is combat like, therefore the relationship between stressors and various outcomes in recent military studies is of relevance.

Marshall et al (2000) conducted a review of the scientific literature pertaining to stress and the Gulf War. The authors examined both non-Gulf War-related scientific literature concerning the link between stress and health as well as the body of empirical studies bearing directly on the link between stress and health problems experienced by Gulf War veterans.

Studies concerning stress and Gulf War veterans had to include a measure of stress exposure as defined by self-report or documented exposure to potentially stressful conditions (eg graves registration duty). Studies that relied solely on a comparison of deployed versus nondeployed personnel were not included because associations would be confounded by the multiple other exposures experienced by deployed personnel.

Their conclusions in relation to the empirical literature were as follows:

> *"... exposure to stressful events, including combat or war-zone exposure, can contribute to various psychological or bodily symptoms. Relatively common symptoms include depression, anxiety, fatigue, impaired memory and concentration, headaches, back and neck aches, gastrointestinal complaints, and breathing difficulty. More severe forms of psychiatric disorder, including PTSD, have also been linked to exposure to stressful life events. The onset and duration of these problems vary, with some individuals reporting delayed onset of symptoms or delayed treatment-seeking. Although they generally dissipate over time, it is not uncommon for symptoms of psychological or bodily distress to persist for years. In many instances, what appears as delayed onset of symptoms may be more aptly characterised as delayed help-seeking.*
>
> *The empirical literature also suggests that stress exposure acts as a contributing risk factor for a broad range of physical illness and disease, although the strength of the evidence is generally modest and varies depending upon the disorder in question. Some epidemiologic studies, a few of which are large and well-controlled, are consistent with the possibility that combat or war-zone exposure may contribute to greater prevalence of self-reported chronic health problems, perceived poor health, and higher levels of help-seeking behaviour. Less evidence implicates combat or war-zone exposure in actual physical disease.*
>
> *The empirical literature indicates that self-reported health complaints in the absence of objectively verifiable disease is relatively common in the general population. Some evidence suggests that stress exposure and perceived stress, as well as psychological and social processes, may contribute to both medical help-seeking behaviour and the experience of oneself as ill, even in the absence of objective evidence of disease."*

Mortality

There are few studies which examine exposure to stress or stressors as risk factor for mortality in its own right. The Australian Vietnam Veterans' Mortality Study looked at mortality rates by corps groupings. The groupings were determined by expert opinion according to the stress and danger to which men in the corps would have been exposed in Vietnam. Up to 1994, the standardised mortality ratios were not significantly different between the corps groupings (Crane et al, 1997).

As stated above, submariners have group and operational characteristics which are similar to the SASR. However, follow up studies of the mortality of US and British submariners have not revealed increased mortality rates or negative long-term effects on physical or mental health in former submarine crew members (Charpentier 1993, Inskip 1997).

Psychiatric Disease

At the Repatriation Medical Authority's 1998 conference, the consensus was that the psychiatric illnesses that may be associated with exposure to stressors were: PTSD, acute stress disorder, panic disorder, major depressive disorder, dysthymic disorder and alcohol dependence.

The Australian Gulf War Veterans' Health study found that, although veterans did not have poorer physical health than the comparison group, veterans did have an increased risk of psychological disorders in the post-Gulf war period. These disorders included depression, anxiety, post-traumatic stress disorder (PTSD) and substance use disorders. They were strongly associated with reported military service experiences that occurred in the Gulf War, especially the threat of attack. Adjusted odds ratios for selected psychological disorders which were newly present post Gulf-war are summarised below (adjustment was made for service type, rank, age, education and marital status).

Table 15 Adjusted odds ratios for psychological disorders newly diagnosed post Gulf War

CIDI defined DSM-IV disorder	N (veterans/comparison group)	OR (95% CI)
Any affective disorder	250/164	1.7 (1.3-2.1)
Major depression	225/152	1.6 (1.3-2.0)
Any anxiety disorder	105/40	2.9 (2.0-4.2)
Generalised anxiety disorder	10/3	2.9 (0.7-16.4)
PTSD	73/19	3.9 (2.3-6.5)
Alcohol dependence/abuse	209/125	1.5 (1.2-2.0)
Drug dependence/abuse	50/24	1.9 (1.1-3.2)

A 1990 cross sectional survey (Chemtob et al) looked at the prevalence and risk factors for PTSD in 57 Special Forces soldiers who had served in Vietnam, all of whom had had combat experience. The study found a prevalence of PTSD of 25%, based on a PTSD scale, with PTSD categorised as both a dichotomous and continuous variable. However, the response rate to the survey was only 51% so this estimate may be subject to selection bias and also depends on the validity of the instrument. The survey found that as well as combat events, post-service social interaction and pre-

service characteristics are also predictors of PTSD. Factors which increased the likelihood of a diagnosis of PTSD or a higher PTSD score were: poorer family relationships, less family closeness, having a friend missing in action, guilt over the death of a friend, number of times wounded, being wounded after R&R, not being emotionally prepared to leave Vietnam and not being able to discuss feelings upon return. Pre-service variables accounted for about one third of the variation.

The 2000 review of stress and the US Gulf War by Marshall et al concluded that all of the 27 studies that examined the link between exposure to stressors during the Persian Gulf War and symptoms of PTSD found evidence of a positive, albeit modest, relationship between these two factors. However the authors note that many of the PTSD studies did not document exposure to a traumatic event, though a definitive diagnosis of PTSD requires linkage of symptoms to a specific traumatic event.

The review also looked at mental health outcomes other than PTSD. Of 10 studies focusing on objectively verifiable stress exposure or self-reported combat-related exposure, seven provided at least some evidence of a significant relationship between stress exposure and psychological distress. The strength of these associations, although significant, tended to be modest (correlations between 0.06 and 0.27), suggesting that factors other than stress exposure also play a role in determining psychological distress.

The authors reported that the studies they reviewed were limited by the methodological flaws identified above, as well as by issues with sampling. Sampling problems included convenience sampling, low or unreported response rates, inadequately described comparison groups, under-representation of some groups of military personnel and failure to compare characteristics of participants and non-participants.

Cardiovascular Disease

The 1998 RMA consensus conference concluded that sudden cardiac death and cardiac arrhythmias may be associated with exposure to stressors.

There are several postulated pathways for stress/cardiovascular disease association:

- Excessive direct sympathetic stimulation of the heart and noradrenaline spill over.
- Hyperactivity of the noradrenergic nervous system accompanying chronic arousal.
- Alterations of the neuro-chemical modulation of the cardiovascular system induced by abnormalities in the hypothalamic pituitary axis (the "adrenaline hypothesis").
- Lifestyle patterns: smoking, lack of exercise, alcohol consumption, poor nutrition.
- Poor compliance with management of cardiovascular disease.

The National Heart Foundation published a review of systematic reviews (using only evidence from prospective studies) in March 2003. It concluded that there is strong and consistent evidence of an independent causal association between depression, social isolation and lack of quality social support and the causes and prognosis of coronary heart disease (CHD). The report also concluded that there is no strong or consistent evidence for a causal association between chronic life events, work-related stressors (job control, demands and strain), Type A behaviour patterns, hostility, anxiety disorders or panic disorders and CHD.

Hypertension

The link between stress and hypertension was examined both at the 1998 Consensus conference, and by the RMA as part of a recent investigation. Both concluded that the body of evidence regarding the association between hypertension and exposure to job strain was not indicative of a causal association. The link between depression, anxiety and hypertension was also examined in the investigation into stress and hypertension. The RMA concluded that there is sufficient sound medical-scientific evidence to support a reasonable hypothesis of an association between hypertension and suffering from a clinically significant anxiety or depressive disorder for the six months immediately before the clinical onset or clinical worsening of hypertension.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The Statements of Principles in which exposure to stressors is a factor are listed in Table 16. In some cases (impotence, irritable bowel syndrome), exposure to a stressor is an indirect cause of disease, via a specified psychiatric condition or substance use disorder for which stressors are contributing risk factors.

Table 16 Statements of Principles concerning exposure to stressors

STATEMENTS OF PRINCIPLE.	INSTRUMENT NO
Acute Stress Disorder	5 & 6 of 99 amended by 56 & 57 of 99
Adjustment Disorder	57 & 58 of 96
Alcohol Dependence Or Alcohol Abuse	6 & 77 of 98
Asthma	85 & 86 of 2001
Anxiety Disorder	1 & 2 of 2000
Bipolar Disorder	128 & 129 of 96
Cerebrovascular Accident	52 & 53 of 99 amended by 57 & 58 of 2003
Depressive Disorder	58 & 59 of 98
Drug Dependence Or Drug Abuse	78 & 79 of 98
Gingivitis	3 & 4 of 2002
Impotence	97 & 98 of 96
Irritable Bowel Syndrome	103 & 104 of 96
Ischaemic Heart Disease	53 & 54 of 2003
Panic Disorder	9 & 10 of 99 amended by 58 & 59 of 99
Personality Disorder	143 & 144 of 95 amended by 13 & 14 of 97
Post Traumatic Stress Disorder	3 & 4 of 99 amended by 54 & 55 of 99
Psoriasis	56 & 57 of 2002
Schizophrenia	132 & 133 of 96
Subarachnoid Haemorrhage	48 & 49 of 99
Sudden Unexplained Death	99 & 100 of 96 amended by 185 & 186 of 96
Suicide Or Attempted Suicide	71 & 72 of 96 amended by 177 & 178 of 96

8. PRESSURE EFFECTS ASSOCIATED WITH DIVING

SUMMARY AND RECOMMENDATIONS

Diving is a potentially hazardous activity even in controlled circumstances with adverse effects ranging from mild and self-limiting to fatal. Long term injuries can also occur as a result of diving, including damage to the middle and inner ear and possibly also long term neurological effects. There were many reports of diving accidents. The conditions of some of the diving exercises undertaken by SAS veterans suggest that the probability of injury may have been increased.

The Expert Panel recommends that:

10. The RMA should develop Statements of Principles for certain diving related medical conditions not currently covered, including decompression illness, pulmonary barotrauma and dysbaric osteonecrosis and should consider diving and pressure effects in relevant Statements of Principles.

INTRODUCTION

There are two principle ways pressure can cause adverse effects, barotrauma from the uncontrolled expansion of gas within gas-filled body compartments and decompression illness from expansion of dissolved gases due to rapid return to atmospheric pressures. This chapter will focus on the pressure effects associated with diving, but, because other diving related problems are described in the submissions, these are also discussed briefly.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of the potential exposure to blast and overpressure among members of the SASR was made on the basis of the following sources of information:

i) Submissions from individual former members of the SASR

ii) Submission by the Australian Special Air Services Association Counter Terrorist and Special Recovery Support Group

iii) Submission entitled Report of the Risks associated with the Australian Army Special Air Service Counter Terrorist Offshore Assault Team 1982 by Claudio Gino Ferreri.

SAS EXPOSURE TO DIVING

Diving is an activity practised by water troops in readiness for incidents both inside and outside Australia. In July 1980 the SASR was directed to establish a capability to retake offshore oil platforms in Bass Strait that may come under the control of terrorists. This capability was known as the Offshore Assault Team (OAT), which operated on land and at sea. The role at sea involved an assault on an offshore oil platform at night and submarine swimmer release. Bass Strait was a key training area and was visited every six weeks for about two weeks each time. Bass Strait has seas

with 20 to 30 foot swells, strong winds and deep, cold water inhabited by seals and white pointer sharks. Assault swimmers were capable of insertion into the maritime environment by parachute, submarine, surface vessel, helicopter, surface or underwater swimming.

The purpose of these operations was reconnaissance and surveillance of enemy strategic areas or assets and conducting raids to rescue prisoners, capture key enemy personnel or destroy enemy assets. Only three support staff were available for 25 swimmers, which was said to be inadequate, especially for underwater activities. The submissions report that operational requirements meant that the Manual of Army Safety could not be adhered to because it would have limited the ability of the OAT to achieve its objectives. Particular safety breaches cited were reduction of standby diver numbers, reduction of safety craft, operating in sea states above those allowed, mother craft unavailability due to weather conditions and lack of availability of an on site recompression chamber. Buoyancy vests were provided for underwater operations but were not capable of lifting swimmers to the surface in the event of an emergency.

The apparatus used for underwater operations in 1982 was the Drager LAR V, which was a closed circuit breathing apparatus incorporating a soda lime canister. The principal reason for using this apparatus was that it did not release bubbles and hence made an underwater approach to a target invisible to the adversary. When swimmers reached the oil platform the apparatus was removed while still underwater and the swimmer made a free ascent to the surface. This entailed a risk of entanglement with the line during ditching of the apparatus (which did result in a near drowning on one occasion) and a risk of burst lung during ascent.

Underwater swimming was reported to involve risks of carbon dioxide poisoning, oxygen poisoning, soda lime inhalation from water entering the canister, loss of consciousness, drowning, burst lung, underwater entanglement and attack from marine life. During swims, seals continually harassed both surface and underwater swimmers.

It was stated that carbon dioxide poisoning caused swimmers to lose consciousness on several occasions, thereby requiring them to be rescued and creating the additional risk of burst lung when taking the unconscious diver to the surface. Suggested reasons for the occurrence of carbon dioxide poisoning were: the soda lime canister not being packed properly so that exhaled gases were not scrubbed adequately, and water flooding the soda lime canister and neutralising its carbon dioxide absorbing capacity. Another suggested cause of loss of consciousness was inert gas hypoxia. There was one fatality from a burst lung during exercises in Bass Strait due to a rapid ascent.

It was stated that several swimmers had to be recovered from swims due to inhalation of caustic soda lime solution. This problem occurs when water enters the soda lime canister, and it was suggested that this could have been due to a faulty rubber seal on the lid or fractures in the mouthpiece or canister.

An additional specialised diving related activity was submarine swimmer release. This technique was developed to deliver a team of swimmers onto a target covertly from a distance over the horizon. It involved placing swimmers in the forward area of the

submarine under the casing and in the top of the fin. The swimmers breathe air from cylinders strapped to the submarine hull but they also wear self contained underwater breathing apparatus (SCUBA) gear. After reaching the target, swimmers left the submarine and switched from the submarine air supply to SCUBA. Sometimes the submarine descended below the acceptable depth, causing ear squeeze and increasing the risk of nitrogen narcosis, decompression sickness and running out of air. Due to the limited availability of the submarine, this operation was often rushed and swimmers had to work long hours with inadequate rest. In Bass Strait and Jervis Bay personnel operated for about 16 to 20 hours a day on a continuous basis.

ADVERSE HEALTH EFFECTS FROM PRESSURE EFFECTS ASSOCIATED WITH DIVING

DIVING PHYSIOLOGY

Many of the adverse effects of diving can be attributed to the changes of pressure experienced by divers. As depth increases, pressure increases but the gradient of pressure change is greatest near the surface. According to Boyle's Law, at a constant temperature the volume of gas is inversely proportionally to pressure (Russi 1998). Thus, as the diver descends, gas filled spaces within the body are compressed and the reverse occurs on ascent. The gas filled sites within the body are the middle ear, the eustachian tube (when open), the sinuses, the thorax and the gastrointestinal tract. Temporary gas filled spaces within the teeth may be caused by caries and other pathology.

Water is non-compressible and pressure increases linearly at a rate of one atmosphere every 10 metres (Becker and Parell 2001). Because the gradient of pressure is greater near the surface, the rate of gas expansion is correspondingly greatest near the surface. A volume of gas at 30 metres will double at 10 metres and double again at the surface. For a diver to avoid barotrauma the pressure in air filled spaces must be equalised to ambient pressure. Without equalisation, on descent the surrounding hyperbaric pressure will force blood and tissues into air filled spaces while on ascent expansion of air will cause pressure on tissues surrounding air filled spaces.

Intrapulmonary and environmental pressures can be equalised by exhalation during ascent, but if the ascent is too rapid for this too occur, expanding gas in pulmonary alveoli causes them to rupture. Air can then escape into the thorax, subcutaneous tissues, mediastinum and the arterial bloodstream. In the upright ascending diver, air emboli will tend to direct towards the cerebral circulation via the carotid arteries.

The other gas law relevant to diving is Henry's Law, which states that the amount of a given gas dissolved in a liquid is directly proportional to the partial pressure of that gas (Russi 1998). This law explains the cause of decompression illness and nitrogen narcosis. Most divers breathe air, though in some specific situations they breathe special mixtures of nitrogen and oxygen (nitrox) or helium and oxygen (heliox). Air is 21% oxygen and the rest is primarily nitrogen gas. Oxygen is metabolised but nitrogen is inert and remains in the body. Inhalation of air under pressure causes nitrogen to be absorbed into the tissues. The more time spent at depth and the greater the depth, the more nitrogen is absorbed. During ascent, nitrogen comes out of solution and is exhaled. If the pressure decreases too quickly for exhalation of gases,

nitrogen forms bubbles in intravascular or extravascular tissue. Once formed, because of Boyle's Law, the bubbles grow during ascent. Unlike arterial gas emboli, these bubbles are primarily venous.

This disease, now called decompression sickness (DCS), was initially known as "the bends" from the stooping posture assumed by caisson workers with affected joints. Decompression sickness may also be called decompression illness (DCI), when arterial gas emboli are included in the definition (Spira 1999). The site of bubble formation determines the symptoms experienced by the diver. Factors that lead to DCI include too rapid an ascent rate, diving for too long or at too great a depth beyond the limits of no-decompression diving limits, exercise, dehydration, age, obesity, previous injury, alcohol and cold (Spira 1999). Nitrogen bubbles can occur anywhere in the body for up to 72 hours after the last dive (Taylor 2000). Generally the sooner the onset of symptoms after a dive, the more serious the case. Boarding an airline less than 12 to 24 hours after a dive can cause bubble formation as standard cabin pressures are lower than atmospheric pressure.

Dive tables and dive computers are used to estimate the amount of time needed to stop at various depths in order that nitrogen can come out of solution and be breathed out. Unfortunately divers often misuse or ignore the information provided by these tools.

DECOMPRESSION ILLNESS

Decompression illness or sickness is often divided into the milder DCS 1, defined as having skin rash or muscle/joint pains only, and the more serious DCS II in which there are neurological, cardiopulmonary or vestibular abnormalities (Spira 1999). Both types of DCI may appear simultaneously as they are actually part of a spectrum of bubble induced disease.

Musculoskeletal symptoms are the most common feature of DCI and often presents as vague, poorly localised pain near a synovial joint without redness, swelling or tenderness. The pain is usually asymmetrical and may change in character over time. The body parts most commonly affected are the shoulders, followed by the elbows and arms. Dermatologic findings usually include pruritis and diffusely mottled erythematous patchy rashes, lividity and marbling. Skin lesions are often seen on the shoulders and upper thorax. Bubbles blocking the lymphatics may cause local pitting oedema.

Clinical presentations of the more serious forms of DCI include paraesthesia, hypoaesthesia, paresis, paraplegia, hemiplegia, urinary retention, visual disturbance, impaired consciousness, coma, ataxia, seizures and death. Spinal cord involvement is the site of most frequent neurological involvement, with the lower thoracic spine affected most often, followed by the lumbar and cervical spine. Chest pain and cough may occur with intrathoracic intravascular bubbling ("the chokes"), which is uncommon but very serious. It has been suggested that repeated subclinical DCI from cerebral bubbles may lead to a condition analogous to multi-infarct dementia (Spira 1999). Inner ear DCI is also called vestibular DCI ("the staggers") and presents with vertigo, tinnitus, hearing loss and nausea.

Dysbaric osteonecrosis is a necrosis of long bones, thought to be due to nitrogen gas expanding in bone marrow adipose tissue causing occlusion of blood supply

(Coulthard 1996). It may occur months or years after exposure, and is more common in occupational deep sea divers (Davidson 1989). Bone necrosis typically occurs in the heads of the humerus or femur and in the shafts of the femora and tibia. Lesions next to the joint surface cause the most symptoms and may progress to secondary osteoarthritis.

A patent foramen ovale predisposes to DCI because the lungs, which filter most of the small bubbles formed in the venous system during a dive, are bypassed. Approximately 20% of the population are estimated to have a patent foramen ovale (Spira 1999). The risk of all DCI in divers with PFO increases by 1.93 times compared to divers without PFO (Saary and Gray 2000).

DCI may present immediately or with a delay of minutes to hours. Affected divers often delay seeking treatment, and treatment delays reduce the chance of making a full recovery. Recompression in a hyperbaric chamber with oxygen is the primary treatment for arterial gas embolism (AGE) and DCI as it reduces bubble size and eliminates vascular obstruction and tissue distortion. Patients with suspected DCI should ideally be transported at altitudes less than 300 metres. Repeated treatments until no further improvements occur are often necessary and treatment after delays of weeks or months from exposure can still be helpful.

Residual symptoms may last week, months or years or be lifelong. Recurrent symptoms have been more often reported in association with sleep deprivation, long work hours, alcohol, prolonged immobilisation and psychiatric morbidity (Spira 1999).

BAROTRAUMA

Barotrauma is the damage or injury from a pressure gradient between the environment and air-containing body cavities. During ascent the pressure within the body cavities is greater than the surrounding environment, so it is important for divers to ascend sufficiently slowly for the pressure to be equalised. Uncontrolled ascents to the surface due to panic are a common cause of diving injuries and fatalities.

Pulmonary barotrauma

Pulmonary barotrauma is uncommon but has serious consequences. It includes thoracic squeeze (when the lungs are compressed below residual volume), diffuse alveolar haemorrhage, and pulmonary overpressurisation syndrome (“burst lung”). The latter presents clinically as arterial gas embolism, pneumomediastinum, pneumothorax and subcutaneous emphysema (Spira 1999). It is caused by gas expansion during ascent which exceeds the lung’s elasticity and leads to alveolar rupture. Pathological conditions of the lungs which can increase the likelihood of rupture, include chronic obstructive pulmonary disease, asthma, bronchitis, pulmonary blebs or bullae and restrictive lung diseases.

Arterial gas embolism is the second most common cause of death in divers after drowning and usually presents within 10 minutes of ascent as bloody froth at the mouth, chest pain, dyspnoea or collapse. Many cases occur while still submerged. Cerebral arterial gas embolism (CAGE) occurs when emboli lodge in the brain, resulting in unconsciousness, vertigo, paraesthesias, seizures, paralysis, paresis, visual disturbances, headache, confusion, cardiovascular accidents and death. Arterial gas

embolism may be hard to differentiate from DCI (Clenny and Lassen 1996, Russi 1998). The former diagnosis is suggested by a history of rapid ascent, the occurrence of symptoms within minutes of surfacing and other symptoms of pulmonary barotrauma. The diagnosis of DCI is suggested by more gradual onset of symptoms after a dive requiring decompression stops.

Other barotrauma

The middle ear is the most common body part affected by barotrauma (Spira 1999). Middle ear squeeze occurs during descent when the surrounding water pressure exceeds the air pressure in the middle ear. Air pressure within the middle ear can be equalised by opening the eustachian tube, but this may not be possible due to poor technique, inflammation, infection or anatomical abnormalities. If middle ear pressure remains excessively negative in relation to the pressure on the opposite side of the tympanic membrane, the result can be vascular congestion, haemorrhage, pain and rupture of the tympanic membrane. The risk of drum rupture is increased by stiffness of the tympanic membrane from scarring.

A reverse squeeze can occur upon ascent resulting in excess air in the middle air and outward bulging of the tympanic membrane. However, middle ear barotrauma of ascent is less common because expanding air during ascent tends to passively open the eustachian tube (Becker and Parell 2001). Excessive air in the middle ear upon ascent relative to the other ear may cause vestibular stimulation and vertigo (alternobaric vertigo).

Inner ear barotrauma is less common than middle ear barotrauma, but potentially more disabling. Inner ear barotrauma includes cochlear haemorrhage, a tear of the labyrinthine membrane and rupture of the round or oval window leading to a perilymphatic fistula (Sheridan et al 1999). This is often precipitated by forceful attempts to relieve negative middle ear pressure resulting in raised pressure within the perilymphatic space (Clenny and Lassen 1996, Pullen 1992). Ascending from depth can also cause rupture of the round window from increased pressure within the middle ear (Pullen 1992). Symptoms of middle and inner ear barotrauma include vertigo, nausea, pain, vomiting, deafness and tinnitus.

Obstruction of the external auditory canal by cerumen, tight fitting hoods or ear plugs, may cause external ear squeeze upon ascent or descent, resulting in pain, haemorrhage or tympanic membrane rupture.

The sinuses are second most common site of barotrauma, with the frontal sinuses being more affected than the ethmoids and maxillary sinuses (Spira 1999). Most sinus barotrauma occurs during descent, when negative pressure causes tearing of mucous membranes from the sinus walls. The main symptoms of sinus barotrauma are pain and epistaxis. Rarely, expanding air during ascent may cause fracture of the sinus walls (Becker and Parell 2001).

Air within teeth from tooth pathology may cause tooth pain (barodontalgia) and implosion or explosion of teeth. Gastro-intestinal barotrauma may result from expansion of air in the intestines upon ascent, producing symptoms of discomfort, colicky pain and increased eructation and flatulence after the dive. Rarely, a hollow viscus may rupture (Cramer et al 1982). Distension of the tissue at the gastro-

oesophageal junction may cause tearing, similar to tears caused by violent vomiting, resulting in haematemesis (Novomesky 1999). Mask squeeze can cause orbital haemorrhage (Butler and Gurney 2001).

OTHER DIVING HAZARDS

Hazards of diving gases

Nitrogen under pressure has a narcotic effect ("rapture of the deep"), which can cause dangerous errors of judgement underwater. Martini's Law of diving equates the effect of nitrogen as being equal to a single martini for every 50 feet of depth (Spira 1999). Symptoms disappear upon ascending.

Carbon monoxide and carbon dioxide may contaminate scuba tanks. Carbon monoxide poisoning can be fatal because it binds to haemoglobin in preference to oxygen, preventing oxygen delivery to tissues. This can result in dizziness, altered mental status and arrhythmias underwater. Higher than normal blood carbon dioxide levels can also be caused by failure of rebreathing apparatus, dead space in the regulator, slow breathing, restriction of chest movement from equipment and increased partial pressure of gases at depth. Symptoms include headache, rapid breathing, dizziness and confusion.

Excess oxygen causes CNS and pulmonary toxicity. CNS toxicity tends to occur after short exposure to high oxygen levels and presents as seizures. Hyperoxia is unlikely in dives up to 50 metres but is more likely when oxygen enriched air is being used or when 100% oxygen mixtures are given in recompression. Exposure to levels of oxygen above normal for hours or days affects other parts of the body, particularly the lungs. It manifests as chest pain, coughing and decreased vital capacity as well as paraesthesia, headache, dizziness and nausea (Hamilton and Silverstein 2001).

Hypoxia can occur in diving operations due to the use of inappropriate breathing mixture with too low a proportion of oxygen, malfunctioning equipment or running out of air. Mild degrees of hypoxia cause impaired judgement, inattentiveness, motor incoordination and occasionally euphoria, and major hypoxia leads to unconsciousness (Harrison's 1998).

Pulmonary oedema

There have been rare reports of pulmonary oedema occurring in otherwise healthy divers and surface swimmers, particularly in colder waters. It has been reported to occur in Special Forces swimmers during combat training (Mahon et al 2002). The mechanism is unknown, though capillary stress failure due to increased central vascular volume and pulmonary vascular resistance is postulated. All known cases have recovered fully, although the condition is potentially serious (Pons et al 1995, Slade et al 2001).

Headache

Headache in divers is common. Benign causes include tension or migraine headaches, exertion, exposure to cold, mask or sinus barotrauma, sinusitis and a tight face mask. Even benign causes can be distracting and pose a safety hazard. More serious causes include cerebral decompression sickness, arterial gas embolism, otic or sinus

barotrauma, arterial gas embolism, carbon monoxide or carbon dioxide poisoning and oxygen toxicity (Newton 2001, Cheshire 2000).

Long term neurological effects

There is some debate about whether divers may suffer long term damage to the central and peripheral nervous system. Some studies have shown evidence of persistent neurological damage in occupational divers (Todnem et al 1990, Vaernes et al 1990, McQueen et al 1994) and others have not (Hulst et al 2000, Andrews et al 1986, Curley 1988, Bast-Pettersen 1999). Most of these studies have been small and possibly subject to selection bias. Long term damage is more likely if there is a history of acute neurological injury (Vaernes and Eidsvik 1982, Todnem et al 1991) but has been demonstrated without such a history (Todnem et al 1990).

Attempts have been made to test for CNS abnormalities in divers in comparison to non-divers. In a small study of 20 divers and 20 age matched controls, magnetic resonance imaging showed a higher but non-significant difference in the number of brain lesions in the diving group (Tetzlaff 1999). In this study morphological change did not correlate well with functional deficit although brain lesions were significantly correlated with diving exposure. Cerebral perfusion tests have been suggestive of perfusion deficits, but only small groups were studied and they have not conclusively demonstrated brain abnormalities in divers compared to controls (Wilmshurst et al 1993, Shields et al 1997). Such tests may not be sufficiently discriminating to measure subtle deficits. Todnem et al (1991) found that divers had more electroencephalogram abnormalities than controls, although 33% of the divers had a history of severe neurological DCI. Murrison et al (1994) were not able to show a difference in somatosensory evoked potentials between divers and non divers with clinically normal lower limbs.

Symptoms in groups of divers with regular exposure to diving have included difficulties in concentration and problems with long and short term memory. Objective neurological findings have included signs compatible with dysfunction in the spinal cord and nerve roots and polyneuropathy (Todnem et al 1991, Tetzlaff 1999, Calder 1992). The consensus from two international workshops on the issue was that the changes are in most cases minor and do not influence the diver's quality of life (Molvaer 2000). Exposure to deep diving and a history of DCI is more likely to correlate with long term neurological symptoms (Todnem et al 1991, McQueen 1994)

Long term prospective studies are needed to determine whether or not diving results in persistent neurological damage in divers with no history of DCI. Normal neuropsychiatric tests in Royal Netherlands Navy mine-clearance divers compared to Navy controls (Hulst et al 2000) demonstrate that careful medical assessment and conservative decompression procedures are important in contributing to the long term health of divers.

Submarine escape training

Submariners in the Royal Navy were required to practise submarine escape techniques. Training is undertaken in water filled towers to which subjects gain entry at air locks at various depths (Saywell 1989). Trainees practised ascent with buoyancy aids or within specially designed immersion suits in which the head is enclosed in a hood filled with breathable air (Benton et al 1999). This exercise differs from that

described by the SAS for submarine swimmer release in two major respects; the ascent rate of submarine escape trainees appears to have been very rapid (about 3 metres/second) and the SAS had the capability to switch to SCUBA and would ascend using this apparatus.

Benton et al (1999) reported on incidents occurring during a 22 year period of training from 1975 to 1997, involving 115,090 ascents. There were 53 incidents in which the trainee required hospitalisation or recompression or both, of which 10 were due to pulmonary barotrauma. Donald (1991) reported two cases of DCI in human escape trials carried out by the Royal Navy between 1945 and 1970.

Closed circuit oxygen diving

Most recreational diving uses an open circuit apparatus, in which exhaled gases go out through the regulator into the water in the form of bubbles. In closed circuit oxygen diving a rebreather allows the diver to breathe his own air over and over again. This is accomplished by using a "scrubber" or canister of sodium hydroxide to remove carbon dioxide. Consumed oxygen is replaced with a small tank of pure oxygen. A microprocessor is used to monitor the partial pressure of gases and keep the partial pressure of oxygen constant.

This apparatus has particular usefulness for the military because it produces few or no bubbles. Other advantages are less wastage of gases, lighter weight and less decompression because there is less nitrogen in the system. These advantages have spurred the commercial development of such systems, prompting a call for industry guidelines and minimum requirements for the specifications of the electronic sensors which monitor and maintain the correct gas mixtures (Kirk 1998). As well as advances in electronics, another potential technological advance being researched is the use of differential permeability membranes to control gas composition (Wells 1998). The latest equipment should therefore be lighter and safer than earlier apparatus.

Good equipment does not substitute for appropriate training and divers need training specific to this type of apparatus. Particular risks associated with this equipment are central nervous system oxygen toxicity, hypoxia, carbon dioxide toxicity and inhalation of soda lime particles or caustic soda solution (Butler and Thalmann 1986, Crosson and Youngblood 1996, Nuckols 1996, Eynan et al 2003, Neubauer et al 2000). Some divers are particularly susceptible to oxygen toxicity (Butler and Knafelc 1986) and hypercapnia is associated with increased risk of oxygen toxicity (Eynan et al 2003, Arieli and Ertracht 1999). An experimental study which measured soda lime particles in the diving apparatus showed that some soda-lime dust does get through the filters into the air intake loop and may potentially contribute to chronic airway inflammation (Neubauer et al 2000).

INCIDENCE OF DIVING RELATED INJURY

The incidence of diving related injury varies according to whether divers are occupational or recreational. In addition, because of the highly specialised equipment used and the types of tasks undertaken, military divers experience conditions which other occupational divers do not. There are no data on the incidence of diving illness in SAS members and exposure to diving will vary among SAS members, depending on whether they are have belonged to the "water troops". However, there was one

death from “burst lung” reported and the numerous incidents described above suggest that diving related accidents were common.

Drowning is reported to be the most common cause of death among divers, but arterial gas embolism as a complication of pulmonary barotrauma ranks second (Russi 1998). An analysis of DCI and diving fatalities in a US military facility found a rate of 13.4 DCI events per 100 000 dives and 1.3 fatalities per 100 000 dives (Arness 1997). Violations of no decompression limits accounted for only 26% of all DCI incidents.

Murrison et al (1991) looked at ten years of diving related illness in the Royal Navy (January 1980 to December 1989). In the ten year period there were 244 injuries or 1.9 injuries per 1000 dive hours. The most common injuries were DCS I (21.7%), DCS II (10.2%), oxygen toxicity (9%), pulmonary barotrauma (7%) and CAGE (5.7%). Other injuries were carbon dioxide toxicity, non-pulmonary barotrauma, hypoxia, hypothermia and near drowning. Pulmonary barotrauma was a particular hazard for inexperienced divers.

A later analysis (Benton 2000) of diving injuries in the Royal Navy based on computerised records collected between 1995 and 1999, again showed that decompression illness was the commonest injury (rate 16/100 000 dives) for military air dives, followed by pulmonary barotrauma (7.5/100 000 dives). The incidence rate for DCI among recreational divers using air was much less than for the military (7.6/100 000 dives), but underreporting meant that the true rate might have been 25-50% higher. This is slightly more than the incidence rate for DCI of 0.001% to 0.004% estimated from the records of the US Divers Alert Network (Spira 1999). Two fatalities occurred during the study period (rate 1.9/100 000 dives). All eight cases of pulmonary barotrauma occurred after shallow dives and there were five cases of neurological decompression illness after shallow dives. This illustrates the danger of shallow water dives, particularly when there is poor buoyancy control and large swells, which can cause large and sudden pressure changes.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles contain factors which are relevant to diving and pressure effects (see Appendix K for details of factors).

Table 17 Statements of Principles concerning pressure effects and diving

STATEMENTS OF PRINCIPLES.	INSTRUMENT NO
Acute sinusitis	328 & 329 of 95
Chronic sinusitis	21 & 22 of 2003
Conductive hearing loss	19 & 20 of 1996
Otitic barotrauma	27 & 28 of 2001
Otitis externa	73 & 74 of 2001
Otitis media	1 &2 of 2003
Sensorineural hearing loss	29 & 30 of 2001
Sinus barotrauma	316 & 317 of 95
Tinnitus	25 & 26 of 2001

9. THE SYNERGISTIC EFFECTS OF THE ABOVE EXPOSURES

The Expert Panel has been unable to find any sound medical-scientific evidence as to the synergistic effects of the above exposures. It is in the nature of scientific enquiry that agents are initially studied in isolation, so as to be certain that any effects are due to that agent alone. There are sometimes additional studies performed if there is reason to suspect that synergistic effects are a possibility. Such studies are uncommon and there were none found for any of the many potential combinations of agents considered in this report.

In view of the complexity of potential interactions and the lack of clear evidence, the Expert Panel considers that further review would be uninformative on decision making and has made no recommendations in relation to this term of reference.

10. THE POTENTIAL FOR GENETIC ALTERATION ASSOCIATED WITH THE ABOVE EXPOSURES

SUMMARY AND RECOMMENDATIONS

Genetic alterations to human cells, as measured by chromosomal aberrations, occur spontaneously and potentially by exposure to various environmental chemical and physical agents. SAS veterans were concerned that the various chemicals to which they had been exposed might have produced changes in their DNA which may in turn have had long term consequences to their health and that of their offspring. Based on the review of the evidence of the genotoxicity of the exposures reported by the SAS and the likely level of exposure, it is highly unlikely that these exposures would produce adverse health effects.

The Expert Panel recommends that:

11. There is no indication for or benefit from testing all SAS veterans or their offspring for chromosomal aberrations.

12. In view of the possible concerns arising in the context of the previous genetic testing performed on some SAS veterans, those veterans and their families should be provided with the opportunity to receive genetic counselling and, if appropriate, chromosome studies at an accredited laboratory.

INTRODUCTION

Concern has been raised about the possibility of genetic alteration due to exposure of members of the SASR to various chemicals during training activities. There has also been concern about a report of structural chromosome aberrations in cytogenetic tests conducted on ten former members of the SASR.

STRUCTURAL CHROMOSOMAL ABNORMALITIES AND HEALTH

All of us have a background rate of DNA damage in our cells. Some of this damage occurs spontaneously during cell division while other damage is the result of exposure to endogenous or exogenous mutagens. The vast majority of this DNA damage is repaired soon after it occurs as our cells have excellent DNA repair mechanisms. If DNA damage is not repaired the damaged cell may die or it may survive with altered DNA. In some cases this DNA damage leads to gross changes in one or more chromosomes and these changes can be visualised using cytogenetic techniques. In rare cases, surviving cells with DNA/chromosome damage can progress to become cancerous. If the DNA/chromosome damage occurs in the germ cells of the testes there is the additional theoretical possibility that the mutated sperm may cause a birth defect in the man's offspring or may increase the chance of cancer in the offspring.

GENETIC ALTERATIONS IN CANCER

It is now widely accepted among cancer researchers that cancer originates from changes in the genetic makeup of cellular tissue. Furthermore, cancer is regarded as a genetically unstable disease, and the accumulation of many genetic abnormalities is a common source of malignant progression. Accumulation of mutations occurs in those genes that directly control cell birth or cell death. In a small subset of cancers, the instability is observed at nucleotide sites along chromosomal arms, without necessarily resulting in detectable gross chromosomal structural change. However, in other cancers, the instability is observed at the chromosomal level, resulting in losses or gains of whole chromosomes, or large portions thereof.

Lengauer et al (1998) reviewed genetic instabilities in human cancers. They noted that numerous genetic alterations that affect growth-controlling genes had been identified in neoplastic cells over the past 15 years, and that these genetic alterations could be divided into four major categories: subtle sequence changes, alterations in chromosome number, chromosome translocations and gene amplifications.

DETECTION OF AND SIGNIFICANCE OF GENETIC ABNORMALITIES

It has been known for many years from examining chromosomes in culture that breaks, rearrangements and abnormalities in chromosome number may be seen in a low proportion of cells in otherwise normal people and that their frequency increases with age.

Some of these changes result from technical aspects of culturing cells in vitro and the frequency can be increased by using different culture media or varying the length of time in culture. Some aberrations may reflect damage accumulated over a lifetime in the individual. Factors known to play a role include age, smoking and alcohol habits, recent infections, vaccinations, medications or drug use, exposure to X-rays as well as gender and occupation (Roessner et al 1998, Testa et al 2002). Some individuals have reduced ability to repair DNA damage.

Researchers have published a variety of methods to judge if toxic chemicals have produced changes or mutations in DNA/chromosomes. Most studies published to date have been conducted in mice or in transformed cell lines and so the relevance of these results to humans is inconclusive. To test an exposed group of individuals, their cells need to be studied and aberration rates measured against a matched control group of persons. However, each individual method has limitations. Also, the amount of chemicals an individual is exposed to, the duration of exposure and the lag between exposure and tests need to be taken into account.

Changes in DNA in humans may be studied using several different methods. To detect subtle sequence changes molecular techniques are needed to identify gene sequences. There are several methods of measuring changes at the chromosomal level. These include chromosome aberrations in peripheral blood lymphocytes, exchanges between chromosomes in cells (sister chromatid exchange) and looking at stained white cells for micronuclei in the cytoplasm. Some of these tests are more useful for detecting changes due to recent chemical exposure and others are more appropriate for studying the individual's cumulative exposure to mutagens.

There is debate about the interpretation of such chromosomal changes in terms of whether they are actually good predictors on an individual's future risk of adverse health effects. It is still too early to say that a high level of chromosomal aberrations in peripheral blood lymphocytes is associated with an increased risk of future cancer. For a detailed discussion of methods of detecting cytogenetic abnormalities and the implications of abnormal findings see Appendix H.

CYTOGENETIC TESTS CONDUCTED ON SAS VETERANS

The testing of a group of ten former members of the SASR was initiated by them and was performed in a private laboratory not accredited by NATA (Australia's Government-endorsed provider of accreditation for laboratories). The testing offered was the chromosome analysis of banded chromosomes from peripheral blood lymphocytes. This test detects all types of structural chromosome aberrations including symmetrical ones such as translocations.

Each subject had 50 cells examined for chromosome abnormalities. Four of the men had no abnormal cells, three had 1 abnormal cell, one had 2 abnormal cells, and 2 had 6 abnormal cells. Therefore, out of 500 cells tested, a total of 17 (1 in 29.4) were abnormal, including 10 cells showing chromosomes with structural alterations.

The author of the report concluded that there was a significantly high rate of chromosomal abnormalities in the SAS veterans, compared to background rates (the author calculated that 1/28 cells were abnormal compared to 1/115 abnormal cells in the control group). However, the analysis involved a comparison of the proportion of abnormal cells with controls generated from laboratory data. Few details were given about this control group in the report but, from the total number of cells analysed (27,050), they appear to be the same as the control population mentioned in a published paper (Ford et al 1998). In this published paper the control population data used was the stored results of chromosome studies performed on individuals on other occasions and investigated for other reasons such as infertility, miscarriage, a relative with a congenital abnormality and mental retardation.

Most well designed studies use control samples matched for age, gender and other relevant variables. However, the control population did not appear to be matched to the SAS veterans for age, gender or smoking habit. Further, it is likely that there is a difference in age and gender between the SAS veterans and the controls. The controls had a mean age of 30 years, while the mean age of 4 of the veterans of the SAS was 49 (at the time of testing in 1999). The controls were a mixture of males and females while all SAS veterans were male. The genetic investigation of the SAS veterans used only one of the available cytogenetic tests although a testing protocol of several methods is recommended to conclude that an exposed group has had some genotoxic damage.

There were several other methodological limitations in the SAS report. There was disparity in the number of cells examined between the SAS subjects and the control subjects. 50 cells were examined for each SAS subject as opposed to only 15 cells in the control subjects, except for a subset of control subjects with mental retardation where a further 100 cells were scored for the fragile X syndrome. The latter was a targeted scan looking specifically at the X chromosome and so other aberrations may

have been overlooked. The reader of the genetic test was not "blinded" to the SAS status of the individuals. Therefore, observer bias was a possibility.

These methodological limitations raise serious doubts about the validity of the results in the report.

REPRODUCTIVE EFFECTS OF PATERNAL EXPOSURE TO GENOTOXIC AGENTS

The only way in which the children of members of the SASR could be affected by paternal exposure to genotoxic agents would be if the SAS men had been exposed to chemicals that had damaged their germ cells. If the germ cells in a man (spermatozoa and precursors) are exposed to genotoxic chemicals it is theoretically possible for DNA/chromosome damage to occur in these germ cells. If the damaged sperm then participates in the formation of an embryo, the pregnancy may result in the birth of a child with a congenital abnormality or the child may have an increased risk of cancer.

Although such an effect has been demonstrated in animal studies following exposure to highly toxic genotoxic chemicals (eg cyclophosphamide) (Trasler et al., 1985; Jenkinson and Anderson, 1990) a similar effect has not been observed in human studies (Li and Jaffe, 1974; Li et al., 1979; Byrne et al., 1988; Green et al., 1991; Nygaard et al., 1991; Hawkins, 1991, Dodds et al., 1993). The human studies involved survivors of childhood cancer. Such cancers are frequently treated with highly toxic chemicals which often have genotoxic properties. While these chemicals may successfully kill the cancer cells there has been concern that they may concurrently cause DNA damage in the patients' germ cells. However, a number of studies of pregnancy outcome after chemotherapy have failed to detect any increase in birth defects or other adverse outcomes.

There is information that men who father children as they become older are more at risk of having a child with abnormalities resulting from point mutations. Presumably this is due to accumulated mutations resulting from both increasing age and the rapid cell turnover in the testes. The best known of these and the most common is achondroplasia, a form of dwarfism. The Expert Panel has no documentation that SAS veterans have fathered children with achondroplasia.

CYTOGENETIC EFFECTS OF SAS EXPOSURE TO CHEMICALS

In order for a hazardous chemical to have an adverse health effect there must be sufficient exposure of an individual to that substance. The degree of exposure required to cause harm (ie frequency, duration and amount) will vary according to the inherent toxicity of the chemical and the genetic susceptibility of the exposed person.

From the information supplied to the Panel it is apparent that the SAS personnel were exposed to both lead and CS Agent at various times without the use of protective clothing and therefore there may have been significant exposure to these substances. There are also claims of exposure to other chemicals but from the information provided it appears unlikely that the SAS members would have been exposed to a degree sufficient to cause harm.

The Expert Panel has no way of knowing the actual level of exposure for many of the chemicals used during SAS training and can only describe the reported health effects of the chemicals of concern. In the event of a claim by a veteran for compensation, the level of exposure would need to be demonstrated. There are no known genetic alterations associated with exposure to stressors, physical trauma, blast and overpressure or diving, so these exposures have not been considered.

Lead has been shown to be carcinogenic in rats and mice at high doses (renal tumours) but the evidence for carcinogenicity in humans is considered inadequate. Studies that evaluated lymphocytes for genotoxic effects (chromosomal aberration, sister chromatid exchange) in humans occupationally or environmentally exposed to lead produced contradictory results. Likewise, inconsistent results regarding chromosomal aberration and sister chromatid exchange, were found in studies with human lymphocyte cultures exposed *in vitro* to lead. High blood lead levels in men have been associated with decreases in fertility, decreases in sperm count and motility and increased percentage of abnormal spermatozoa but other studies have not found this association. There is no evidence of birth defects in humans resulting from paternal exposure to lead.

CS Agent has been shown to cause chromosomal damage *in vitro* but similar results were not seen in studies in live mice. Carcinogenicity studies in rats and mice were negative. There have been no carcinogenicity studies in humans. There are no reports of birth defects in humans resulting from paternal exposure to CS. The Expert Panel considers that the available evidence does not support the hypotheses that CS is a carcinogen or that it is likely to cause genotoxic damage to germ cells.

The Expert Panel was also provided with evidence that the SAS personnel were exposed to a number of other chemicals including those in hexachloroethane smoke, red and white phosphorous smoke, various coloured smokes and asbestos. Some of the chemicals in these smokes are highly toxic and show genotoxic properties in animals but there is inadequate evidence concerning the carcinogenicity or genotoxicity of these smokes in humans. There is strong evidence that asbestos can cause certain cancers, particularly mesothelioma of the pleura and peritoneum, and lung cancer, but no evidence that it is genotoxic. As noted above, it is unclear from the information provided to the Expert Panel whether the SAS personnel had prolonged, heavy exposure to these chemicals without protective clothing during normal operational use. In the absence of such exposure, the relevance to SAS personnel is low.

Overall, the evidence suggests that the SAS personnel have not received significant exposure to chemicals that are likely to be genotoxic. Hence any observed increases in chromosomal aberrations in the lymphocytes of SAS personnel are unlikely to be due to occupational exposure to harmful chemicals.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS of PRINCIPLES

In the Statements of Principles for the relevant cancers, specific carcinogens are included as factors where the scientific evidence supports this.

11. THE INTERPERSONAL RELATIONSHIP, BEHAVIOUR AND LIFESTYLE ALTERATION THAT MAY BE ASSOCIATED WITH THE ABOVE EXPOSURES

SUMMARY AND RECOMMENDATIONS

The interpersonal relationship, behaviour and lifestyle alterations that may be associated with the above exposures are difficult to establish because the issues are complex and the literature is still at a relatively primitive stage. In addition, most of the published studies are not specific to high intensity units such as the SAS and there are particular aspects of SAS life that are both potentially positive and negative compared to regular service life.

The Expert Panel recommends that:

13. The programs to facilitate transition to civilian life currently being piloted by the ADF should be further evaluated and, if shown to be effective, disseminated as per usual practice.

INTRODUCTION

This chapter of the report will consider the hazards of military life as a unique type of exposure and the effects of military life on the family, divorce, interpersonal violence, suicidal behaviour, smoking, drinking and substance abuse.

The Expert Panel heard reports of the existence of problems in the SAS relating to family and lifestyle but has no other evidence that this is a major problem. The relevant, available literature is sparse and methodologically flawed and mostly pertains to the US military as a whole. There is no literature specifically about the SAS in relation to these matters. A scarcity of systematic data collections to measure the true incidence of most of these family and lifestyle problems means that comparisons between rates in different units of the military or in veterans cannot be made. From this limited information the Expert Panel raises a number of issues for consideration.

SOURCES OF REPORTED SAS EXPOSURE

Assessment of potential effects on interpersonal relationships, behaviour and lifestyle among SAS veterans and members was made on the basis of the following sources of information:

i) Submissions from individual former members of the SASR.

ii) Minutes of consultations with Australian Special Air Services Association Counter Terrorist Special Recovery Support Group.

SAS EXPOSURE

A number of former members of the SASR stated in their submissions that they felt that their service had had a negative impact on their families. As identified in the chapter on stressors, SAS veterans have reported that counter terrorist training involves being on call, separations from family for training exercises and deployments, working long hours (70 to 90 hours per week), extreme physical demands and a high risk of injury. The extremely long working hours and the need to remain on call meant that "the ability to maintain a normal family life was impossible". Exercises were held every month and would last between four to five days. Sleep deprivation was common during these exercises.

The difficulties of military life in general are well recognised and include constant changes in postings, a rigid social hierarchy, absence of serving family member, and direct threats of injury or death to the serving family member (Ursano 1989). These hazards are mitigated to some extent by other aspects of military life, including a wide range of formal and informal social networks, free or low cost access to health, recreational and other services, a stable income and being a member of a cohesive, respected group. Furthermore, some of these factors are not unique to military life. Work/life conflicts are common in many modern households, especially when partners are both employed. Non-military personnel can also be required to undertake frequent and/or extended absences from home, including long distance drivers and workers who travel overseas for business or government purposes.

The SASR is permanently based in Perth, so that families are not required to move to new postings every two years. On the other hand, regular military units normally receive some warning of impending moves or deployments, giving soldiers and family members time to adjust. SAS members are required to be available at two hours' notice which means that they cannot be far from barracks for the duration of the on call period, which obviously also imposes restrictions on the soldiers' families.

If the soldier is called up, the family may have no idea whether or not the soldier is on a training exercise or on deployment. One submission stated that, in the early days of counter terrorist training, members of the SASR were forbidden to contact their families until they returned from deployment. This practice was modified after an occasion on which a death was reported on the news before the family members could be notified.

When regular military units are deployed, it is usually for a fixed term, whereas SAS members do not always know the length of time they will need to be on operation. This uncertainty about length of deployment and the dangers that will be faced is also starting to affect the regular forces through their increasing involvement in peacekeeping activities.

Blount, Curry and Lubin (1992) describe three different phases of separation: predeployment, survival and reunion. Each of these phases brings stressors to families. Remaining spouses must take on new responsibilities and tasks, including being the sole provider of discipline if there are children. Increased responsibilities may mean that reduced time is available to spend with children. Older children may also be expected to take on new tasks. These increased demands have the potential to

induce both stress and personal growth. Stress itself stimulates adaptation by motivating individuals to enlist support from others (Rosen et al 1988).

During separation, both spouse and children may fear for the deployed member's safety, reinforced by vivid and ubiquitous television coverage. On reunion, family roles and responsibilities again have to be redefined. Change requires adjustment, whether or not it is welcome, and adjustment induces stress (Holmes and Rahe 1967). Reunion may also create stress by bringing back issues of past unresolved problems.

ADVERSE HEALTH EFFECTS

The veterans reported that they suffered adjustment difficulties, rage attacks, difficulty sleeping, difficulty socialising, alcohol abuse, stress on families, divorce and suicide. While on counter-terrorist training SAS members were said to become aggressive and insensitive to the suffering of self and others.

In addition to the effects on families during the period of service, many also reported difficulties adjusting to civilian life after discharge, especially if they had mental or physical disabilities. The impact of retirement on well being is difficult to assess as it encompasses both positive and negative change and is dependent on the circumstances surrounding the retirement, the economic consequences and how individuals perceive it. Self-discipline and determination, two characteristics of SAS soldiers, appear to assist retired military men to start over again in civilian life (Berkey 1972). Successful adaptation is more likely if there is appropriate planning (Lo and Brown 1999, Moen 1996).

Several veterans reported that their options for employment had been restricted because of disabilities sustained during SAS training. One former veteran with degenerative injuries of the spine and various joints stated that "I was very worried that I wouldn't be able to provide for my family if my medical problems kept worsening". Another stated that "when soldiers are injured they are hidden away or posted out of the unit and left embittered that they have wasted their life for little or no reward". He also stated that, because of delays in receiving compensation "your wife has to return to work....and on top of this...is left to cope with a mentally and physically disabled partner".

DVA is currently piloting a Defence Transition Scheme (DTS), which commenced on 6 November 2003 in Townsville and will run for a minimum of 12 months. The DTS is available for all ADF personnel on a voluntary basis. The pilot involves the appointment of a DVA transition coordinator working with Lavarack Barracks and RAAF Base Townsville. The DTS coordinator is the link between the Defence discharge cell, community and employment services, and the Transition Management Service for medical dischargees. The co-ordinator will offer detailed advice and support in relation to compensation, superannuation, employment, financial and housing services. The co-ordinator will also manage access to the VVCS's new "Stepping Out" Program, which concentrates on lifestyle/emotional impacts during and following transition. This program is scheduled to commence in February 2004.

Chronic pain from disabilities was mentioned by several veterans as a factor which affected their families due to the resultant effects on mood and frustration levels. One

former soldier stated that "my family has suffered because of my chronic pain, bad moods, frustration and aggressive way of living". Other service related factors mentioned in the submissions which could have the potential to impact on relationships were poor sleep and inability to hear other people due to hearing loss.

Although there are no data on the effects of service on families of SAS members, there are some data available on the caseloads of social workers from the Defence Community Organisation, which provides mental health services to ADF personnel and their families. Of these cases, 57.3% were members and 37.3% were member's partners (Draft ADF Health status report 2002). The presenting problems were very broadly categorised, with the top two being family issues (50.6%) and member issues (17%). ADF members can also access mental health services from doctors, chaplains, the Vietnam Veterans' Counselling Service and the Defence Force Psychology Organisation.

Many of the behaviours and problems reported by SAS veterans are not unique to the military and it is important to assess whether or not military families are at increased risk compared to the civilian population. There are many problems with the available data which make it difficult to come to firm conclusions about the effect of military life on the family and on behaviour and lifestyle. There are no data on these issues among Special Forces in particular, so it is necessary to have regard to the literature on the military in general. Most of these data are only available on the US military, which has different characteristics to the Australian military. In addition, studies on military families are often done using clinical or self-selected populations.

Not all studies which compare civilian and military populations control for confounding due to differences in socio-economic status and different age/sex distributions between military and civilian populations. A US study comparing probability samples of military and civilian populations found that the military sample was younger, better educated and more likely to be male or black (Bray 1985). In the US, younger age and being black are characteristics which are associated with greater levels of substance use, although higher education is generally associated with lower levels of substance use.

EFFECTS ON RELATIONSHIPS

Some researchers have studied whether or not the families of armed services personnel are more susceptible to psychological disorders than their civilian counterparts (the "military family syndrome"). Although some initial research was suggestive (Lagrone 1978), this work was based on case records and subsequent studies have not supported this idea. Studies have not shown an increase in the prevalence of psychiatric symptoms in the families of US military personnel compared to the general population (Morrison 1981, Terr 1992, Fernandez-Pol 1988, Jensen 1991, Zeff et al 1997).

In families of members who have a diagnosed psychiatric condition, such as depression or PTSD, relationship distress is more likely. Riggs et al (1998) examined the quality of the intimate relationships of male US Vietnam veterans. Over 70% of PTSD veterans and their partners reported clinically significant levels of relationship difficulties compared to only 30% of the non-PTSD couples. Hiley-Young et al (1995) also found that participation in warzone violence in patients admitted to

hospital for PTSD predicted postmilitary violence to self, spouse and others. Veterans may use drugs of addiction or alcohol as self-medication for psychiatric problems (Mintz et al 1979), which can further compound relationship difficulties.

Separation is stressful for some military wives and children, as evidenced by their attendance at mental health services (Lagrone 1978, Blount et al 1992). Separations affect the family "system", that is, the individual family members, each with an established role, tied together by complex dependencies, interdependencies and balances (Breger 1984). A healthy flexible system is able to withstand the stress of repeated separations. Factors which might affect the ability of the family system to adapt and the likelihood of behavioural, emotional or mental health problems in wives or children can include:

- the remaining caretaker's perception of stress and psychological characteristics
- parent's and children's cognitive appraisal of the situation (the "meaning" of the separation and the level of fear for the serving member's safety).
- pre-existing child or parental psychopathology
- the status of the relationship between parents
- the relationship between the parents and children
- the number and type of non-military life stressors
- the age, sex and number of children
- military rank and socio-economic status
- the coping capacities and resources of the family

(Breger 1984, Gabel 1992, Blount 1992, Knapp and Newman 1993, Jensen et al 1990, Desivilya and Gal 1996).

It is difficult to differentiate the effect of these factors from the effect of separation. While most emotional and behavioural problems are likely to be mild and temporary, absences of greater length or frequency or under combat conditions are more likely to have persistent effects (Jensen et al 1992). There may be certain families which are vulnerable to dysfunction for these or other reasons and levels of vulnerability may change over time (Fernandez-Pol 1988, Jensen et al 1986).

Longitudinal studies are needed to determine which families are at "high risk" for dysfunction. Ensuring support in the form of social, legal and health services has been advocated but not well evaluated (Jensen et al 1986). Social support has been found to have a buffering effect on the stress of separation in military wives (Rosen 1988). It has been recommended that support for families should include facilitating communication with absent serving members, providing education on what feelings and problems to expect before, during and after a separation (Blount 1992) and providing both spouses with skills in problem-solving and conflict resolution (Desivilya and Gal 1996).

Divorce

Divorce can be an indicator of psychosocial dysfunction, but there are a paucity of data to show whether military families have a higher incidence of divorce than non-military families. If anything, the literature suggests that the incidence of divorce is lower in US military families (Williams 1976 in Jensen et al 1986, Morrison 1981, Lester 1993).

A study examining the social and behavioural effects of combat intensity on a random sample of the American Legion suggested that the level of combat intensity, not just service experience, should be taken into account when measuring psychosocial effects (Stellman, Stellman and Sommer 1988). In this cross-sectional study with self-reported outcomes, there were higher levels of divorce in South-East Asian veterans than non-South East Asian veterans (36.7% vs 28.4%) and men who had faced high levels of combat intensity were at significantly greater risk of divorce than non-South East Asia veterans. The odds ratio of ever being separated or divorced to ever being married when these groups were compared was 3.86 ($p < 0.001$). Stellman et al also found a significant and negative effect of medium and heavy combat compared to low combat on the marital well being and sexual satisfaction scales.

EFFECTS ON BEHAVIOUR AND LIFESTYLE

Interpersonal violence

There is a view that military families may be at risk of misplaced aggression from spouses who have returned from deployments (Werkman 1992 in Terr 1992). The limited empirical evidence slightly favours this contention, although further work must be done before definitive conclusions can be made. Recently there have been anecdotal reports in the press of violence to family members by returned servicemen in the US. However, the Expert Panel must consider the peer reviewed literature and at this stage there is no new evidence beyond what is presented here that the rates of interpersonal violence are different to that of the general population.

McCarroll et al (2000) studied the length of deployment and the risk of moderate or severe spousal aggression. Surveys were administered to a random sample of 26,835 deployed and non-deployed married active duty US Army men and women in the US between 1990 to 1994. Measurement of conflict was based on self-report. There were no significant increases in the risk of moderate aggression by deployment status or length of deployment. However, the probability of severe aggression was significantly greater for soldiers who had deployed in the past year compared to soldiers who had not deployed and the probability of aggression increased with length of deployment. The rates of severe aggression were 3.7% to 4.1% for no deployment and 5% for a deployment from 6 to 12 months.

Comparison of rates of spousal aggression in the military with civilian populations is difficult because the US Army only counts married people as spouse abuse victims, whereas as people who are co-habiting are often also included in civilian studies. Depending on whether the incidence of spousal aggression is more or less in co-habiting couples compared to married ones, the incidence of violence in military would tend to be under or overestimated. There is also lack of control for confounding by socio-economic status, which is a factor that does appear to affect the risk of family violence. Finally, there may be selection bias if Army recruits have pre-existing risk factors which are different to the general population.

Two national surveys of US civilians showed rates of self-reported spousal aggression of 3.0 to 4.6%, which are similar to the rates in the military reported by McCarroll (Straus and Gelles 1986). Heyman and Neidig (1999) compared only married, employed civilians with the US Army sample and showed significantly higher age and race-adjusted rates in the military (2.5% compared to 0.7% in civilians). Jensen et

al (1986) reviewed three studies on the incidence of child abuse in military families compared to civilian studies which indicated that the incidence of abuse was lower in military settings compared to surrounding civilian communities.

Suicide

Suicide rates among serving military personnel and veterans tend to be reported separately. In studies comparing death from suicide in serving military personnel or veterans of various conflicts and the general population, the "healthy soldier" effect needs to be considered. Those with pre-existing mental health problems are less likely to be selected into the military. In addition, being part of a cohesive group may protect against suicide, at least during the period of service (Wong et al 2001).

The available epidemiological evidence suggests that suicide rates in current members of the military are not elevated compared to the overall population (Sentell et al 1997, Hourani et al 1999, Wong et al 2001). While there was a slightly elevated standardised mortality ratio in the Australian Vietnam Veterans' Mortality Study (SMR 1.21, 95% CI 1.02-1.42), there was no elevation of suicide rates in US Vietnam veterans in comparison to non-Vietnam veterans (Breslin et al 1988). Retrospective cohort studies of mortality among UK (Macfarlane et al 2000) and US (Kang and Bullman 1996) Gulf War veterans have not shown an excess of deaths recorded as suicide. In the Australian Gulf War Veterans' Health Study the numbers were too small to calculate a relative risk for suicide.

Figures from the 2003 draft ADF Health Status Report are consistent with the US data. The average rate of suicide between 1997 and 2001 for male ADF personnel was 17.2 per 100,000 compared to 31.5 per 100,000 in the community. However, suicide is a leading cause of death in ADF personnel and in recognition of this a Suicide Prevention Initiative is being implemented as part of the new Mental Health Strategy (Draft ADF Health Status Report 2002). Some components of this initiative are the development of psychological autopsies, research into the risk factors and protective factors which pertain to the Defence Forces and promotion of opportunities to enhance resilience, problem solving, well-being and cohesion.

Analysis of risk factors for suicide suggests that there are some exposures, demographic characteristics and psychiatric disorders which increase the risk of suicide in certain vulnerable subsets of military and veteran populations. Recent research in military populations confirms that psychiatric disorders (especially major affective disorders), alcohol dependence and personality disorders are associated with an increased risk of suicide (Thompson et al 2002, Kausch and McCormick 2002, Waller et al 1999, O'Toole and Cantor 1995).

A review of suicide risk factors among veterans identified the following additional factors: male gender, older age, homelessness, unmarried status and availability of firearms (Lambert and Fowler 1997). O'Toole and Cantor (1995) examined suicide risk factors among Australian Vietnam era draftees. The factors that were associated with suicide were intelligence test score, postschool education, going AWOL and history of diagnosis and treatment of psychological problems. Service in Vietnam was not associated with suicide. To the extent that military experience increases the

likelihood of having one of these risk factors, then the risk of suicide will also be indirectly increased.

The risk factors listed in the Statement of Principle for suicide and attempted suicide are: being a prisoner of war, exposure to traumatic events or stressors, alcohol dependence, abuse of psychoactive substances, and suffering from schizophrenia, depression, a borderline personality disorder or PTSD.

Smoking, alcohol and drug consumption

It can be argued that there are many aspects of the military environment which are conducive to adopting smoking and/or drinking behaviours. These include a culture of drinking and smoking, desire for group acceptance, stress, cheapness and accessibility, lack of other forms of recreation, boredom, fear and family separation (Jensen et al 1986, Klevens et al 1995). These factors may increase on deployments and cause changes in smoking and alcohol patterns. The SASR is a highly trained, highly selected group which is very focussed on fitness and it is not clear that smoking or drinking rates in the military as a whole are generalisable to members of the SASR.

Against these influences are military policies or programs directed at reducing consumption of these substances, at least while on duty. In the US, all of the Services prohibit smoking on base except in designated smoking areas and offer smoking cessation programs to encourage smokers to quit (Kroutil et al 1994). In the Australian Army there is no formal policy on smoking, apart from the general Commonwealth wide ban on smoking within the workplace (Colonel Glenn Wells, personal communication). There are no cheap cigarettes available within Australia to the military, but cigarettes and alcohol can be obtained duty free on deployments. Alcohol obtained from the mess is cheaper than at bars and hotels because it is sold at cost price. In the last five years the annual health assessment has included questions about alcohol and cigarette consumption and any concerns are followed up.

A table summarising studies on the prevalence of smoking and alcohol consumption in military and veteran populations is available in Appendix J and the main findings are summarised below.

Smoking

Ex-smokers have reduced risk for smoking related outcomes compared to current smokers, but the rate and length of time over which the risk returns, if ever, to that of never smokers, depends on the amount smoked and the disease in question (McElduff et al 1998). Smokers also increase the risk of certain adverse health outcomes to their families through the effects of passive smoking.

Studies comparing smoking habits of civilian and military populations that have been appropriately standardised or that compare within the same age group show a higher prevalence of smoking in the military (Lewthaite and Graham 1992, Bray et al 1985, Ballweg and Li 1989). Two studies suggested that smoking is adopted quite quickly after recruitment (Schei and Sogaard 1994, Cronan and Conway 1988). The higher proportion of ex-smokers in studies of veterans also attests to higher rates of smoking in the military (Haddock et al 1994, Klevens et al 1995). Smoking prevalence has

declined over time in the civilian population and smoking trends in the military appear to mirror this (Kroutil et al 1994, Klevens et al 1995).

There is conflicting evidence as to whether smoking prevalence declines upon retirement from the military. One study found that smoking rates and alcohol use among military retirees and dependents were similar to those reported in surveys of the general senior population (Haddock et al 1994) but this study suffered from selection bias. One large study found that veterans are 1.9 times more likely to be current smokers than non-veterans (Klevens et al 1995) and another found that veterans were more likely to be heavy smokers (Fiegelman 1994). The prevalence of smoking in veterans may in part be related to age (Klevens et al 1995) and level of combat experience (Stellman et al 1988).

Alcohol

There were relatively few studies identified in relation to alcohol consumption patterns and most were not very recent, but the available literature suggests that drinking behaviour is more common in the military than in civilian life, although for the majority drinking levels are consistent with light to moderate social consumption. In two studies there was a higher proportion of heavy drinkers in young adults currently serving in the US military than in civilians (Bray et al 1985, Ballweg and Li 1989). One US study showed a higher proportion of heavy drinking in those older than 26 years (Bray et al 1985).

Accurate baseline data on ADF alcohol usage are not currently available, although the 2002 Draft ADF Health Status Report states that estimates from the last Health Status Report (2000) suggest that misuse of alcohol has been slightly higher among ADF members compared to the Australian community (17% in the ADF compared to 10-15%). An Alcohol Management Program has been developed to provide prevention strategies and programs for surveillance and research, education and management of alcohol misuse.

Stellman et al (1988) found that the average weekly consumption of alcohol in Vietnam veterans increased with increasing levels of combat, as did the odds ratio of ever having had a drinking problem. It is possible that there are different reasons for the heavy drinking observed in younger age groups of currently serving members and the heavier drinking observed in veterans who had experienced high levels of combat. The former group may represent a subset of young men who are characterised by high risk taking behaviours (Williams et al 2002). Among veterans, heavy drinking may be associated with psychological disturbance due to combat experience.

The factors listed in the Statement of Principles for alcohol dependence or alcohol abuse include: experiencing a severe stressor in the two years [one year BOP] before the clinical onset or worsening of alcohol dependence or abuse; and suffering from a psychiatric disorder at the time of clinical onset or worsening of alcohol dependence or abuse.

Other drugs

Studies of rates of nonmedical drug use have found that rates are generally comparable to civilians of a similar age group and era (Lanphier and Macauley 1982,

Needleman and Romberg 1989, Ritter et al 1985). One study showed that use of marijuana declined after entry into the military (Needleman and Romberg 1989). Marijuana and heroin are the only two drugs for which usage rates are remarkably higher among Vietnam veterans compared to non veterans (O'Donnell 1976 cited in Ritter et al 1985) and the availability of heroin in Vietnam may have been a factor promoting its uptake during military service (Robins 1993). As many as 45% of Army enlisted men reported trying narcotics and while in Vietnam as many as 20% felt that they were addicted. However, addiction did not persist for most veterans; in the first year after return only 5% of those who had been addicted in Vietnam were addicted in the US (Robins 1993). Most of those addicted recovered without treatment and only 6% became readdicted

The continuing use of drugs of dependence in a small subsection of veterans may reflect underlying psychopathology. Stellman et al (1988) found that men who had served in SE Asia were more likely to be regular users of sleeping pills or tranquillisers than those who had not served in SE Asia (6% vs 3% $p< 0.05$), but the use of amphetamines, uppers or marijuana was similar. The Australian Gulf War Veterans' health study found a slightly higher odds ratio of drug dependence/abuse than the non-deployed comparison group (OR 1.9, 95% CI 1.1-3.2).

Drug dependence can develop in patients who have been prescribed a course of opioid, sedative, hypnotic or anxiolytic medications for a medical or psychiatric condition, and this is recognised in the SoP for drug dependence and drug abuse.

REPATRIATION MEDICAL AUTHORITY'S STATEMENTS OF PRINCIPLES

The following Statements of Principles contain smoking, passive smoking, substance abuse or alcohol factors. The individual dose and induction period varies between different SoPs and readers are referred to the full SoP for doses and definitions.

Table 18 Statements of Principles concerning smoking, substance abuse and alcohol

STATEMENTS OF PRINCIPLES & INSTRUMENT NO.	**FACTORS**
Acquired Cataract 37 & 38/2001	smoking
Acute Myeloid Leukaemia 169 & 170/96	smoking
Acute Pancreatitis 45 & 46/97	alcohol
Adenocarcinoma Of The Kidney 87 & 88/2001	smoking
Analgesic Nephropathy 56 & 57/94	chronic analgesic abuse
Aortic Aneurysm 66 &67/98	smoking
Asthma 85 & 86/2001	smoking
Atherosclerotic Peripheral Vascular Disease 65 &66/2002	smoking
Atrial Fibrillation 19 & 20/2003	alcohol
Bipolar Disorder 128 & 129/96	alcohol, cocaine
Buerger's Disease 73&74/95	smoking
Cardiomyopathy 19 & 20/98	alcohol
Carotid Arterial Disease 9 &10/2003	smoking
Cerebrovascular Accident 52 & 53/99	smoking (for cerebral ischaemia only), alcohol
Chronic Bronchitis And Emphysema 73 & 74/97	smoking
Chronic Myeloid Leukaemia 15 &16/2003	smoking

Chronic Pancreatitis 57 & 58/2001	alcohol
Chronic Sinusitis 21 & 22/2003	smoking
Cirrhosis Of The Liver 35 & 36/98	alcohol
Cluster Headache Syndrome 69 & 70/99	alcohol
Colorectal Adenoma 62 & 63/2002	smoking; alcohol
Diabetes Mellitus 82 & 83/99	smoking
Drug Dependence Or Abuse 78 & 79/98	prescribed drugs of dependence
Epilepsy 79 & 80/96	alcohol
Gastro-Oesophageal Reflux Disease 52 & 53/2002	Smoking; alcohol
Gingivitis 3 & 4/2002	smoking
Goitre 21 &22/2000	smoking
Gout 11 & 12/2000; 43 & 44/2003	alcohol
Haemochromatosis 5 & 6/97	alcohol
Hepatitis B 41 &42/95	parenteral drug use
Hepatitis C 43 &44/95	parenteral drug use
Hepatitis D 45&46/95	parenteral drug use
Hypertension 35 & 36/2003	alcohol
Impotence 97 & 98/96	smoking, alcohol
Inflammatory Bowel Disease 21 & 22/2001	smoking
Intervertebral Disc Prolapse 130 7131/96	smoking
Ischaemic Heart Disease 53 & 54/2003	Smoking, passive smoking
Macular Degeneration 29 & 30/97	smoking
Malignant Neoplasm Of The Larynx 27 & 28/95	smoking; passive smoking; alcohol
Malignant Neoplasm Of The Lung 35 & 36/2001	smoking ; passive smoking (other than typical carcinoid tumour of the lung)
Melioidosis 27 & 28/2003	alcohol
MN Of The Bladder 23 &24/2000	smoking
MN Of The Breast 53 & 54/97	alcohol
MN Of The Cervix 41 & 42/97	smoking
MN Of The Colorectum 58 & 59/2002	smoking; alcohol
MN Of The Lip Epithelium 41 &42/2001	smoking
MN Of The Liver 171 & 172/96	Smoking; alcohol
MN Of The Nasopharynx 167 & 168/96	smoking
MN Of The Oesophagus 11& 12/9	smoking; alcohol
MN Of The Oral Cavity Or Hypopharynx 113 & 114/96	smoking; alcohol
MN Of The Pancreas 55 & 56/97	smoking
MN Of The Penis 340&341/95	smoking
MN Of The Renal Pelvis And Ureter 155 7 156/96	smoking
MN Of The Stomach 7 &8/2003	smoking
Motor Neuron Disease 65/2001	smoking
Non-Aneurysmal Aortic Atherosclerotic Disease 68 & 69/98	smoking
Osteoporosis 67 & 68/2002	smoking; alcohol
Peptic Ulcer Disease 21 & 22/99	smoking
Periodontitis 1 & 2/2002	smoking
Peripheral Neuropathy 79 & 80/2001; 13 & 14/2003	alcohol
Porphyria Cutanea Tarda 19 & 20/2001	alcohol
Psoriasis 56 & 57/2002	alcohol
Pulmonary Thromboembolism 3 & 4/2000	smoking
Renal Artery Atherosclerotic Disease 39/98 & 33/99	smoking
Secondary Parkinsonism 38 & 39/2002	alcohol
Seizures 81 & 82/96	alcohol; sudden withdrawal of a sedative drug
Sleep Apnoea 39 & 40/97	alcohol
Subarachnoid Haemorrhage 48 & 49/99	smoking; alcohol
Sudden Unexplained Death 99 & 100/96	alcohol
Suicide Or Attempted Suicide 71 & 72/96	alcohol