

Proceedings of a joint RMA, DVA & ESO forum held in response to recommendations of the Pearce Report

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ISBN 0 642 39932 8

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Health Threats to Navy Personnel since the Vietnam War

Dr M.J. Flynn CDRE RANR
Formerly: Director-General of Strategic Health
Office of the Surgeon-General
Australian Defence Force

Currently: Counter Disaster & Olympic Planning Branch
NSW Health Department

Working Conditions in the RAAF – an occupational medicine perspective

Dr Warren Harrex
Department of Veterans' Affairs, Canberra

Factors Influencing Post-Traumatic Stress Disorders in the ADF

Commander Alison McLaren, RAN
Staff Officer Medical Clinical Policy
Defence Health Service Branch, ADF

Injury in the ADF

Lieutenant Colonel Steve Rudzki
Commanding Officer
Canberra Area Medical Unit
Duntroon, ACT

Human Research in Defence

Commander Kevin Donovan
Director of Health Capability Development
Defence Health Service Branch, ADF

Opening Address to the Canberra Forum

Senator Eric Abetz

Professor Ken Donald and members of the RMA, Keith Lyon and Paul Stevens from the Repatriation Commission, representatives from the veteran community, the Surgeon-General, Professor John Pearn, and other members of the Defence Forces, and our distinguished guests, the epidemiologists, Professor Kaldor and Dwyer, ladies and gentlemen, good morning and welcome to you all.

This morning it's my duty, albeit a very pleasant duty, to open this forum, a forum between the Repatriation Medical Authority, the Department of Veterans Affairs and the Veteran community joined by several senior medical officers from the Defence Forces. Let me say at the outset that there are a number of people who would have liked to have been with us today but were required to be elsewhere. Suffice to say that the Minister for Veterans Affairs, Bruce Scott, is in Belgium representing Australia to celebrate the cessation of hostilities 80 years ago, the armistice in relation to World War I. Similarly, the President of the Repatriation Commission, Dr Neil Johnston and the President of the RSL, Major General Peter Phillips. All of these gentlemen are representing Australia at this important international commemorative event.

This forum arose out of the recommendations of the review of the Repatriation Medical Authority and the Specialist Medical Review Council that was undertaken by Professor Denis Pearce and assisted by Professor Darcy Holman. This report was commissioned to honour the Government's pledge, when it was elected in 1996, to undertake a review of the authority. The review was completed in October 1997. In his report, Professor Pearce found that the changes that were made to the Veterans' Entitlements Act in 1994 had resulted in a system that is more equitable, more efficient and less adversarial than the system that was in place prior to the changes being made.

However, he also found that the system could be improved. After the release of the Pearce/Holman report, the Minister implemented a program of broad

consultation with the Ex-Service community. During this process of consultation, many, but not all, organisations and individuals within the Ex-Service community expressed support for the majority of the recommendations contained within the report. After this process, the Minister decided to accept the central recommendations of the Pearce/Holman report and made the implementation of the recommendations of that report part of the election platform on which the coalition went to the last Federal election.

There are three recommendations that are of particular relevance to us today, and I note that they are in fact referred to in the brochure or pamphlet that you have detailing the agenda of today's conference. The first of these recommendations was that the Department and the Authority should discuss with the veteran community the possibility of hosting a conference during which medical and scientific experts with service experience could share their knowledge of this experience.

After the Minister's acceptance of the report, these discussions did take place and this forum today was the outcome of those discussions. I'm sure that the discussions in the days ahead will shed light on, and a better understanding of, the manner in which service life is different to most other occupations.

The second recommendation was that the RMA should prepare a publication that sets out its understanding of the powers and functions that it exercises under the Veterans Entitlements Act, the general principles that it follows in addressing causality, a description of how the Authority works and the ways in which individuals can bring matters before the Authority.

Although the publication will remain the Authority's document, the members of the Authority would like to develop this in consultation with the Ex-Service community. This forum is an important opportunity for the veteran community to contribute to the development of this document.

The third recommendation that is of relevance to this forum relates to the collection of data that may be used for research purposes. The report of Professors Pearce and Holman noted that information that has been collected for particular purposes may not be used without consent for other purposes. It's suggested that it might be appropriate to consider different ways of collecting data in the future so that this data might be useable for research questions.

There are difficult ethical questions associated with a collection of data that could be used for a variety of unforeseen research projects in the future. I hope that this forum will move some way towards resolving these issues, or perhaps might move to some way towards outlining structures and processes that could lead to appropriate resolution of these problems.

The forum also has a mutually educative role. This will involve representatives of the Ex-Service community learning more about evidence based medicine. The members of the Authority will learn more about the conditions of service.

Professor Donald, the aims and objectives that this forum has set are ambitious. At the end of these three days, it may seem to you that you have made only limited progress towards your goals, however, given the difficult nature of the tasks that are before this forum, even limited progress in your eyes has the potential for tremendous benefits for the veteran community.

I would like to express my hope that the next few days will provide for a surprising amount of progress in developing agreement to

appropriate answers to these difficult questions. In attempting to move towards solutions to these difficult questions, you will be assisted in your deliberations over the next few days by two distinguished epidemiologists, Professor John Kaldor and Professor Terry Dwyer.

On behalf of you all, I would like to extend our thanks to these two imminent individuals for giving so freely of their time to assist the work of the Authority and the Department and the Ex-Service community. In fact, at this time, as we're moving on towards 11 November and we think of the voluntary service that a lot of Australians undertook for the benefit of our country, I could not help but think that the spirit of the volunteer lives on even with specialist epidemiologists, and I'm sure that we are all very thankful for their assistance in this forum.

I would like to thank the members of the Authority, the Repatriation Commission, and the representatives of the Ex-Service community for the opportunity to open this forum. I am not sure how frequently I will be, as Parliamentary Secretary to the portfolio, involved in events with the veteran community, but can I say if they're all as pleasant as last night and this morning, I hope that when they do the allocations within the portfolio, I am given some slice or some area which will allow me to continue an ongoing involvement with the work of repatriation and with the veterans community.

In formally declaring this forum open, I wish the forum every success and trust that your deliberations will be of great benefit to those that you are seeking to assist. Thank you very much and all the best.

The RMA's Understanding of the Powers and Functions it Exercises Under the VEA and the General Principles it Follows in Addressing Causality.

Professor Ken Donald

Chairman of the Repatriation Medical Authority

9 November, 1998

[This document was prepared from the audio transcripts of the Forum and has been edited for clarity and continuity only.]

**PROFESSOR KEN DONALD,
Repatriation Medical Authority**

This is going to be the first of my two attempts at communication this week, one today and on Wednesday I'm going to come back with another session, and then we'll have some questions and hopefully some answers from me.

If you remember, the reason we're here is first of all the Pearce report, but it also emerged during the consultations following the Pearce report that the veterans were prepared to accept the present legislation provided they understood it better than they had up to this point in time, and that they got an understanding of our decision making process.

I remember at the conference after the release of the Pearce report, it was pointed out that the veterans knew the old system pretty well and they didn't really know the nuts and bolts of this system, and that whilst you were relatively comfortable with the outcome, that is the SOPS, and that is not a claim that you agree with every SOP or with every factor in every SOP because my mail and my telephone calls make it quite clear that you don't. But that you in general agree that we've got the standard about where you expected it, and irrespective of some discussion and disagreements we still have about some SOPS, some factors and some matters, you are prepared to accept the SOP system provided that under the legislation there isn't the opportunity for it to be dramatically changed or significantly changed in the future.

And during those discussions, we were asked would we attempt to codify, and I just saw our Australian Government Solicitor who tries to keep me out of gaol, shudder when I mention the word "codification". Since the word "codification" came up at that conference, we've had a lot of discussions about what it means. We can enter into those discussions on Wednesday or at any time when there's questions along the way.

What we're going to try to do is we're going to do some presentations about how we do this thing remembering that nobody had ever done

this before. So, we were presented with some legislation and a clean sheet of paper. So there wasn't a precedent for how to do this. We're going to go through and try to let you see how we do it. I'll try to point out, as we go along, the decision making points that the RMA reaches where decisions could be made differently, and discuss that with you over three days with the background science provided by other experts to assist you to understand those points.

The presentations and slides will all be available in a small booklet. What I say is being recorded. We will type it up, edit it and we'll put it out as part of the book too so that you will have a record of the three days which we think will take us a long way towards a combined understanding of where we're at. Whether it's what you expected at the end of those Pearce review discussions, and whether it in fact satisfies your requirement to understand the legislation the way you want to, only time will tell. So, I suspect we'll be back in to discussion in the future about parts of it that you may or may not be satisfied with.

With that introduction, can I go to our first overhead. This is going to be a record of just a simple view of how the RMA goes through the process of getting to a SOP. On Wednesday, I will come back and fill in one or two of these boxes in substantially more detail, particularly the box about causal inference.

[Refer to Attachment 1]

The RMA takes requests for developing statements of principle from a number of sources; from ESOs, from ourselves, from the Repatriation Commission, from a claimant, and that may be a war widow, and veterans. So, I think you will agree that Ian McLennan's secretariat is extremely open to veterans initiating processes inside the RMA to either develop a new SOP or to review old SOPS or do formal or informal investigations.

That is a deliberate decision of the RMA. It is a decision which could be changed. I wouldn't want to be Chairman of the RMA if the organisation decided to close that door. It would be a very uncomfortable position because I know what you would do. But nevertheless, it is an open door policy, and it is in fact a policy that is deliberate and could be changed. It won't be as long as the present personnel are in place, but it is one that could be.

The second thing that the RMA does is when it's faced with a condition, it asks the question, "Is it a disease under the VEA?" If the answer to that is, "No", no statement of principle follows. That's a deliberate decision. There are a number of things like obesity which we have decided are a risk factor, not a disease in their own right. And there is a substantial discussion that goes on inside the RMA about a number of conditions, symptoms, signs, laboratory tests, risk factors which in themselves are not diseases.

Now, changes to the decisions about what is a disease and what is not a disease could again change the nature of the system. The experienced Counsel who gives us advice in these matters has told me that under the wording of the legislation, when asked "what is a disease?", he responded, "It is what you think it is" i.e. expert opinion of the meaning of the legislation. The legislation is actually that open. So, the RMA does make decisions about what are diseases and what are not diseases within the substantial latitude of the framework of the legislation. Those decisions again could be changed and would make a difference to the system.

If we decide that the issue or condition under consideration is a disease, the next question we ask is, "Is there published peer-reviewed literature which becomes evidence in relation to the disease?" If the answer to that is "No", there is no published peer-reviewed literature on the disease, we then move to this other part of the legislation which says we could use our clinical judgment. So the fact that there is no peer-reviewed published literature does not prevent a SOP being made.

Now, that is a decision which I think probably could be changed. That part of the legislation, in my view, was put there for that purpose, amongst others, and we use it for that purpose, and RMA may or may not be obliged to use it for the purpose in the future, but we certainly are on the record as using it for that purpose. That's another decision step that the RMA makes that could be changed.

So, by either pathway, we come up with a range of factors potentially implicated in causation of the disease. To do this, we nearly always have a list of potential factors that is longer than the ultimate list that comes out in the SOP. We require our staff to assist in outlining the possible associations indicated in the literature. This task was originally started by the DVA who, if you remember, did the drafts of the early SOPs for us and they in fact taught us how to do this.

In its submissions the DVA always produced a list of potential factors for a SOP which was longer than those that stood the test of causation. And that is an important step for the RMA and its medical officers. We put in submissions, things that pretty clearly when we put them in are not going to stand the test of causation, but we put them all in so that at the end of the day, we are unlikely to miss any that do stand the test of causation.

Now, that's another decision that the RMA makes which is one that the DVA in fact taught us to make, and we have pretty significant mechanisms in place to make sure that we don't miss factors that should be in a SOP by starting off with more than were actually going to finish up standing up to the test of causation.

Now, we go through a process of causal inference. I'm not going to deal with that in any detail this morning. I will come back to that on Wednesday afternoon. In the meantime, Dick Heller and John Kaldor and Terry Dwyer and others will take you over two days of coming to grips with the processes of causal inference in scientific and epidemiological terms. So, by Wednesday, you will be, we hope, much more informed about the processes of causal inference in science, and then I'll

come back to this box here and I'll deal with it in much more detail.

And I know that many of you have your major interest in this box in the questions about what is the standard of proof of reasonable hypothesis; what is the standard of proof of balance of probabilities; what are the comparisons between the outcomes in this system and the previous High Court decisions about definition of reasonable hypothesis? In that particular box I expect a substantial amount of interest on Wednesday afternoon, and probably quite a lot of questions, and about which the document at the end of the day that we exchange with you will need to be in substantial detail. But let's put it aside for the moment. I think it requires a couple of days preparation for us to deal with that box effectively.

If we decide that a particular factor does not stand the test of causal inference under the standard for reasonable hypothesis, then that factor is not included within the RH SOP. The next thing that the RMA does is that we then decide the reasonable hypothesis factors. I don't quite know why we do that. We have always decided the reasonable hypothesis factors first. I suspect it's because they're the ones that we're most interested in. I suspect that they're the ones that affect most veterans. I suspect they're the ones that when we first began, we spent most time coming to grips with.

We spent a lot of time learning about the history since 1920. We spent a lot of time considering second reading speeches over decades in the Parliament. We spent a lot of time listening to the Department of Veterans Affairs who gave us many briefings about reasonable hypothesis. We read court cases, we read High Court decisions. We listened to you, sometimes whether we wanted to or not, and so spent an awful lot of time and energy in the early days of the RMA getting fixed into our mind what everybody thought a reasonable hypothesis in the history of this system is.

Then we tried to translate that into a decision making mode that produced an outcome at what we believed was the intentions of

Parliament captured by the words of the legislation, but I think it's fair to say that the history, the briefings we had, the second reading speeches, the reasons for decisions by the High Court, and the correspondence that we had from you, the floods of correspondence that we had from you were all as important as the words of the legislation in setting the standard that we took.

In the early days we also had difficulties working out how we were going to be consistent with that standard. We had all sorts of internal tricks. We had a star system at one stage, one to five stars, and we had one star decisions and two star decisions and three star decisions, etc. When we made a decision, we'd go back and look at other two star decisions for example and see whether we've done the same thing again.

So, I don't know that you were aware that we took as much trouble as we did in those first months to understand what the intention was and to try to calibrate ourselves to make decisions consistently at that level. And the word "calibration" is going to come back on Wednesday. Where you are pushing us to codification, I'm uncomfortable that, one, I know what that means, and, two, I can legally do it under legislation. But what I can talk about at this point in time is how we calibrate the RMA; how we try to make consistent decisions at that level of proof which reflects the intention of Parliament; the history of the system, etcetera, etcetera, etcetera; as well as the words of the legislation.

A number of you have said to me that you think it might be possible with the words of the legislation to get a different set of outcomes. I think that's true; I think that's possible. The RMA has made a habit of combining on the one hand, the history culture, second reading speeches, etcetera, and the words of the legislation, to get the standard that we have been running.

The RMA decides the factors for inclusion in the reasonable hypothesis SOP. We then have to set a dose for each factor. This is where we have enormous trouble because most of the epidemiological literature was never assembled and written for the purpose

that we are now using it. It was written for all sorts of purposes: public health, advancement of people's careers; all sorts of reasons. But it was not written specifically to be used for this sort of social purpose.

Actually this exercise, as I move around the world now talking, this exercise is beginning to reveal that there are many more social purposes for the use of epidemiology than epidemiologists thought of when they were building up this literature, and actually I think that we're dealing with a model here that's going to become used and used and used over the decades ahead.

When we go out to set dose, we find that the literature uses all sorts of different definitions of "dose" which make it very difficult to compare one study with another, and frequently the literature deals with just proving something is causative, not actually concerning itself about what dose. So, quite frankly, we may have sketchy evidence on which to set doses. And when we set the doses, we are conscious of that and we are also conscious of being generous in setting those doses in the context of the history, culture, etcetera, of the legislation that I talked about before.

On occasion, the decision that the RMA makes about dose may perhaps be a "fuzzy-logic" decision and I think one which in different circumstances might be different. The one that really struck home for us in the history of this was smoking and lung cancer where those of you who remember the history will remember that we kept on reducing the dose of smoking for the factor in lung cancer. Now, the reason we did that was that at the time we were basically holding debates internally about how to set dose. And every time we held those debates, the dose for smoking and lung cancer got lower. So, we're down to half a pack year, if I recollect now. So, that reduction in dose there, was a reflection of the debate that was going on inside the RMA about how to use this fairly sketchy, non-comparable type of literature to set a dose in this environment. and that could be changed.

Once we've set a reasonable hypothesis factor we then go to the balance of probabilities factor and you will notice from looking at the SOPs that the biggest difference between RH SOPs and BOP SOPs is in the dose. It is not common for us to eliminate a factor in the BOP SOPs. It is common for us to significantly change the dose. That's not giving away any secrets, you just need to look at the SOPs to see that and I'm sure you do.

Sometimes, however, there are factors in the reasonable hypothesis SOP that do not stand the more rigorous test for BOP and the whole factor disappears. Can I go to the next overhead please? I want to just quickly run through a few of those things now.

[Refer Attachment 2]

Just to remind you what the words of the legislation are about: a disease. In fact, when you sit down to actually work very carefully through what that means, it gives the RMA substantial latitude in what it defines as a disease in some circumstances. So, it is an area in which a different outcome could occur.

Remembering that anything normal and physiological doesn't satisfy. I think the most classic example we've had of this recently is in the stress and hypertension area where many of you attended our international conference on stress and "everything", and in the stress and hypertension area, there's substantial confusion out there in the community and the media. Stress does put up blood pressure, but as soon as the stress stops, the blood pressure goes back to normal eg. whilst the person is asleep.

So, therefore, stress does not cause hypertension. The physiological response of human beings to stress is that their blood pressure goes up. But as soon as that stress stops, the blood pressure comes back down again, and it's normal and there's no evidence that it does any harm. So, we therefore don't call that sort of response a disease. That is a normal physiological response to a normal physiological activity of humans. So, those

are the sorts of decisions that come out of these sorts of words.

The RMA provides a very precise definition of diseases. Now, that's another decision process. By changing the wording in some of those SOPS where we define the disease, the system could be changed in quality. It could be more difficult to actually meet the definitional requirements of the SOP. So, a future RMA starting to change the definitions of diseases could change the standards of the system. It's a decision area in which that could happen.

One area, in particular, where you have nagged us a lot is about clinical onset. There have been a number of your representatives who have pushed us very hard to define clinical onset. We have not done so, and we don't intend to do so. We have always pointed out that it's an example where, as soon as we write down a definition for clinical onset, we will make the system more restrictive. And I think it took some of your representatives quite a while to come to grips with that.

As it is operationalised now, a clinical onset means what the English language usage of those words means. It means the first time the veteran noticed anything to do with the disease. Clinical onset is not when it's diagnosed, not when the first laboratory test or X-ray is done. Clinical onset in its ordinary English usage means the first time the patient notices anything to do with the actual disease. Now, that is a much more generous interpretation than anything we could write in terms of laboratory tests or diagnostic processes, therefore we have refused to codify. We've refused to write it down.

It also fits in better unwritten with the other part of the legislation about developing a reasonable hypothesis. Remember that the reasonable hypothesis involves an assessment of the whole claim. It involves an assessment of the likelihood that the veteran's claim about what happened at war or as a result of war is true. It revolves around when the disease started in relation to the war service. It revolves around meeting the template of the SOP, so that the reasonable

hypothesis is a total package, and we make a template in the SOP end.

The more we were to stray into making definitions about disease onset, the more we would go into that other part of the reasonable hypothesis which are the questions about what happened to the veteran in war which, in fact, is none of our business. But another RMA, if it decided to push the legislation could begin to make written pronouncements about clinical onset - remember that once we have written something and it sits on both tables of Parliament for two weeks, it becomes law. So, it then may become a more restrictive template.

So, once we write it, once it becomes the law, and if an RMA was to start pushing towards that other end of reasonable hypothesis, towards when did the disease come on, how do you define when the veteran first got sick, it's not a big step to then saying, when did the etiological factor occur, and that RMA is already then dabbling in the other end of reasonable hypothesis where, in our view, it has no place. We believe we are restricted to the factors that cause disease. We are not involved in the rest of the construction of a reasonable hypothesis.

So, I've always seen the push from you to get us to define clinical onset as being a bit of an invitation for us to move into an area of the construction of a reasonable hypothesis where I don't think you want us to be. And that's the reason why we always resisted doing it. So, if you get a new RMA in the future, I wouldn't push them in that direction. I would not push them in going in that business of defining clinical onset.

[Refer Attachment 3]

Again, this slide says that we don't make biochemical tests a disease. A simple rise in the biochemical parameter is not defined as a disease by us. And we don't have broad spectrums like kidney disease and heart disease. We act much more specifically than that. So, that we have got some history, some

track record of how we handle this part of the legislation which is clear to you.

[Refer Attachments 4 and 5]

You'll notice that we've used the word "information". We collect a lot of information. We don't collect sound medical scientific evidence at the first step. We throw our net much wider than that. We collect a lot of information and then we decide which of it is sound medical scientific evidence that we can use.

Now, if you did that the other way around, I mean to say, if what the staff or you or the DVA put in front of the RMA was only already processed information that had been defined as sound medical scientific evidence by the Act, and we'll go through in the next two days about how the sound medical scientific evidence decisions are made, but remember the RMA gets a lot of information in front of it which is not yet sound medical scientific evidence, that's an important decision because it means we look at a lot of background material without the restrictions of that part of the legislation.

We can't use it for making the decision about causal inference for the factors later, but we use it to make sure we don't miss factors. We use it to make sure we get a broad feel for information about the disease, and we get that from all sorts of sources. So, again, this RMA has used a wider net to assemble the material that ultimately becomes sound medical scientific evidence than the words of the legislation themselves require us to use. I believe that is quite legitimate. We only use material that stands the test of sound medical scientific evidence to justify factors, but we look much more widely to get a feel for where we might be going to go with factors.

The rest of this slide sets out a whole lot of things that we do, and they'll be in the papers. Some people that deal with us seem to lack confidence that we do thorough searches, and so people will ring us up or write to us and say, "Oh, have you really looked at this or have you really looked at that?" And I think people are surprised when

they look at our files on these diseases to find the enormous extent to which our staff go to make sure that we do not miss any significant published material on a particular disease. It is something that we do extremely carefully so that we go through all of these steps, we look at all sorts of places to find material that might bear upon the disease.

We use all sorts of places. We even access the Internet. Now, a lot of the stuff on the Internet could never stand up to being sound medical scientific evidence, but it's information that we use in trying to assemble what ultimately is sound medical scientific evidence. So, a list of sources of information will be provided as part of your papers. There's no need for me to go into it in great detail. There is a distinction between information and sound medical scientific evidence when we finally come to make the decisions.

[Refer Attachments 6 and 1]

And the last slide I want to use this morning is an introduction to where we will go on Wednesday. On Wednesday, we will, in much more detail, look at that box in our processes which talks about causal inference, and there we will talk about it in three headings roughly. We'll talk about it in the heading of acting lawfully and the sorts of things that we do because we are bound to act lawfully. We have a permanent appointee from the Australian Government Solicitor's office to see that we do, and Cherrie puts her hand up every time she thinks we're not acting lawfully, no matter whether that's to the advantage or to the disadvantage of the veterans, or what it is. She simply tells us as soon as she thinks we're not acting lawfully. So, we are very careful to make sure that we act lawfully.

We will also talk to you about the way in which we go about making causal inferences, and we'll go into that in some considerable depth, over the next day or so, and there will be a significant number of papers about that. And then I will talk to you about how we calibrate the RMA and how

we try to get consistency in our decision making. We'll talk about the processes that we've used to try to get the current set of SOPS which cover about 91 percent of claims.

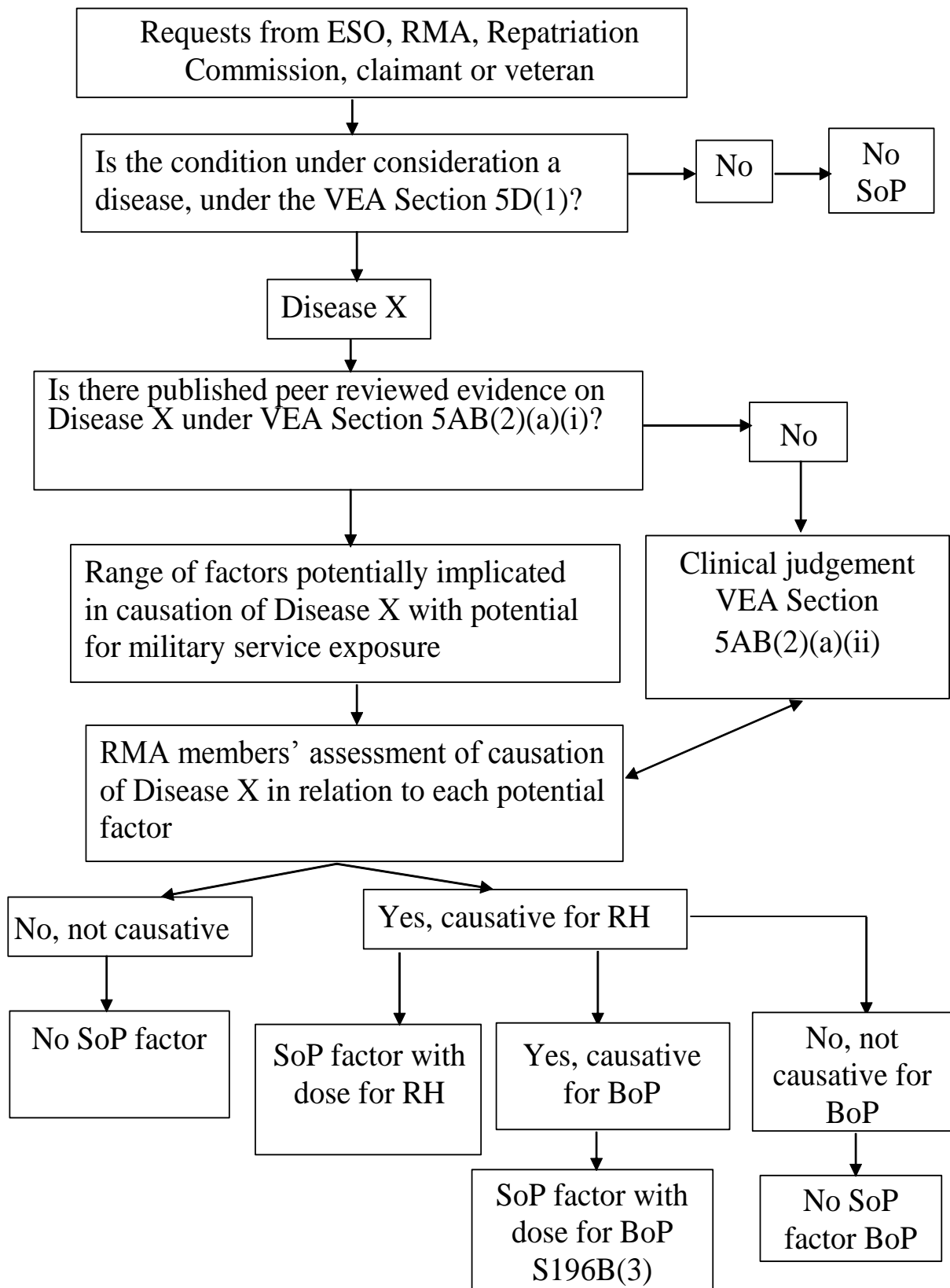
We're hoping to continue to push that up till we get to the 100 percent of claims covered by a statement of principle, and the methods by which we've tried to calibrate ourselves to do that at the standard of proof that we think the system intended, the Parliament and the system, and how to do that consistently. But that's going to take another session, perhaps

a bit longer than this one, to go into that in substantial detail on Wednesday.

I hope we can, over the next two or three days, get to understand this system better, and we still think we're learning about it, but nobody has ever done this before, and we are sure that there are bits of it that we haven't thought through properly, and we're looking forward to you telling us things that you think we should be looking at, and we hope we can get you to understand what we've done so far so that you can comment on it and so that you can keep an eye on it in the future. Thank you very much.

Attachment 1.

Determination of Statements of Principles



Attachment 2.

The definition of “disease” (section 5D(1) of the VEA) **is as follows:**

“disease” means:

- (a) any physical or mental ailment, disorder, defect or morbid condition (whether of sudden onset or gradual development); or
- (b) the recurrence of such an ailment, disorder, defect or morbid condition;

but does not include:

- (c) the aggravation of such an ailment, disorder, defect or morbid condition; or
- (d) a temporary departure from:
 - (i) the normal physiological state; or
 - (ii) the accepted ranges of physiological or biochemical measures;

that results from normal physiological stress (for example, the effect of exercise on blood pressure) or the temporary effect of extraneous agents (for example, alcohol on blood cholesterol levels);

Attachment 3.

Further considerations regarding “disease”

The RMA provides a precise definition of each disease which is the subject of a Statement of Principles. This is undertaken in accordance with the VEA.

The definition contains both a description in words and the ICD code/s applicable to the disease. The ICD coding alone is considered inadequate for this purpose.

For the purposes of SoP development the RMA generally does not classify as disease processes,
symptoms,
signs,
results of biochemical or haematological tests, or
potential risk factors for subsequent disease.

Additionally, broad classifications such as kidney disease, heart disease or liver disease, which each cover an enormous mantle of individual pathologies, are not currently the subject of SoPs.

Attachment 4

Collection of Information Once a Statement of Principles on a particular kind of injury, disease or death is to be made (consistent with section 5AB(2)(b))

Submissions from interested parties considered and references sought.

Literature Search for Factor Development

Ovid Medline database search

This database, operated by the National Library of Medicine in the USA is comprehensive and dates back to 1966.

Range of search techniques re disease/exposure.

Epidemiology/etiology/chemically induced/

Military/etc

English language+

5-10 years+

Specific searches

Textword and author searches

Attachment 5

Collection of Information Once a Statement of Principles on a particular kind of injury, disease or death is to be made **(consistent with section 5AB(2)(b))**

(cont'd)

Other sources of information as necessary

Referenced texts (eg Harrison's and Scientific American Medicine),
Monographs (such as NAS, US Surgeon General, IARC, NIH),
Internet
Other databases eg psychlit, sociofile, drug related databases used.

Note the distinctions between information and "sound medical scientific evidence" as defined in section 5AB (2) of the VEA.

Strategies for considering non-MESHed literature include review of evidence from reports of large prospective cohorts already on file which may be examined where applicable (eg British Physicians Study and results for mortality with smoking and alcohol exposures).

RMA Members' Assessment of Causation

Acting Lawfully

- ◆ Legislation
- ◆ Body of information considered
- ◆ Design, quality and results of individual studies

Causal Inference

- ◆ Use of Bradford Hill and similar criteria
- ◆ Judgement
- ◆ Personnel

Calibration & Consistency

- ◆ SoPs themselves
- ◆ Further considerations

How the RMA Goes About the Performance of its Functions.

Professor Ken Donald

Chairman of the Repatriation Medical Authority

11 November, 1998

[This document was prepared from the audio transcripts of the Forum and has been edited for clarity and continuity only.]

PROFESSOR KEN DONALD
Chairman of the Repatriation Medical Authority

Yesterday afternoon while you were doing the factors exercise, I re-read the legislation, and by five o'clock yesterday afternoon I understood it perfectly. Unfortunately, as a result of dinner with a number of you, I can no longer remember all I understood. However, I am pretty safe because I know that you can't either.

[Refer Attachment 1]

What I want to do is put up Monday's slides again because I understand the background we had on them was not very visible at the back of the room, and just very quickly recap on some of the crucial issues out of them that I want to carry forward into today's discussion. I have interpreted your request of today's session, both in terms of your writing to us and copies of correspondence that you have had with the Minister which you have been kind enough to give me copies of. From that I have understood that what you really want to do today is to understand how the RMA goes about its business so that that can be, in a sense, put on record so that at least in the future, if there is a new RMA membership you will have some record of how we did our business in respect of getting to the Statements of Principles and the factors.

What I can't tell you is what goes on in John Duggan's mind when he agrees to put a factor in. I don't know what goes on in John Duggan's mind, or Dick Heller's, or Beverley Raphael's, or John Kearsley's. So I can't explore that point of decision-making for you, and I don't know what goes on in the mind of a High Court Judge when they create definitions either. So that's not what I want to talk about.

What I want to talk about is the process we go through and the steps we take. Now, this is being recorded and we will give you

a formal version of it with the papers for the conference. I will reserve the right to edit it because I am not sure that I am not about to talk a bit of nonsense here and there, but we will give you a document based on the overheads that I am about to run through and a fair summary of what I say today. I know from past experience that you will make sure that it is just that.

So what things to look out for? Well, I think the first thing that you need to look out for is that the RMA has established a dialogue which is not dependent upon the formal powers of investigation under the legislation to look at issues that you raise. Ian McLennan and our medical staff receive a large number of phone calls and letters of things that veterans want to have looked at, and we look at them all. I think that a sign that you would need to watch out for is if the RMA was only prepared to respond in the formal sense of the legislation to investigations.

We believe that the informal process of looking at things raised by a telephone call rather than a formal legal request has been a very important part of the process. It's also done, to some degree, what yesterday's session took forward, that is it has given us an understanding and a feel for the things that the veterans are concerned about. So an RMA that did not keep that avenue opened would be one that I think you would need to keep a close watch on. So that's that first step.

Now, reviewing the first OHP, while we actually consider disease, injury or death for simplicity here we will consider disease. Is the condition under consideration a disease under the VEA legislation? On Monday I talked about the fact that we don't use risk factors or laboratory tests as diseases, but there is another important element in that, and that is that we have the track record of SOPs which shows that for nearly every disease in the book or in the codes, the RMA has decided that they could be war-related.

There are some countries where that decision has not been taken, and it is an

important decision. It means that about the only diseases that aren't included to be considered as war-caused are genetic diseases. We have, of course, got SOPs about genetic diseases because genetic diseases may be made worse by war. But apart from genetic diseases, I can't remember us ever making a decision that a particular disease could not be war-caused.

The factors or events that caused the disease may have happened directly in a war, however war may also change behaviour. The RMA recognises that war may cause mental health disease, and we have factors for that, but war may also change behaviour without creating mental illness as such, and that changed behaviour can be the chain to diseases that are related back to that war because most of the modern chronic diseases have a lifestyle factor as part of their causation process.

We don't look for one cause for any disease. Most diseases or the vast majority of diseases have a multiplicity of causes, sometimes acting together and sometimes acting separately. And those lifestyle changes that follow as a consequence of behaviour change without mental illness from war is a pathway that this country has accepted. There are precedents for it having been accepted in the old system. We have never questioned the precedent, for example, that cigarette smoking can be related to behaviour change in war service.

There are some countries where that is not an accepted pathway. So it is very important under this consideration, "Is it a disease under the VEA?", and further down in the legislation, "Can it be related to military service?". That is an important decision which the track record of this RMA is clear about and if it were changed there would be a dramatic change in the Statements of Principles, and you would soon notice.

Can I go on to this one "Is there published peer reviewed evidence?" Now, this brings us into 5AB(2). I think from the last couple of days you now understand, pretty much,

what we actually do. I don't know how you define that in legislative terms, and I'm not actually going to try to do so, but you know what we do. We look at all the information we can get our hands on, and we assess it. We look at its design. We do all those things. If you like, we prioritise it, we put it through a sieve.

At the end of that process we finish up with a bundle of material called sound medical scientific evidence that we then use to make a decision about causality. So our view is that this is the business of the peer reviewed literature and our clinical judgment. Then based in our clinical judgment are things that we already know which we can't avoid knowing, things that people tell us which may not be part of peer reviewed literature. It is impossible for us to escape our experience and clinical judgment when we are deciding what is sound medical scientific evidence and what is causal.

So that constellation of having this linked to the peer reviewed literature was deliberate, in my view. I was around when that was done, when DVA put that in for the legislation, and in my view my recollection of it is that it was put in for just the purpose I have described, and it was put in because, as the Minister said in the Second Reading Speech, there was no intention to change the standard of proof. It was put in so that the RMA was not bound by solely the numbers calculation as a purely statistical exercise; it was put in so the RMA could make judgments, expert judgments.

I am perfectly comfortable about the way we use it. I don't know how many "meanings" there are of 5AB(2) about – I am aware of several – but that is what we do, and I believe that what we do reflects what was intended by the Parliament, what was intended by DVA when they wrote the legislation, and what was intended by the Minister in the Second Reading Speech, and in fact what was intended by both the Full Court of the Federal Court and the High Court in a number of decisions in which they made statements about the standard of proof.

So I don't actually believe that we have changed the standard of proof, and I don't believe Parliament asked us to, and I think the legislation allows us to do that, and I think there are other words in the legislation which I will mention as we go by that I think give us the irrefutable authority to do that. When we come to this box, I didn't cover that on Monday, and that's what I want to go on to in a moment.

I remind you that we make the reasonable hypothesis SOP first, and then having made the reasonable hypothesis SOP we then make the BOP SOP and in each of these Statements the question of dose is considered. So I wanted to reiterate those particular issues about Monday's slides to bring us to the start of the next phase which is the RMA members' assessment of causation.

The fact that you have now had the last two days experience is, I think, extremely valuable for this following discussion. It would have been, I think, inappropriate for me to discuss the RMA members' assessment of causation on the first morning. I think now that you have shared at least some of the feeling and some of the process of our experience, this particular set of slides is going to be more meaningful to you and I might say more meaningful to us. As Dick Heller said to me yesterday afternoon "We've learnt a lot from the last two days as well." By the way, having done something like this for the first time, even four years into it, we are still learning how to do it; we're still learning some of the nooks and crannies that we hadn't expected to be there. So for us this has been a learning experience, too, and that's another reason why doing it late in this session is more valuable. I think I understand what I am supposed to do better.

[Refer Attachment 2]

I am going to deal with these under these headings "Acting Lawfully" and the "Causal Inference Process", and then some

questions about calibration and consistency that the RMA tries to achieve. How do we calibrate ourselves and how do we try to get a consistent program? A couple of quick points I want to make here is that you will notice that we have used the words "body of information considered". Now, the legislation uses the word "information". The legislation uses the word "information" as a preliminary step to distilling out the sound medical scientific evidence that will be used finally to make the decision.

You have gone through some of that process with us when you have looked at the design, quality, and results of individual studies. Two days ago you went through that process of looking at the information and prioritising it so that it was being made ready to be used as sound medical scientific evidence. Not discarded, but being made ready to be used as sound medical scientific evidence. Now, with, if you like, priorities or weightings upon it as a result of that process that you did in the last two days where you looked at how we go about deciding the quality of the studies.

So we don't throw them away; we assess them; and we figure out how much we can rely upon them to make decisions, but we use them to make the decision. So I think that is what we do, and I don't notice any members of the RMA actually shaking their heads this way. I think what I have enunciated is what we actually do.

Now, as far as we are concerned, that's not a legal debate. John Kaldor put it well to me at breakfast this morning when he said his view of it now, having had his experience with it, is we have a body of evidence over here, and we have a decision over here to be made, and we turn that body of evidence into a decision, and the legislation enables that. So I don't think it is necessary for us to enter into any sort of debate at all about what words in the legislation might or might not mean in a legal context.

If a Judge somewhere tells us what they mean some day, then we will clearly behave lawfully and look at that, and take

advice. I want to digress for a minute. We have had the experience recently where some legal decisions on cases have, in our view, remade those decisions that we make. That is, they have remade the medical decision. Now, this legislation specifically precludes the courts from remaking our medical decisions. So when that has happened, we have gone back to the Statement of Principle and we have revised it. We have revised it to make it absolutely clear what we meant.

That is exactly what Parliament does in exactly the same circumstance. That is, if Parliament is in a situation where the known facts are explicit, the legislation is in place, and a court makes the decision which does not reflect the intention of Parliament in that legislation, the Parliament will say "Oops, we must not have been clear in what we wrote; we will rewrite the legislation".

One of the comments I would like to make about such revisions; and it came up fairly substantially at the Maroochydore RSL conference earlier this year, and there has been another example of it recently; is that when that happens, it appears that when we go back to review the SOP we may make it more restrictive. If I can give you an example, where we in a factor use the words "for example" and then give a list of things that we think are "for example", Our legal adviser for sometime has been saying to me "That is from the legal process point of view, a dangerous thing to do" because that list of "for example" can be extended by the courts who will make medical decisions possibly not in the same way or at the same level as we intended, and therefore when the courts do that, we will almost inevitably have to take out the "for example" and restrict the list to only those things that we can think of.

We put the "for example" in there as scientists. We didn't believe that we could necessarily be sure that we could think of everything that should be on the list, so we put "for example" which is the sort of thing a scientist does in that circumstance. But we hadn't realised that in doing that, we actually turn over the decision-making

process to the legal system who may or may not make the decision at the same level or in the same way.

This is one example where we are learning things about the system as we get to handle it more. The outcome of that has been, in one case, the removal of the example of medical intervention in injury from the definition "trauma to the relevant joint". When we took that out because the court had extended the "for example" to a degree that we believe was not in line with what we had intended.

Remember, we have responsibilities to be guardians of the credibility of the system. So we don't want this system to be criticised in time because it is inconsistent or because it has in it unjustifiable elements. So in our role of protecting the credibility of the system we took medical intervention completely out of the definition. We then got a flood of correspondence saying "Oh, that's too tough", and we have had a look at it again and quite clearly it is fair to say that we over-reacted, I think, to that particular issue, so we will revisit it and we will put back something but not the words "for example"; that's gone forever. We will probably have to get rid of "for examples" out of nearly everywhere we have used them in due course, but we will put in a list which is what we intended the first list to be.

So those sorts of events are happening to us along the way and as I say, they are things that we are learning about this process that I wasn't aware of six or twelve months ago. I was looking for an opportunity to put that digression in because I did say I would talk about it.

Can I just go over here and before we go through the slides point out that when we get into causal inference, we have got three dot points under there. It says, "use of Bradford Hill and similar criteria". The legislation and the Second Reading Speech do not bind us to the Bradford Hill criteria alone, and they don't require us to match any particular number of the Bradford Hill criteria even when we are using them. It also, under "causal

inference", has elements of judgment and that is very strongly linked to the personnel of the RMA, their backgrounds, their clinical judgment, their experience, their calibration of where they are coming from in this system, not where they are coming from in some other system.

I think we saw a very good example of that during John Kaldor's presentation. I do accept John Printz's view that Dick Heller is really after my job, but there is another possible interpretation of the Heller phenomenon which is that - and Dick may be embarrassed by this and I haven't checked his permission to actually talk about it, but it happened and we all saw it - I think Dick did that because he was calibrated differently in this environment.

That is why I have said a few times now that the issue is how you calibrate the RMA, by the Second Reading Speech, the readings of the High Court decisions, the interaction with the veterans, understanding of the culture, and the briefing by the Department of Veterans' Affairs. Contrary to what some people sometimes think, a great deal of credit for the calibration of the RMA in taking on this task lies with the DVA. Officers of the Department have taken a great deal of time to participate in calibrating the RMA.

We were new boys on the block. None of us knew anything much at all about the veterans' compensation system. So we went through a period earlier on when we were calibrating the RMA. We still hadn't finished doing that when some of the legal argument started to emerge, and I didn't understand what the legal arguments were about. I knew I was trying to calibrate myself and calibrate the organisation, but I wasn't too keen on a debate about what words meant in bits and pieces of legislation.

So we went through a period where we were in that process, and I repeat that there were a number of influences on the RMA that made that process, and the DVA was certainly strongly one of them. So I think Dick's view on that difference was because he was thinking in RMA mode.

Now, I think if you took him into another environment, if he spent a week in San Francisco with a whole lot of epidemiologists and having some hard-nose discussion about things in that scientific context where proof is much more difficult to get your colleagues to agree to, I think you would get a different outcome.

So I think it's a question of the judgment, the personnel of the RMA, and how they are trained and calibrated to this system that is important. We are conscious that if the government asked us why we put a factor in there, we have got to be able to point to sound medical scientific evidence defined by us, and we have got to be able to point to some applicable epidemiological criteria that we used. So you are not our only masters; we have got to be able to justify to Parliament our activities as well.

So we have to act lawfully, and I think the process I have just outlined, in our view does. I don't believe the legislation is any impediment to doing what we do as we do it, but if somebody decides somewhere with proper authority at some time that that's not the case, we will obviously have to listen.

Now, calibration and consistency are other things that I will deal with again towards the end. Much of this you have now already been through. Dick Heller was saying this morning that with the way you have appreciated the concepts of confounding and various study designs, it has been fairly astounding. You have actually caught on, haven't you, very quickly, to these sorts of issues so that I think you do understand much more now the processes we go through.

[Refer Attachment 3]

Remember that we look at the military and non-military literature, and most of what we look at is non-military literature. Now, if we didn't use that literature, there would be very few factors and very few SOPs because that's where most of the factors come from. For many diseases there's not

much or nil military literature upon which we can draw.

We try to go back to the primary papers rather than other people's assessments of them. So an RMA in the future should make sure that it doesn't just take another group of scientists review of the literature as its own. The RMA should go back to the primary data, the primary research, and make its own judgment of them, for fairly obvious reasons I think you would now agree.

Reviews and meta-analyses are however still helpful. They are sign posts for us. When I first started this job I can remember saying that I thought that meta-analysis would be a very important part of this process. It hasn't turned out to be so. In fact, meta-analysis as a methodology has some difficulties with it anyway, but in this context meta-analyses are a guide post to us because the process that scientists might have used in writing a meta-analysis is not necessarily the process we are using under this legislation.

So meta-analyses are a part of the information, if you like. They are no more part of the sound medical scientific evidence than anything else in the information that we start with because they are somebody else's view of the literature. They're not our view of the literature. So they may or may not become sound medical scientific evidence for our final decision, but they're part of setting our clock, if you like.

When we look at study design, and quality, as you have done with us now, we give it a sort of soundness rating. With us, soundness is not a black or white decision; soundness is a quality rating. When we have a lot of good sound evidence, we will make decisions with confidence that we would not make without the evidence being good and sound, and I will come back to that in a little while.

[Refer Attachments 4 and 5]

Now, issues relating to consistency and calibration, the setting of the standard of the RMA for factor inclusion and development. Body of evidence: these words, I think, are very important. We use the Bradford Hill and similar applicable criteria. I think the key word in there is "applicable". Applicable means, in my mind, what we think is applicable. The legislation doesn't tell us what is applicable. I doubt whether any court can tell us what is applicable. I think Parliament could, but it would have to do so openly and in formal terms, or by legislation change. It may be able to write to us. But I don't think anybody else, other than Parliament by a formal process, can tell us what that word "applicable" means.

I think it means we can use epidemiological criteria that are relevant in our mind to the data base we have at any time and the decision we are trying to make. So we don't have to use the same formal list of criteria on every occasion. And the list we will use or the applicable criteria that we will use will, to a large degree, be affected by the quality and amount of the sound medical scientific evidence we have got, its characteristics, and our own clinical judgment of the likelihood that this is a reasonable hypothesis.

Can I remind you at this point of decisions by the full Federal Court and the High Court. I think I can give my own fair summary of what they have endorsed over the years. In a reasonable hypothesis they appear to me to require three elements. They have said not too tenuous, not too remote, not too fanciful. They have said it must be more than a possibility. They have said that in very clear terms. They have said that in exactly those words, as I understand it. And they have said it must be consistent with the known facts, ie not too remote or not too fanciful, more than a possibility, and consistent with the known facts.

Now, I think if you combine those words, I do not argue with the intention. They are the words of the High Court. They seem, from a scientist's point of view (not a lawyer's point of view) to be a sensible

definition of reasonable hypothesis in this context. It might not be the definition I would use of "reasonable hypothesis" in a scientific research project— but that's not the context. In this context, I don't have any argument with it.

So I think that the crucial elements that lead the RMA currently to make the decisions in the reasonable hypothesis SOPs that it does are its choice of applicable criteria which is within its own power, the quality of the sound medical scientific evidence that it has distilled from whatever information is available to it, and due cognisance of the High Court's definition of "reasonable hypothesis". So I think in my mind, at the kernel of the decision-making process, is that constellation of, if you like, issues or statements or whatever.

Now, as a consequence of that, it is plainly on the public record, just by looking at the SOPs and by looking at the evidence, sound medical scientific evidence if you will, that we have in our files, it is clear that we will respond for reasonable hypothesis at a relative risk very close to one, not infrequently 1.1 but very occasionally below 1.1. Now, you heard from Terry Dwyer and John Kaldor the other day that in some other context where the constellation of events isn't the ones I just outlined a moment ago, scientists would be more likely to respond at a relative risk of 2, and for some purposes even at 3 or more depending upon the consequences of their decision.

So the idea that scientists always make decisions at the same level of proof is actually not true. They make decisions at various levels of proof depending upon what they're using as sound medical scientific evidence, what the definition of the hypothesis is that they are working to, and what the purpose or outcome of the decision will be. Now, they're just ordinary people. They can't avoid the influence of any of those inputs any more than you can.

So the idea that this is black and white is not accurate, which is one of the reasons why calibration rather than codification is

what we can talk about. However, in your view, this may or may not be codification. As far as we are concerned this is the way we do business. So, yes, it is a matter of public record that we will put in a factor pretty close to a relative risk of 1 under circumstances where the data, our judgment, and the definitions that surround us come together. There are times we don't and if the data is poor or conflicting, and the studies come up with ambivalent results which are all over the place, under those circumstances we are less likely to go close to 1.1. That's because when our mind is set, when our mind is in decision-making mode, the doubt raised by the poor evidence influences the decision. You can't avoid that. You couldn't avoid it yesterday in the consideration of factor development. We can't avoid that either.

Personnel of the RMA: I think it has been an enormous strength of the RMA. First of all, can I say they have been four of the most pleasant human beings I have ever worked with, so I think we have been very lucky in the way in which the selection process came out. We got a group who I believe work extremely well together. We have had enormous good luck with our Secretariat who participate and have no hesitation in offering advice that we frequently listen to. We have been very lucky to get on board some very good medical staff.

It is the combination of the five members of the RMA that actually makes the final decision, and as I say in a way which I don't understand everybody else's exact thought processes. We never vote; we always talk it through until we get consensus. That was one part of the process that you didn't see yesterday or the day before.

John Kaldor demonstrated how a group of experts, or for that matter a group of non-experts, come up with a scatter of opinions at the end of part of this process. What you didn't see was the consensus process that then follows. John alluded to it. He said with continuing discussion some of the people down in that bottom rung of the decision-making module would move up

and some of the high ones would move down.

So we have had some decisions sit on our plate for three, four, five meetings. We have discussed some things for a year before we are comfortable that the five of us, and our support staff, are in agreement. So there is another process beyond what you saw the other day which is vitally important, and as I say it's at that point that I don't really understand what is going on in John Duggan's mind, and he doesn't understand what is going on in mine when we are going through that consensus process. I don't know which pieces of information or which pieces of legislation influence his final decision. We just talk about the data, talk about standard of proof, we talk about the decision, and ultimately we look around the table and we finish discussion and a decision is made. There is no more discussion to be had.

So that is another important part of the process. The judgment, as I have just said, takes into account all of that. I will reiterate it takes into account the definition that the High Court made of "reasonable hypothesis". Again, I have mentioned beforehand that we have made a decision that most diseases have the potential to be war-caused by a variety of pathways. That's been a deliberate decision. We have talked about it a number of times, and we have always come back to the conclusion that only genetic diseases can really be left out, except where war makes them worse.

Now, what are the issues that are important in this process of calibrating the RMA for this specific purpose that it's meant to do rather than for some other purpose and to get it into a consistent level of decision-making. You have picked occasions when you have written to us and said "That decision isn't consistent with that one" and we have looked at them and they haven't been. So it's not something that you can do perfectly, but the attempt to be consistent is important, and the attempt at calibration of the organisation is important.

What are the things that I think are highly relevant to maintaining that? Well, I think the very first thing is the outcome. The SOPs are there. They are a measure of the calibration and the consistency of our efforts. Now, they are instruments that can be readily changed. We can change them if we look at them and say we got that wrong, and scientists do that. It's the way we work. In fact, it's much of the way science makes progress. So we're quite comfortable, I think to the surprise of some legal organisations who wrote to the Pearce Report and said we change our mind too often. We have no problem with changing our mind. It's what scientists do.

So we can change a SOP if we come to the conclusion it is wrong. We can change a SOP if we come to the conclusion that the courts have misunderstood it, and we can change a SOP if the evidence changes, the literature changes, new studies are done. So, yes, they're readily changeable instruments. But nevertheless, they are a record of what a reasonable hypothesis is. They represent a standard, and I think part of this process for us, and it was a very sensible recommendation of Pearce, is that you can understand that too.

Secondly, the RMA actually asks the question "Can a factor be included?". In my view, if you ask the question routinely "Can a factor be excluded", it would have an effect on the standard of proof. I think if an RMA asked "Can a factor be excluded?" as its routine question, you would get a different outcome, so I think that's an important issue. Also, there is no deduction with respect to age. In other words, we don't routinely, as many scientists would, ask the question "Now that somebody is 80 what is the real possibility that something that happened when they were 20 is still actively contributing to a disease process?" When I presented material on the RMA to my own University Department, the first question that our statisticians asked me was "What discount do you make for age?". That was the very first question they asked me.

I said "none". Under different legislation, under different circumstances, under

maybe a different RMA – I don't know, I doubt it – but under different circumstances that question might be asked in making the decision. “How likely is it that somebody aged 80 is still being influenced by an event that happened when they were 20?” Not asked. I believe that the intention of Parliament was that that question should not be asked. I think that is something about which the Australian people, by their representatives, have indicated a clear decision. I think the speeches that go right back into the 1920's and the speeches in parliament since, reflect the view that that question is not on the agenda.

Dose estimates: Much of the epidemiological literature has not been written to quantify dose. Some of it has, most of it hasn't. This is the most difficult part of the exercise because here the evidence is least reliable, least available, and we make decisions about dose that I believe are at a very generous level. In some of the reviews of us by either SMRC or Pearce, there have been comments to that effect. I have no objection to those comments; they are true. We make dose decisions at a very generous level.

I believe we are meant to. I have no compunction about it. I have no concern about it, it is a fact. The Holman calculations I think are an important piece in the puzzle. Not everybody would totally agree with Darcy's methodology for calculating it, but Darcy is a very experienced, very capable epidemiologist, but as you are well aware in methodological debates there would be schools of opinion that wouldn't use the same methodology. It is not meant as a criticism of Darcy. His calculation that when a pension is awarded using a SOP with its attendant dose as part of the reasonable hypothesis chain, that you can make a calculation which has got several pages of formulae behind it, that our track record is that that will happen with a chance of somewhere between 5 and 10 per cent of being a true causal chain.

The government has accepted that as a reasonable standard. I think that's the standard, and I am not saying that I believe

Darcy got it right, I am not prepared to go that far – that's just because I don't think I should – but if that calculation is correct, the government has accepted it, I don't have any problem with it, and it fits the High Court definition of a reasonable hypothesis. It is more than a possibility. It's not too remote or too fanciful, and it's consistent with the known facts. It has to be consistent with the known facts. There's three pages of calculations and formulae, so there must be some facts going into the assumptions.

In fact, I think as I pointed out at the meeting following the Pearce Report, it even is in line with one of the High Court Judges who said "a reasonable hypothesis is something around a 20 to 1 chance". It fits that. So I don't actually see, to be honest, too much of a problem in any of Darcy Holman's comments. I think we have got a number of elements pointing to the fact that we are at about the right standard. We seem to fit with what the High Court said. Darcy's review is another method of looking at it; it comes up with the same thing. We, as an RMA, have a view that we have understood "reasonable hypothesis". We have a view that we have been well calibrated to what it means in this context by a whole range of people of whom I have named some before, including yourselves.

So I actually am fairly comfortable that we are running this thing at about the standard that the Parliament expected. As I said, we have had no complaints from the Parliament. Not one of our instruments has been challenged by the politicians. They have all been through Parliament. Parliament has the opportunity to challenge them; it has not done so. But more than that, the government has endorsed the Pearce Report, and the Pearce Report talks about the standard of proof, and the government has, in our view, put a tick on that standard.

I think the last thing that I want to say is that in looking at future RMAs, and in dealing with them, it is very important that the RMA attends ESO meetings, veterans' meetings, and forums like this. If you run

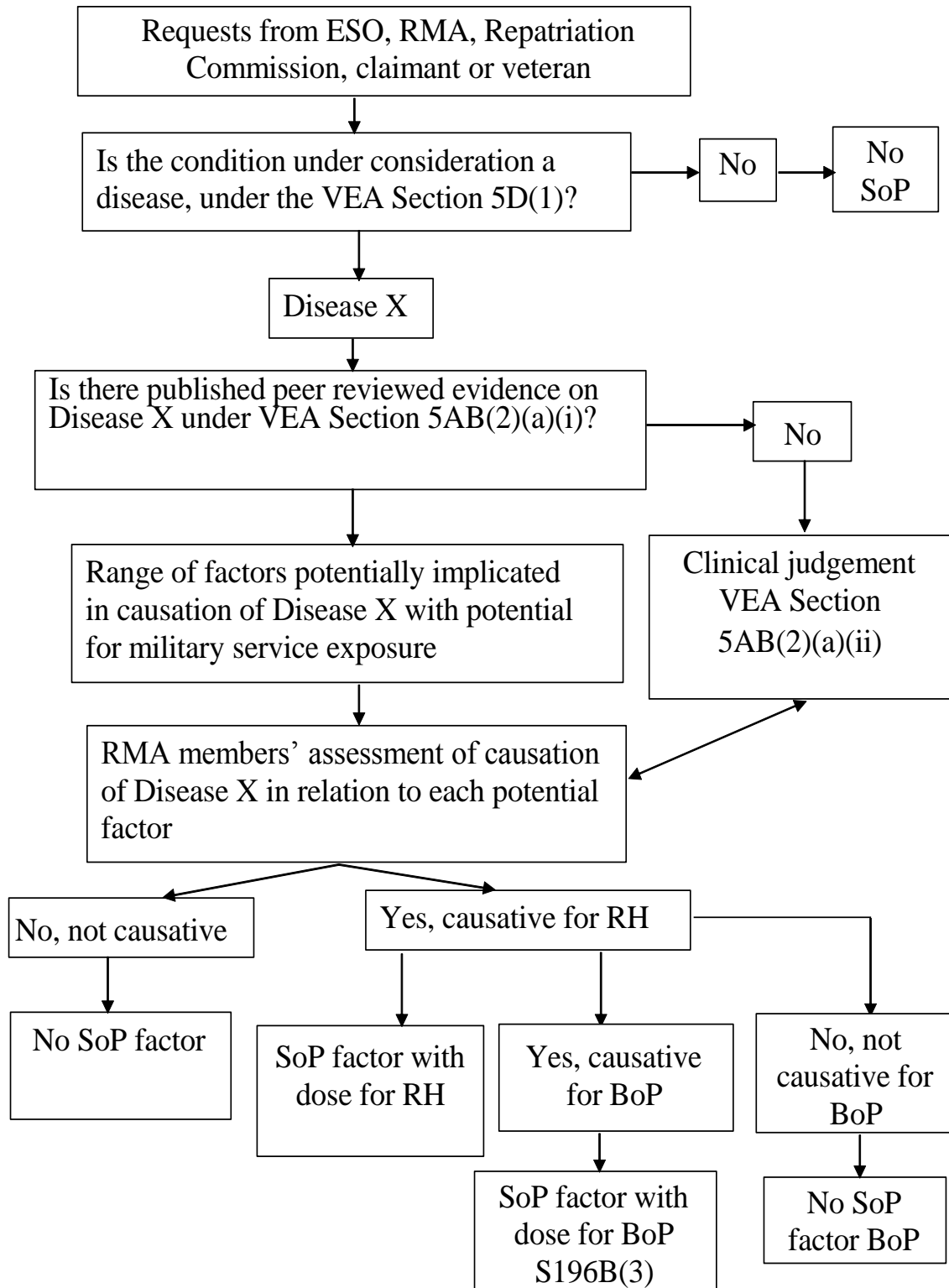
into an RMA that retreats behind the legislation and is not prepared to come to dinner with you and justify – not necessarily justify, not even defend – but face up to the times it says no, then I think that would be an occasion for me to start thinking about it because if a group of scientists is doing what they think is an honest job, they will have no hesitation in coming to dinner with you, and they will have no hesitation in saying to you "Yes, we said no".

They might not try to justify why they said it, but they will have no hesitation in keeping on coming back. So I would take that as a sign in a future RMA if people decided to hide behind the legislation, that you need to be cautious of the outcome. What I am trying to do is to take you through what we do as I see it without entering into semantics about what the legislation means. I believe that what we do is lawful. I can see nothing in the legislation that changes it. I think some of those key words that I have pointed to clearly, in my mind, make this process a sound interpretation of what the legislation and the Parliament intended. Until a court tells me otherwise, that's the way we're going to do it. Thank you very much.

N.B. - In the context of the "calibration" of the RMA, Professor Heller, in comments after the presentation, highlighted that the RMA reviews Statements of Principles and that this was a method used to maintain quality control similar, in principle, to that used in the calibration of laboratory measurements. As new RMAs come, they will go back and review Statements and repeat the assessment of the evidence. This process would assist to maintain the calibration and to establish continuity for future RMAs.

Attachment 1.

Determination of Statements of Principles



Attachment 2.

RMA Members' Assessment of Causation

Acting Lawfully

- ◆ Legislation
- ◆ Body of information considered
- ◆ Design, quality and results of individual studies

Causal Inference

- ◆ Use of Bradford Hill and similar criteria
- ◆ Judgement
- ◆ Personnel

Calibration & Consistency

- ◆ SoPs themselves
- ◆ Further considerations

Attachment 3.

RMA MEMBERS' ASSESSMENT OF CAUSATION

INDIVIDUAL STUDIES

Military and non-military literature considered

Primary research

Reviews and meta-analysis

Study design and quality (“soundness”)

Results

Chance

Bias

Confounding

Attachment 4.

RMA MEMBERS' ASSESSMENT OF CAUSATION

Issues relating to the consistency and calibration of factor inclusion and development

Legislation (and requirement to act lawfully)

Body of Evidence

Bradford Hill and similar “applicable criteria”

The RMA do not require all BHC to be satisfied before inclusion of a factor

Not all criteria are of equal weight

RR (>1.0 to ?)

Personnel of the RMA

drawn from many medical disciplines with extensive and broad experience

Judgement

Scientific and medical judgement, and also

Culture/history/second reading speeches/High Court decisions

Possible relevance for Veterans/defence force personnel

Some genetic disorder exclusions

Otherwise no disease or factor excluded because service may lead a range of exposures and may also alter behaviours

Attachment 5.

RMA MEMBERS' ASSESSMENT OF CAUSATION

Issues relating to the consistency and calibration of factor inclusion and development

Further Considerations

The outcomes of the calibration are the SoPs (RMA's standards)

The RMA asks
"can a factor be included?", and not "can a factor be excluded?"

There is no deduction with respect to age

Only harmful and not beneficial effects are considered
(this differs substantially from Public Health practice)

Dose estimates are generous

Holman calculations have been accepted by Government
as reasonable in outcome

RMA attends ESO meetings, veterans' meetings and Forums
like this

Causation in Medical Science

John Kaldor
Professor of Epidemiology
University of New South Wales

Paper presented at the RMA Forum
November 1998

Introduction

Cause sounds like such a simple, practical concept in its everyday usage. “The wet road *caused* the car to crash”; “Fred’s drinking *caused* him to lose his job”; and “the baby’s crying *caused* the dog to start barking” are all statements that would be well understood by the average person. So why do scientists, and in particular epidemiologists who study human disease causation, have such a problem with deciding what is a cause and what is not?

In fact even these everyday examples are not as straightforward as they might seem at first glance. The wet road may have made driving conditions more hazardous, but the driver’s actions surely made an essential contribution to the accident, and perhaps the crash would have occurred even if the road had not been wet. Maybe Fred’s drinking was the excuse his employer was looking for to cut staff when business was slow. And as for the dog, he may have been about to bark anyway.

The questions raised by these examples are not unlike some of the issues that scientists have to consider when trying to decide about disease causation. In fact such questions have been at the heart of intense philosophical debate about the very nature of scientific knowledge, for several hundred years. My objective in this paper is to try to explain how the notion of cause is defined and understood in medical science, without going into too much technical detail. Holman, in his Appendix to the Pearce Report, provides a comprehensive review of criteria for assessing causality that is aimed at the more specialised reader.

Defining a factor to be “a cause of a disease”: the theory

Medical science frequently deals in statements such as “high blood pressure causes stroke”; “high levels of alcohol consumption cause cirrhosis of the liver”; or “sun exposure causes lymphoma”. These statements propose a linkage between exposure to a specific factor, and the

subsequent occurrence of a disease. As such, they correspond directly to the kinds of relationship embodied in the RMA’s “Statements of Principle” (SOPs).

In the popular view, something that is worthy of the name “cause” could be expected to produce its “effect” reasonably frequently, but the situation in disease causation is often very different. For all the factor/disease combinations noted above, there would be many exposed people who never develop the disease. For this reason, cause must be interpreted in terms of probabilities rather than certainty of disease occurrence.

*A factor or agent is defined to be a **cause** of a particular disease if exposure to the factor results in a person having a higher risk, or probability, of developing the disease than a person who is not exposed.*

This definition has a number of immediate consequences. It allows for the possibilities that people exposed to a particular cause will never develop the disease, and that, conversely, the disease can develop in people never exposed to that cause. The definition also permits a factor to be defined as a cause even if it increases the risk of disease by a rather small amount, as long as there is a *real* increase in risk. A further implication of the definition is that a disease can be associated with multiple ‘causes’.

How do we determine whether or not a factor is a cause?

The work of the RMA is largely focused on making such determinations. Like any other body charged with identifying causes, the RMA starts from a definition of cause comparable to the one presented above. Although the definition may seem straightforward from a conceptual point of view, particular challenges arise when we try to apply it in practice:

- *How do we measure a person’s “risk of developing the disease”?* Probability is not like blood pressure or cholesterol levels. We cannot simply carry out a

test on an individual person to measure his or her “risk”. In fact risk can only be measured through *epidemiological studies on groups* of people, in which case it translates into the proportion of people in the group who develop the disease. A factor can then be defined as a cause of a disease through an epidemiological study if proportionately more cases of the disease are observed to occur in a group of people exposed to the factor, than in a corresponding group who are not.

- *If there are more cases in a group of people exposed than in an unexposed group, couldn't this just happen by chance?* Indeed it could, and for this reason, it is important to pay attention to the magnitude of the difference, and its *statistical significance*.
- *If exposed people do have a higher risk than unexposed people, how do we tell whether the difference is due to the factor in question, rather than something else that distinguishes the two groups?* Exposed people may differ from unexposed people in ways other than the presence or absence of the factor. This phenomenon, known as *confounding*, may not even be recognised by the investigators. The only way that confounding can be avoided with any confidence is through random allocation of the factor between two groups, an approach that is not really practical for most of the agents that are under consideration.
- *If no epidemiological studies have ever been done comparing the amount of disease between people exposed and unexposed to a factor, is it possible to define the factor as a cause? What if studies have been done but they are of poor quality?* In this situation, we must go beyond the evidence provided by the human epidemiological studies. Biological hypotheses, animal studies and arguments by analogy with other factors can all play a role, but are generally considered to provide weaker evidence for human disease causation than epidemiological studies.

Until these issues have been worked through systematically, it is not really

possible to state that a factor is a cause of the disease in question. Even if they have been dealt with, some doubt may remain as to whether the factor is a cause or not. For some factor/disease combinations, the evidence becomes overwhelmingly clear, and any inconsistent piece of information raises suspicions about its validity, rather than the truth of the causal relationship. If a study of cigarette smokers did not detect raised levels of lung cancer, questions would be raised about the quality of the study rather than the validity of the causal association.

On the other hand, for many factor/disease combinations, the evidence is of a lower quality, or contradictory, and it is not possible to make a definitive statement about the factor being causal. In this situation, those responsible for assessing the evidence (and living with the consequences of any decision about causality) would need to decide how well a causal relationship had been established.

This decision ultimately becomes somewhat subjective, and cannot be reached by any standard formula. Over the last few decades, a number of public health researchers have compiled checklists of criteria to be satisfied before a factor/disease relationship may be considered to be causal. Figure 1 illustrates the Bradford-Hill criteria, which are widely regarded as the first serious attempt to find a rigorous framework for defining causal factors in medical science. Although these criteria are seen as a useful way to consider the requirements of a causal factor, there has never been any attempt to check their ultimate value, and it is hard to think how such a validation might be carried out. The criteria are certainly satisfied by factors that the devisors of the checklist considered to be causal; But how did they decide that the factors were causal in the first place? Again, we are thrown back to a reliance on human qualities such as ‘experience’ and ‘judgement’.

Quantification of causal relationships

From the above discussion emerge two quite different aspects to the quantification of causal relationships.

First is the *strength of the evidence* that a factor/disease relationship is a causal one. At one extreme, we could assert with virtually 100% certainty that “alcohol consumption is a cause of liver cirrhosis”. At the other extreme, the evidence surrounding the relationship between sun exposure and lymphoma is far weaker, but it would be difficult to put a number to its strength. To say that there was “5% certainty that sun exposure was a cause of lymphoma” would be a rather limited vote of confidence in the causal relationship. In betting terms, it would mean that we would be prepared to take a 1 to 20 bet against the relationship really being causal.

The second dimension is the *strength of the association* between the factor and the disease. This dimension is generally only considered if there is a fairly high degree of confidence in the evidence for causality. If we are not convinced that a factor increases the risk of disease in the first place, it would not make sense to try to quantify the strength of its association with disease. The index commonly used to quantify the strength of a causal relationship is the *relative risk*, or the ratio between the respective probabilities of disease occurrence in the exposed and unexposed groups. For a factor which is causal, this index describes the extent to which disease occurrence is influenced by the factor.

Figure 2 shows two examples of relative risk. In the first case, the risk of disease in people exposed to the factor increases by 25%, or a factor of 1.25 compared to unexposed people. In the second, it increases by a factor of 5.

The figure also illustrates another index of the strength of association, known as the *attributable risk*. This index is defined by the proportion of people in the exposed group whose disease can be attributed to exposure. For the relatively weak factor,

with a relative risk of 1.25, the proportion of cases of disease added in the exposed group is $0.25/1.25$, or 20%. For the stronger factor, with a relative risk of 5, the proportion of exposed cases that can actually be attributed to the exposure is $4/5$, or 80%.

There is clearly some degree of circularity between the strength of an association and the strength of evidence for causality. The observation of a very strong association between a factor and disease (in other words, a large increase in disease occurrence among people exposed) is often used as strong evidence that the association is truly causal, thereby permitting the strength of the association to be quantified!

To what extent can we determine the cause of disease in a given person?

Perhaps the most frustrating practical consequence of the causality definition above is that even if an agent has been determined to be a cause of a particular disease, it cannot generally be stated that the factor is *the* cause of disease in an individual exposed to factor. For example, observation may have shown that drivers on wet roads have significantly more crashes than drivers on dry roads, and in this sense, wet roads are *a cause* of car accidents. Nevertheless, if a car accident does occur in wet weather, it will not be possible to absolutely attribute the wet road as *the* cause, because the car may have crashed no matter how little water there was on the road.

Although epidemiological science cannot generally determine in absolute terms whether a specified factor was or was not the cause of an individual's disease, it can nevertheless estimate the likelihood, or probability of causation in an individual case. The estimation generally proceeds in three stages, which are illustrated in Figure 3. As noted above, the work of the RMA does not generally go beyond the first stage, in that it does not attempt to attribute causality (or otherwise) in individual cases.

Stage I: An assessment must be made as to whether the factor is a cause of the disease, in the terms defined above.

Stage II: If the factor is assessed, on the basis of epidemiological and other evidence, to be a cause of the disease, an estimate must be obtained of the relative risk associated with the level of exposure experienced by the individual.

Stage III: Having obtained this relative risk, an estimate can then be derived of the *attributable risk*, or the probability that an exposed person's disease was actually caused by the exposure.

It is important to emphasise a number of aspects of this process. First, it depends on external information to determine whether or not the factor may be considered to be a cause. Second, this information must also be used to derive the relative risk associated with the exposure that the person experienced. Crucially, the conjunction of exposure and disease in the person for whom causation is being assessed says absolutely nothing about the likelihood that the factor was the cause of disease.

It is in the area of attributing cause at an individual level that medical science and the law can come crashing into each other, and the outcome is often highly unsatisfactory. Courts, tribunals and other legislatively empowered bodies must make determinations as to whether the disease that occurred in a particular person was, or was not, caused by a specified factor. To further complicate matters, the criteria for making the determination are often qualified by expressions such as "beyond reasonable doubt", "on the balance of probabilities" or "based on a reasonable hypothesis" (the latter two of which appear in the Veterans' Entitlement Act). Although it may be valid to leave these terms rather vague in the interest of judicial flexibility, it is also fair to say that there is little common understanding of the probabilistic meaning of such expressions.

An example of attributing cause

To illustrate the process of attributing causality, consider the following hypothetical scenario:

Three well- conducted epidemiological studies have been carried out to examine the relationship between exposure to a particular kind of solvent and the development of asthma. The studies all find statistically significant increases in the amount of asthma among people exposed to the solvent compare to those unexposed, and no other confounding factor can be shown to explain these increases. The solvent is known to have irritating effects on the lung when inhaled, and causes long-term pulmonary damage in animal experiments. The relative risks estimated from the three studies were 1.7, 2.0 and 2.3

In *Stage I*, the evidence presented here would generally support the attribution of causality to solvent exposure. The studies have found significant increases in risk, and they are all described as good studies. There is no known confounding, and the animal and physiological data are consistent with the proposition that the solvent is toxic to the lung in some way. The strength of the evidence is high, and most experts would be confident in asserting that the solvent causes asthma.

For *Stage II*, a combined estimate of the strength of association derived from these studies would be around 2.0, and the attributable risk for *Stage III* would then be $\frac{1}{2}$ or 50%. In other words, if a person who was exposed to the solvent develops asthma, there is a 50% chance that the disease was due to the exposure.

Obvious causes and self-fulfilling causes

Any discussion of causality in medical science would be incomplete without reference to the idea of the 'obvious' causes such as the bullet wound/death association discussed in the Technical Appendix to the Pearce Report. If the relative risk associated with the exposure is very high (the probability of a person's heart stopping following a direct hit by a bullet is several thousand times the probability of cardiac arrest in a person who has not just been shot) or if there is essentially no risk in the absence of the

exposure, the attributable risk will also be extremely high. Effectively there is 100% probability that death in a person who has just sustained a bullet wound to the heart was caused by the bullet wound.

Another issue that can cause consternation in discussions about causality is the fact that some diseases, or more often 'syndromes', are actually defined by a prior exposure, and any discussion of the relationship between exposure and disease becomes tautologous. For example, AIDS is defined by the presence of HIV, and post-traumatic stress disorder is defined by a prior traumatic experience. A study that tried to look at whether or not HIV was a

cause of AIDS would be stymied by the fact that every person with AIDS has HIV infection, by definition! The question that could be sensibly addressed is whether a person with HIV infection was more likely than a person without HIV infection to develop the cluster of symptoms and illnesses that are known as AIDS *when they occur in conjunction with the presence of HIV infection*. Similarly, an assessment of the role of traumatic experiences in the cluster of symptoms that define post-traumatic stress disorder would need to be carried out in terms of these symptoms, defined independently of prior exposure history.

Figure 1.

The Bradford-Hill criteria for causation

1. **Strength:** Size does matter.
2. **Consistency:** If it causes disease in them, it should also cause it in us.
3. **Specificity:** It can't cause too many different types of disease.
4. **Temporality:** What came first, the factor or the disease?
5. **Biological Gradient:** Is more of a bad thing even worse for you?
6. **Plausibility:** It doesn't sound too far-fetched.
7. **Coherence:** There is no major conflict between the strands of evidence.
8. **Experiment:** What happens if we remove the factor?
9. **Analogy:** We have seen something along these lines before.

Figure 2.

Strength of association for a causal factor

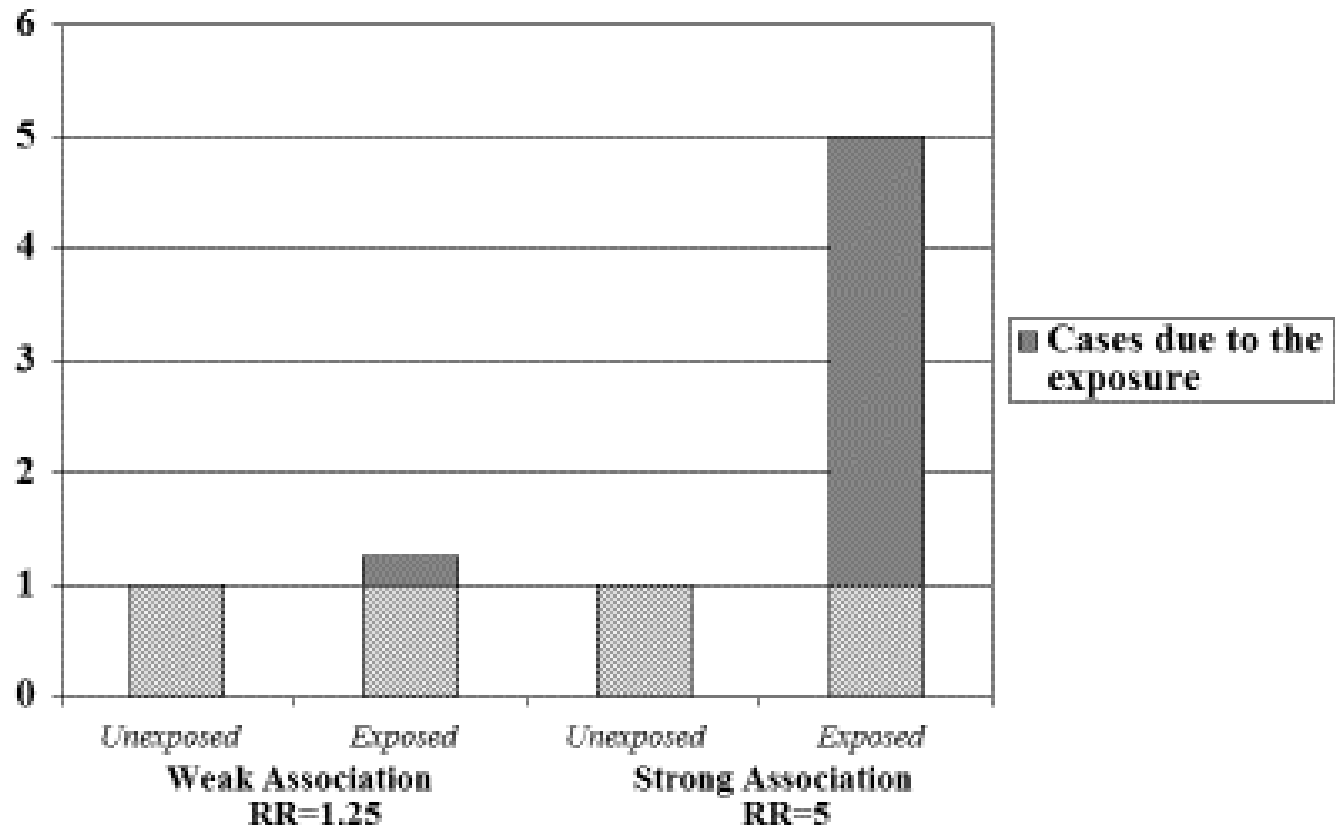


Figure 3.

Case: A person develops disease D, and was previously exposed to factor F. How likely is F to have been the cause of D?

Stage	Question	Action
I	Is F a cause of D?	Assess epidemiological and other external evidence
II	At the exposure levels to F experienced by the person, what is the relative risk of developing D?	<ul style="list-style-type: none">• Assess person's exposure level• Compile information on relative risk at this exposure level
III	What is the probability that F caused D?	Convert relative risk to attributable risk

Background Terminology: Introduction To Some Epidemiological Terms

Dr Alex Bordujenko

Repatriation Medical Authority, Secretariat

EPIDEMIOLOGY

This is the study of variations in disease frequency among population groups, and the factors that influence these variations. The principle objective of epidemiology has been to determine factors which may cause or contribute to disease processes in humans, so that preventive measures may be applied.

Epidemiologic observations have a long history, with much work developed through the study of acute epidemic diseases such as cholera and typhoid. The discipline has burgeoned over the latter half of the Twentieth century, with interest in the study of the cause, treatment and prevention of cancer, cardiovascular and other chronic disease, and of course the advent of computer storage and analysis systems.

APPROACHES FOR EPIDEMIOLOGICAL STUDY

Hennekens and Buring (1987) define epidemiology as “the study of the distribution and determinants of disease frequency in human populations.”

“Humans” distinguishes the approach from research using animal or other systems in experiments. “Populations” contrasts the practise of individual investigation as in clinical research. “Frequency” indicates the quantification of disease occurrence and the risk attributable to various potential causes. The term “distribution and determinants” points to the two major approaches of epidemiology:

1. examination of the distribution of disease frequency in populations (this can produce hypotheses about the causes of disease) known as descriptive studies; and
2. analytical studies which test these hypotheses by reviewing personal characteristics or exposures among individuals within the populations.

Descriptive studies use population based statistics on mortality, disease incidence, and survival. Other registries for example hospital based disease registries, may also be useful. Obviously the studies concern populations and not individuals and measures of any exposures are usually broad and may be subject to confounding or interfering factors. Selection of free living populations may introduce biases and confounding into the calculations. Examination of national and international trends, migrant studies and time trends has provided valuable insights into the causation of a number of chronic diseases for example breast, prostate and lung cancers.

Analytical studies have provided much useful information concerning the discovery and/or confirmation of a number of lifestyle and other environmental exposures as causes of chronic disease, including cancer. Examples of these include cigarette smoking, where, for smokers of 40 or more cigarettes per day there is a risk of lung cancer of more than twenty times that of a non-smoker. Another well documented example is occupational exposure to asbestos and the development of mesothelioma, where the relative risk is well over 100 fold that of the unexposed population. Analytical studies from several international sources in the last decade have also demonstrated that both the incidence and recurrence of neural tube defects can be greatly reduced by maternal folate supplementation in early pregnancy, even in the absence of maternal folate deficiency.

In chronic disease epidemiology, the types of analytical studies encountered are:

A. Cohort studies identify groups of individuals with and without a particular exposure, and follow them over time to examine disease incidence and/or mortality rates. These may be current or past exposures. An association is suggested when rates of disease or death differ between the groups. These are able to directly measure incidence and mortality

rates related to a particular exposure (especially with prospective design) but they require large numbers of exposed individuals particularly when considering uncommon diseases, before significant differences may be noted.

B. Case-control studies or case-referent studies identify people with a particular disease (case), and a group of people without the disease (controls), and then collect information about past exposures, for example by interview or questionnaire. They provide a method of studying rare diseases but may be subject to recall and other biases, and difficulty in measuring past exposures.

Data Presentation and Interpretation

The odds ratio (OR) is a measure of association used in case control studies to estimate the odds of exposure in cases to the odds of exposure in controls. This approximates, but is not synonymous with, the “relative risk” (RR) the measure of association used in cohort studies. The term relative risk (RR) is used to describe the comparison of the risk of a known exposed group versus a known unexposed group developing a specific condition. Thus if the relative risk is one the risk is the same for both groups and exposure is not seen to be associated with the development of the particular condition that is, there is no increase in the risk of a studied outcome with the exposure of interest. If the RR (or OR) is 1.5 then the risk for the studied outcome in the exposed versus the unexposed group is increased by 50%. An RR (or OR) of 2 implies a doubling of risk, and an RR (or OR) of less than one implies a reduction of risk. Problems in decision making occur when the described increase in risk is weak (under a two to three fold increase) and particularly when the relative risk is close to one, for example 1.1 (10% increase) or 1.3 (30% increase) rather than the 20 fold increases for heavy cigarette consumption and the incidence of lung cancer and the much greater increases seen with

occupational asbestos exposure and the incidence of mesothelioma. Many epidemiologists are reluctant to accept as real, increases in risk of less than 100% ($RR \leq 2$) as likely to be causative unless the “Bradford Hill” types of criteria are stringently applied to the body of evidence pertinent to the putative association, and overall, a considered case can then be made to support causality.

Another term, the “confidence interval” (CI), is used to describe the range of relative risk (or odds ratio) rates within which the actual result lies, to within, for example, a 95% probability. Thus, if the confidence interval includes one then the result could have occurred due to chance and no true effect may exist. If the 95% confidence limits exclude one it does not exclude the possibility of a chance result, rather it indicates that chance would explain the observed (or a greater) risk estimate only one out of 20 times.

Selected Measures of Disease Frequency

As well as the relative risk and odds ratio a number of other measures of disease frequency need to be considered. A consideration of the basic concepts of these measures includes the formulae used to calculate such measures. In its simplest form data from a two-by-two table from a case-control or cohort study with count denominators would appear as

	Disease		Total
	Yes	No	
Exposure Yes	a	b	a+b
Exposure No	c	d	c+d
Total	a+c	b+d	a+b+c+d

a= the number of individuals who are exposed and have the disease
 b= the number who are exposed and do not have the disease
 c= the number who are not exposed and have the disease
 d= the number who are not exposed and who do not have the disease

As stated above for cohort studies the term relative risk (RR) is used to describe the comparison of the risk of a known exposed group versus a known unexposed group developing a specific condition, that is the incidence of the disease in the exposed divided by the incidence in the unexposed I_e/I_o or the cumulative incidence of the disease in the exposed divided by the cumulative incidence in the unexposed CI_e/CI_o .

The formula for calculating relative risk for cohort studies with count denominators is thus:

$$\frac{I_e}{I_o} = \frac{CI_e}{CI_o} = \frac{a/(a+b)}{c/(c+d)}$$

(where a,b,c,d are derived from the 2x2 table outlined above).

For case control studies with count denominators the odds ratio is expressed as:

$$\frac{a/c}{b/d} = \frac{ad}{bc}$$

(where a,b,c,d are derived from the 2x2 table outlined above)

The odds ratio is said to provide a valid estimate of the relative risk for case-control studies where the cases are newly diagnosed, where prevalent cases are not included in the control group and where the selection of cases and controls is not based on exposure status.

Attributable risk is the measure which provides information about the absolute effect of the exposure and is the excess risk of disease in those exposed compared with those who are unexposed to a specific factor. This measure is defined as the difference between the incidence rates in the exposed and unexposed groups and may be calculated in cohort studies as

$$AR = CI_e - CI_o = a/(a+b) - c/(c+d)$$

(where a,b,c,d are derived from the 2x2 table outlined above)

The attributable risk percent (AR%), attributable rate percent attributable

proportion or etiologic fraction is calculated as the attributable risk divided by the rate of disease among the exposed and is said to represent the proportion of disease in that group that could be prevented by absence of the exposure.

$$AR\% = \frac{AR}{I_e} \times 100 = \frac{(I_e - I_o)}{I_e} \times 100 = (1 - I_o/I_e) \times 100 = (1 - 1/RR) \times 100 = \frac{(RR-1)}{RR} \times 100$$

Population Attributable Risk (PAR) is the measure used to estimate the excess rate of disease in the total study population of exposed and unexposed individuals that is attributable to the exposure. The PAR is calculated as the rate of disease in the population (incidence rate in total population = I_t) minus the rate in the unexposed group (I_o):

$$PAR = I_t - I_o$$

or by multiplying the AR by the proportion of exposed individuals in the population (P_e):

$$PAR = (AR) \times (P_e)$$

Population Attributable Risk Percent (PAR%) is represented by:

$$PAR\% = 100 \times (P_e) \times \frac{(RR-1)}{1+(P_e) \times (RR-1)}$$

EPIDEMIOLOGIC STUDIES NEED CAREFUL EXAMINATION

(Refer to:- Darzins PJ, Smith BJ and Heller RF (1992). *How to read a journal article*. The Medical Journal of Australia, Vol 157 pp 389-394.)

The size of the population studied is important - the larger the sample size the greater the power (or ability) to detect a specified risk, the smaller the sample size the weaker the power. Negative results from small studies may not be conclusive as only large studies may confidently exclude or include low to moderate levels of risk.

When examining any study results, consideration of the possibility of a non-causal association is necessary. The observed association between exposure and disease may result from bias, confounding, chance, or cause-and-effect.

Bias is the term used for any systematic error in a study and may occur during study selection, information gathering or in reporting of the assessment of the exposure or outcome under investigation. Confounding bias is the possibility of the observed effect being due to other variables not adequately considered in study design or analysis of the results. Many types of study bias have been described including selection, information, recall, and interviewer bias. Confounding bias or confounding is due to variables which may themselves account for all or part of an apparent association between an exposure and a disease. They may also obscure an association. Chance is considered previously in the discussion of study power and Confidence Intervals.

Study Types

Study design has an effect on the quality of evidence which may be gained and a

recognised 'hierarchy' of study types exist. In developing the following the "US Preventative Services Task Force: Guidelines for Quality of Evidence" (Fisher, 1989) have been considered. Given the specific needs of the RMA some modification has been undertaken. In this instance the level of evidence available is at best observational (cohort or case control studies). The following is broadly the division of available study designs and how these may be considered in the information gathering.

Analytic Studies:

- 1 Intervention Studies
 - 1a Randomised Controlled Trial
 - 1b Controlled Trial
- 2 **Observational Studies**
 - 2a Cohort-Prospective
 - 2b Cohort-Retrospective

3 Case Control Studies

Descriptive Studies:

- 4 **Population (Correlational)**
- 5 **Individual**
 - 5a Cross Sectional Surveys
 - 5b Case Series
 - 5c Case Reports

Where the numbering 1-5 refers to the grade assigned to the quality of the evidence. Quality refers here to study design rather than individual study merit that is that the evidence from cohorts is graded as higher than that from case control studies - this is the method used by the US Preventative Services Task Force.

While the RMA places emphasis on primary research published in the leading peer reviewed journals of either broad or discipline specific type; published, peer reviewed, reports on the epidemiology of disease such as those produced from time to time by the International Agency for

Research into Cancer, the National Academy of Science, or the Surgeon Generals' Reports concerning to smoking related disease; are considered appropriate sources for examination. Published reports from sources such as the National Health and Medical Research Council and other expert committees are also be considered where contemporary, applicable material is available.

CONSIDERATION OF INDIVIDUAL STUDIES

(Refer to:- Darzins PJ, Smith BJ and Heller RF (1992). *How to read a journal article*. The Medical Journal of Australia, Vol 157 pp 389-394.)

In the absence of interventional studies such as randomised controlled trials most reliance is placed on well designed and reported cohort and case control studies and Professor Heller provides a method of considering these. This forms a mental check list in consideration of materials.

The following questions may be specifically addressed.

1. What is the research question?
2. What is the study type?
3. What are the outcome factors and how are they measured?
4. What are the study factors and how are they measured?
5. What important confounders are considered?
6. What are the sampling frame and sampling method?
7. How many subjects reached follow-up?
8. Are statistical tests considered?
9. Are the results clinically/socially significant?
10. What conclusions did the authors reach about the study question?

After determining these features a decision on adequacy of methods and clarity of results is made considering:

bias - are the results biased in one direction. If so, what is the direction and magnitude of bias

confounding - are there any serious confounding or distorting influences? Has an attempt been made to deal with these and has this been adequate?

chance - is it likely the results occurred by chance? Consideration of the statistical content of the study.

It is recognized that for many putative factors evidence may only be available in descriptive studies. This is often the case for case reports or case series of disease associations or drug reactions.

ASSOCIATION AND CAUSATION

Association is the term used to describe the statistical dependence between two variables. In epidemiology it is the degree to which the rate of disease in persons with an exposure of interest is either higher or lower than the rate of disease among those without that exposure. Such an association does not mean, or even imply, that the observed relationship is one of cause and effect (Hennekens and Buring, 1987).

Making judgements about causality from epidemiologic data involves a logical process which addresses two major areas:

1. Whether for any individual study, the observed association between an exposure and disease is valid. An assessment of validity requires a consideration of the likelihood of alternative explanations for the results and chance (the luck of the draw), bias (any systematic error in the study for example in subject selection, information gathering or reporting), or confounding (the observed effect being due to other variables not

adequately considered in study design or analysis of the results); and

2. Whether the body of the evidence considered supports a judgement of causality. In this process standard epidemiological criteria are used (Hennekens and Buring, 1987).

Epidemiologic criteria used to assist in the assessment of causality

The RMA considers the individual studies with respect to the above and then, in considering the available evidence uses standard epidemiological criteria to make a judgement regarding causality with regard to the reasonable hypothesis and balance of probabilities standards of proof. The Bradford Hill criteria (Bradford-Hill, 1965), and more contemporary versions, are widely accepted in the interpretation of epidemiological studies for the purpose of assessing the possibility of a causal association.

Consideration of the body of evidence available for each contention against the current epidemiologic criteria will result in a judgement regarding causality. As Professor Holman (1997) notes, more than 30 different systems of causal verification have been described. In his technical appendix to the Pearce Report he outlines ten criteria for classification of evidence of causality, based on work by Mervyn Susser. The RMA has considered a number of such systems including those of Bradford Hill, Susser and those co-authored by Professor Holman in "*The Quantification of Drug Caused Morbidity and Mortality in Australia, 1995*" (English and Holman, 1995). The RMA recognises the underlying similarities which underpin these systems.

The exact description of these epidemiologic criteria varies between authors and the RMA recognises the need to consider both internal study validity (for individual studies) and factors important in

the body of evidence (the applicable evidence available from epidemiological, clinical, toxicological and other research) in these criteria.

Sir Austin Bradford Hill, as well as other prominent statisticians and epidemiologists, including Mervyn Susser and Kenneth Rothman, have described how the subjective likelihood (or the correct judgement) of a causal relationship is increased when evidence relating to an association meets criteria devised to consider the available evidence. The Bradford Hill (Bradford-Hill, 1965) criteria are as follows:

1. Strength of Association
2. Consistency
3. Specificity
4. Temporality
5. Biological Gradient
6. Plausibility
7. Coherence
8. Experimental evidence
9. Analogy

The criteria used by the Expert Committee on Herbicide Exposure and Spina Bifida (1996) further refined the criteria to explicitly include consideration of bias and confounding in the criteria:

1. Statistical significance (that is the possibility of chance being responsible for an apparent association; and study power)
2. Strength of association
3. Consistency of association between studies
4. Possibility of bias in measurement of exposure or outcome
5. Possibility of selection or confounding bias
6. Time sequence
7. Dose response
8. Biological plausibility (including aspects of theoretical coherence, biological coherence and factual coherence)

1. Statistical significance and power

If the criterion of statistical significance is satisfied then the evidence is supportive of an association. The failure of a test to reach statistical significance in the presence of adequate statistical power provides evidence against the association, however in the absence of adequate statistical power it may not necessarily detract from the association.

2. Strength of association

The greater the strength of association the more likely it is to be causal. Confounding is less likely to explain a strong association because the strength of the association between the confounding variable and the outcome must also be strong. While a strong association is supportive of causality, a weak association may not necessarily detract from the evidence of causality however adequate consideration of potential confounding or bias is essential.

3. Consistency of replication

Consistency of the evidence or the lack of evidence in the face of study diversity in time, place, circumstances and population, as well as research design, strongly supports or detracts from a causal hypothesis.

4. Possibility of bias in measurement of exposure or outcome

Consideration of any systematic error in the study in information gathering or in reporting of the assessment of the exposure or outcome under investigation. Absence of bias in the studies considered to show a positive association supports the existence of a putative association. The presence of bias detracts from the conclusions which may be drawn from the information.

5. Possibility of selection or confounding bias

Consideration of any systematic error in the study in subject selection; or the possibility of the observed effect being

due to other variables not adequately considered in study design or analysis of the results. Absence of bias or confounding in the studies considered to show a positive association supports the existence of a putative association. The presence of bias or uncontrolled confounding detracts from the conclusions which may be drawn from the information.

6. Time sequence

The exposure must precede the disease or injury. This criterion is compatible with, but does not necessarily support causality. Reversal of the order of exposure and disease or injury is the most persuasive basis available for rejection of causality.

7. Dose response

A response which is in proportion to the level of exposure is strongly persuasive of a causal relation. However, its absence does not necessarily detract from the association.

8. Biological plausibility

(aspects of theoretical coherence, biological coherence and factual coherence)

Theoretical coherence: Findings plausible in terms of pre-existing theory are supportive of the association. Conversely, findings that are implausible in terms of pre-existing theory detract from the evidence.

Factual coherence: Compatibility of a new result with pre-existing facts is supportive of the association. Incompatible pre-existing facts strongly detract from evidence of causality.

Biological coherence: Pre-existing knowledge which identifies a mechanism by which the chemical exposure may produce the disease or injury is supportive of case for the association being causal. Observations from species other than humans may also be used to support the potential

mechanism of action. Incoherence between biological knowledge and study observations detracts from the case for a causal association.

As Rothman and Greenland (1997) eloquently acknowledge inductively oriented causal criteria are not sufficient within themselves and require sound scientific judgement to traverse the path for which the criteria are “the road map through complicated territory”.

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FUTURE STUDIES:
(Pearce Recommendation 10)

Professor John Duggan

Repatriation Medical Authority

Professor John Duggan chaired the discussion relating to future research. He considered that the forum participants needed to discuss what might be done in the future about research which would be of particular relevance to war and defence force veterans. He considered that there is much that could be done for research out of the data bases that do exist and that discussing these would assist in dealing with a number of outstanding issues of concern to members of the RMA. A number of specific issues were outlined on overheads (Refer Attachments 1-3).

The relationships between heavy physical exertion and osteoarthritis and spondylosis were considered; as was the necessity of using civilian and military studies and the paucity of relevant military literature in this area where exposures may not be identical between civilian and military life. The potential effects of EMF (Electromagnetic Field) radiation on radar operators and exposure to large G forces and risk of haemorrhoids were other areas which were seen as of interest and which could be addressed by relatively simple studies of military personnel. For example in the last case a comparison could be made between the incidence of haemorrhoids in fighter pilots and aircraft refuellers with the exposure of interest being the differing exposure to G forces. Assessment of potential confounders would also need to be considered.

The role of post trauma counselling in the potential for prevention of PTSD (Post Traumatic Stress Disorder) was of importance for current military forces and it was seen that a study could be designed to determine the need for such counselling and the most appropriate style and duration of such counselling to minimise the effects of significant stressor experience.

Additionally, the issue of cohort and nested case control studies on current and future military personnel was canvassed, particularly with regard to the need for blood sample collection and storage. This

would entail taking blood at the beginning of service and at the end; and then storing these samples over time. This would allow large scale investigations of exposures and disease as well as accurately indicating for the individual, levels of service related exposure to a range of infectious and chemical agents.

The potential uses of medical service record linkage were considered and audience members suggested a number of potential sources of data. There was a need to ensure the common structure of records across the Australian Defence Forces.

Systems must allow prospective data collection. Issues of compatible data systems, confidentiality and study ethics, the role of the ADMEC (Australian Defence Medical Ethics Committee) and potential benefits and negative effects for personnel involved were touched upon.

Participants suggested that prospective studies may be of more benefit given the variable collection and quality of past records. Past records were focussed on the individual and without an accurate database covering the occupation or exposures in question it would be difficult to retrieve data. With regards to retrospective data sources it was suggested that consideration be given to the National Acoustics Laboratory in North Sydney with regards to assessment of acoustic trauma in certain military personnel and the RAAF Institute of Aviation Medicine Edinburgh in South Australia with regard to a variety of matters affecting air crew fitness.

It was suggested that the military has no incentive and no requirement to consider this form of research at the moment. Department of Defence (DoD) has no responsibility for paying the costs of compensation for what happens after people leave service. Therefore its systems are not engineered to actually look at that because there is no incentive for them to collect that group information and use that then to feed back in to reduce their costs.

Current military practice was said to have both positives and negatives with regard to research. On a positive note it was suggested that the three Services are now very clearly moving towards common data collection systems and have a common outpatient consultation form. Classifications differ between the forces however within each arm of the forces the classifications should be comparable. Caution was then raised given the progressive move for down-sizing and multi-skilling which blurs occupational boundaries and the proposed changes to military career paths with defence and civilian activities combined.

The forum participants supported a number of broad actions as follows:

- **Agree on comparable data collection systems between Army/Navy/Air Force where they are not already in place.**
- **Establish these collection systems retrospectively/prospectively with**

an aim to predict outcomes in relation to baseline characteristics and exposure during service.

- **Continue follow-up data collection on morbidity/mortality after discharge to link to exposures during service.**
- **Identify risks that suggest needs for prevention to avoid future service related illness and develop preventive intervention trials.**

As an initial step in this process the forum participants anticipate that a working party, as outlined in Recommendation 10 of the Pearce Report, will be established to examine ways in which data relating to service personnel and service conditions relevant to the RMA's functions might be assembled. In accordance with the Pearce Report recommendation the working party would consist of DVA, RMA, DoD and ESOs representatives.

Appendix 1

The members of the RMA have noted with interest the developments which have recently taken place in the United States (US). An examination of recent Institute of Medicine (IOM) publications indicates that a number of interlinked health related military databases already exist and further improvements are planned. Consideration is underway for a US military health surveillance database which would include a recruit survey (including risk factors identified before service) and periodic individual assessment and environmental monitoring during service. Further, the National Academy of Science (NAS) is currently undertaking a feasibility study for the development of a "Centre for Post War Illness" focussing on the diagnosis, treatment, education and research regarding the health of the US military forces.

Considerable impetus for these developments has occurred in the wake of the Persian Gulf War (PGW) when the US Congress directed the secretaries of DVA and DoD to seek an agreement with the Medical Follow-up Agency of the Institute of Medicine to review existing scientific, medical, and other information on the health consequences of military service in the Persian Gulf theatre of operations during the Persian Gulf War.

United States Institute of Medicine

The committee was charged to assess the effectiveness of actions taken by the secretaries of DVA and DoD to collect and maintain information that is potentially useful for assessing the health consequences of military service referred to in subsection (a) of Public Law 102-585 (PG theater of operations during the PGW); to make recommendations on means of improving the collection and maintenance of such information; and to make

recommendations as to whether there is a sound scientific basis for an epidemiological study or studies of the health consequences of such service and the nature of the study or studies. The findings of the IOM committee are contained in their publication "*Health Consequences of Service During the Gulf War: Recommendations for Research and Information Systems*" National Academy Press, Washington, D.C. 1996.

The recommendations of this committee are as follows and a number clearly are of relevance to Australian military research:

Recommendation 1. The Department of Defense (DoD), the branches of the armed services, and the Department of Veterans Affairs (DVA) should continue to work together to develop, fund, and staff medical information systems that include a single, uniform, continuous, and retrievable electronic medical record for each service person. The uniform record should include each relevant health item (including baseline personal risk factors, every inpatient and outpatient medical contact, and all health-related interventions), allow linkage to exposure and other data sets, and have the capability to incorporate relevant medical data from beyond the DoD and DVA institutions (e.g., U.S. Public Health Service facilities, civilian medical providers, and other health care institutions). Appropriate consent and protection of individual privacy must be considered for information obtained and included.

Recommendation 2. The DoD and DVA should conduct further studies, with appropriate statistical and epidemiological support, to identify risk factors for stress-related psychiatric disorders among military personnel (active and reserve) and to develop better methods to buffer and ameliorate the psychiatric consequences of modern training, deployment, combat, demobilization, and return to daily living.

Recommendation 3. Studies being conducted by DoD and DVA that have included longitudinal follow-up of the mental health of veterans who served in the PG should be supported with continued follow-up, after appropriate peer review of study methods. Follow-up in these studies should be sufficient to provide at least a decade of information comparing the mental health status of those deployed with those not deployed.

Recommendation 4. The DoD should ensure that military medical preparedness for deployments includes detailed attempts to monitor natural and man-made environmental exposures and to prepare for rapid response, early investigation, and accurate data collection, when possible, on physical and natural environmental exposures that are known or possible in the specific theater of operations.

Recommendation 5. Research is needed to determine whether differences in personal characteristics or differences in policies and procedures for mobilization, deployment, demobilization, and return of reserves, National Guard, and regular troops are associated with different or adverse health consequences. If there are associations, strategies necessary to prevent or reduce these adverse health effects should be developed.

Recommendation 6. The mortality experience of PG veterans should continue to be monitored for as long as 30 years, on a regular basis, including comparisons with that of PG-era veterans. (PG-era veterans have been defined as those in military service at the time of the PGW, but assigned or deployed elsewhere) Research investigators should focus on the reported excess mortality from unintentional injury, on mortality from specific illnesses, and on evidence of elevation (or reduction) in the risk of death from other causes.

Recommendation 7. The DVA should exert greater effort to improve understanding of the reasons for excess mortality from unintentional injury. Detailed

evaluation is needed beyond death certificate data concerning the circumstances surrounding fatal injury, through more focused case-control studies to identify both individual risk factors and remediable causes.

Recommendation 8. The Defense Medical Epidemiological Database System should be continued, expanded as planned, expedited to develop the proposed integrated information management system, linked to other key systems, and evaluated regularly.

Recommendation 9. The DoD should complete development of information systems to expeditiously and directly pinpoint unit locations at a high level of disaggregation in space and time (that is, fine detail) and to document local environmental conditions, including appropriate data quality checks, with direct data entry into the system. There is likely to be a need for a similar information system during and after any future conflict, and DoD should prepare and continually update plans for such a non-paper system. A manual for use of the information systems by research investigators should be compiled, with the strengths and limitations identified.

Recommendation 10. For every specific question posed to the current TEAM (Troop Exposure Assessment Model), DoD should assess the strengths and limitations of the TEAM as a resource for evaluating the health significance of geographically defined exposures of troops, including those in the PGW and those in conflicts that may develop in the future. Evaluations and recommendations for possible modification of the TEAM should be reported to the PG Coordinating Board Research Working Group.

Recommendation 11. The DoD and DVA should ensure that studies of the health effects of deployment, including effects on PGW veterans, include evaluation of the exposures, experiences, and situations of both women and men, with attention to their age, prior military service, marital and

parental status, and other gender-specific parameters.

Recommendation 12. The DoD and DVA should conduct studies of the health consequences of assigning men and women to serve together in combat or under the threat of enemy action. Such work should be undertaken with focus on prevention and amelioration of any added stresses.

Recommendation 13a. The Naval Health Research studies in San Diego should be completed and results published as designed and scheduled.

Recommendation 13b. The DVA National Health Survey should be completed and results published as designed and scheduled.

Recommendation 13c. Evaluation of predictors of enrollment in the DVA PGHR (Persian Gulf Health Registry) should be promptly completed and results published. Included, if possible, should be information on type of care requested, required, and received.

Recommendation 14. The epidemiological capabilities of the armed forces should be

strengthened rather than reduced. The command structure should be kept informed about the reasons for and the results of this recommendation and its relevance to military preparedness and effectiveness, and should be encouraged to support appropriate epidemiological work in the theater of operations and in the post-deployment period.

Recommendation 15. The DoD and DVA should adopt a policy that internal and contract-supported reports on health research will be submitted for publication in the peer-reviewed scientific literature in a timely manner.

Recommendation 16. The Congress, DVA, and DoD should adopt a policy that unless there are well-specified, openly stated reasons to the contrary, requests for proposals for research related to unexplained illnesses or other needed health-related research will be publicly announced and open to the scientific community at large, that proposals will be reviewed by panels of appropriately qualified experts, and that funding will follow the recommendations of those experts.

Attachment 1.

Problems for Future Studies

- ❖ Spondylosis
 - ❖ Arthritis
 - ❖ PTSD – post stress counselling
 - ❖ Tumours and electro magnetic radiation
 - ❖ Haemorrhoids – air crew and G forces
 - ❖ Blood sample storage
- exertion, trauma, vibration

Attachment 2.

Issues to Consider

- ❖ Some problems insoluble in civil life
 - Spondylosis SoP based upon 1950 miner's study

- ❖ Service life ideally suited for some studies
 - Post battle counselling
 - G forces and haemorrhoids
 - Environmental smoke
 - Chemical exposure

- ❖ Non exposed controls readily (?) available

- ❖ Stored serum sample

- ❖ Logistic/ethical/legal issues

Attachment 3.

Logistic/Ethical/Legal Issues

- ❖ Medical/service record linkage
- ❖ Common structure of records e.g. Employment Classification No.
- ❖ Systems to allow prospective data collection
- ❖ "Survey fatigue"
- ❖ Confidentiality of data
- ❖ Will personnel gain or lose?
- ❖ Who researches – DoD, DVA, contractors
- ❖ Who pays?

SUMMARY of ISSUES RAISED

Standard of Proof

ISSUE: Concern was raised that subsequent Repatriation Medical Authority could interpret or have a different view of the “standard of proof” applicable to the definition of “sound medical-scientific medicine” from that interpretation or view held by the current RMA. That is why a codification/calibration document is seen as an important guide for future RMA membership to assist with the continued consistency in the standard of proof applied.

RESPONSE: Professor Donald acknowledged that there is room for future members of the RMA to read the legislation and make a different interpretation from that interpretation held by the current members. However Professor Donald did confirm that a document would be produced detailing his address to the forum and that this document would include the calibration issues. This publication would then be a public document and kept on the files of the RMA.

Professor Richard Heller (RMA member) highlighted that the RMA has a regular review program for SoPs. This concept of regular review is in line with what would be done if undertaking an epidemiological study where you're trying to maintain constant quality control or maintain any laboratory measure. In other words you keep calibrating against the standard and you repeat things.

Relevance of Evidence

ISSUE: The lack of good quality, detailed and specific research from the military and veteran arenas.

RESPONSE: Professor Donald agreed that it would be helpful if such research in the military area was available. Professor

Donald acknowledged that the quality of the evidence produced by epidemiological and other science is quite frequently not adequate when applied to a specific social purpose. However, he also stressed that such research could be a double edged sword and does not mean that factors wouldn't get tougher than they are at present as one of the outcomes of research is that you have to accept the outcome.

Further, Professor Donald noted that “Governments seem to have become aware that they (governments) have more responsibility to soldiers before, during, and after wars than they have exercised in the past. If we can be part of that, then that's another social purpose for an organisation like the RMA. So I would fully endorse there being better and more research that's relevant to these issues, and that's the reason why the session is on this afternoon.”

Clinical Onset

ISSUE: Has the RMA given consideration to incorporating a general definition for “clinical onset” in SoPs

RESPONSE: Professor Donald pointed out that the concerns raised in the case history detailed, related to evidentiary matters of a particular veterans case which, of course, are not within the RMA's area of responsibility; evidentiary matters are a responsibility for the DVA. Mr Bill Maxwell from DVA expressed concern at the broad interpretation that commission delegates apply to the term “clinical onset”. He informed the forum participants that his understanding of the term “clinical onset” is the same as that given by Professor Donald in his earlier address to the forum and that through their training courses, the Department was, and will continue to instruct delegates accordingly.

Interpretation of SoP contents

ISSUE: Has the RMA given consideration to the range of interpretations which may be made for the description of the factors within the SoPs

RESPONSE: Professor Donald responded that in SoPs the words used should have their plain English usage, that is the RMA tries to say "these words don't have any other meaning than their plain English meaning". Some difficulties occurred with the earlier use in SoPs of the words "before" and "for example". The RMA is learning how they will be interpreted and are trying to make the language clearer and clearer and more and more precise. The problem with that is the more precise you get the more restrictive you are likely to be. So there's a balance. We're not unconscious of it and we welcome people bringing things to our attention, which the Department frequently does.

Identified trauma to a joint (injury) versus occupational overuse (wear and tear) in spondylosis statements

ISSUE: Combat type service is rarely if ever of a duration which could qualify for the occupational exposure factor; and injuries occur and become aggravated in such situations, why does the occupational exposure factor require such a prolonged history of exposure?

RESPONSE: Professor Donald responded that injury which occurs as a result of eligible service is generously defined in the Statements to require only the trauma to the relevant area and resultant symptoms for 7 (or 10 days with BoP) days. Occupational overuse in the absence of such injury is a case of "wear and tear" and the literature suggests that in certain circumstances of work and loading, it is a long period of time before irreversible joint changes occur. Indeed for wear and tear to damage a joint it has to happen for

decades and 10 years is an enormously generous interpretation of the decades that it takes to wear out a joint.

Departmental representatives noted that the injury factor in the relevant Statements of Principle is quite generous and that it was important to carefully clarify the claimant's history and other evidence to ascertain the presence or absence of such an event.

The Departmental representatives also noted that a number of military personnel have served 10 years. Some of these personnel such as road plant operators and Engineers, have had their osteoarthritis or lumbar spondylosis accepted on the basis of 10 years of occupational work. It was commented that almost all who had that type of occupational history will also most likely have had an injury history that would be much more easily accepted.

Use of data from civilian and military studies and determination of dose

ISSUE: When looking at dose, does the RMA put any sort of weighting factor as the intensity of the work or the effort in the civilian study vis a vis Defence Force?

RESPONSE: Professor Donald responded that where there is published peer reviewed sound medical scientific evidence, the RMA is bound by the legislation to take notice of it. When the dose is considered, it is pushed to the lowest limits. Reasonable hypothesis requires the factor as a minimum which must exist and the real reason why the legislation requires the RMA to push the dose to its limits is that the Parliament expects the case of veterans to run a generous standard.

Sharing of data between DoD, DVA & RMA

ISSUE: Need to foster co-operation between the DVA, RMA, and the Armed

Forces medical services to provide statistical information on injury, particularly orthopaedic type injuries.

RESPONSE: Professor Donald responded that this was an issue which would be canvassed further in the discussions relating to research, to be chaired by Professor Duggan.

Representatives from the defence forces commented that, at the moment there is only imperfect liaison between DVA and Defence and that a number of factors have interplayed to contribute to this.

The Vietnam Veterans' Health Study

ISSUE: Does the RMA have confidence in the VV Morbidity Study?

RESPONSE: Professor Donald responded that the Vietnam Veterans' Morbidity Study highlighted a number of diseases for which factors exist in current statements. They are either covered by a Vietnam factor already or they are covered by other factors that would apply equally to the war in Vietnam as to anywhere else, like trauma to joints for example. The Vietnam Veterans' Morbidity Study forms part of the "information available" to the RMA and some parts of it will influence decisions by becoming sound medical scientific evidence, and some parts of it will be part of the information that sets the

scene that tells the RMA where to look and what to look for, but doesn't give us answers that make things beyond a possibility.

It's not in itself frequently likely to be the sole causative sound medical scientific evidence that we have got. The RMA has read it. We know what's in it. We know that we have got a significant number of the diseases covered, either in a Vietnam factor or in various other ways. So we will not ignore it, but it won't always be sound medical scientific evidence when it comes to the decision-making, but we will certainly check it to make sure that the factors that we have got do cover all the possibilities that are available to us for Vietnam veterans as well as anybody else.

The use of the "Vietnam factor"

RESPONSE: Professor Donald responded that the Vietnam factor can only be justified in this legal context really because it acts as a surrogate for Agent Orange exposure where no better surrogate or direct measure is available. If a better or more direct measure of Agent Orange was available, a factor such as the Vietnam factor would become illegal. There is no evidence that Agent Orange causes osteoarthritis. It is trauma that causes osteoarthritis. So in that context there is no difference between the Vietnam veterans and any other group of veterans in terms of joint injury.

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Duggan, Professor John	RMA Member
Heller, Professor Richard	RMA Member
Kearsley, Professor John	RMA Member
Lyon, Mr Keith	Repatriation Commission, Deputy President
Stevens, Maj. Gen. Paul	Repatriation Commission, Commissioner
Bordujenko, Dr Alex	RMA Secretariat
Cameron, Ms Cherrie	RMA Secretariat
Hickey, Dr Kym	RMA Secretariat
McLennan, Mr Ian	RMA Secretariat Registrar
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Kelley, Dr Jon	DVA Qld State Office
Killer, Dr Graeme	DVA National Office
Lewis, Mr Alan	DVA National Office
Maxwell, Mr Bill	DVA National Office
Reece, Mr Peter	DVA National Office
Dwyer, Professor Terry	Guest Epidemiologist - Tas
Kaldor, Professor John	Guest Epidemiologist - NSW
Smith, Dr Wayne	Guest Epidemiologist - ANU Canberra
Alexander, Mr Peter	AVDSC, NSW
Debnam, Mr GC	AVDSC, Tas
Gabriel, Mr & Mrs JD	AVDSC, WA
Griffiths, Rear Adm GR	AVDSC, NSW
Hall, Mr Brian	AVDSC, SA
Zwar, Mr R	AVDSC, SA
Keep, Mr Gerard	BCOF, Tas
Lang, Mr George	KOREAN VETERANS, Qld
Barton, Dr DE	LEGACY, NSW
Clarke, Mr Norm	LEGACY, Qld
Everts, Ms Anna	LEGACY, NSW
Grierson, Mr JD	LEGACY, Vic
Harrison, Mr Arthur	LEGACY, Tas
Scales, Dr WT	LEGACY, SA
Swinbourne, Mr & Mrs T	LEGACY, NSW
Gilbert, Mr Cyril	POW, Qld
Dowsett, Mr Michael	RDFWA, ACT

Mellor, Mr KB	RDFWA, ACT
Philip, Mr John	RDFWA, ACT
Carey, Mr Peter	RSL, NSW
Craig, Col Arthur	RSL, ACT
James, Maj Gen 'Digger'	RSL, Qld rep. & presenter
Kelly, Mr Peter	RSL, ACT
McLaughlin, Mr Noel	RSL, ACT
Naughton, Dr Mike	RSL, Qld
Printz, Mr John	RSL, Vic
Quinliven, Mr Marcus	RSL, ACT
Rogers, Mr Neil	RSL, Qld
Sheldrick, Brigadier John	RSL, ACT
Sinclair, Group Capt Doug	RSL, NSW
Doust, Mr Colin	TPI, NSW
Youl, Mr AT	TPI, Tas
McKenzie, Mr Brian	VVAA, Tas
Trevor-Hunt, Mr Geoff	VVAA, Vic
Grady, Mr Frank	VVFA, NSW
McCombe, Mr Tim	VVFA, NSW
Walker, Mr Graham	VVFA, NSW
Donovan, Commander Kevin	"Realities of Service" presenter
Flynn, Commodore Mike	"Realities of Service" presenter
Harrex, Air Cdre. Warren	"Realities of Service" presenter
McLaren, Commander Alison	"Realities of Service" presenter
Pearn, Maj. Gen. John	"Realities of Service" presenter
Rolfe, Brig. Bill	"Realities of Service" presenter
Rudzki, Lt. Col. Steve	"Realities of Service" presenter