SOUTH AUSTRALIAN TASK FORCE ON MULTIPLE CHEMICAL SENSITIVITY

Responding To The Environmental Public Health Crisis Of Chemical Injury

● Postal Address: PO Box 3308 Port Adelaide 5015 ● Telephone (08) 8240 5084 ● Email: satfmcs@optusnet.com.au

Submission to the
Office of Chemical Safety /
National Industrial Chemicals Notification and Assessment Scheme
Scientific Review of
Multiple Chemical Sensitivity:
Identifying Key Research Needs
November 2008.
By
Peter Evans, RN (formerly) Grad Dip Soc Sc
Convenor
SA Task Force on MCS

ABOUT THE AUTHOR

Peter Evans is a former registered nurse and health counsellor who began experiencing symptoms of multiple chemical sensitivity (MCS) in 1984 following exposure to phenolic disinfectants and formaldehyde in the health care setting. He was diagnosed with chronic fatigue syndrome in 1990. In 1994 his chemical sensitivity and associated disability were acutely exacerbated by sick building syndrome while working in a newly constructed, tightly sealed building. Following a period of disabling illness requiring total chemical avoidance, he recovered sufficiently to return to working life. But in 2000, after being exposed to a combination of organophosphate pesticides used in the South Australian government's fruit fly eradication program, he became too chemically sensitive and permanently disabled to engage in paid employment. Subsequently his health declined further following sensitisation and severe reactivity to herbicides used by local governments in suburban weed control programs. Peter has a significant family history of MCS with all of his adult siblings disabled with the condition. In 2001 he became involved in the community response to MCS and established the SA Task Force on MCS. He was medically diagnosed with MCS in 2004. Peter is a community representative with the interagency MCS Reference Group convened by the SA Dept of Health.

COMMENT ON THE EXISTING TEXT

The Review of MCS by the Office of Chemical Safety and the National Industrial Chemicals Notification and Assessment Scheme is a welcome and necessary development. Australia has lagged well behind other developed nations in its response to this major and growing public health and disability access problem. Proper focus on MCS in Australia is well overdue.

Unfortunately the Review does not go nearly far enough to ensure appropriate action will be taken to address the public interest in MCS. Many of the observations, conclusions and recommendations in the Review were made over a decade ago by the US Interagency Workgroup on Multiple Chemical Sensitivity (1998). Many of the public comments in response to this US report reflect the same concerns that MCS community groups have already expressed, notably that the Review is biased towards vested

interests and psychological explanations of MCS (National Center for Environmental Health, Centers for Disease Control and Prevention, 2000).

Ten years on from the US Report, little has changed with respect to MCS and this Review now appears to be replicating the same mistakes. Importantly, there remains no coordinated research effort to investigate the cause/s or effectively treat the symptoms of MCS. People with MCS continue to be widely misunderstood and discriminated against, conspicuously due to the lack of public education on MCS and the maintenance of existing international standards in chemical regulation. Consequently, it is understandable that people with MCS might have developed a certain amount of cynicism with respect to the policy response to MCS.

It is hard to see how this Review might change this situation without significantly altering the content and general approach of the text, as well as identifying exactly who in Australia would be responsible for developing a coordinated MCS research program together with a national MCS prevention, medical management and disability access strategy, similar to that for HIV/AIDS and hepatitis C.

As two of the principal authorities in chemical regulation in Australia, NICNAS and OCS have a responsibility to ensure overall public safety and also to strongly assert the basic disability right of people living with MCS to avoid involuntary chemical exposures. The current draft of the Review has failed in both of these areas. The general tone of the document is at times sceptical and defensive and has not adequately considered the complex sociological influences that have driven medical and scientific opinions on MCS. The Review, along with much of the quoted research, confuses precedents and antecedents in the course of MCS illness resulting in false and misleading assumptions about its aetiology.

The Review should establish MCS as a high priority public health and disability access area. However, in its current form, it is likely to cause more harm than good by perpetuating the misinformation and discrimination surrounding MCS. The psychological theories in this Review that are often promoted by the chemical industry and its medical associates are neither sufficient nor necessary to explain MCS. There is more than enough evidence for the existence of MCS as a physiological illness and a serious public health problem to require a robust precautionary response. The Parliamentary Inquiry into MCS in South Australia (Social Development Committee, 2005) concluded that "MCS is very real and that many individuals experience considerable suffering, particularly in light of the lack of recognition surrounding this condition." The inquiry into MCS by the Danish Ministry of the Environment (2005) concluded that "Most international experts within the field agree, on the basis of epidemiological data, that MCS is a reality." Both these inquires have made recommendations for a regulatory response to MCS.

Despite these conclusions it is entirely questionable if chemical regulatory authorities are even capable of adequately responding to MCS without legislative changes that would most likely need to be achieved through sustained public protest action similar to that seen in Australia in the asbestos debacle. However, given the state of isolation experienced by many people with MCS, it is difficult to imagine how this scenario might occur. Meanwhile the industrial juggernaut moves unstoppably on, relatively indifferent to this emerging issue in environmental public health and human rights.

There has been little consideration given in the Review to the very real and considerable human suffering involved in MCS. Consequently, it cannot be said to be complete without reference to research data detailing the sociological impacts of MCS, together with comprehensive MCS disability access guidelines such as those available through the US National Institute of Building Sciences (http://ieq.nibs.org/).

1. Comment on "Executive Summary"

The experience of people living with MCS is that the condition can be extremely debilitating. It is not unusual for people with severe MCS to be confined to bed or to a wheel chair due to pain, fatigue and

many other symptoms. Referring to MCS as "quite debilitating" in the opening paragraphs of the Executive Summary does not do justice to the extreme suffering and disability of those people who experience severe forms of the illness. MCS is not a benign condition and can be life threatening in severe cases. It must be given equally serious weight to similarly debilitating public health issues such as untreated AIDS or end stage coronary artery disease, both of which can be compared to MCS in its severe form. The descriptive language used in the Review should reflect this fact.

It is acknowledged that the lack of consensus surrounding the definition of MCS has impeded recognition and research. The Review has identified the research need to establish agreed MCS diagnostic criteria. However, international attempts to do this over the last several decades have failed. The Review should therefore clarify exactly how agreed MCS diagnostic criteria might be established and promulgated in Australia.

In 2006 the US Chemical Injury Information Network conducted an international workshop to develop a new definition of MCS. The workshop also identified laboratory tests that were promising as diagnostic tools. Although not complete at this stage, this attempt to more clearly define MCS may be useful and could be referred to in the Review (see the Environmental Health Association of Nova Scotia article on these developments at http://www.environmentalhealth.ca/developing%20definition.htm).

The Review has claimed that symptoms of MCS cannot be distinguished from overlapping diagnoses such as chronic fatigue syndrome. However, people with CFS do not necessarily experience loss of tolerance to chemicals previously tolerated, which is a diagnostic criterion for MCS (Bartha, 1999). This critical diagnostic distinction should be noted by the Review. It is highly unlikely that a diagnostic laboratory marker will be developed for MCS in the near future. Interim acceptance of the 1999 clinical criteria for the diagnosis of MCS would greatly assist in overcoming the shortcomings of self-diagnosis referred to in the Review.

The Review has focussed on the need to identify the "underlying cause and triggers of MCS". But the discussion surrounding this subject has confused causes with mechanisms and focussed on the characteristics of MCS subjects rather than event-driven studies that identify the chemicals associated with MCS. The Review demonstrates bias and lacks real credibility due to its failure to examine evidence of the exogenous causes of MCS.

Determining "the relative contributions of toxicodynamic and psychogenic mechanisms in the process of the disorder through the use of appropriately blinded challenge tests" has been established as a priority task by the Review. However, the use of challenge studies has proven to be highly problematic when investigating MCS. Most of these studies are poorly designed and make psychologically biased conclusions based on inaccurate assumptions about MCS. The claim that "it is the smell or odour of a triggering agent, rather any of its pharmacological or toxicological properties *per se* that elicit MCS symptoms" is not consistent with the reported experience of people with MCS who are usually able to tolerate many smells except those associated with chemicals that trigger symptoms. It is also inconsistent in circumstances where the incitants are odourless or below the subject's odour threshold. Challenge studies need to take a far more sophisticated approach than previously. Given the complexities of MCS, any assumptions about a psychological aetiology based on challenge studies are premature.

Longitudinal studies have been recommended by others in the past and by this Review and probably represent one of the best approaches to the study of MCS subjects. However, very few longitudinal studies have been undertaken to date. It may be many years, even decades, before the mechanism or mechanisms underlying MCS are fully elucidated, particularly given the general lack of medical interest and scientific investigation surrounding the condition. It is not reasonable for people with MCS to be made to wait for this to occur before precautionary public health policy measures are adopted and the disability access rights of people with MCS are respected. Some comment in the Review on this dilemma

is necessary, particularly with respect to the inadequacies of the toxicological models currently used in chemical regulation.

The Review has correctly proposed medical education and training in MCS but has not adequately identified people with MCS themselves as key stakeholders in this process. There are already concerns within the MCS community regarding the results of the Review's clinical meeting of health care professionals held in Sydney, to which no MCS community representatives were invited. This kind of paternalistic approach to the provision of health care belongs firmly in the past. If the Review is to carry any credence within the MCS community itself, then it must promote the concept of shared responsibility amongst people with MCS and their treating clinicians. This is particularly important with respect to psychological care as people with MCS will undoubtedly reject treatment models which consider their illness to be psychosomatic. The Review should also identify exactly how general medical education on MCS might occur given that the subject is not currently covered in mainstream medical training.

With regard to MCS treatment/management strategies the review has taken an extremely conservative approach. There is a body of medical knowledge within clinical ecology and environmental medicine that can assist patients with MCS. However, medicine is historically resistant to new ideas and the existing prejudices of mainstream medicine will need to be addressed if these techniques are to be made readily available to patients with MCS. Any medical education program will need to address these issues which have impeded the medical management of people with MCS for many decades. Opposition to the recognition of MCS by vested medical interests needs detailed discussion in this Review.

A national population study to verify the prevalence of both medically and self-diagnosed MCS is a relatively simple project that would assist in quantifying the extent of the problem and providing a baseline for further studies. The Review should discuss possible standardised questions to assist in data comparisons and identify exactly which area of the Department of Health and Ageing might be responsible for conducting this national survey.

A national public education campaign on MCS is well overdue and would be a welcome development in Australia considering the current widespread ignorance. Although MCS is occasionally covered in the Australian media, the approach taken is usually simplistically sensational and rarely offers any in-depth analysis of the issues.

2. Comments on "UNDERSTANDING MULTIPLE CHEMICAL SENSITIVITY".

2.1 Comments on "WHAT IS MULTIPLE CHEMICAL SENSITIVITY?

The word "putative" with respect to a diagnosis of MCS should be removed as it carries an implication of scepticism and denial of MCS. MCS is being diagnosed by doctors around Australia and the world. The SA Inquiry into MCS found 0.9% of South Australians already medically diagnosed with MCS. This figure is highly likely to be an underestimation of the true incidence of the illness and some comment on this fact in the Review is warranted. In North America between 2% - 6% of Canadian and United States citizens have been medically diagnosed with MCS. The diagnosis is clearly being applied medically to large numbers of people; most often by highly qualified and clinically experienced doctors. The diagnostic label of MCS needs to be respected by the Review.

2.2 Comments on "WHAT ARE THE SYMPTOMS OF MCS?"

The Nova Scotia Environmental Health Centre, a state sponsored, purpose built facility for the care of people with environmental illness, has researched the prevalence of major symptoms (Joffres, 2001). The

study concluded that "The type and consistency of symptoms experienced after exposure to triggering substances may not fit a purely psychogenic theory."

2.3 Comments on "IS MCS RELATED TO OTHER DISORDERS?"

The Review refers to the overlap between MCS, CFS, FM and PTSD. However, there are numerous other medical conditions that are also associated with MCS. Lacour et al (2005) have identified over 40 diseases and disorders that may overlap with MCS, including HIV/AIDS. In the case of HIV, patients sometimes acquire chemical hypersensitivity following the commencement of antiviral medications after a prolonged period of deterioration of the immune system, usually in conjunction with one or more AIDS defining illnesses. Other people with HIV/AIDS report losing tolerance to chemicals previously tolerated following an identifiable exposure to MCS initiating agents such as solvents used in renovations. In such cases HIV clinicians do not consider their patients' chemical hypersensitivity to be psychological but rather a consequence of altered immune system function. Another possibility not usually considered is that antiviral medications themselves may have initiated MCS.

Apart from the syndromes listed in Table 2, the Review must acknowledge the wide variety of medical conditions that are risk factors for MCS. It also needs to recognise the clinical observation that people with various chronic illnesses are at much greater risk of chemical hypersensitivity than previously supposed.

2.4 Comments on "WHAT TRIGGERS THE SYMPTOMS OF MCS?"

The Review asks the question: "are there additional triggers identified in MCS?"

Ashford and Miller's seminal WHO prize winning text "Chemical Exposures Low Levels and High Stakes" (1998) has examined hundreds of research articles on MCS and related subjects. Ashford and Miller have identified pesticides, organic solvents, new building materials, petrochemicals and harsh cleaning agents as the most common MCS initiators. This observation has been consistently confirmed through international research.

Ashford and Miller's description of MCS as occurring in two stages – 1) initiation followed by 2) broadening of sensitivities - is an important distinction that requires further discussion in the Review as it is consistent with the experience of many people with MCS and helps to throw light on the exogenous causes of MCS.

The population study by Caress and Steinemann (2003) contains lists of common chemical agents reported to initiate MCS and also trigger symptoms in established MCS.

The Review states "research reports suggest that there is likely to be a psychogenic component in the aetiology of MCS". Much research suggesting a psychological aetiology for MCS confuses precedents with antecedents with respect to the psychological sequelae of the illness. Caress and Steinemann (2003) found that, while 37.7% of MCS subjects reported depression, anxiety or other emotional problems, only 1.4% reported a history of such problems prior to the onset of MCS. Depression and anxiety are common conditions in modern society. The finding that many people with chronic and disabling symptoms of MCS experience these conditions should not be surprising and is consistent with findings in many other chronic health conditions, particularly where alienating societal factors are involved. The researcher-based confusion between psychological precedents and antecedents needs elaboration in the Review.

In addition to the precedent/antecedent confusion much research positing a psychological aetiology for MCS ignores the many physiological abnormalities that have been repeatedly well documented, an

example of which is Ziem and McTamney's study "Profile of Patients with Chemical Injury and Sensitivity" (1997). This study is particularly important as it reflects the proposition that MCS may not in fact be a single disease entity with only one aetiological pathway. Ziem (2001) has also identified metabolic, biochemical, immunological, nutritional, and infective abnormalities in MCS. Further examples of study findings of physiological abnormalities in MCS such as MRI and nasal abnormalities, damaged detoxification pathways, and changes in EEG, SPECT and PET scans are cited by Watanabe et al. (2003). In a study adapted from Kreutzer and Neutra (1996), Ashford and Miller (1998) summarise laboratory testing in MCS subjects which found abnormalities in immunologic, respiratory, cardiovascular, neurological, psychological, behavioural, and social function.

The Review needs far more discussion of the physiological abnormalities associated with MCS.

2.5 Comments on "CAN MCS BE CLINICALLY DEFINED?"

As with some other medical conditions, MCS can be clinically defined without diagnostic laboratory markers, despite some disagreement on exactly how this should occur. The kind of taxonomical debate seen in MCS is relatively common in medical science but does not usually impede progress in the way that it has with MCS.

The real problem in defining MCS is not in identifying the inclusion criteria but rather in dealing with the vested interests and associated financial stakes impeding the recognition of MCS. If billions of dollars were not involved it is highly unlikely that the commonly accepted diagnostic criteria for MCS, such as those identified by Bartha et al (1999), would attract much attention. Objections to a clinical definition of MCS often come from industry associated sources, such as Ronald Gots, who is cited by the Review as opposing a clinical definition of MCS. Gots is infamously associated in the USA with the Environmental Sensitivities Research Institute (ESRI), a sophisticated front for the promotion of chemical industry interests and the denial of MCS as a legitimate medical condition, and whose board is entirely composed of industry and trade association interests (Ashford and Miller, 1998; McCampbell, 2001). In such circumstances it is unlikely that these industry sources would be satisfied with any definition of MCS as it clearly threatens their vested interests.

The Review appears to be politically naïve in its lack of ability to identify and comment on research and opinions that may be guided by vested commercial interests. The public health lessons of industry subterfuge in tobacco and asbestos must not be forgotten. The influence of industry's MCS disinformation campaign on medical, legal, research and governmental institutions (McCampbell, 2001) should not be underestimated and needs further discussion in the Review.

Entrenched but unproven opinions amongst some medical professionals have undoubtedly impeded advances in the definition and recognition of MCS. Objections to a clinical definition of MCS from medical associations, such as the cited American Academy of Allergy and Immunology, are often connected with partisan anti-MCS medical professionals who are philosophically opposed to the legitimate practice of clinical ecology in medicine and who frequently appear as defence witnesses in MCS related compensation claims. Similar circumstances exist in Australian medical associations such as the Australasian Society of Clinical Immunology and Allergy.

The review has cited calls for double blinded, placebo controlled challenge testing of MCS subjects. There are significant difficulties with such testing that must be addressed before meaningful data can be extracted from such trials. The vast majority of challenge studies to date have been poorly designed and show little understanding of the complexities of MCS. Some of the difficulties associated with challenge studies are:

- Confused subject inclusion criteria may actually be looking at populations of people who suffer from intolerance to fumes and smells or psychosomatic disorders rather than MCS.
- MCS itself may not be a single disease entity. Consequently investigations using simplistic challenge testing methods may not be very fruitful.
- Challenge testing relies on subjective clinical observations which may or may not be accurately recorded. Challenge studies looking at brain function, such as SPECT scans, are more likely to provide useful data.
- Subjects' sensitivity reactions to incitants may be delayed for hours or even days.
- Exposure to chemicals in people with MCS may result initially in a feeling of excitation and well being, notably with solvents, which is followed by more negative physical symptoms over time. An analogy here is the initial stimulus of drinking alcohol followed by the illness of a hangover. Such complex reactions would be hard to assess in short term studies.
- According to clinical ecology theory, MCS is characterised by a phenomenon known as masking whereby reactive symptoms are reduced in daily life due to continual exposure to chemicals. Subjects must first go through a period of detoxification and unmasking through chemical avoidance, preferably in an environmental medical unit, in order to clearly observe symptom triggering. Challenge studies performed to date have not adequately considered this possible complication.
- People with MCS usually experience fluctuations in the severity of their sensitivity reactions, which are often dependent on the type and/or duration of recent chemical exposures. Even though they may not experience obvious sensitivity reactions during challenge testing, they remain vulnerable to both acute high level and prolonged low level chemical exposures, most notably to MCS initiating agents such as solvents and pesticides.
- People with severe MCS would be unlikely to participate in any experimental challenge testing as they would not want to experience symptoms associated with chemical exposures. Consequently challenge testing to date is likely to be observing subjects with relatively mild MCS.
- Challenge testing on subjects with severe MCS might be judged as unethical if properly considered by ethics committees.
- The design of conventional challenge testing to date is highly flawed. Subjects are usually exposed to one or two chemical agents that may or may not trigger symptoms in the subjects' daily lives. MCS is similar to classical allergy in that different people react differently to a variety of incitants. Expecting all MCS subjects to react to one or two specific chemicals is like expecting everyone with an allergy of any type to react to bee venom or peanut protein simply because it is a potential allergen. Clearly in the case of allergy this approach is not realistic. An experiment to assess for the presence of allergy, if designed like the current challenge testing for MCS, which usually involve small numbers of subjects, might conclude that allergy is a psychological phenomenon because none of the subjects reacted to bee venom or peanuts. Similarly, current experimental challenge testing designs are not realistic with respect to MCS.

A 2008 review of MCS challenge studies by Goudsmit and Howes found "a number of methodological weaknesses which do not appear to have been given due consideration by the authors when interpreting the findings". The review claims that the role of psychological factors in the aetiology of MCS may have been over-stated (Goudsmit and Howes, 2008). Ashford and Miller (1998) have also been critical of the design and implementation of challenge tests and have cited vested interests and psychological bias as serious deficiencies in such tests. For example, Staudenmayer (1993), who is a psychologist and frequent defence witness in MCS cases and whose psychogenic views on MCS have been widely published, has not indicated the source of his study population. The referral source is important as MCS subjects who can clearly identify a specific initiating chemical exposure are much less likely to have premorbid psychiatric conditions that those subjects who cannot identify an initiating exposure. If drawn from Staudenmayer's own psychology practice, this study population may have shown a significant bias

towards premorbid psychiatric conditions. Comments on the shortcomings of current challenge study design and researcher bias need to be included in the Review.

An interesting Japanese challenge testing model by Saito et al (2005) is reported in the Goudsmit and Howes (2008) review. MCS subjects were tested in their daily lives for chemical sensitivity, with ambient air samples being taken when subjects experienced reactions. In the great majority of cases (11 out of 12 subjects) potentially reactive chemicals were identified in the air samples at the same time that sensitivity reactions were reported. Saito et al. concluded that "MCS patients do not have either somatic or psychologic symptoms under chemical-free conditions, and symptoms may be provoked only when exposed to chemicals." Reference to this study together with some comment on developing more appropriate challenge study designs is needed in the Review.

References in the Review to the 1996 Berlin workshop on MCS must stop spreading misinformation that the term idiopathic environmental intolerance (IEI) has been endorsed by the World Health Organisation. According to Ashford and Miller (1998) the Berlin workshop, which was organised with the assistance of the International Program on Chemical Safety (IPCS) and WHO, engaged in a highly biased process which ignored European Commission recommendations that both proponents and critics of MCS should be included in proceedings. The only non-government representatives invited to the workshop were all full time employees of BASF, Bayer, Monsanto and Coca Cola. The highly biased Ronald Gots of the Environmental Sensitivities Research Institute (see above) was invited to give the US perspective on MCS. The term IEI was proposed at an ad hoc meeting by industry representatives and other observers already involved in a major lawsuit surrounding wood preservatives associated with MCS. Subsequently, the paper in which the term IEI appeared was specifically embargoed for public distribution, review, abstraction, or quotation without the written permission of IPCS. A statement subsequently issued to workshop participants by WHO advised that: "A workshop report to WHO, with conclusions and recommendations, presents the opinions of the invited experts and does not necessarily represent the decision or the stated policy of WHO." It goes on to say that "with respect to 'MCS,' WHO has neither adopted nor endorsed a policy or scientific opinion" (WHO, 1996 in McCampbell, 2001). Despite the publication embargo, the paper containing the term IEI was published anonymously in the journal Regulatory Toxicology and Pharmacology, with an acknowledgment that the publication was made possible with a grant from Ronald Gots' Environmental Sensitivities Research Institute. The term IEI is a sophisticated and relatively successful attempt by the chemical industry to remove the term MCS from use.

This is just one example of the unethical lengths to which industry is prepared to go to squash any references to MCS. The false claims surrounding the term IEI continue to be repeated by vested interests and even, more recently, by government institutions. A summary of the circumstances surrounding the development of the term IEI can also be found in Ann McCampbell's publication "MCS Under Siege" (2001). McCampbell, a medical doctor who chairs the MCS Task Force of New Mexico, rightly states that "the use of the term IEI is like a tracer dye that immediately alerts the reader, patient, or constituent that the person or organization using the term is biased against MCS."

An account of the Berlin workshop in an inquiry into MCS by the Danish Ministry of the Environment (2005) concluded that: "A final document from the workshop has never been published, because the participants could not agree on the conclusions. 80% could not support the main conclusions."

The origin and status of the term IEI should be accurately explained in the Review and the unethical and inaccurate claims that IEI is endorsed by WHO should cease.

As confirmed by the Review the 1999 consensus criteria (Bartha, 1999) are commonly used internationally and have successfully been used in Australia. These criteria have proven to be useful in

both the research and clinical settings. In the absence of established alternatives, they should be promoted more strongly by this Review.

2.6 Comments on "DOES MCS HAVE A DISEASE CLASSIFICATION?"

The opening line in this section that "MCS is not recognised as a classified disease identity in any country in the world" is Orwellian double-speak at its best. It is entirely baffling how this claim can be made when MCS is indexed in the German version of the World Health Organisation's "International Statistical *Classification* of Diseases and Related Health Problems (ICD-10-SGB-V)" (italics are the author's). Significantly, MCS is classified physiologically as T78.4 under allergy in Section T15 – T98, which is allocated to poisoning and certain other consequences of external sources. This physiological classification should be noted in the Review. Correspondence from the German Institute of Medical Documentation and Information (DIMDI) to Sylvia Muller, President of the CNS-Chemical Sensitivity Network, Germany, confirming the classification of MCS, together with an English translation of the relevant German text of the ICD-10-SGB-V is attached to this submission. This classification allows documentation of an MCS diagnosis in the hospital setting in Germany.

According to the World Health Organisation:

> *The ICD is the international standard diagnostic classification for all general epidemiological, many health management purposes and clinical use. These include the analysis of the general health situation of population groups and monitoring of the incidence and prevalence of diseases and other health problems in relation to other variables such as the characteristics and circumstances of the individuals affected, reimbursement, resource allocation, quality and guidelines.*
> *It is used to classify diseases and other health problems recorded on many types of health and vital records including death certificates and health records. In addition to enabling the storage and retrieval of diagnostic information for clinical, epidemiological and quality purposes, these records also provide the basis for the compilation of national mortality and morbidity statistics by WHO Member States.*
> (WHO, http://www.who.int/classifications/icd/en/)

Personal communications between OCS/NICNAS and DIMDI are not sufficient to deny validity of the ICD-10-SGB-V classification of MCS. This is a clear example of the persistent defensiveness and denial of MCS that is evident in governmental institutions and amongst regulatory authorities. The Review's distorted claims denying MCS as a classified disease entity are neither accurate nor in the public health interest and should be withdrawn. If the objection to the classification of MCS as a disease in the ICD-SGB-V is being made on the basis of the rarefied distinction between a disease and an illness then evidence for this should be offered.

The Review has commented on the National Centre for Classification in Health's refusal of community-based proposals to index MCS in the Australian version of the ICD-10 (ICD-10-AM). However, the Review has made no comment on the internal machinations that led to this refusal. The SA Task Force on MCS was one of the community groups that submitted applications to NCCH for the indexing of MCS. According to personal communications with NCCH staff, after consideration of the submissions and further discussion with other stakeholders, NCCH was initially inclined to index MCS but first sought advice from the Australasian Society of Clinical Immunology and Allergy (ASCIA). ASCIA rejected the proposal on the basis of the justifications listed on Page 16 of the Review, which are clearly open to points of debate such as:

- There are numerous clinical and laboratory markers indicative of pathophysiology in MCS (Ziem and McTamney, 1997; Ziem, 2001, Ashford and Miller, 1998, Watanabe, 2003a, etc).
- There are consensus diagnostic criteria for MCS (Bartha et al., 1999).
- The use of these criteria can enable an acceptable delineation between subjects with MCS and those with intolerance to smells and fumes (McKeown-Eyssen 2001).
- While there is crossover in the symptoms of MCS, CFS and FM, the latter two conditions do not always exhibit loss of tolerance to low levels of chemicals previously tolerated, which is a diagnostic marker in MCS (Bartha et al., 1999).

The potential benefits of the inclusion of MCS in the ICD-10-AM are many. Unfortunately ASCIA's biased claims on MCS appear to have been accepted unquestioningly by NCCH.

ASCIA is a medical association that is supported financially by the pharmaceutical and pesticide industry, for example their pharmaceutical sponsor AstraZeneca also produces the herbicide paraquat (Akha Heritage Foundation, 2008). The pharmaceutical industry's efforts to suppress MCS, together with its links to pesticide manufacture, have been previously documented (McCampbell, 2001). ASCIA is well known to the MCS community as a partisan organisation that is pedantically opposed to the recognition of MCS and the legitimate medical practice of clinical ecology in environmental illness (ASCIA, 2007). Their principal protagonist in these matters is Dr Robert Loblay, who routinely appears as a defence witness in MCS compensation claims. His unreferenced and highly debateable submission to the SA Inquiry into MCS on behalf of ASCIA is attached (Loblay, 2004) but appears to have been recently removed from ASCIA's website. Recent correspondence from 10 MCS-related community groups to ASCIA questioning its role in MCS, including with respect to reasonable disability access accommodations, has not received a response. Copies of this correspondence have been sent to OCS and the federal Minister for Health.

ASCIA's negative advice to NCCH with regard to the indexing of MCS in the ICD-10-AM is a good example of the subtle yet highly influential role of vested interests in the MCS debate. As mentioned above, the Review could benefit from a more candid discussion of industry's role in MCS, particularly given that medical services in Australia are largely based on a business model. The Review should publicly clarify ASCIA's role in NCCH's refusal to index MCS. Given its close associations with vested interests, ASCIA cannot ethically be allowed to be the final arbiter on the recognition of MCS in Australia. The Review should recommend that the Department of Health and Ageing approach NCCH with the view to opening unbiased discussions on the indexing of MCS in the ICD-10-AM following the German model. This indexing would assist in the collection of vital data on MCS in Australia.

2.7 Comments on "Do INDIVIDUALS WITH MCS SHARE COMMON CHEMICAL EXPOSURES?"

People with MCS are often able to identify a particular chemical exposure that initiated the onset of their loss of tolerance to chemicals previously tolerated and associated symptoms. Hileman (1991, cited in Ashford and Miller, 1998) has reported that some people with early onset MCS have recovered from the condition provided they were given advice to avoid initiating chemical exposures before chronic symptoms became well established. This suggests that MCS is a preventable condition. Given the overlap between MCS, CFS and FM, people with the latter conditions would be well advised to avoid chemical exposures that potentially might initiate MCS. There are significant public health implications to the possibility that MCS is preventable that merit further discussion in the Review.

The Review's claim that "Currently, there are no epidemiological data that link MCS subjects or those who may be susceptible to MCS with particular chemical exposures or lifestyles" is incorrect. Although epidemiological studies into MCS are relatively limited there are certainly population and clinical data

which throw light on the origins of MCS. The population study by Caress and Steinemann (2003) identifies pesticides, solvents, building materials, and petroleum products as initiating or causing MCS. This study also identifies chemicals that commonly trigger symptoms once MCS has been established. A study by Ross (1997) from the Environmental Health Center, Dallas, Texas, found that exposure to renovations at work or at home resulted in symptoms of MCS in 30% of subjects while pesticides accounted for 24%. A Japanese study by Itoyama et al., (2006), of Yokohama National University, reported that new constructions and renovations in homes and buildings accounted for the most common causal onset in cases of MCS (41.7%), with other causes such as pesticides, fungicides, and termite treatment (16.2%), workplace chemicals (9.0%) and outdoor air pollution and exhaust gas (5.4%) accounting for other causes. Glutaraldehyde is also reported to cause MCS (Ziem and McTamney, 1997). Ashford (1999) identifies pesticides and solvents as being consistently reported as causing MCS across North America and Europe and highlights the need for better medical documentation with respect to MCS initiating chemical exposures.

Ashford and Miller (1998) discuss an exposure-driven study by Miller and Mitzel (1995) which hypothesises that, as pesticides are usually more toxic than solvents, MCS subjects exposed to pesticides should experience more severe symptoms than those exposed to solvents in renovating. Conversely, if MCS is a psychological phenomenon, symptom severity should be similar in both groups. Notably the pesticide exposed group experienced higher levels of symptom severity that the renovating group. The authors concluded that MCS has a biological basis with a distinct yet undefined pathophysiology. Other similar epidemiological studies reported by Ashford and Miller (1998, Pages 211-218) are worthy of evaluation by this Review.

The absence in the Review of comment on available epidemiological studies into the event-driven causes of MCS indicates a strong bias against these data that appears to be problematic in regulatory institutions.

The Review reports on a study by Henneberger (1995) where “54% of the non-MCS patients worked in industries considered to have a greater potential for hazardous chemical exposures than other occupational settings. In contrast, only 26% of the MCS patients were employed in the more hazardous industries.” This observation can be explained by the fact that people who develop chronic symptoms associated with occupational chemical exposures tend to leave those occupations where chemicals are involved. This type worker migration is a commonly documented occurrence of in the occupational setting (Ashford and Miller 1998).

The Review advises that the Australian Safety and Compensation Council have developed the Australian Hazard Assessment (AHEAD) database for workplace chemical exposures. It will be interesting to follow this development as workers who develop MCS following occupational chemical exposures are invariably denied proper recognition and compensation for their injuries. Often these workers are severely disabled and compromised in their ability to work. Some may never work again. Industry has a long history of opposing proper compensation for injuries and diseases acquired by workers in the occupational setting. Proper compensation for chemical injury is a necessary cornerstone of public policy to address MCS (Ashford and Miller, 1998).

3 Comments on “WHAT CAUSES MULTIPLE CHEMICAL SENSITIVTY?”

The Review concentrates on proposed physiological or psychological mechanisms in MCS but does not undertake a thorough examination of exogenous environmental causes. The failure to consider existing epidemiological data has already been raised.

The Review appears to have confused proposed mechanisms with causation in MCS. In his article “Low-level chemical sensitivity: implications for research and social policy”, Ashord (1999) comments on the illogical approach that attends much of the scientific literature and lay comment on MCS. He then

attempts to clarify a more logical approach to the problem that underscores the value of focussing on event-driven studies, rather than attempting to characterise MCS subjects themselves with their varied and complex exposure histories. Ashford states:

> *"What is disappointing in much of the literature is the continuing failure to distinguish causes and symptoms of the condition and the unjustified drawing of conclusions from successes or failures of possible interventions".*

The Review would benefit from a discussion of the practical insights and potential solutions that are brought to the study of MCS by Ashford's article, which includes advice on clinical study design, the application of the precautionary principle to prevent chemical injury, and compassionate disability access accommodation in social policy.

It is also worth noting Winder's (2002) comments:

> *"With regard to the mechanisms of low dose toxicology, and more specifically, MCS, a range of models has been proposed to explain these phenomena, including the immunological, neurological, toxicological and sociological models. None work adequately in isolation, and the medical or scientific explanation of polysymtomalogy is yet to be established, although working definitions and a diagnostic label (MCS) have been defined.*
> *However, when low level chemical effects occur, the question that should be answered is not "does this effect correspond with identifiable medical conditions or pathological correlates" or "why does no one else seem to be affected by what do not appear to be high levels of exposure?" but more "would the symptoms have occurred if the person had not been exposed?" Subjects with the chemical exposures that precipitate symptoms of MCS suffer from a syndrome of disability from which they may never recover from adequately and .because of a temporal relationship between exposure and effect, are legitimate cases to consider as being chemically associated"*

Based on information from investigations across nine European countries a 2005 report into MCS from the Danish Ministry for the Environment (2005) identified the following as initiating MCS.

Amalgam / Mercury
Anaesthesia gasses
Carpets and glue
Diesel exhaust
Formaldehyde
Hair-care chemicals
Indoor climate
Degreasing agents
Methylmethacrylate
New, renovated buildings
Organic solvents
Paint, lacquers
Pentachlorophenol, etc.
Pesticides
Drugs
Printing materials
Stress – psychosocial factors

The Danish report notes that solvents and pesticides are most frequently cited as MCS initiators and added that the basis for notifications that stress and psychosocial factors can elicit MCS is unclear (Danish Ministry of the Environment, 2005)

Pall (not dated) has identified seven classes of chemicals reported to initiate MCS, all of which stimulate NMDA activity consistent with the NO/ONOO-cycle aetiological model for MCS.

An examination of the endogenous causes of MCS by this Review would be a welcome change from the history of regulatory indifference to these data demonstrated in the past. Whatever the underlying mechanisms of MCS might be, the Review lacks credibility due to its failure to adequately discuss the relationship between causative chemical exposures and the onset of symptoms of MCS. There are certainly more than sufficient published data to enable this discussion.

3.1.1 Comments on "Immunological Dysregulation".

There are clinical data indicating that CFS subjects experience Th1/Th2 immunological imbalance (Cheney, 2000) and this is anecdotally reported to be true in MCS. The Review could benefit from a literature search on this topic. It is certainly the case that some chemical exposures, such as the controversial bisphenol-A can result in alterations to Th1/Th2 function (Shin Yoshino 2003).

3.1.2 Comments on "Respiratory disorder/neurogenic inflammation".

The Review reports on Staudenmayer et al.'s (1993) study where participants "did not show a reliable response pattern". However, the US Interagency Workshop on Multiple Chemical Sensitivity (1998) in Section III, Theories of Causation and Mechanisms, found that "the results of this study may have been influenced by the choice of placebos used in the experiment, the use of masking, and the outcome measures that were used." These factors need to be included in the Review. Researcher and subject selection bias have been raised previously with regard to this study.

The Review reports on odour perception studies which found odour thresholds in MCS subjects unchanged compared to controls (Doty, 1994; Hummel, 1996; Dalton and Hummel, 2000). This finding should not be surprising if olfactory dysfunction plays no causative role in MCS.

There is physiological evidence of increased neurogenic inflammation in MCS that has not been reported in the Review but should be. A study by Kimata (2004) found "Plasma levels of substance P, vasoactive intestinal peptide and nerve growth factor, but not histamine, were elevated in patients with self-reported multiple chemical sensitivity (sMCS). Exposure to volatile organic compounds (VOC) increased plasma levels of all parameters in these patients, while it had no effect in normal subjects or patients with atopic eczema/dermatitis syndrome (AEDS). Exposure to VOC also enhanced skin wheal responses induced by histamine in patients with sMCS, while it failed to do so in normal or AEDS subjects. These results indicate that exposure to VOC may enhance neurogenic inflammation with concomitant enhancement of histamine-induced responses."

Potential deficiencies in challenge study design and researcher bias have been raised previously with regard to any conclusions on the psychological characteristics of MCS subjects.

3.1.3 Comments on "Limbic kindling/neural sensitisation."

The Review has reported on early studies of neurological function that generally failed to identify abnormalities in people with MCS, with a more recent study finding decreased activation of the olfactory centre. However, alterations in brain imaging are confirmed.

In a study of fibromyalgia subjects, Kwiatek et al, (2000) found reduced blood flow in the thalamus and, newly, the pontine tegmentum. Abnormalities were identified by state of the art SPECT scan and computer analysis. Abnormal changes are statistically significant but represent less than 3% difference from controls. The author has speculated that these findings may be diagnostic of FM. The identified abnormalities are located adjacent to the olfactory centre and similar SPECT scan examination of brain function in MCS subjects may be useful.

3.1.4 Comments on "Elevated nitric oxide, peroxynitrite and NMDA receptor activity".

Martin Pall's NO/ONOO cycle theory of MCS has received significant international attention and merits further investigation. Prof Pall has advised of some inaccuracies and the need for corrections in the current draft of the Review. The NO/ONOO cycle theory offers a resolution to many of the conundrums that have surrounded MCS, including the numerous types of chemicals reported to initiate MCS, the phenomenon of masking, the triggering of symptoms by odourless chemicals, the wide variation in symptoms and symptom severity between individuals with MCS, the acute sensitivity to low dose exposures, and the multi-organ presentation of symptoms. The model also confirms highly plausible reasons for the need for chemical avoidance in the treatment of MCS and strongly refutes psychogenic theories in MCS.

3.1.5 Comments on "Toxicant-induced loss of tolerance (TILT)".

The TILT model suggesting that MCS represents an entirely new category of diseases related to chemical exposures is consistent with the variable multi-symptom presentation between MCS subjects. As mentioned earlier, Ashford and Miller's observation of MCS as a two stage phenomenon of initiation and triggering is also consistent with the experience of people with MCS, as is masking. These phenomena merit further discussion in the Review.

3.1.6 Comments on "Behavioural conditioning."

Individuals with MCS frequently report marked variations over time in the severity of their chemical sensitivity reactions, often based on the type and duration of recent chemical exposures, particularly with MCS initiating agents – e.g. solvents, pesticides, petrochemicals, and harsh cleaning agents. Given this experience it may be possible that some individuals with MCS might develop a form of behavioural conditioning with respect to some chemical exposures. However, this experience is certainly not universal in all MCS subjects. The possibility that behavioural conditioning might exist in some MCS subjects does not mean that MCS of itself is a conditioned response. Over time, most people with MCS do recover some chemical tolerance following the acute initiating exposure, particularly if they avoid symptom triggers. However, they usually retain a permanent vulnerability to MCS initiators and subsequent further loss of tolerance.

The popular saying "once bitten, twice shy" is probably the most apt explanation for avoidance strategies amongst people with MCS who have recovered some tolerance for chemical exposures but nevertheless continue to maintain their avoidance strategies. This is not some form of unconscious conditioning but rather a deliberate, sensible and pragmatic adaptation to the problem of living with MCS.

Although some medical conditions can induce hyperventilation, the theory that it may play a role in MCS is fanciful and unlikely to be confirmed by research. According to Watanabe (2003b); "There are currently no data on the association of hyperventilation with MCS/IEI symptoms." This observation should be included in the Review.

As noted in the Review, behavioural conditioning does not explain the wide variety of symptoms associated with MCS. It should also be noted that behavioural conditioning does not explain the wealth of data showing physiological abnormalities in MCS subjects.

3.1.7 Comments on "Psychiatric disorders."

The Review rightly questions whether psychiatric manifestations in MCS are a *cause, effect, predisposing factor,* or *co-morbid* occurrence. The experience of people who actually live with MCS is that the illness is most definitely not psychogenic but is caused by common chemical exposures. Far more credence needs to be given to this experience if the conundrums of MCS are to be solved. Most notably, psychiatric explanations of MCS ignore the multiple physiological abnormalities that are evident in people with MCS.

Despite wide variations in symptoms amongst different individuals with MCS, overall symptom presentation is remarkably consistent within the broad patient group. The Review emphasizes concepts such as belief systems, somatisation, psycho-physiological stress and anxiety responses, to present a psychogenic aetiology for MCS. But psychological theories cannot explain the complex symptomatology, the diversity of the chemicals that result in physiological toxicological effects in MCS, the exquisite sensitivity in MCS subjects, or the reactions to incitants that are odourless or below the subject's odour threshold. Nor can they account for the genetic variations seen in MCS, notably the reduced ability to detoxify chemicals and heavy metals. While there are undoubtedly neuropsychological sequelae to MCS, a purely psychogenic aetiology should be rejected as a primary cause of the illness. More discussion of why a psychogenic aetiology is not appropriate for MCS should be included in the Review.

For very practical reasons, it is vital that any psychiatric bias is removed from the Review. While this bias exists, people with MCS risk being taken inappropriately into psychiatric care, at times forcibly, where they are denied access to proper medical investigations and interventions which may bring them some relative health and independence. Also, psychosomatic assumptions in MCS open the real possibility that physical ailments unrelated to MCS will go undiagnosed and untreated.

The Review has cited studies that found the majority of people with MCS have a pre-morbid psychiatric history. However, in a clear example of psychiatric bias, it has not cited studies where the opposite has been found. For example, the population study by Caress and Steinemann (2003) concluded that "Only 1.4% [of MCS subjects] had a history of prior emotional problems, but 37.7% developed these problems after the physical symptoms emerged. This suggests that MCS has a physiologic and not a psychologic etiology." This study is significant in that it represents a randomly selected sample, rather than one generated from clinical practices where selection bias towards a pre-morbid psychiatric history may have been a feature. In the interests of an unbiased Review, Caress and Steinemann's finding that the vast majority of MCS subjects had no pre-morbid psychiatric history should be included. A thorough reading of the literature will reveal other studies that make similar findings.

There are numerous publications which do not concur with MCS being a psychogenic illness. For example:

- Bartha et al. (1999) recommended that MCS be formally recognized on the basis of its high prevalence as a medically diagnosed condition together with the 1994 consensus of the American Lung Association, American Medical Association, U.S. Environmental Protection Agency, and the U.S. Consumer Product Safety Commission that "complaints [of MCS] should not be dismissed as psychogenic, and a thorough workup is essential.
- Miller and Mitzel (1995) found the features of MCS subjects are inconsistent with a somatoform disorder. Although both conditions share multi-system complaints, the MCS group has far more severe cognitive dysfunction than the somatoform group, where this feature may not appear at all.

Also, the onset of somatoform disorders almost always begins before the age of thirty, whereas MCS most commonly occurs after that age.

- In their review of challenge studies Goudsmit and Howe (2008) concluded: "In light of the various shortcomings described above, we concluded that the findings of the various studies should be interpreted with caution and, given that the theory attributing MCS to expectation, fear and conditioning is largely based on generalization and speculation, we consider it to be simplistic and unconvincing."
- The real life challenge study by Saito et al. (2005) concluded that symptoms of MCS were more likely to be caused by chemical exposures than depression, anxiety, or somatoform disorders.
- Ashford (1999) has pointed out that the confused inability to distinguish between causes and symptoms in MCS has led to inappropriate psychological interventions rather than advice to avoid future exposures as a treatment modality. He concluded that "Inasmuch as great uncertainty continues to characterize this condition, these views are premature and perhaps even harmful to the patient."

Many of the psychiatric assumptions made about MCS are incapable of proof. Psychiatry is an uncertain medical practice which lacks objective correlates. With perhaps the exception of behavioural science, it can be questioned whether psychiatric theory is truly a scientific discipline at all, or more aptly a belief system. Psychological theories are not sufficient to explain MCS. Winder (2002) advises that the pathological changes caused by chemical injury "should not be ignored in the absence of measurable pathological changes, injury or disease."

Given these factors, a far more cautious and precautionary approach to psychiatric explanations of MCS should be adopted by the Review.

Ashford and Miller (1998) have cited a proposed cholinergic model for depression by Dilsaver (1986) where subjects with depression have a hypersensitivity to acetylcholine, the same neurotransmitter affected by many pesticides. Pesticide exposure has been found to be associated with depression (Beseler, 2008). It may be that people with depression are at higher risk of developing MCS. Given the high incidence of depression in modern society this proposal has significant public health implications for more stringent pesticide regulation. A growing number of jurisdictions internationally have adopted reforms to reduce non-essential pesticide use in the environmental public health interest (Kassirer et al, 2004; Halifax Regional Municipality, 2000, European Parliament, 2007). Australia has yet to embrace similar reforms.

There is an increasing understanding of the chronic neuropsychological effects of chemicals implicated in MCS, particularly solvents and pesticides. The symptoms of chronic solvent exposure are remarkably similar to MCS. Ziem and McTamney (1997) have described multiple physiological and neuropsychological abnormalities that are consistent with solvent exposure in patients with chemical sensitivity. A study of MCS subjects by Maschewsky of Hamburg University of Applied Sciences (2002) found that "MCS subjects mostly did not have unusual psychosocial stress, but an unfavourable work environment, characterized by indoor-air pollution and/or chemical exposure. Neurotoxic exposures (mainly solvents) prevailed." Davies et al. (2000) have identified chronic neurobehavioural deficits in around 10% of workers regularly exposed to organophosphates, where depression and suicidal ideation are key features of chronic organophosphate induced neuropsychiatric disorder (COPIND). Again, the presentation of COPIND, for which the diagnostic criteria appear below, is remarkably similar to MCS.

Diagnostic criteria for COPIND.

1. Repeated exposure to organophosphates
2. At least four of the following:

    1. personality change and destabilisation of mood
    2. impairment of concentration and memory
    3. impaired exercise tolerance
    4. reduced tolerance to alcohol
    5. heightened sensitivity to organo- phosphates.
3. At least three of the following:
    1. exacerbation of ‘dippers' flu’
    2. impulsive suicidal thinking
    3. language disorder
    4. heightened sense of smell
    5. deterioration of handwriting

Dunstan and Phil (2001) have commented on organophosphate exposure syndromes including 1) the intermediate syndrome following OP poisoning, 2) the organophosphate induced delayed neuropathy, and 3) the chronic organophosphate induced neuropsychiatric disorder (COPIND). Symptoms of organophosphate poisoning include depression, anxiety and irritability.

Apart from being associated with chronic illness and disability, the neuropsychological manifestations of MCS are also consistent with those of chemical brain injury. Similarities between the symptoms of MCS and those resulting from acute or chronic exposure to pesticides and solvents should be noted in the Review.

Winder (2002) has proposed that these symptoms, when considered as being on the extreme end of an extended bell curve within a sufficiently large sample population, can be understood without requiring a shift in the conventional dose-response toxicological paradigm. According to a submission to the Review from Professor Martin Pall, this dose-response hypersensitivity in MCS is consistent with the findings of “Fiedler and Kipen (Ann NY Acad Sci 2001; 933,24) which show in Figs 4 and 5 of their study, fairly reproducible dose-response studies in MCS patients that are substantially left-shift as compared with those of normal controls, suggesting these patients are on the order of 1000 times more sensitive than normals.”

The suggestion that MCS is somehow iatrogenic confuses precedents with antecedents. There are simply too many people who have developed MCS independently from any medical or even media knowledge of the illness to give any credence to iatrogenic theories. In trying to explain their unusual and debilitating experience with chemical exposures people with MCS seek medical explanations. In general, the theories proposed by clinical ecology or environmental medicine are those that most closely fit the experience of actually living with MCS. They are also the medical modalities most likely to bring some health benefits. Consequently people with MCS may adopt an understanding of environmental medicine based on their own experience. It is absurd to suggest that these people are somehow being harmed mentally or conditioned psychologically by practitioners of environmental medicine. These claims are usually promoted by vested interests in industry and by allergists and immunologist who are philosophically opposed to the practice of environmental medicine. Such iatrogenic claims cannot be taken seriously, have no supporting research data, and should be removed from the Review.

Osterberg’s (2006) assumptions surrounding the presence of anxiety in MCS subjects confuse the causes of MCS with symptoms and subject characteristics. Numerous personal characteristics have been identified in MCS subjects. Bell (1994) found they had higher scores than controls on standardized tests for shyness. This does not mean that these characteristics are causative of MCS or that MCS is a psychological illness. People with heart disease have also been shown to be more likely to have certain personality traits but this does not mean that heart disease is a psychological illness. The relationship

between cause, effect and correlate has not been sufficiently established to warrant psychogenic conclusions on these data and the Review should highlight this fact more strongly.

The Review has focussed on proposed psychological mechanisms in MCS with an unspoken assumption that somehow a psychological basis for MCS makes the problem less real. But whatever the aetiology of MCS, the considerable pain and suffering caused by chemical exposures to people with MCS is real and disabling and there is no cure, psychological or otherwise. Rather than the theoretical approach presented to date, this Review needs to make a far more pragmatic analysis on what is actually going to be done to address the problem, particularly from the regulatory and disability access perspectives.

3.1.8. Comments on "MCS as a 'belief system.'"

The proposal that MCS is some form of belief system does not stand up to scrutiny. As noted above there are just too many people who have developed MCS without any prior knowledge of the condition for such a proposal to have any credence. There is evidence to support this position that is outlined by Ashford and Miller (1998) in a study by Davidoff and Key (1996) in which three subject groups of twenty people with MCS were matched against controls. The first group attributed their illness to solvents, the second to pesticides and the third to renovations. In addition, the study also looked at a fourth group of ten individuals exposed to chlorine dioxide and chloroform who subsequently reported chemical intolerances. All MCS subjects shared similar illness characteristics whether or not they had seen a clinical ecologist. By comparison, none of the chlorine dioxide group had seen a clinical ecologist, belonged to a patient support group, or claimed to have "MCS". Nevertheless, this group reported symptoms and intolerances that were congruent with the three MCS groups. Based on these findings the authors concluded that MCS "is not a figment of the clinical ecologists' collective imagination."

The behavioural and social conclusions on MCS reached by Mayou et al., (2005), Das-Munshi et al., (2007) and Osterberg et al. (2007) where patients are conditioned by iatrogenic influences and disabled by prolonged avoidance of chemical exposures are fanciful suppositions at best that have no real evidence base and are incapable of objective proof. The fact that Das-Munshi's conclusions are based on poorly designed challenge studies and are published in the Journal of Allergy and Clinical Immunology, a publication linked to medical interests that are routinely opposed to MCS, immediately suggests bias. By comparison the study by Davidoff and Key (1996) above provides real evidence that MCS is not some form of cult phenomenon influenced by belief systems or iatrogenic conditioning. Hausteiner et al.'s (2007) recommendation that MCS be treated as a somatoform illness is highly inappropriate and potentially harmful to MCS patients. As noted above in the study by Miller and Mitzel (1995), MCS is not consistent with the general characteristics of somatoform illness. The proposal by Sparks (2000b) that MCS is a "belief system" best managed by the "prevention of illness behaviour" is also potentially harmful to people with MCS. These potential harms should be raised by the Review.

Either a summary of Davidoff and Key's (1996) study should be included in this section of the Review or the reference to a "belief system" should be deleted.

Unprovable theoretical models proposing a psychogenic aetiology together with psychological treatments for MCS persist despite evidence that psychological interventions to cure MCS are relatively ineffectual. In a study of the perceived efficacy of treatment modalities for MCS Reed-Gibson et al. (2003) established that less than 5% of subjects found psychotherapy to cure MCS to be "very useful", with just over 15% finding it "somewhat useful". By comparison, 65% found that it had "no noticeable effect", with nearly 7% finding it "somewhat harmful" and 8% "very harmful". Based on these findings it is clear that any conclusions of psychogenesis in MCS or recommendations for psychotherapeutic treatments in MCS are premature and potentially harmful to MCS patients and must be approached with great caution. The most effective treatment for MCS found in this study was chemical avoidance. Reed Gibson's

(2003) data on the poor results of psychological treatments to cure MCS need to be included in the Review.

In summary, psychological factors are neither necessary nor sufficient to explain MCS. There is more than enough evidence of the harm caused to people with MCS by common chemicals to require a robust precautionary response. The promotion of MCS as a psychological condition is highly irresponsible, threatens the health and safety of people with MCS and delays the public health and disability access reforms required in chemical regulation and usage. Treatments that focus on psychiatric interventions without first ensuring a proper environment for MCS patients that is free from symptom triggers are, firstly, potentially harmful and, secondly, an abuse of the patient's basic human rights. The general view amongst people with MCS that their illness is not of psychological origin must be properly considered by this Review.

3.1.9 Comments on "Odour perception."

Many of the studies used to suggest that odour perception plays a role in MCS were not conducted on MCS subjects. Consequently the conclusions reached cannot reasonably be applied to MCS with its complex symptomatology and associated physiological abnormalities. Theories of odour perception in actual MCS subjects rely heavily on flawed challenge studies which have been discussed above. Both of these points should be referred to in the Review.

As noted by Goudsmit and Howes (2008), people with MCS report severe reactions to odourless chemical incitants. Odour perception is inconsistent with reported experiences of MCS initiation where the chemical involved has no odour yet exposure at levels assessed as acceptable for the general population results in severe and permanently disabling symptoms with associated loss of tolerance to chemicals previously tolerated. Goudsmit and Howes' (2008) reports of sensitivity reactions to odourless chemical should be referred to in the Review.

Existing research does not appear to support theories of olfactory involvement in MCS. According to Spencer (2008); "Although not extensive enough to wholly refute MCS, research that undermines the Olfactory Threshold Sensitivity Theory; for example, has been done". This comment should be included in the Review as the results of further research in this area are unlikely to be fruitful but will undoubtedly be used to deny the physiological basis of MCS.

With regard to the conclusions reached by Donoghue and Cullen (2007) on perceptions of health risks within the community surrounding the Alcoa refinery at Wagerup, Dr Donoghue is an employee of Alcoa whose opinion cannot be said to be entirely disinterested. An independent survey of residents living near the refinery conducted by the Telethon Institute for Child Research (2008) concluded: "For symptoms potentially related to chemical exposure, elevated rates were found for most symptoms for residents of Hamel/Wagerup/Yarloop and also for residents of Cookernup. Many of these effects were statistically significant." The reported symptoms of headache, breathing difficulties, sore or irritated eyes, difficulty concentrating or remembering, stuffy nose or sinusitis, skin irritation or rashes or eczema, cough or sore throat, fatigued after a good night's sleep, felt weak or dizzy, nosebleed, and nausea are consistent with chemical sensitivity. Most significantly nose bleeds in the subject population were three times higher than the rest of Western Australia. Attributing these symptoms to community "perceptions of risk" is a very convenient way of avoiding responsibility for the harm caused by industrial pollution. Residents surrounding the Alcoa refinery also experience higher rates of cancer.

References to Wagerup in the Review that attempt to support odour perception theories indicate an unreasonable bias towards an industry sponsored view of MCS. The bias should be addressed or the reference removed.

3.1.10.1 Comments on "Altered metabolism."

Genetic predisposition has been identified as a factor in MCS (McKeown-Eyssen et al., 2004). Pall (not dated) also highlights evidence of genetic predisposition as confirming the physiological basis of MCS. Watanabe (2003b) reports that nearly 30% of people with MCS have relatives with the condition. Watanabe's comments are consistent with genetic factors playing a role in MCS and should be noted in the Review.

3.2 Comments on "COMMENTARY ON THE PROPOSED MODELS OF MODES OF ACTION."

People with MCS are generally quite aware that their experience with low level chemical exposures is not consistent with current toxicological paradigms. MCS presents a significant challenge to medicine, science, industry and society but its manifestations cannot be explained by psychogenic theories alone. The emergence of MCS amongst significant numbers of the population requires a new paradigm in toxicology that is better able to assess the relative risks of low level chemical exposures.

The failure of MCS subjects to react to their triggers in challenge testing may not suggest a psychological aetiology. This observation can equally be explained by the concept of masking and adaptation or it may reflect variations in the physiological mechanisms governing the intensity of sensitivity reactions. As noted previously, challenge testing has not adequately taken into account the possibility of masking. For a better understating of the concepts of adaptation and masking see Ashford and Miller's text Chemical Exposures Low Levels and High Stakes, (1998), Chapter 2, Key Terms and Concepts, a summary of which should appear in this Review.

The Review should confirm that any future challenge studies ensure that MCS subjects have been through a period of de-adaptation and unmasking prior to challenge testing, preferably within an Environmental Control Unit, where exposure to incitants can be strictly controlled. The US Interagency Workgroup on MCS (1998) reported that: "For research purposes, ECUs may offer the possibility of learning whether many of the etiologies and mechanisms suggested for MCS can be validated. Some scientists and physicians believe that valuable information could be gained by the proper use of an ECU." Similar recommendations for the use of ECUs should be made by this Review. At the present time Australia does not have an environmental control unit, although such facilities have existed in the private health care sector in the past.

Using single-blinded placebo controlled trials Joffres et al. (2005), of the Nova Scotia Environmental Health Centre, have attempted to account for variations in adaptation to test conditions amongst MCS subjects. They concluded "This study shows the importance of using an adaptation period in testing individuals with reported chemical sensitivities and, despite small numbers, raises questions about underlying mechanisms and level of reactivity to low-level chemical exposures in sensitive individuals." The use of adaptation methods is important in MCS studies.

Despite the lack of convincing evidence, the Review appears to be biased towards perceptions of odour in MCS and the inappropriate psychogenic conclusions based on related studies. According to Pall's NO/ONOO model, odour and the olfactory centre play no role in MCS. Reports of odourless triggers and initiators have been raised previously. This should be noted in the Review.

The Review's claim that "no evidence of accumulation of toxic chemicals in MCS subjects has been found" is not necessarily correct. According to Watanabe (2003), Rea (1992) has found pesticides in the blood of MCS subjects. As further studies are sought in this area, the Review should consider the study by Dunstan et al. (1995) which showed significantly higher levels of chlorinated hydrocarbons in the blood serum of subjects with chronic fatigue syndrome. The authors concluded: "The results suggest that

recalcitrant organochlorines may have an aetiological role in CFS. The role of low-level organochlorine bioaccumulation in the development of CFS symptoms requires further investigation." A further CFS study by Dunstan et al., to assess bioaccumulated hydrocarbons and red/white blood cell parameters concluded that: "Those patients with unexplained and persistent fatigue had significantly higher levels of DDE compared with the controls and had different specific blood cell responses to organochlorines compared with control subjects." Given the significant overlap between CFS and MCS, the studies by Dunstan et al. are significant and should be reported in the Review.

The fact that standardised measures of body burden chemicals have not been adequately conducted on MCS subjects indicates that some of the most basic research projects have not been undertaken in this area.

3.3 Comments on "FURTHER RESEARCH TO IDENTIFY POTENTIAL CAUSATIVE MECHANISMS OF MCS"

With regard to further research the Review once again appears to have illogically confused causation with mechanisms. The research focus remains on the characteristics of MCS subjects themselves rather than observations of event driven studies. This needs to change. Many public health problems, for example lead, asbestos and tobacco, have only been identified by close observation of exposure events and not by attempting to fully understand the associated disease mechanisms in the affected subjects themselves. The same kind of approach is required with MCS. A discussion of the need for event driven studies is vital to this Review and Ashford's (1999) insights in this area are worthy of comment.

There is little doubt that the MCS community would support research that prioritises physiological investigations into MCS. Further research into odour perception and psychological factors contributing to long-term disability in MCS is unlikely to provide useful data, assist in meeting the pragmatic needs of people with MCS, or find support within the MCS community.

Unlike the USA, Australia has no national project to monitor the chemical body burden of its population. Australian research into chemical body burden and bioaccumulation in MCS is required and should be discussed in this Review.

4 Comments on "DIAGNOSIS, TREATMENT AND MANAGEMENT OF MULTIPLE CHEMICAL SENSITIVITY."

4.1 Comments on "DIAGNOSIS AND PREVALENCE OF MCS"

It is clear from national and international data that the extent of the chemical sensitivity problem is already large. The South Australian Parliamentary Inquiry into MCS (Social Development Committee, 2005) recommended that "the Department of Health (DoH) monitors the prevalence of MCS in SA and compiles comparative data on the incidence of MCS to enable trend analysis."

Population studies to monitor the national prevalence of MCS are appropriate. This task would be assisted by standardized questions on medically and self-diagnosed MCS, the interim use of the 1999 consensus criteria (Bartha et al, 1999) for the diagnosis of MCS, and the indexing of MCS in the ICD-10-AM.

The Department of Health and Ageing has a clear responsibility to take a leadership role in MCS. As noted earlier, the Review should clearly identify which area of DOHA might be responsible for accumulation population data on MCS.

4.2 Comments on "MCS CASE DEFINITION AND PREVALENCE DATA."

Disagreements on diagnostic criteria for MCS should not be used by this Review to avoid taking responsible action on MCS. Total consensus for these criteria may never be achieved, particularly given the existing opposition to MCS by industry and vested medical interests. As noted above, the 1999 consensus criteria should be recommended for use in population studies.

For the reasons outlined above, notably due to broad fluctuations over time in both the presence and severity of sensitivity reactions amongst individual MCS subjects, strict reliance on double blinded challenge testing in MCS may not be as reliable as supposed as a diagnostic tool. Anecdotal reporting amongst people with MCS suggests that Winder's (1994) observation is correct that early detection and intervention, including minimizing exposure to triggers, results in improved outcomes. When such improvements occur, chemical sensitivity reactions may be significantly reduced to the point where challenge testing may not give reliable results. However, most patients retain some daily symptoms of MCS and remain vulnerable to acute and chronic MCS initiating exposures and loss of tolerance to chemicals previously tolerated. An accurate patient assessment would also need to rely on clinical diagnostic criteria and a thorough patient history, including documentation of any known chemical exposures. This is currently not occurring in the clinical setting and some discussion of this omission is appropriate in this Review.

Longitudinal studies would be appropriate to help throw light on the natural history of MCS in Australia. Sears (2007) in "The Medical Perspective on Environmental Sensitivities" published by the Canadian Human Rights Commission, has noted that "susceptibility to sensitivities will be life long."

4.3 Comments on "TREATMENT FACILITIES"

Lack of access to informed, chemical-free medical services is a critical problem facing people with MCS. The entrenched and often acrimonious philosophical divide between allergists on the one hand and practitioners of environmental medicine on the other has been a constant obstruction to health care for people with MCS. Despite the Review recognising the need to avoid symptom triggers in MCS, there are very few medical services in Australia that have adopted this MCS treatment and disability access strategy. The reluctance amongst medical practitioners to provide MCS disability access accommodation has been noted by the New Mexico Report to the Legislature on Multiple Chemical Sensitivity (Governor's Committee on Concerns of the Handicapped, 1996).

A draft MCS hospital protocol based on the Royal Brisbane Hospital model is presently in development by the South Australian Department of Health in consultation with the interagency MCS Reference Group. Once completed, the protocol will be adopted by all South Australian public hospitals. However, this protocol applies to hospital in-patients only. Despite recommendations from the SA Inquiry into MCS for the development of practical measures to assist people with MCS with disability access to services in the community (Social Development Committee, 2005), there remains no overarching strategy to provide people with MCS with disability access to health care and allied services, including aged care, in the community.

There are currently no medical services in South Australia providing dedicated care at any level to people with MCS. What limited services that are available rely on a handful of general practitioners who have maintained an interest in the area. Those GPs who do have knowledge of the diagnosis and management of MCS are usually fully booked and cannot take on new patients without extended waiting times of many months. People with MCS in South Australia must seek medical consultations interstate in order to access informed specialist care.

Despite the lack of dedicated services, people with MCS report a relatively large number of medical consultations and are more likely to seek assistance from conventional and complementary health care

services compared to other chronic illnesses (Park and Knudson, 2007). Dedicated MCS treatment facilities, similar to the state-sponsored Nova Scotia Environmental Health Centre in Canada, that are capable of providing informed medical services together with reasonable disability access accommodation are urgently needed. A recommendation for such services should be included in the Review.

The inclusion of chemical sensitivity in the Australian Human Rights Commission Guidelines; *Access to Buildings and Services: Guidelines and information*, is a welcome development. However, these guidelines do not contain sufficient detail on MCS disability access and actually refer to US documentation from the Job Accommodation Network. This situation reflects the general lack of policy development on MCS in Australia. An example of the extensive work done in the USA on MCS disability access is their National Institute for Building Sciences' guidelines entitled "Indoor Environmental Quality". A copy of this document, which includes MCS hospital protocols, can be accessed at http://ieq.nibs.org/. Reference to the NIBS document should be included in the Review.

4.4 Comments on "TREATMENT OF MCS."

The lack of recognition of MCS amongst mainstream medical professionals presents serious difficulties for patients with MCS. Management of the MCS patient depends on individual clinicians but rarely is medical advice given to avoid further chemical exposures and a thorough patient history of suspect chemical exposures is almost never taken.

Referral to a conventional allergist is unlikely to result in much progress as many of these professionals are actively opposed to the recognition of MCS. Also, MCS does not fall within the current paradigms of occupational medicine and simply baffles these clinicians. All too often, when conventional investigations show few or no laboratory abnormalities, the patient is inappropriately referred to psychiatric services.

There is a body of knowledge within the practice of environmental medicine that is able to assist people with MCS and possibly bring them improved health and independence. However, while conventional medicine continues to be philosophically opposed to these practices they are unlikely to be made generally available to patients with MCS. A discussion of how to heal the medical divide on MCS would be a useful contribution to this Review.

MCS support groups are mainly comprised of people with MCS. These community groups are extremely limited in the services they can provide and, unlike many groups associated with more conventional medical diagnoses, are not linked into a particular medical specialty or the benefits this can provide. The cooperation and assistance of mainstream medical services together with medical education on MCS is urgently needed. The Review's set of general principles in the management of MCS may help this situation somewhat but only if they are broadly distributed amongst medical professionals. The Review should detail exactly how this medical education might occur.

Comments on "MCS Clinical Management Principles."

The principles outlined in this section are likely to provide some support for patients but they are extremely medically conservative. The statement: "Recognise and explain that no specific therapy has yet been proven to be of benefit" leaves little hope for the future in patients with MCS. While it might be true that there are no proven treatments for MCS, this may well be due to the lack of research into those MCS treatment modalities that are reported to offer benefits. It should be recognised that a great deal of conventionally accepted medical practice is based on clinical experience and accrued knowledge that has not been proven. This does not mean that this clinical knowledge is not valuable in medical practice.

Medical practice in MCS must be conducted ethically without exposing the patient to unreasonable costs, false hopes or treatments that have been proven to be bogus. However, to simply say to the patient that there is no hope of any treatment that might improve their circumstances might not always be the correct approach. Some management strategies are worth exploring. Strategies involving environmental medicine may are widely reported to have some benefit despite the philosophical objections of conventional allergists and immunologists. Any treatment strategy should confirm the need for avoiding symptom triggers.

An example of medical treatment for MCS based on Pall's NO/ONOO model and Ziem's clinical experience is available on the MCS America website at http://www.mcs-america.org/index_files/mcsmedicaltreatment.htm.

In the UK, some members of the British Society for Ecological Medicine are recognised by the General Medical Council as having a specialist standard and are funded by the National Health Service in the treatment of serious chemical sensitivity (Dunn and Coe, 2005).

Some reference to the potential benefits of non-conventional treatment programs is appropriate in the review.

5. Comments on APPENDIX 1.

5.1.4 Workshop

As noted above, no representatives from MCS community groups were invited to participate in this workshop. This kind of exclusive behaviour by public health institutions belongs firmly in the past and should not be repeated in future. A truly cooperative approach between patients and clinicians is needed if the extensive public health problems associated with MCS are to progress beyond the current impasse.

FURTHER COMMENTS

Regulatory Risk Assessment Models in MCS

Given that OCS is the principal chemical regulator in Australia, it is surprising that no comment has been made in the Review on potential risk assessment models that may assist in the regulation of chemicals associated with MCS. The development of a new paradigm in chemical regulation is a vital part of the response to MCS. The present chemical negligence must not be allowed to continue.

Increased sensitivity to cholinergic agents has been proposed by Overstreet and Djuric (1999 and 2001) as a mechanism in chemical intolerance and MCS. The authors claim that the use of selectively bred Flinders Sensitive Line (FSL) rats, which exhibit increased sensitivity to organophosphates and are also "sensitive to a variety of drugs unrelated to the cholinergic system and to cholinergic- and allergen-induced bronchoconstriction", may be useful in the study of MCS and asthma. FSL rats show similar behavioural characteristics reported in chemically intolerant human populations. The authors conclude that: "An abnormal cholinergic system may therefore contribute to both MCS and asthma," and also that "An elucidation of these mechanisms may provide useful clues to those involved in chemical intolerance in humans."

Rowat (2000) has considered mechanisms and measurement in chemical susceptibility, injury and reactivity with emphasis on developmental and receptor-ligand considerations. The author proposes several models involving genetic variability and molecular mechanisms that may be useful in risk

assessment analysis of chemicals in chemical injury and MCS. This paper also has an extensive bibliography of research related to this subject.

Abou-Donia et al. have published numerous studies showing toxic chemicals administered in combination, and in combination under stress, leads to measurable neurological damage and neurobehavioural deficits in rats and hens. These studies require further scrutiny for their risk assessment potential.

Some comment in the Review on potential regulatory models in response to MCS together with a frank discussion of the inadequacies of the current regulatory system is appropriate. Further discussion is also needed on what can be done immediately from a regulatory perspective. For example, adequate label warnings on products associated with MCS, as recommended by the South Australian Parliamentary Inquiry into MCS (Social Development Committee, 2005).

Sociological Impacts

The Review is incomplete without an assessment of the sociological impacts of MCS caused by the ongoing inadequacies in chemical regulation.

The sociological impacts of MCS are substantial. Although no sociological data has been published in Australia to date, international statistics paint a bleak picture for people with MCS. There is no reason to believe that this situation would be any different in Australia. In fact, circumstances in this country may actually be worse due to the lack of recognition surrounding MCS.

Based on state department of health prevalence data it can be estimated that approximately 1% to 3%, or somewhere between 200,000 and 600,000 people, have been medically diagnosed with MCS or chemical sensitivity in Australia. However, if the Parliamentary Inquiry into MCS in South Australia is correct that "Interstate and overseas research has shown that up to 6 percent of the population may have MCS", the number of people with MCS in Australia could actually be more that one and a quarter million. Other states apart from SA and NSW have no prevalence data. A national survey of the prevalence of MCS is required.

Although the relative percentages may be small, the actual numbers of people with MCS are large and increasing and undoubtedly represent a major public health problem of similar proportions to asthma and diabetes. These statistics alone should alert regulatory authorities to the urgent need for action on MCS.

People with MCS are significantly disadvantaged and discriminated against in all walks of life.

With regard to employment, Caress and Steinemann (2003) have shown that close to 2% of the population have lost their jobs due to MCS, a figure which compares with 2% in California (Kreutzer and Neutra 1996). Kassirer and Sandiford (2000), in a study of the socio-economic impacts of environmental illness in Canada, have estimated that between 1% and 3% of the Canadian population is unemployed due to environmentally-related conditions. The overall cost of MCS to Canada, including loss of income for people with MCS, is estimated to be around 13 billion dollars annually, a figure which is likely to be comparable to Australia. Gibson et al. (1996) found that 205 of 268 people with MCS lost or were forced to quit their jobs due to intolerable chemicals in the workplace, with three quarters of these people eventually seeking disability support payments.

With regard to disability discrimination in the workplace Vierstra et al. (2007) have compared the employment discrimination experiences of Americans with MCS in comparison to those in a general disability group with allergies, asthma, HIV, gastrointestinal impairment, cumulative trauma and

tuberculosis. People with MCS were found to be more likely to allege discrimination related to reasonable accommodation, to file complaints against employers, and to receive non-merit based resolutions of complaints under the *Americans with Disabilities Act.*

Reed Gibson (1999) has provided a summary of the individual and social consequences of failing to accommodate workers with environmental illness.

Housing for people with MCS is an area of extreme disadvantage due to ever present chemical barriers in construction, renovation and maintenance materials. People with MCS in Australia are frequently homeless or living in substandard conditions. There are no formal MCS disability access policy guidelines in any public housing services in Australia. In South Australia, people with MCS, despite qualifying for priority public housing in all other criteria, are routinely denied applications on the basis that there is no suitable housing stock. Reed Gibson (2003) reports that, of a given US sample population of MCS subjects, 10% were homeless, with another 10% living in substandard conditions in tents, caravans and shacks. Only 41% of respondents report living in safe housing.

Numerous authors have commented on the difficulties people with MCS experience in accessing informed health care. Of note is the extended number of years between the onset of chemical intolerance and the acquisition of a medical diagnosis of MCS. According to a study by Park and Knudson (2006), people with MCS are more likely than other people with chronic illness to seek help from both conventional and complementary health care providers. The authors suggest that this may be due to multiple referrals but it is just as likely to be due to the lack of knowledge of MCS amongst medical professionals which results in the patient continuing to seek answers to their multiple health problems. Reed-Gibson (1997) notes.

> *Early stages of MCS are often marked by a frantic search for medical care. Respondents in my ongoing study saw a mean of 8.6 physicians, and only a quarter of these were deemed to have been at all helpful by patients. Participants reported spending exhorbitant amounts of money, experiencing considerable iatrogenic harm, and being perceived as not credible by both medical and psychological providers. Access to medical care is limited because of the perfumes, pesticides, and other chemicals present in medical facilities. This means that MCS patients are often unable to find help either for the MCS symptoms or for unrelated health problems should they occur. Respondents in my research reported difficulty finding physicians educated about MCS, safe offices, or someone to take their symptoms seriously. Consequently, many avoided any medical treatment, and 20 persons delayed treatment until their conditions had become medical emergencies.*

Ashford and Miller (1998) have referred to this experience as a "medical odyssey". The associated costs to the health care system due to the ongoing ignorance and denial of MCS are substantial.

Pamela Reed Gibson, Professor of Psychology at James Madison University, Harrisonburg, VA, has published extensively on the sociological impacts and disability access requirements of MCS:

- Chemical and Electromagnetic Exposures as Disability Barriers: Environmental Sensitivity, (2009), In Press at *Disability & Society*, 24(2): 187-199. http://www.mcsresearch.net/journalpapers/chemicalbarriers.pdf
- Sickness Related Dysfunction in Persons with Self-Reported Multiple Chemical Sensitivity at Four Levels of Severity, (2009). *Journal of Clinical Nursing*, 18, 72-81. http://www.mcsresearch.net/journalpapers/sicknessrelateddysfunction.pdf
- Work Accommodation for People With Multiple Chemical Sensitivity (2007) *Disability & Society*, 22 (7), http://www.mcsresearch.net/journalpapers/workaccommodation&mcs.pdf

- Disability-Induced Identity Changes in Persons With Multiple Chemical Sensitivity (2005), *Qualitative Health Research*,15(4): 502-524. http://qhr.sagepub.com/cgi/content/abstract/15/4/502
- Understanding and Accommodation People with Multiple Chemical Sensitivity in Independent Living (2005), published by Independent Living Research Utilization http://www.ilru.org/html/publications/bookshelf/MCS.html
- Hope in multiple chemical sensitivity: social support and attitude towards healthcare delivery as predictors of hope, (2001) *Journal of Clinical Nursing*, 8(3):275-283 http://www.ncbi.nlm.nih.gov/pubmed/10578750
- Social support in persons with self-reported sensitivity to chemicals, (1998) *Research in Nursing & Health* 21(2): 103-115. http://cat.inist.fr/?aModele=afficheN&cpsidt=2196146
- Multiple Chemical Sensitivity, Illness, and Culture: On the Margins of Health Care. Paper presented at the annual convention of the American Psychological Association, August 15-19, l997, Chicago, IL. http://www.mcsresearch.net/Conferencepapers/marginsofhealthcare.pdf
- Chemical Sensitivity/Chemical Injury and Life Disruption, (1997), Paper presented at the Association for Women in Psychology, March 6-9, Pittsburgh, PA. http://www.mcsresearch.net/Conferencepapers/lifesatisfaction.pdf
- Multiple Chemical Sensitivity, Culture, and Delegitimization: A Feminist Analysis, (1997), *Feminism & Psychology*, 7(4): 475-493. http://fap.sagepub.com/cgi/content/abstract/7/4/475

Other publications of interest are:

- Chircop, A., and Keddy, B., Women Living with Environmental Illness, *Health Care for Women International*, (2003) 24(5): 371-383. http://www.ingentaconnect.com/content/routledg/uhcw/2003/00000024/00000005/art00002
- Lipson, G., We are the Canaries: Self Care in Multiple Chemical Sensitivity Sufferers, *Qualitative Health Research*, (2001) 11(1): 103-116. http://qhr.sagepub.com/cgi/content/abstract/11/1/103?ck=nck
- Spencer, T., The Challenge of Multiple Chemical Sensitivity, *Journal of Environmental Health*, (2008) 70(10): 24-27.

This Review cannot legitimately avoid commenting on the sociological impacts of MCS by suggesting that it is a scientific analysis alone. Much of the psychological assumption and published opinion in the Review is based on unscientific psychological theories that cannot be objectively assessed. By contrast much of the wealth of sociological data accumulated on MCS is based on sound research principles in social science. A more holistic scientific approach that includes an analysis of the sociological impacts of MCS is required if this Review is to have validity, particularly within the context of current standards in chemical regulation.

Involvement in the Review of the Australian Pesticides and Veterinary Medicines Authority

Given that a significant proportion of MCS is initiated by pesticide exposure, it is disappointing and possibly negligent that APVMA has not been directly involved in this Review. This omission reflects the inability of various departments to work cooperatively on MCS within the confused and overlapping responsibilities of the chemical regulatory structure in Australia

In a recent decision the UK High Court found its Government and national regulator, the Pesticides Safety Directorate, negligent and unlawful with respect to agricultural pesticide use and the protection of public health (Downs, 2008). The case was brought to the court by a young woman with chronic fatigue syndrome whose illness was severely exacerbated, and probably caused, by routine exposure to pesticide spray drift and residues near her rural home. In the final judgment the court stated:

> *"The result of this judgment is that the defendant must think again and reconsider what needs to be done. It is not for me to specify any particular action he needs to take. He must take steps to produce an adequate assessment of the risks to residents. In addition, he must carefully reconsider whether the existing conditions of use are adequate. The need to inform residents of intended spraying and of the composition of the pesticides to be used is I think clear. Voluntary action is not achieving this. Equally, I think there is a very strong case for a buffer zone, such as incidentally already exists to avoid spraying too close to watercourses in order to minimise the risk of pesticides entering groundwater"* (Downs vs Secretary of State, 2008).

The APVMA is aware of these developments and is apparently developing a response. Unfortunately APVMA senior bureaucrats have not yet responded to questions on this issue from SATFMCS.

Given these developments it is vital that this Review include some comment on national policy and public health with respect to agricultural pesticides and MCS/CFS. The APVMA's current spray drift policy is not adequate to protect either public health or the disability access rights of people with MCS.

Disability Access

The Review has a moral responsibility to include reference to comprehensive guidelines on MCS disability access such as those available through the US National Institute of Building Sciences at http://ieq.nibs.org/.

The Australian Human Rights Commission recognises MCS as a disability under the *Disability Discrimination Act*. Regulatory authorities such as OCS and NICNAS provide a public service to ensure the safe use of chemicals for the entire public. Although not technically acting unlawfully, regulatory authorities are morally guilty of discriminating against people with MCS on the basis of their disability by refusing to provide them with services in chemical regulation that are available to the majority of the population without the disability and by requiring them to adhere to exposure standards in the community with which they are not able to comply. This administrative malfeasance is reprehensible and deserves the strongest condemnation. The national policy of regulatory indifference and denial of MCS must change.

Conclusion

The Review attempts to delegitimize MCS by overly emphasising unprovable psychogenic theories, despite clear evidence to the contrary, and by focusing on the characteristics of people with MCS rather that the real causes of the illness. Regulatory institutions are too closely associated with vested interests in industry and appear incapable of responding to the environmental public health and disability access crisis that MCS presents.

Anne McCambell, MD, Chair of the MCS Task force of New Mexico, put it succinctly.

> *The manufacturers of pesticides, carpets, perfumes, and other products associated with the cause or exacerbation of chemical sensitivities adamantly want MCS to go away. Even though a significant and growing portion of the population report being chemically sensitive, chemical manufacturers appear to think that if they can just beat on the illness long enough, it will disappear.* (McCampbell, 2001)

MCS is not going to disappear because it is physiologically based with predisposing genetic determinants and is caused by many toxic chemicals found commonly in the modern environment. MCS is real and governments need to take urgent action to combat this public health crisis. The numbers of people with

MCS are already large but evidence to the SA Parliamentary Inquiry into MCS advised that the incidence of the condition is increasing in the community. At what stage will regulatory action be taken to address this problem? How many people must become sick and disabled by toxic chemicals before anything is done?

References

Akha Heritage Foundation, Astra Zeneca Death, 2008, http://www.akha.org/content/chemicalhazards/astrazenecadeath.html

Ashford, N., Low-level chemical sensitivity: implications for research and social policy, *Toxicol Ind Health* (1999) 15:421-427. http://dspace.mit.edu/bitstream/handle/1721.1/1578/ChemSens_Tx&In.pdf?sequence=1

Ashford, N. and Miller, C., *Chemical Exposures Low Levels and High Stakes*, Van Nostrand Reinhold, New York, 1998.

Australasian Society of Clinical Immunology and Allergy, Unorthodox Techniques for the Diagnosis and Treatment of Allergy, Asthma and Immune Disorders, 2007, http://www.allergy.org.au/pospapers/unorthodox.htm

Caress, S. and Steinemann, A., A Review of a Two-Phase Population Study of Multiple Chemical Sensitivities, *Environmental Health Perspectives*, (2003) 111(12):1490-1497. http://www.ehponline.org/members/2003/5940/5940.pdf

Danish Ministry of the Environment, *Multiple Chemical Sensitivity, MCS*. Environment Protection Agency, 2005. http://www.aeha-quebec.ca/pdf/87-7614-549-2.pdf

Das-Munshi J, et al., Multiple chemical sensitivities: A systematic review of provocation studies. *J. Allergy Clin Immunol* (2006) 118: 1257-1264. http://www.jacionline.org/article/S0091-6749(06)01696-4/abstract

Davidoff, A., and Keye, P., Symptoms and Health Status in Individuals with Multiple Chemical Sensitivities Syndrome from Four Reported Sensitizing Exposures and a General Population Comparison Group, *Archives of Environmental Health*, (1996) 51(3):201-231.

Dilsaver, S., Cholinergic Mechanisms in Depression, *Brain Research*, (1986) 11:385-361.

Downs, V., Press Release, (2008), http://www.pesticidescampaign.co.uk/documents/PRESS%20RELEASE%20-%20Historic%20Victory%20in%20Landmark%20High%20Court%20Action%20over%20Pesticides.pdf

Downs vs Secretary of State for Environment Food and Rural Affairs, Before Mr Justice COLLINS, Royal Courts of Justice, Strand, London, 14 November, 2008. http://www.bailii.org/cgi-bin/markup.cgi?doc=/ew/cases/EWHC/Admin/2008/2666.html&query=title+(+downs+)&method=boolean

Dunn, R., and Coe, T., Tired or Toxic? Understanding Multiple Chemical Sensitivity, *Inter Action*, May 2005, 19-22. http://www.afme.org.uk/res/img/resources/IA%2052%20MCS%20and%20ME.pdf

Goudsmit, E. and Howes, S., Is multiple chemical sensitivity a learned response? A critical evaluation of provocation studies, *Journal of Nutritional &Environmental Medicine*, (2008) iFirst Article, 1–17.

Governor's Committee on Concerns of the Handicapped, Report to the Legislature on Multiple Chemical Sensitivity, Legislature of the State of New Mexico, 1996.

Hausteiner, C., et al., Dysfunctional cognitions in idiopathic environmental intolerances (IEI) – An integrative psychiatric perspective. *Toxicol Letters* (2007) 171:1-9.

Hileman, B., Multiple Chemical Sensitivity, *Chemical and Engineering News*, (1991) 69:26-42.

Interagency Workgroup on MCS, A Report on Multiple Chemical Sensitivity (MCS), 1998.

Itoyama, K., et al., Characteristics and Causes of Self Reported Idiopathic Environmental Intolerances, 2006.

Joffres, M.R, et al., Environmental sensitivities: prevalence of major symptoms in a referral center: the Nova Scotia environmental sensitivities research center study Abstract - see Environmental Sensitiviites - Prevalence of Major Symptoms in a Referral Centre, *Environ Health Perspect.* 2001 Feb;109(2):161-5.

Kassirer, J., et al., The Impact of By-Laws and Public Education Programs on Reducing the Cosmetic / Non-Essential, Residential Use of Pesticides: A Best Practices Review, Jointly Prepared by: The Canadian Centre for Pollution Prevention and Cullbridge Marketing and Communications.

Kassirer, J., & Sandiford, K. (2000). Socio-economic impacts of environmental illness in Canada. Prepared for Judith Spence, President, Environmental Illness Society of Canada. Ottawa, Ontario, CANADA: Cullbridge Marketing and Communications.

Kreutzer, R. and Neutra, R., *Evaluating Individuals Reporting Sensitivities to Multiple Chemicals,* Agency for Toxic Substances and Disease Registry, National Technical Information Service, Springfield, V.A., (Publication #PB96-187646) June, 1996.

Kwiatek, R., et al., Regional Cerebral Blood Flow in Fibromyalgia, *Arthritis & Rheumatism* 2000; 43(12):2823-33.

Lacour, M. et al., Multiple Chemical Sensitivity Syndrome (MCS) – suggestions for an extension of the US MCS-case definition. Int. J. Hyg. Environ.-Health (2005) 208:141–151

Loblay, R., "Multiple Chemical Sensitivity", Submission to the Social Development Committee of the Parliament of South Australia, on behalf of the Australasian Society of Clinical Immunology and Allergy, 2004.

Mayou R, Kirmayer LJ, Simon G and Sharpe M Somatoform disorders: Time for a new approach in DSM-V. *Am. J. Psychiatry* (2005) 162:847-855.

McCampbell, A., Multiple Chemical Sensitivity Under Siege, *Townsend Letter to Doctors and Patients*, (2001) 210.

McKeown-Eyssen G. et al., Multiple chemical sensitivity: discriminant validity of case definitions. *Arch Environ Health* (2001) 56(5):406-12

McKeown-Eyssen G., et al., Case-control study of genotypes in multiple chemical sensitivity; CYP2D6, NAT1, NAT2, PON1, PON2 and MTHFR. *Int J Epidemiol* (2004) 33:1-8

Miller, C. and Mitzel, H., Chemical Sensitivity Attributed to Pesticide Exposure versus Remodeling, Archives of Environmental Health (1995) 50(2):119-129 cited in Ashford, N. and Miller, C., Chemical Exposures Low Levels and High Stakes, Van Nostrand Reinhold, New York, 1998.

National Center for Environmental Health, Centers for Disease Control and Prevention, Summary of Public Comments Received for the Multiple Chemical Sensitivity Report, 2000.

Ojima M, et al., Odour perception in patients with Multiple Chemical Sensitivity. *Tohoku J Exp Med* (2002) 198:163-173

Overstreet, D., and Djuric, V., A genetic rat model of cholinergic hypersensitivity: implications for chemical intolerance, chronic fatigue, and asthma. *Ann N Y Acad Sci*. (2001) Mar;933:92-102.

Overstreet, D., and Djuric, V., Links between multiple chemical sensitivity and asthma in a rat model of cholinergic hypersensitivity: a brief review, *Toxicology and Industrial Health*, (1999) 15(5):517-521.

Pall, M., Multiple Chemical Sensitivity: Toxicological Questions and Mechanisms, (not dated).

Park, J., and Knudson, S., Medically Unexplained Physical Symptoms, *Health Reports*, (2007) 18(1); 43-47.

Rowat, Chemical Susceptibility, Injury and Reactivity: Mechanisms and Measurement with emphasis on developmental and receptor-ligand considerations. 2000.

Rea, W., et al., Considerations for the diagnosis of chemical sensitivity. In: National Research Council editor. Multiple chemical sensitivities. Addendum to biologic markers in immunotoxicology. Washington DC; National *Academy Press*, (1992): 169–192.

Reed Gibson, P., Multiple Chemical Sensitivity, Illness, and Culture: On the Margins of Health Care. Paper presented at the annual convention of the American Psychological Association, August 15-19, l997, Chicago, IL. http://www.mcsresearch.net/Conferencepapers/marginsofhealthcare.pdf

Reed-Gibson, P. et al. Multiple chemical sensitivity/environmental illness and life disruption. *Women & Therapy*, (l996): 19, 63-79. http://www.mcsresearch.net/Conferencepapers/lifesatisfaction.pdf

Reed Gibson, P. et al. Perceived Treatment Efficacy for Conventional and Alternative Therapies Reported by Persons with Multiple Chemical Sensitivity, *Environmental Health Perspectives* (2003) 111(12): 1498-1504.

Ross G.H., Clinical characteristics of chemical sensitivity: an illustrative case history of asthma and MSC. *Environmental Health Perspectives*, (1997) 105: 437–441 cited in Watanabe, M., et al., Multiple Chemical Sensitivity and Idiopathic Environmental Intolerance (Part One), *Environmental Health and Preventive Medicine* (2003) 7:273–282.

Sears, M., The Medical Perspective on Environmental Sensitivities, Canadian Human Rights Commission, 2007.

Social Development Committee, Inquiry into Multiple Chemical Sensitivity, Parliament of South Australia, 2005.

Saito, M., et al. Symptoms profile of multiple chemical sensitivity in actual life. *Psychosom Med* (2005) 67:318–325 cited in Goudsmit, E. and Howes, S., Is multiple chemical sensitivity a learned response? A critical evaluation of provocation studies, *Journal of Nutritional &Environmental Medicine*, (2008), iFirst Article, 1–17.

Watanabe, M., et al., Multiple Chemical Sensitivity and Idiopathic Environmental Intolerance (Part Two), *Environmental Health and Preventive Medicine* (2003a) 7:273–282.

Watanabe, M., et al., Multiple Chemical Sensitivity and Idiopathic Environmental Intolerance (Part One), *Environmental Health and Preventive Medicine* (2003b) 7:264-272.

Winder, C., Chemically related chronic fatigue syndrome: *Int. J Occup Med Toxicol* (1994) 3: 253-278

Winder, C., Mechanisms of multiple chemical sensitivity, *Toxicology Letters*, (2002) 128:85-97.

World Health Organization, Note to invited participants in "MCS" workshop, 21-23 February 1996, Berlin, Germany, 6/7/96 cited in McCampbel, A., Multiple Chemical Sensitivity Under Siege, *Townsend Letter to Doctors and Patients*, (2001) 210.

Ziem, G., Patients with Chemical Injury and Sensitivity, Presentation at the 2001 MCS Conference, Santa Fe, N.M., August 13-15, 2001.

Ziem, G. and McTamney, J., Profile of Patients with Chemical Injury and Sensitivity. *Environmental Health Perspectives*, (1997) 105 (2).