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AUSTRALIA - MEDIDAS QUE AFECTAN A LA IMPORTACIÓN DE MANZANAS PROCEDENTES DE NUEVA ZELANDIA

Transcripción de las actuaciones en el marco de la reunión del Grupo Especial con los expertos[*]

De conformidad con el procedimiento para la reunión del Grupo Especial con los expertos y las partes y para la segunda reunión sustantiva del Grupo Especial, adoptado por éste el 11 de junio de 2009, la transcripción de las actuaciones en el marco de la reunión del Grupo Especial con los expertos científicos únicamente estará disponible en formato electrónico.

[*] La transcripción de la reunión del Grupo Especial con los expertos científicos está disponible inicialmente en el idioma original. Cuando se disponga de recursos, se facilitará una traducción al español y al francés.

ANNEX B-2

TRANSCRIPT OF THE PANEL'S MEETING WITH EXPERTS

Tuesday, 30 June 2009

Chairman

1. I would like to welcome the experts and the Parties to this meeting of the Panel on *Australia – Measures Affecting the Importation of Apples from New Zealand*. The experts with us today are Dr Cross, Dr Deckers, Dr Latorre, Dr Paulin, Dr Schrader, Dr Sgrillo and Dr Swinburne. The Panel greatly appreciates their presence and assistance in this case. I would also like to welcome the public who is following this meeting by closed-circuit TV broadcast.

2. My name is Attie Swart and I am Chairman of the Panel. I would like to introduce to you Ms Kirsten Hillman on my left, and Mr William Ehlers, who are the other panel members. The WTO Secretariat staff assisting the panel consist of Ms Christiane Wolff. She is the Secretary of the Panel. And we have Mr Jorge Castro. We have Mr János Volkai at the end there. We have Mr Adit Pujari, who is an intern in the Agriculture Division. We also have Mr Eric Gillman, an intern with the Legal Affairs Division, assisting the panel.

3. By inviting the public to view this meeting from another room, the Panel has accepted a request made by both Parties in this case. I would like to thank the Information and External Relations Division and Security and the Conference Office of the WTO Secretariat for their assistance with the preparation and organization of the public viewing of this meeting. May I remind all delegations, as well as all members of the public viewing the meeting, that any type of recording, filming, and photography during the meeting is prohibited. Also I'd like to request you to switch off your cell phones or to put them into silent mode during the meeting. Any persons causing any disruption whatsoever to these proceedings may be requested to leave the room and be prohibited from further participation in the meeting. I would like to add that this rule includes both the Panel and the Secretariat. Finally, if there is any disruption of the proceedings, the Panel reserves the right to suspend the meeting at any time and to continue it under closed session.

4. To respect the rules of confidentiality, no information, in particular exhibits designated as confidential by a Party, shall be addressed during this meeting. The Panel reserves the right to suspend the meeting at any time, on its own initiative, or at the request of a Party, either Party, if there is any risk of breach of confidentiality or of disruption of the meeting. If the meeting is suspended by the Panel for any reason, the Panel may decide to resume it in a closed, confidential session. would like to ask if any of the Parties, if one of the Parties believes that a speaker is addressing confidential information, please raise your flags so that you'll alert the Panel or the Chair to this. I note that, with the agreement of the Parties, this meeting will be conducted in English only, without simultaneous interpretation.

5. I would now like to invite the Heads of Delegation, the Parties, to introduce their respective delegations. And if you have not already done so, I would ask you to submit updated delegation lists to the Secretary of the Panel. Please also ensure that you use the microphones when addressing the Panel. This is necessary to ensure that the Secretariat can prepare a transcript of the meeting, as we've agreed, based on the tape recordings being made. It is also necessary to enable the public in the other room to hear what you are saying. So if I can ask New Zealand first, if you'd like to introduce your delegation. Ms Fearnley.

New Zealand (Clare Fearnley)

6. On my left is Professor Don McRae; beside him is Alex Smithyman from the Ministry of Foreign Affairs and Trade; Dr Rob Beresford from HortResearch New Zealand; Alice Tipping from

the Permanent Mission here in Geneva; Dr Stuart MacDiarmid, Ministry of Agriculture and Forestry Biosecurity New Zealand; David Evans, Ministry of Foreign Affairs and Trade; Dr Carolyn White, Ministry of Agriculture and Forestry Biosecurity New Zealand; Anais Kedgley Laidlaw, Ministry of Foreign Affairs and Trade; Dr Jim Walker, HortResearch; Dr Rob Taylor, Scientific Consultant; Jason McHerron, legal consultant; and Rob MacFarlane, Ministry of Agriculture and Forestry Biosecurity New Zealand. Thank you.

Chairman
7. You have noticed that there are earphones if you want to hear. Australia.

Australia
8. My name is James Baxter, I am head of the Australian delegation at this meeting. If it's acceptable, I'd like to ask the members of my delegation to introduce themselves.

Chairman
9. Please do.

Australia
10. Henry Burmester, Consultant Council with the Australian Government Solicitor. Bill Roberts, Principal Scientist with Biosecurity Australia. Mark Jennings, Senior Counsel, Office of International Law Attorney-General's Department. David Heinrich, Biosecurity Australia. Simon Barry, Team Leader, Commonwealth Scientific and Industrial Research Organization. Mike Robbins, Australian Quarantine and Inspection Service. Jasmine Tsen, Senior Legal Officer at the Office of International Law in the Attorney-General's Department. Paul Schofield, WTO Trade Law Branch, Department of Foreign Affairs and Trade. Julia O'Brien, WTO Trade Law Branch, Department of Foreign Affairs and Trade. Ben Cas, Trade Law Branch, Department of Foreign Affairs and Trade. Bruce Bowen, General Manager, Trade and Market Access Division, Department of Agriculture, Fisheries and Forestry. Caroline McCarthy, Department of Foreign Affairs and Trade. Elizabeth Bowes, Australian Permanent Mission to the WTO. Lisa White, Trade Law Branch, DFAT.

Chairman
11. As you know, the Panel was established by the Dispute Settlement Body on 21 January 2008, at the request of New Zealand in document WT/DS367/5. The Panel has standard terms of reference, namely to examine in the light of the relevant provisions of the covered agreements cited by New Zealand in document WT/DS367/5, the matter referred to the DSB by New Zealand in that document, and to make such findings as will assist the DSB in making the recommendations or in giving the rulings provided for in those agreements.

12. The Panel is mindful that, under the WTO Dispute Settlement Understanding, "[t]he aim of the dispute settlement mechanism is to ensure a positive solution to a dispute. A solution mutually acceptable to the parties to a dispute and consistent with the covered agreements is clearly to be preferred." So, accordingly, the Panel is willing to provide Parties with adequate opportunities to develop a mutually satisfactory solution, if that is possible. We, as a Panel, certainly encourage you to do so.

13. With regard to expert consultation, after its substantive meeting, the Panel decided to consult with experts who have specialized scientific expertise on the issues arising in the dispute. The working procedures adopted on 26 March 2008, after consultation with the Parties, contained rules for expert consultation in paragraph 17 of those procedures. Subsequently, the Panel has also adopted special working procedures for this week's meetings after consultation with the Parties. The Panel received suggestions for experts from the Secretariat of the International Plant Protection Convention, IPPC, and from the Parties. Following consultations with the Parties on the candidate experts, the Panel appointed Dr Cross, Dr Deckers, Dr Latorre, Dr Paulin, Dr Schrader, Dr Sgrillo, and

Dr Swinburne, to serve as scientific experts in this dispute. In accordance with the working procedures, and after having considered the Parties' comments, the Panel sent questions to the experts, and the experts were expected to reply in writing, and these replies were communicated to the Parties. Comments and counter-comments received from the Parties on the expert replies were provided to the experts, and the experts also received a copy of the Parties' rebuttals submissions.

14. So if I can turn to the conduct of the meeting today. I think the purpose of this meeting with the experts is for the Panel to obtain clarification on some of the factual scientific issues relevant for this case. In particular, the meeting should allow the experts to elaborate and clarify the written responses submitted to the questions that were posed by the Panel, and to respond to the comments made to those responses by the Parties. As well as to allow the Panel, and the Parties, to pose questions to the experts in order to seek any elaboration or clarification on issues that are relevant to the case. The Parties shall have the opportunity to pose direct factual questions to the experts. However, they must please refrain from making statements or posing argumentative or leading questions to the experts.

15. The Panel has noted the due process and procedural concerns raised by Australia in its written submissions. The Panel takes these issues seriously, and will fully consider them in its deliberations. However, it is imperative that we use the limited time we have available in this meeting with the experts for its intended purpose, and not lose focus by concentrating on concerns that might be better addressed in the second substantive meeting with the Parties, and in the responses provided by the Parties to the subsequent questions posed by the Panel. I think we'll have full opportunity tomorrow and the day after, and then with what follows. To that end, the Panel will manage its time with the experts carefully, concentrating on the most important issues within each area of enquiry, and moving the proceedings forward when it deems necessary.

16. The Panel notes that its role is not to conduct a *de novo* review of a party's risk assessment, but to make an objective assessment of the matter before it, including, among other things, an objective assessment of the facts of the case. The Panel needs to assess, for example, the sources that support the scientific basis, as well as the scientific methodological rigour of a risk assessment. The role of the experts is to assist the Panel in this assessment by clarifying scientific matters within their expertise. As such, and in accordance with the procedures adopted by the Panel for this meeting, experts should only answer questions that they feel competent to answer. Since factual issues and issues of law are closely linked in this case, experts' responses may cross into an area of competence of the Parties or the Panel. We recognize this. If a Party has a concern over a particular response, it is kindly asked to bring its concern to the attention of the Panel, and briefly note the basis of this concern. When preparing its report, the Panel will decide whether or not it is appropriate to rely on information provided in a specific response by an expert, considering also its relevance and usefulness.

17. Then, the logistics of the meeting. I think we suggest that the meeting proceeds in the following manner. Before beginning with the examination of the specific scientific issues under consideration, the Panel will give an opportunity to each expert to introduce him or herself and to make some brief general comments on his or her responses, in particular, in light of the written comments made by the Parties. Sort of, some general comments. Afterwards the Panel will group the discussion into six subject areas, and organize the discussion accordingly. These subject areas will be as follows: firstly, general terms and definitions; secondly, risk assessment techniques; then, fire blight; European Canker; apple leafcurling midge; then finally, other. For each of these areas the Panel will organize the discussion so that both Parties, and the Panel, have the opportunity to pose questions on the various issues. The Panel will normally start with questions, and then allow New Zealand the opportunity to pose its questions to the experts, followed by Australia. I'll go into a little bit more detail later on, on this. Throughout the discussion of each issue, the Panel may pose questions before, during or after the Parties have posed their own questions. The Panel may also allow

the Parties to come back with new questions, or follow-up questions. Once the Panel has decided that a particular issue has been sufficiently explored, the Panel will give the experts an opportunity to make some concluding remarks on the specific issue, if they so wish. Throughout the meeting with the experts, the Panel will endeavour to ensure that sufficient time is devoted to the discussion of the different relevant issues. In order to achieve this objective, the Panel may decide at any point that there is a need to move on to a different issue. The Parties are reminded that the time we have available in this meeting with the experts is rather limited. The experts will be asked to provide their responses individually and should please refrain from conferring during the course of the meeting.

18. Before we begin, I would like to recall that the purpose of today's meeting is to take advantage of the experts' presence, and to allow the Panel to gain a better understanding of the scientific issues before us. These experts have been selected, were selected, after extensive consultations, and again, the Panel appreciates their contributions and their presence today. Expert consultation is an important aspect, and asset, for many WTO dispute settlement panels, who are asked to make findings in disputes that frequently involve sophisticated technical issues. I'm confident that the Parties will also make the best of their expertise during this meeting. I kindly request that the experts attempt as much as possible to explain their responses in a way – and listen carefully to this – to explain their responses in a way that even those of us who do not have the benefit of their expertise, can fully understand them. Experts should feel free to respond to a question or to make comments, even if the question is addressed to another expert, bearing in mind you should feel competent to respond to that particular question. The WTO Secretariat will prepare a transcript of the proceedings of the Panel's meeting with the experts, and for the purpose of reviewing the transcript's accuracy, the Panel will send a copy of the transcript to the experts and to the Parties. The transcript of the proceedings of the Panel's meeting with the experts, and the compilations of written replies of the experts, will be part of the record and referenced in the Panel's report.

19. I have given you more or less the flow of the meeting. What we intend to do is have the Panel ask, under each of those headings, its questions. But after each specific question, we will allow New Zealand and Australia, after the experts have responded to, on that point, elaborate, if there's anything you want. After the Panel has exhausted its questions under that heading, under those six headings, we will then offer New Zealand the opportunity of asking its questions, followed by Australia asking its questions, if that's acceptable to you. We'll see how it goes. Also, I think we have consulted with you on the timing for today, and we felt, if we could, it would possibly be better if we try to deal with the experts meeting today, but looking at the number of questions it may well be that we need more time. So I think that the arrangement that we are suggesting is that we break a little early for lunch and have an hour's lunch, in other words stop at 12.30, start again at 1.30, and then continue, if necessary, until 7pm. In looking at the spread of our own questions, it seems to me that the bulk of the questions will be on the issue of risk assessment, and then the three pests. So my suggestion is that we'll try and give about an hour and a half on each of those, but we'll see how it flows, and we'll try and cover as much as we possibly can, but there will be a little bit of give and take. So that's our suggestion as to how to proceed. Hopefully we'll get through all of our and your questions during the course of the day. If absolutely necessary we have tomorrow morning, but we might be able to conclude the expert consultations today. If that's all right with you, I'd like to proceed to the experts and ask them to give us their brief introductory statements. Would you like to take the floor to make such brief comments and then I'll also just offer you the opportunity in the order that you are, Dr Cross, if you'd like to…

Dr Cross

20. I'm an entomologist and I'm an expert person on the apple leafcurling midge. Clearly there will be many questions about this pest and its biology and I hope I can help you with some guidance on that. I don't have a general statement to make.

Chairman
21. Dr Deckers.

Dr Deckers
22. I'm Tom Deckers from the Fruit Research Station PCFruit in St. Truiden, Belgium. I am working in a pomology department, and one of the items we have since more than 25 years is the fire blight research in our country. As a pomologist, I am also aware of the other important problems that are in the discussion here. I don't have either a big comment to make in advance.

Chairman
23. Dr Latorre.

Dr Latorre
24. I am Bernardo Latorre, from Chile. I'm working at the Catholic University in Santiago at the present time, primarily on fruit diseases and apple diseases as well. So I'm here to be able to help you in technical aspects of European canker.

Chairman
25. Dr Paulin.

Dr Paulin
26. I am Jean-Pierre Paulin from *Institut National de la Recherche Agronomique* in France. I work in Angers. I have been working for long on fire blight and maybe the most useful experience I have had which can be relevant to this meeting is the fact that I have followed, I would say, the spread of the disease of fire blight in Europe and the Middle East. So I have seen places without fire blight which became blighted. So this is maybe the experience which can be useful today. I have no special things to tell you, except that I am a scientist, not a legal person, so some aspects of this meeting, as probably for my colleagues, are new for me.

Chairman
27. Dr Schrader.

Dr Schrader
28. My name is Gritta Schrader. I am from the Julius Kuehn-Institute, in Braunschweig in Germany, and I am the pest risk analysis coordinator for Germany, so I have commented mostly on the questions regarding pest risk assessment. My experience with pest risk assessment is now about nine years. I am involved in many issues dealing with pest risk assessment. For example, I am a member of the panel on plant health in the European Food Safety Authority, and I was involved in drafting the standards of IPPC No. 2 and No. 11 as well. I was also involved in preparing the training material for doing pest risk analysis by IPPC. So I hope I can help you with any experience I have with doing pest risk analysis, being very much aware of difficulties with doing such analysis, because especially the lack of data is a big constraint to doing a PRA and this is also why we have now started, in Europe, a large project, with 15 partners, involving Australia and New Zealand as well, on how to improve the methods of PRA.

Chairman
29. Dr Sgrillo.

Dr Sgrillo
30. I am Ricardo Sgrillo from Brazil. I am an engineer agronomist and work for the Brazilian Government. I have a master in nuclear energy and a doctorate in entomology, a strange mixture! I have been working with mathematical modelling for the last 30 years and, since 1997, I have been

working with the IPPC in the development of international standards, three or four different standards. And I hope I can help you resolve these problems.

Chairman
31. And, finally, Dr Swinburne.

Dr Swinburne
32. I am a retired gentleman of leisure. But I have been publishing papers on apple cankers since about 1961. I think I enjoy… I deserve the leisure I am now enjoying. But I have published continuously on various aspects of this disease, right up until last year.

Chairman
33. I think we'll then turn to consideration of the different areas and I'd like to start with general terms and definitions. We have, as Panel, four questions. And I am going to, against the advice of my Panel members, start with one which I don't want you to answer immediately, but to think about. And then I'll ask three specific questions and come back to the first one last. The Panel would like to ask the experts to explain three terms. The one is "disease severity"; the second is "disease prevalence"; and the third is "disease incidence". I'll give you a bit of time to think about those. I would like to then pose firstly to Dr Sgrillo, if I may, a more specific question. You sometimes use the phrase "the scientific evidence does not guarantee that this is true". The Panel would like to know whether you are using this word "guarantee" in the sense that it is your view that the evidence does not sufficiently prove that something is true, or in the sense that it does not support the issue. If you could perhaps explain your use of the word "guarantee".

Dr Sgrillo
34. Well, in the case of assigning numbers to probabilities, I guess this phrase was used mostly commenting on the numbers that were assigned to the probabilities. What I would expect in this case is that you have a hypothesis, and then you have to validate this hypothesis. And in the case of assigning numbers to probabilities, the numbers you are assigning represent a hypothesis about the real process in the world. And when you assign these numbers you should be based on numbers from sampling of the reality. Otherwise you are elaborating on the subject. One note only: when I refer to guesses, it's not a pejorative term. I'm sorry, because English is not my native tongue. But you can discuss with your colleagues and find out about a number that you and your colleagues find is a good number, okay? Nevertheless, if you don't have numbers that were sampled in the real world, you have no guarantee that the numbers that you are assigning are representative. It's a hypothesis, after all. A common sense hypothesis, an elaborated hypothesis, but it follows that it's a hypothesis, no guarantee. This is the sense where the term "guarantee" was used.

Chairman
35. Dr Latorre.

Dr Latorre
36. Maybe I can explain. "Disease severity", "disease prevalence"…

Chairman
37. Before we go on to that question, I am going to ask New Zealand or Australia, whether you had any specific issue around the question that we've just asked. And then I'm going to ask two more questions directly, and then we can come back to the first question I asked, if that's all right with you. New Zealand, are you satisfied? Australia, please.

Australia
38. If the Australian delegation could just thank Dr Sgrillo for his clarification of the use of the "guess" word, because we did comment on it, and we fully appreciate that clarification.

Chairman
39. I'd like to then move to a second, more specific, question. Not that definitional one that I asked you just now. There seem to be differing views in expert's responses to Question 46 that we had sent to you regarding Australia's evaluation of the likelihood and implications of New Zealand apples being repacked at rural packing houses in close proximity to orchards. And this is then for all three pests. Would any of the experts like to comment or further clarify on this matter? I am wondering whether it's sensible, obviously, to give you some time to think about this. Is it useful posing a few questions, then coming back to specific questions? Or shall we just take them one by one? I am asking, really, the experts. I don't want to confuse issues, but it might be useful. Would anybody be ready to answer this specific question I've posed now, about the different views, expert responses, to that question? Dr Sgrillo?

Dr Sgrillo
40. Yes. I reproduced the model, I mean the quantitative model of Australia, in my computer and I have played around with it. This is a case of the sensitivity of the model. The model has not… The sensitivity of the model to adjust this parameter is very low. This means that you can assign different numbers to this parameter, I mean, different percentages of the repacking and the effect in the general system, I mean in the end result, representing the percent of infestation of apples, would not change too much. This means that the model is not sensitive to this process, because of the mathematical structure.

Chairman
41. Any further comment on this question from the experts. Dr Cross.

Dr Cross
42. Yes. I felt that this issue of the amount of fruit that is handled at these seven wholesalers seems to me, for the apple leafcurling midge, to be quite a critical issue. And I was uncertain how to determine what proportion of apples would be handled there with the different scenarios. So if the fruit is retail-ready, I was uncertain as to whether any of the fruit would have been handled at these wholesalers. I am also uncertain of their location, and whether they are in areas of Australia where apple leaf midge could establish and exist. So it was a complicated question that I found quite difficult to answer.

Chairman
43. New Zealand, do you want to … I see, no more comments from the … Thank you. Can we move on? Could I ask also – I noted your comment, just your statement, about – can I ask that we try and not go that route, because in terms of time, I think we all appreciate the comments that you've made, and you all appreciate that we have you available to us today, but let's try and stick to specific questions. Anything you'd like to raise on this particular issue in terms of the question, not clarification.

Australia
44. Just a clarification, Mr Chairman. I mean, we are happy to talk about that issue over the next couple of days. I think it requires a fairly detailed local knowledge about the apple supply chain system in Australia. And I think it falls into that group of issues where we'd probably be better to focus on in the next two days.

Chairman
45. I tend to agree with you. Okay. Third more specific question and then we come back to those three definitions I've asked you. Several experts use the word "negligible" in their responses. Now I wonder what you could explain what you mean by "negligible" and is there any standard or accepted definition of this term? Can the likelihood of an event that occurs several times in one year be characterized as negligible? Let me add a little tail on this. Is there any international guideline in terms of translating that into a number? If any of the experts have some comment on that … Dr Schrader.

Dr Schrader
46. Well, as far as I know, there are no standard terms, no standard definitions, for it. It's used by every country. Not everybody uses it in its risk assessments. For example, Canada uses also negligible, and gives a certain example for it. But it's not standardized in any way.

Chairman
47. Dr Latorre and then Dr Sgrillo after that. Dr Latorre.

Dr Latorre
48. When I use this term, I try to indicate that it is something that may happen so rarely, in such a low proportion, that it is almost not important. Something close to zero, but certainly it is not zero.

Chairman
49. Dr Sgrillo.

Dr Sgrillo
50. Well, as Dr Gritta said, there is no international standard. However, there is some proposal. One proposal is that "Clearly a negligible risk should be much lower than the maximum tolerable risk for a given class of hazard. We therefore propose defining a hazard as 'negligible' if its associated risk is at least two orders of magnitude smaller than the maximum tolerable risk for this class of hazard". This is a kind of definition that would help very much analysing the pest risk, the importation risk analysis of Australia. I interpret that "negligible" refers to a quantity so small that it can be ignored.

Chairman
51. Could I ask you just to explain again the proposal that there is and to whom is this proposal? Could you just explain again those numbers that you mentioned. It was quite quick.

Dr Sgrillo
52. Okay. You want me to read again?

> "Clearly a negligible risk should be much lower than the maximum tolerable risk for a given class of hazard. We therefore propose defining a hazard as 'negligible' if its associated risk is at least two orders of magnitude smaller than the maximum tolerable risk for this class of hazard."

53. I can give you the complete reference.

Chairman
54. And that proposal is to whom?

Dr Sgrillo
55. This is a proposal of Braband in a paper. I have the complete reference here. Maybe I can write down.

Chairman
56. Any other input from the experts on this question of negligible? If not, thank you very much. New Zealand, thank you, no comment. Fine. Please, Australia.

Australia
57. Just – and we will return to this in the coming days – but I think we will need to place some context around this discussion of what negligible means. We've heard Dr Sgrillo's comments, and we would just refer the experts to Table 12 of our IRA, in which we do in fact define negligible for the purposes of our IRA, and the interval is between zero and 10^{-6}. Though, in terms of the IRA, there is not an issue of definition. We have provided that in the context of Table 12, for reference to the experts. Thank you.

Chairman
58. Okay. Could I then ask that we move back to those three definitions: "disease severity"; "disease prevalence"; and "disease incidence". If – and I think Dr Latorre you were, already, in the blocs to respond to that.

Dr Latorre
59. Okay. Well, "disease prevalence" and "disease incidence" are both relative terms, trying to indicate the proportion of a disease within the population. In the plant pathology literature, both terms are very often used as synonyms. However, they are not. Strictly, "prevalence" means the proportion of the disease in the population at the beginning. And when they refer to "incidence", it is how much this proportion of the disease has changed in time. In other words, 1 per cent would mean one infected tree in 100 (prevalence, at the beginning). But maybe after a few months, you have moved to maybe 10 per cent. This would be an indication of incidence. When we speak of severity, very clearly we try to indicate how heavily is the tree infected. How many leaves of the tree are infected. So that is the definition in general of these three terms, which are very often used epidemiologically.

Chairman
60. Any further comments from the experts on that? You all support that … ?

Dr Deckers
61. I think we could add, for the severity, that it is important to follow the disease development in the plant. And this can be measured, when you say there is a disease development in shoot infection or flower infection, taking all the branch, and so on. So it is more a real, comparable possibility, to speak about the disease severity.

Chairman
62. Okay. New Zealand. Any comment here, please?

New Zealand
63. I'll just make a quick comment. The terms "incidence" and "prevalence" are used in some of the international standards for phytosanitary measures but I don't know that they're defined in a very specific way. And our experience with these terms is that it does vary very much in usage, from expert to expert, author to author.

Chairman
64. Okay. The Panel has exhausted its four questions on "general terms and definitions". Let me offer the floor to New Zealand, if you have any points you'd like to raise in this area. Questions. Not points. Please let me withdraw that word. Any questions you might ask the experts.

New Zealand
65. No questions at this stage.

Chairman
66. Thank you very much, Ms Fearnley.

Australia
67. We do have a couple of questions on this section. Before I begin, I really do need to recall that our view is that individual experts should only be asked to respond to questions which pertain to their particular field of expertise, on which they are appointed to assist this Panel. And just recalling that Dr Sgrillo and Dr Schrader were appointed to advise on risk assessment methodologies. Dr Paulin and Dr Deckers were appointed as fire blight experts. Professor Swinburne and Professor Latorre were appointed for European canker. And Professor Cross was the only expert appointed on apple leafcurling midge. And acknowledging your earlier comments, Mr Chairman, Australia will nevertheless only be directing its questions to those individuals in respect to the field of expertise for which they were appointed.

68. There's just one other matter before I begin the questions on this section. There are a couple of written materials relating to questions we will have on fire blight and canker, which are both things that are already in evidence. But in the interests of saving time, we would like to, if acceptable, provide those to the relevant experts and to you and to New Zealand now, so that they can refresh their memories about those particular things when we come to them. If your preference is, we can wait until later and do that, but it just means that we will probably have to take longer with the responses to our questions.

Chairman
69. Just so I'm clear, these are materials already available as part of the evidence, but you want, you will be asking questions related to those later in the day. That's what you are talking about?

Australia
70. That is correct.

Chairman
71. Let me just confirm… If they are already part of the record, we have no problem with that. Yes, sure. So, how would you like to proceed?

Australia
72. We have a number of copies here to provide to the relevant experts as well as to New Zealand and to the Panel and the Secretariat. And we could do that right now if that's easiest. One is on fire blight and one is on canker.

Chairman
73. That's quite all right. And the Panel has noted the point that you made to start off with, we won't comment on that, I think we've dealt with it in the introduction. And again, point taken, I think it was useful for you to have made that point. And we would like you to continue where, as I mentioned in the introductory remarks, to remind us, where necessary. We will note them. But let's not get into too much detail. And let's also try and avoid statements. I think the point that has been made, is that we will have time tomorrow to take off our jackets on all of these issues. But thank you. Please could we keep that in the background and if you want to continue with your questions, you are welcome to do so. This is now still on this issue of general comments and definitions. Please proceed.

Australia
74. Australia has a question for Dr Latorre and Dr Deckers. We note that in your written response to question 3, you both indicate that "trash", namely leaf stems and other plant organs or junk materials can contaminate fruit. We would be grateful if you could please expand on this point.

Chairman
75. The question is addressed to Dr Deckers and to Dr Latorre, is that correct? While they are thinking, can I just ask, I know you've made the point that you will be asking questions only to the specific members, but in terms of the practice that we will follow, we would like those documents available to all of the experts. But I recognize that you are saying that you will be asking specific questions to specific members.

Australia
76. Okay, would you like us to make additional copies?

Chairman
77. You can do so, if that's easy. Whatever's easiest.

Australia
78. Okay, that's fine.

Chairman
79. Shall we ask the Secretariat?

Australia
80. Yes, thank you.

Chairman
81. Excellent. If and when Dr Deckers or Dr Latorre are ready. Please proceed. Dr Deckers.

Dr Deckers
82. So the question here was, what is the importance of the "trash" here?

Australia
83. Yes, I'll just repeat the question for you. In your written responses to the Panel's questions to the experts, in Question 3 specifically, we noticed that both yourself and Professor Latorre indicated that "trash", namely leaf stems and other plant organs or junk materials, can contaminate fruit. And we were just interested if you would please elaborate on your answer in that regard.

Dr Deckers
84. Okay. I would like to answer here, for fire blight, the infection of flower is not always limited to the flower itself. It often goes into the bourse structure of a fruit cluster. So when this "trash" is consisted of fruit bourses, there is a possibility that these bourses carry the bacterium. So in this way I think it's really important to clarify here that the bourse structure of a fruit, which is sometimes picked with the fruit at harvest, can be considered as a potential contamination source.

Chairman
85. Dr Latorre.

Dr Latorre
86. In connection with European canker, if "trash" means leaves, twigs, stems, these do not represent a problem. Normally, the European canker fungi is not associated to the leaves to the point that it can really contaminate the fruit. If by any reasons you have rotten fruit, that rotten fruit with its sporulation on it, perhaps this type of trash may represent a risk. Nevertheless, rotten fruit are very rare in nature unless you have frequent, very frequent, summer rains.

Chairman
87. Can I ask, Australia, are you satisfied or want to follow up on that?

Australia
88. Just a short follow up for Professor Latorre. So as I understand it, you are referring to leaves. And you mentioned stems, I think, as well. What about twigs?

Dr Latorre
89. Stems and twigs, I don't think they can really represent a problem. When I say "represent a problem", I mean, they cannot carry the fungi, or the fungus.

Chairman
90. Dr Swinburne, I see you have your hand up.

Dr Swinburne
91. In a climate which is highly conducive to apple canker, fruiting spurs often become infected and those dead spurs, produce both conidia and ascospores. The spurs become very easily broken. They snap off. And they can form part of the trash. But this is in a climate which is highly conducive to the development of the disease that you actually see it on the fruiting spurs.

Chairman
92. Can I move… While you are conferring … New Zealand, would you like to come in on this point?

New Zealand
93. I have a question for Dr Deckers and another question for Dr Latorre, but I begin with a question for Dr Deckers. And I note that in Question 3, in the response to Question 3, Dr Deckers comments that – quote – "[f]ruits packed, retail ready can be considered automatically free of trash", and I wonder if Dr Deckers would like to confirm that that remains his view on the issue. Thank you.

Chairman
94. Dr Deckers.

Dr Deckers
95. Okay. I think when the fruit has a normal procedure of preparation, by grading the fruits in water, or whatever else, it is normal that trash is not present when the fruits have passed this chain. I think this is the normal procedure.

Chairman
96. I note, Australia was conferring, so … Are you okay? All right. Are you satisfied with the response or want to follow up, Ms Fearnley?

New Zealand
97. Yes, I'm satisfied with that response and would like to put a question to Dr Latorre. Again relating to a response to Question 3. And I note that Dr Latorre concludes his response on that question with the following comment, and it includes a double negative, but … "[f]ollowing the technical protocol, as indicated in Exhibit NZ-93, there is no risk that 'Class 1 export quality apples', exported from New Zealand will not always be mature, asymptomatic and free of trash", and I'd invite Dr Latorre to comment whether or not that remains his view on the matter.

Chairman
98. Dr Latorre.

Dr Latorre
99. I would like to say that when I answered these questions, I only gave a general definition. Perhaps I was not precise in answering in regard to the European canker. So whatever is said here is

applied to the general situation, and I am going to say, that normally leaves, twigs and stems, they are not responsible for carrying the European canker fungus. I agree with Dr Swinburne that eventually the spurs can be contaminated and that may happen only when you have heavy summer rains or very frequent summer rains and also you have a very, I mean, you may have some problem with the harvesting procedures in terms that the people is not only detaching the fruit but also detaching the spurs.

Chairman
100. Can we move on to your next questions, Australia?

Australia
101. Yes, Mr Chairman. I have a question for Dr Schrader. I'd like to ask you about one of the international standards for phytosanitary management, specifically ISPM No. 10, which deals with the requirements for the establishment of pest free places of production. According to ISPM No. 10, the four main components that should be established and maintained by an exporting country are as follows: systems to establish freedom; systems to maintain pest freedom; verification that pest freedom has been attained or maintained; and finally, product identity, consignment integrity, and phytosanitary security. Would you consider that, if technical justification has been provided for requiring apples only to be exported from a pest free place of production, then ISPM No. 10 would provide justification for whatever specific measures a particular country adopts that are required to practically implement that objective, that is, practical measures to achieve those four components, which are integral to a pest free place of production?

Chairman
102. Dr Schrader.

Dr Schrader
103. So, this is a new point arising, I think, because this was not discussed in the questions given to the experts. So, I would like to. Well, it's very difficult to answer to this now, *ad hoc*. So usually if the requirement of a country is to the exporting country that requirement of pest free area has to be fulfilled, then it has to be shown by the exporting country that this requirement is fulfilled, and this also relies to all of the different issues within ISPM No. 10 because it's not only pest free area but also pest free production site, etc. But I am not sure if that already answers your question.

Australia
104. Thanks for that. I know that it was a little bit different, but thank you very much.

Chairman
105. Anybody want to… New Zealand. Right. Next question, Australia, do you have…?

Australia
106. We have nothing further, Mr Chairman.

Chairman
107. We move on, then, to the area of "risk assessment techniques" and here I have the floor, or the honour, to ask the first question to Dr Latorre. In your response to Question 124 – I think this was about international standards for semi-quantitative methods – you wrote that, in your view, the likelihood values and mid-point values used for Australia's semi-quantitative analysis in the IRA should be validated before acceptance. Could you explain what "validation" means in this context and how it would be done?

Dr Latorre
108. Well, in this context, it only means that we have to have some experiment to provide objective data that allow us to assess the information that there was in there. To have an experimental approach, in other words, in order to know whether this data that was given here are within the acceptable range, that was it.

Chairman
109. New Zealand. Australia.

Australia
110. Can I just follow up on that answer. So, as Professor Latorre, is his comment applied to the choice of methodology and the way the methodologies are applied, or is his answer specific to his views on European canker.

Chairman
111. Dr Latorre.

Dr Latorre
112. Yes. All my questions are thinking on European canker primarily. My answers, excuse me.

Chairman
113. Dr Sgrillo, if I may ask another question on risk assessment. In your response to Question 133 – this is about probability intervals and descriptors – which asks whether the IRA provides objective and coherent explanations for the probability intervals assigned to the qualitative descriptions of events. You write the following, I quote: "The numeric probabilities representing the qualitative descriptors in the IRA are to be interpreted in a *per unit* basis. However they have to reflect the concept of each category (negligible, low, etc) also in populational terms". Could you please explain this a little bit further and also elaborate on your comments about the distortions that appear at the lower part of the categories, if you wouldn't mind.

Dr Sgrillo
114. Well, there is no international methodology for developing this kind of standard. To my view, the beginning is the population. All the risk is proportional to the population. So when you will start a pest risk model, you have to keep in your mind in what population that model will be applied, even defining individual risk. I mean, the individual risk without a population is nothing. There is no individual risk, because you define individual risk in population terms. When you say one in one million to define an individual risk, what is one million? It's a population. So you need the population to give a reference to your individual risk. So I say again, an individual risk without a population as reference, can not be defined. This is why an individual risk should be established, should be given, with the population in mind. If you say that the risk of an event to occur is "negligible", and that event in a population occurs a thousand times in a year, it's not negligible anymore. And that's why appears this "extremely low" with a hundred thousand occurrence in one year. This is not extremely low: a hundred thousand! "Very low", ten million occurrences in one year. Is this very low? So when you will develop a probabilistic model, a kind of Monte Carlo model, I mean, in which you multiply a sequence of probabilities and then you multiply a population by this sequence of probabilities, you have to keep in your mind for which population you are developing. And you have to be careful with the qualitative definition and the meaning that in the population context.

Chairman
115. May I just follow up on this. Are you, I heard you say, I think, that something which is expressed as a probability per unit might be "negligible", but when it is translated in terms of the population, you are saying the qualitative descriptor would be something else than negligible.

Dr Sgrillo
116. Yes. Any of this – any distortions – I mean, you have an interval of probability, from zero to one. This is all the possible probabilities. You divide this interval and you give names for this interval. One, if it always occurs for all the population, it will result in a population size. Zero point 5 it will result in half of the population. Now we have to give names. If you take the population and say that one million will happen of an event, one million times will occur in one year, you have to give a name to that. This is not negligible, this is not very low. It's a kind of criterion taking into account the population size. I have a quote, "most people want to ask, 'One in a million, is that a lot or a little?' There is no good answer to that question. If one focuses upon statistics, it may seem very little. If one tries to focus upon the 250 or so individual deaths that this number implies in a population of 250 million, it may seem like a lot." So it's dependent on definitions, but coherent definitions.

Chairman
117. Maybe just a comment on your concerns about distortions at the lower part of those… What did you mean by distortions?

Dr Sgrillo
118. Well, distortion is that there is no link between the name and the numbers. I am saying that these are distortions. I mean, there is no way to link 10 million occurrences with "low".

Chairman
119. From our side, that's fine. New Zealand, do you want to… Are you happy?

Australia
120. We do have questions to Dr Sgrillo on this point. And it is a very important point. But we'd prefer to hold them for our questions so we don't delay you.

Chairman
121. Sure, okay. Let me ask, then, a next question in this area. New Zealand notes in its rebuttal submission that the numerical ranges for probabilities used in the IRA, are based on Biosecurity Australia's draft guidelines for the import risk analysis, 2001. But that these draft guidelines were not developed with the per apple methodology in mind. And I wondered if any of the experts are aware or familiar with these guidelines and whether you'd like to comment on this point. I have two other questions relating to the same heading, shall I say. Let me ask them as well. Even if you are not familiar with these draft guidelines, could you comment on the relationship between – and I have this question in two ways, because I think they're different perspectives – please comment on the relationship between the object of measurement, namely apple fruit, and the predetermined probability ranges. Do the probability ranges have to be defined with regard to the object at risk in question. I can ask the same question, but change the word "object" into "unit". In other words, does it make a difference? I think that's maybe more obvious, but I'd like to ask that question as well. Whether the unit of measurement, tonnes or units, or whatever, referring to your populational discussion just now, whether that would make any difference. And then finally, is it objective and credible for a risk assessment to use the same predetermined probability range for the different steps and for different pests, or would they have to be derived from the science and the data. So these questions go to any of the experts. Dr Sgrillo.

Dr Sgrillo
122. Well, this is the problem when you don't have enough data to support your choices. You have to rely on qualitative definitions, and this is just a matter of discussion. There is no road to that, there is no established criterion, no recommendation, in international terms, at least as I know. And this is why quantitative models should be applied only when there are sufficient data to support the choice of probability ranges and probability shapes. Otherwise we can go to the end of life discussing, and

there is no way to define this, to prove this, and to choose a range. Or go to the field and make the sampling, or the implementation necessary, and then derive your numbers.

Chairman
123. Dr Schrader.

Dr Schrader
124. Yes, I fully support what Dr Sgrillo was saying and I can also add something to that, because we have, in our project that is currently underway, we have assessed more than forty different risk assessments and guidelines to see how different bodies deal with exactly this point. How to do the risk rating in a standardized way or in a calibrated way. And up to now there is no method which really is reliable for every question for every pest. So expert judgement is something which is still very important in pest risk assessment. And this is also one of the main problems, because you always have to be transparent and up to now you have no good method to compare between the different pests that have the same levels of ratings.

Chairman
125. Did I, Dr Schrader, did I hear you say you have no way of checking for different pests ratings. My question really is, our question is really, I mean, one can translate it, is "negligible", in terms of a risk with regard to pigs, the same as "negligible" in terms of a risk with regard to apples? That would be the one question. And the other question is, can one in the steps use the same numerical probability to define the same qualitative descriptor in each of these steps. So if you are saying it is negligible in step one, would you be able to use the same probability for negligible in step two? Is that what I understood you to say, that there is no commonality there yet?

Dr Schrader
126. There is no commonality, in the one point. And also it's very difficult to do that for different pests. On the other hand, it should be the same, it should be comparable.

Chairman
127. Any comment from New Zealand on this issue? Or, not comment, again, question, elaboration?

New Zealand
128. Not for the moment.

Chairman
129. Australia.

Australia
130. Perhaps my colleague, Dr Barry, might provide a little elaboration. I'll explain Dr Barry's CV perhaps when we ask our questions, but he is, I would say, an eminent statistician in Australia.

Chairman
131. And this is a question, not a comment?

Australia
132. No. It's just to assist this elaboration on what Dr Sgrillo was saying about the intervals, so…

Chairman
133. Again. I mean, I'd like you, Australia, to consider the possibility of dealing with that tomorrow.

Australia
134. Yes. It's just that the way the questions were unfolding there… Well, we'll come to it, we'll just hold off on it.

Chairman
135. Okay, great. Why is the Chair asking all these questions? Well, it's because the Chair put himself into this position. My colleagues will be following. I have two more questions. The first one to Dr Sgrillo. In your response to Question 133, you write that in your view the probability interval seems to have been arbitrarily chosen, and that other ranges could also be used without violating any scientific principle? And, in particular, the range assigned to "negligible", this is the zero to one in a million, would be arbitrary because other ranges could have been used after adjustment of the remaining ranges. In its comments, Australia interprets this as indicating that the choice of one in a million does not violate any scientific principle. New Zealand comments that the value is not supported by the scientific evidence when assessing risk on an apple basis. Could you comment on this. And does "arbitrary" mean that this range is not objectively justifiable, i.e. that it is biased?

Dr Sgrillo
136. Yes. There are two things to consider there. One is that the model uses mostly uniform distribution with this range. I responded to this in another question. Most of the sampling, when you generate random sampling from a uniform distribution within this range, most of the sampling will go to the side of one in one million, not to the side of zero. So, to correct this, they could have considered a triangular distribution with the most probable value zero and the maximum value one times ten in the power of minus six. This will correct the kind of distortion (of bias) in generating random samples in this range. Another thing that can be shown is that negligible in this range, one in one million, in populational terms, can generate 100 or 200 events in one year. To correct this, to be more coherent, to reach a definition of the word "negligible" as a quantity that can be ignored or excluded from consideration, too small and unimportant to be of concern. Another definition: to a degree that is negligible, so small or slight that it may be ignored. I mean, it's there but it may be ignored, not considered. So, it would be much more coherent to choose an upper limit, maybe of ten to the minus twelve, or to the minus 16, or anything like that, as negligible. Or, to change for a triangular distribution with the most probable value being zero. Trying to do, because this has to be by experimentation, and see what are the results, if the results are coherent. So you can really choose the ranges and probability shapes that you are working. Because in the model development, usually you have results, you compare your results with the reality, and then you adjust the model to make it more close to the reality, and then you go in a looping up to a point where you can say "my model is fine, now I can trust it and I can use it". But not before that.

Chairman
137. Okay. Anything from… Any further elaboration on this point by the experts? If not, New Zealand, would you like to… No? My last question under this heading would be, is assigning a negligible event, one that would most certainly not occur, a likelihood in the range of between zero and one in a million, with a uniform distribution and a midpoint of five to the power of -7, based on objective scientific principles? There are two related questions. Would a different type of distribution, and we might have actually heard some answer to this already, would a different type of distribution be more appropriate for events that have a negligible likelihood of occurring, i.e. that it would almost certainly not occur? I think I heard you to say that triangular distribution, but there might be other comments. And the third question, is there a valid scientific basis for including such steps in a pathway model? Is there a valid scientific basis for including such steps – negligible steps – in a pathway model?

Dr Sgrillo
138. Well, considering that negligible is not zero, it should be included in the model. But if it is zero, no. But it should be included with values coherent with the definition of negligible. And

considering another type of distribution, one try could be a triangular distribution, with the most probable value as zero, and the maximum of 1 in 1 million. It can have the minimum and most probable value equal, no problem. Okay. But this is a try, to see what would result.

Chairman
139. New Zealand. No? Australia? Are you satisfied about moving on, as we discussed just now, about this triangular distribution, to deal with that tomorrow, or would you like to make …

Australia
140. Mr Chairman, I think I indicated before that we would not interrupt again. But we reserve the right to come back, and including on that question about a triangular distribution, when we have our questions to Dr Sgrillo. I really just don't want to interrupt you.

Chairman
141. No. I appreciate that, and that's fine. All right. Well, I think that, from the Panel's side, I have exhausted my questions. William, would you like to move on to another question?

William Ehlers
142. In the responses – and this goes to everybody, so feel free – in the responses, several experts note that for different steps in the IRA, data or evidence were insufficient. In its comments, Australia characterizes this as a situation where there is a scientific uncertainty, where experts have to make judgements. In New Zealand's view, Australia misapplies the notion of scientific uncertainty be equating it to situations where the data do not support Australia's conclusions. Would any of the experts care to comment on these contradictions? Yes. I'll repeat. In your responses – those of the experts – several experts note that for different steps in the IRA, the data or the evidence were insufficient. In its comments, Australia says that this is a situation where there is scientific uncertainty and therefore that experts have to make judgements. New Zealand, on the other hand, says that Australia misapplies the notion of scientific uncertainty by equating it to a situation where the data do not support Australia's conclusions.

Dr Sgrillo
143. Well, what I see is that the data does not support, or mostly the data does not support the Australian conclusions, but does not support, also, other conclusions. Because the data really is not adequate to generate the kind of numbers that the people need to proceed with the model. And another consideration is that one would expect the expert judgement in one or two steps in a model. But in this import risk assessment, expert judgement, well, were used in every step of the quantitative model. I think there is not a single step, a single importation step, that the numbers came from sampling, or anything. I think that every step was based on expert judgement and this is why I think that a quantitative model should not be used when you do not have enough data to support the numbers that you have chosen. o that's a problem on the expert judgement. You come out with a number. But we come out with a number and a certain expected error in this number, that comes from the process itself. Another thing. There is a methodology of expert judgement, to aggregate the opinion of each expert, and to come out with a single number from a bunch of opinions. There is a methodology to do that. And there is an error associated. But the error of one expert judgement here will accumulate the error of the next expert judgement, with the next expert, so if you have a collection of expert judgements, we would expect that the error would be quite large in the final, in the end product of the model.

Chairman
144. Doctor, could I ask whether you mentioned that there is a method for dealing with expert judgement? Was this used in your view in this case?

Dr Sgrillo
145. I don't know. I don't know, because there is no mention in the import risk assessment of the methodology for expert judgement.

Australia
146. I promise not to interrupt, Mr Chairman, but I think this is a factual clarification, which is essential to be made at this point. The Australian delegation would direct Dr Sgrillo, the New Zealand delegation, and the Panel, to Appendix 1 of the IRA, part B, around about page 332, where it explains how expert opinion was elicited, in the context of the IRA process, and we're more than happy to come back to that, but that was an essential factual clarification, because there clearly was something in the IRA.

Chairman
147. Thank you, that's useful.

Dr Sgrillo
148. I just want to apologize, because this was my fault, I didn't look for this information. Sorry.

Chairman
149. On this question…

Australia
150. Mr Chairman, sorry. I don't think all the experts were in the room when the question was posed and Australia would be particularly interested to hear Dr Schrader's response to this question.

William Ehlers
151. I'll repeat the question. In their responses, several experts noted, that for different steps in the IRA, data or evidence were insufficient. In its comments, Australia characterizes this as a situation where there is a scientific uncertainty. And therefore that experts have to make judgements. New Zealand, on the other hand, says that Australia misapplies the notion of scientific uncertainty by equating it to situations where data do not support Australia's conclusions. So, we're trying to seek a way to reconcile these contradictions.

Chairman
152. Dr Schrader.

Dr Schrader
153. Well, if there is uncertainty with a question, it has to be lined out fully and explained where this uncertainty is coming from and that may have an impact on the likelihood of a risk to occur. So, if this is fully explained, then it is transparent. But, of course there is a difference if you just don't answer a question because there is no data and you try to, nevertheless, give some estimates on the risk. Or, if you just try to hide some lack of evidence for it.

Chairman
154. Australia, would you like to follow up? New Zealand?

New Zealand
155. Not for the moment, but we may well come back on this issue.

William Ehlers
156. In its comments to experts' responses, Australia has repeatedly argued that in cases of uncertainty, or considerably limited, or varied data, the IRA team decided to exercise its expert judgement to make estimates. Australia has also argued that the possibility of completing the analysis

through the exercise of expert judgement is recognized by ISPM No. 11. Do experts agree with Australia's characterisation of a possibility of a risk assessor completing the analysis through the exercise of expert judgement in cases of uncertainty, or considerably limited or varied data? Are there international standards regarding the exercise of expert judgement, and the limitations to such exercise, under ISPM No. 11 or any other source?

Dr Sgrillo

157. I have a comment on expert judgement, that is in a paper of an Australian – Mike Nunn – and the paper is "Quarantine risk analysis" (1997) – in the *Australian Journal of Agricultural and Resource Economics*. He mentions "However, quantitative risk assessment has also been criticized essentially for a perceived lack of objectivity, resulting from the use of expert judgement that allegedly reflect, not only scientific knowledge, but also factors such as policy values and cultural values. Some commentators have expressed concern that scientific judgement involved in risk assessment is not as objective as maybe purported and that quantitative estimates have a large variability and uncertainty. Particularly when applied to environmental problems." So, again, it is an important problem that could exist when too much expert judgement is applied.

Dr Schrader

158. Well, as far as I know, there are no standard rules or standardized methods to do the expert judgement. There are different approaches on how to do it. In some cases, it is just that the experts meet together and discuss to find consensus on how they would rate a risk. But, generally, this is one of the main constraints of PRA, that if there is a lack of data and you need to judge the risk because you want to protect the country from that pest, so you have to rely on this expert judgement because no other data is available. So there are different kinds to do this. One is to do some modelling, but of course also modelling has some constraints. So you will always have to deal with this uncertainty. If you do the expert judgement you have to explain fully and transparently why you came to this result. If this is reliable, then you have to also use that because it is the only way to address the risk, or to deal with the risk.

New Zealand

159. I wonder, given that we are continuing to explore with the experts the issues around expert judgement, whether the experts have available to them the material referred to as Appendix 1 to the IRA Part B mentioned a few moments ago on page 332. If it is not readily available I could actually read it, if that would be helpful. We then have a follow up question based around that section. The section on page 332 that has been referred to is headed "The elicitation of expert opinion" and reads as follows:

> "There were multiple discussions with the panel and Biosecurity Australia staff about the eliciting of expert opinion. Three considerations were stressed by BRS. They were:
>
> That Biosecurity Australia should be completely satisfied that the bounds of distribution chosen to represent their views would contain the true value.
>
> That the chosen distribution shape should represent expert views. The interpretation of prior distributions in terms of gambling odds was discussed, and the uniform, triangular, and Pert distributions presented at starting points.
>
> It was made clear that qualitative likelihood ranges should not constrain their options and that BRS was available to assist in expressing the experts' views, if necessary.
>
> The issue of expressing divergent opinions was also discussed and different approaches to handling divergence of opinion were canvassed."

160. This would seem to be the extent of the material referenced in terms of eliciting expert opinion. Based on this, I would have a question, particularly for Dr Sgrillo and Dr Schrader, about the outline that is provided here as a "methodology" for the application of expert judgement. In particular, is the conclusion that the interval contains the true value sufficient, especially given that the negligible interval spans many orders of magnitude and a uniform distribution is applied.

Dr Sgrillo
161. It is not explained here how the aggregation of the value could be. This is a very technical issue, so I don't know. You can have a Bayesian aggregation or a classical aggregation. It is a kind of mathematical treatment of the data to converge different opinions to a single number. When I mentioned that I have not seen this, I am referring to that I thought it would be here. I would like to have a more detailed description of the expert opinion, because this is not enough to technically evaluate the process.

William Ehlers
162. Australia?

Australia
163. Dr Sgrillo and New Zealand, I didn't point this out previously to you as an exhaustive explanation of what occurred. This is a summary, in a sense saying what occurred. I would say that we are directly in a position to provide you with much more detail through the province of my colleague, Dr Barry, who actually was intimately involved in these discussions with the IRA team. If you will allow me I might just briefly provide some background on Dr Barry, so that you know what his background is. He has PhD in Biostatistics and heads up a group of 40 statisticians at the Permanent Australian research body, CSIRO. He has extensive experience in risk assessment and he was lead author of the uncertainty chapter in the International Panel on Climate Change report on landscape change and forestry and he is also author of the section on the Monte Carlo method in the encyclopaedia on biostatistics. I would just put this forward so that you are aware of his expertise, and I would invite him, Mr Chairman, to provide some more detail. This isn't in the form of questions or anything but Dr Sgrillo was clearly asking for more detail. We are in a position to provide that now. And with your permission, we will do that.

164. I think the Appendix in the document outlines at the high-level the approach that was taken to elicitation. I think to clarify, in terms of combining the expert opinions, it wasn't a matter that each person in the room had their scientific views elicited and then combined. They came to it as a consensus process. From the last point in the document it talks about how we discussed, if people couldn't come to consensus. If consensus could not be reached, there were ways that could be expressed in the document, so people were not forced to come to consensus. So that option was open to the panel but they never asked for that assistance. In terms of the range, I think picking up on New Zealand's point about ensuring that the negligible range contain the true value. We talked quite extensively over a series of meetings about the requirements that were needed to make their beliefs and their uncertainties expressed through the distributions. What needed to be done is outlined in the summary for the results of the analysis to also be clearly interpretable and so that was done. I would also note that, I think, through the document you will see that there are occasions where data was available or they had a most likely value, or they thought they understood it better. They actually chose distributions beyond the starting point distributions and then we assisted them in developing Pert distributions or triangular distributions and I think there are examples in the document to show that.

William Ehlers
165. In view of this explanation, was that a reasonable thing to do? Do you think that this expert judgment was used in a reasonable manner in the IRA?

Dr Sgrillo
166. In one or three steps it will be acceptable, but applying to 100 per cent of the steps, it would be a risk that the reliability of the outcome from the model will be limited because of the excessive use of expert judgement. Notably, for every importation step, seven steps, almost seven expert judgements.

William Ehlers
167. What level of scientific evidence is necessary in order to assign ranges and shapes of distributions? If they are assigned on the basis of an informed guess or non-validated hypothesis, does this constitute credible science?

Dr Sgrillo
168. Well, there are some rules to accept the result of a sampling. I mean randomness of the errors and technical issues, and representativeness, I mean on place and space. Nevertheless, there are criteria that you apply to say how much I can trust the sample. If your sample pass this criteria, this is a scientific support. This is enough to use the numbers. You have to clearly specify what the criteria are, especially the representativeness in the space and in the time. A single year sample is enough? Who knows? Was that year an average year, or was it an atypical year? And things like that. While sampling from a single space, a single area, the same consideration applies. Also, I mean the structure of the sampling methodology. It would be a random sampling, it would be in blocks, things like that. If the sampling method is described, and it is within your criteria, so you can accept, and this will support your data with no problem. Otherwise you can use part of the data and infer something. Or, you can go to expert judgement when data is not available. It is a valid methodology, but the application is not to be for all the model.

Dr Schrader
169. It is just that the less data you have, the higher the uncertainty. That also refers to the risk.

William Ehlers
170. I would like to move on to the next questions quickly, so we can give at least half an hour for the questions that come directly from the Parties. Australia argues that certain steps in the fire blight pathway are difficult to establish experimentally, either in the laboratory or under orchard conditions and that when comparing such evidence it becomes a case of balancing one type of uncertainty against another. According to Australia, in such situations it would be an error to automatically give more weight to experiments conducted under orchard conditions. Would anyone wish to comment on that?

Dr Paulin
171. It is absolutely true that in fire blight especially, some steps are very difficult to cover experimentally. Well, normally, orchard conditions would represent what is called sometimes a "true life", but if you keep to orchard conditions you could miss some things. Sometimes it is better to go to artificial experimental conditions, i.e. well controlled conditions, and events that you see may be reliable to infer what is happening in the orchard. So the orchard condition may be the best condition in some cases, but it is not a general rule. Sometimes artificial conditions in the glass house, for example, or in the laboratory, may be better in my opinion.

Dr Deckers
172. I would like to add a comment here that we can not only consider the orchard situation as this, as a possible scenario for fire blight disease. We know that some other host plants can play an important role together with the situation in the orchard itself. So I think it is important to mention it here also. Of course, some of the trials are done in controlled conditions in greenhouse or climate chamber, or whatever. This is good for getting information on it, but you can never rely completely.

The situation in the orchard is different, not only due to the fruit trees but also due to the other host plants involved.

William Ehlers
173. Does using hypothetical pathways in a risk assessment provide the necessary scientific and methodological rigour to be considered reputable science?

Dr Paulin
174. It is very difficult, but if you have to consider hypothetical pathways, that means that you don't know about the true pathways. So then you have to go to some hypothesis because you know that some events do happen and you just cannot explain by actual level of science, so I think it is then necessary to go to hypothesis. This is not outside science. It is not experimental science. I would consider that as science.

Dr Sgrillo
175. Actually any path can be included, it depends on the value of the probabilities. You can say, this is equivalent to say that everything can occur. And everything can occur. The probabilities will say how often that thing will occur. So if you have a good reason to include a path, but you have no experimental evidence of that path, I think you can consider that path but with a residual probability, something like negligible, something like that. That would give us a chance of that path of that hypothesis to occur once each many time units.

William Ehlers
176. Let me move on to my last question so that then I can hand back to the Chairman for the Parties' questions. Assuming that the semi-quantitative method could be considered a legitimate methodology, do you believe that the way this IRA applied the risk assessment methodology is objectively justifiable?

Dr Sgrillo
177. Well, I have to say one thing. In the case of this import risk assessment, why is the model semi-quantitative? It is semi-quantitative because the probability of entry, introduction, it is a pure quantitative model, and the consequence assessment is a pure qualitative model, so it's is a semi-quantitative model. But entry, establishment and spread is a quantitative model. The problem is not with the model, the structure of the model is okay, it is acceptable. The problem is with the numbers that you are feeding the model and your model is so reliable as the numbers you input. So you can have a perfect model but responding with very bad things because you feed it with bad things. "Garbage in, garbage out". I am sorry with these terms, but this is a common terminology in computer sciences. The model is fine, but the numbers that you are feeding the model are questionable.

William Ehlers
178. Mr Chairman.

Chairman
179. Australia, would you like to follow up on that?

Australia
180. We just want to be quite clear about what Dr Sgrillo was saying, that the actual model itself that is being represented in the IRA was okay, it was acceptable from your perspective. I think we just need to be clear because I think we need to differentiate between the model and the inputs. And inputs raise a whole lot of other questions which we will come to in the next couple of days.

Chairman
181. I see Dr Sgrillo confirming that.

Dr Sgrillo
182. Yes, yes.

Chairman
183. What we would like to do now is to offer New Zealand the opportunity of a few questions, if you wish, and then Australia a few questions, if you wish. If it works, we will try to finish this section but if it does not, and you have questions, we will continue after lunch.

New Zealand
184. Actually, at this point we are willing to forbear our asking questions, and pass the floor across to our colleagues,

Chairman
185. So New Zealand has at this point, under this subject matter, no questions. We can pass then to Australia, if they have questions.

Australia
186. The first question we would direct is to Dr Schrader. We have had a lot of discussion this morning about expert judgement, but I don't think we have asked the experts yet what they understand expert judgement to be. I think that is a very relevant question and I think Dr Schrader, given her experience with ISPM 11 particularly, would be well placed to provide some answers to what your understanding is, Dr Schrader, of what is meant by expert judgement.

Dr Schrader
187. I can especially refer to how we do it in the EPPO context. That is that the whole risk assessment is divided in many questions. There are about 70 questions in the whole EPPO scheme that have to be answered, as detailed as possible. Usually we do that with different experts, concerning the pest, the crop, also methodologies, etc. So usually we do it in a qualitative way. We refer to every reference that is available, every personal experience with the pest, the crop, etc., as available, and this is discussed and then it is tried to come to a common result on each question. So we don't do it statistically. This is also why I cannot really give answers to all these statistical questions. We try always to find a common result on every question and then also we try to consult experts from outside this working group to do a review on how we dealt with the questions. There is also another step that the plant health panel of the European Food Safety Authority also involved in that process, to go through every question and see if it is reliably answered, every information that is available has been used, and also if the conclusion that was made by the experts is transparent, reliable, etc. So if there are quantitative data available, especially regarding frequency and volume of trade, for example, or amount of damage, etc., then this is included into the risk assessment of course. But in many cases we only have these qualitative data. Does that answer your question?

Australia
188. Just to confirm our understanding of the ISPM No. 11, there is a recognition there that there are uncertainties in plant health risk assessment and that expert judgement does have to be utilized in terms of doing a risk assessment.

Chairman
189. I note that Dr Schrader confirms that that is the case. Australia.

Australia
190. If we could move on to a question that now goes to this issue that Dr Sgrillo has been exploring with us this morning and that is the lack of data that arises in the area of risk assessment. In Australia's comments on Dr Sgrillo's reply to question 124 which concerned, in fact, the IPPC guidelines for risk assessments, we referred to a statement made in a paper that Dr Sgrillo had referred

to, Firko and Podleckis (2000), and that statement was to this: "Ideally, existing data would provide the basis for direct estimation of model inputs. However, scientific experiments are seldom conducted specifically to provide these estimates for risk assessments and results are seldom provided that can be used directly in our models". Could I ask first, Dr Sgrillo, whether in your experience in conducting risk assessment, you have had to confront the issue of a lack of data and how did you respond to that. It is to get some sense of what your experience is of what would appear to be a particularly thorny issue in this area.

Dr Sgrillo
191. Yes, this is true. It is difficult to find out the data needed in the models. Not only models for risk analysis, but mathematical models in general that use parameters. It is difficult many times to find the right data. Fortunately, there are other techniques than quantitative only, so when the lack of data, you see that the data will not support your choice, then you have to move to another approach to solve that.

Australia
192. Could I direct the same question to Dr Schrader, given her experience in the European context.

Dr Schrader
193. Could you explain a little more what exactly you want to know.

Australia
194. Certainly, Dr Schrader. It relates to the issue of the lack of data that may be available and the quote was really, we would say, indicating that this is a common issue in the area we are talking about, which is risk assessment. The question is directed at what is your experience in the conduct of risk assessments and handling the absence of data that might be used in your assessments.

Dr Schrader
195. This is in fact one of our main problems. We try to estimate the answers if there is no data available, but sometimes you can refer to other information, to other pests. For example, where you have some data. You can only make estimates sometimes. Sometimes you even cannot answer the question that is related to this lack of data. Usually, there are other points that you can answer. So sometimes you have to deal with a high uncertainty. In some cases, just to accept that there is a high uncertainty. It depends also on the other points. If there are high indications for risk that may give you the indication, even if you don't know anything about a certain area of the risk assessment, you may nevertheless say that there is a high risk because of this or that point. On the other hand, if you estimate that the risk was low, then you will come to another conclusion because of the lack of data.

Australia
196. Thank you very much for your answer. That was very comprehensive. Could I just clarify, in relation to your reference, to other points in the risk assessment. Would you include within that broad definition of points the likely consequences of entry, establishment and spread of a particular pest that came into a country for the first time? So, you would also look at the consequences side of it as well? If I am understanding you correctly.

Dr Schrader
197. Of course, if you know that there is a very high risk that there will be a large impact in the country where the pest is introduced and you have some open questions regarding entry, then you would be more careful if you already know that the impact would be very high.

Australia
198. One last question from myself and then Dr Barry has a couple of questions. Just to return, Dr Sgrillo, to your comment earlier in the morning when we were discussing the interval $(0, 10^{-6})$. You noted that this interval, when used in the IRA, was applied as a uniform distribution. You suggested that instead a triangular distribution could have been used. Could I just follow that point up, because this was an issue that was raised by the Panel in one of its questions to the experts – Question 135 – where experts were asked for their views on the different types of distributions, uniform, triangular, and so on. The question is this. When experts replied, they indicated that a uniform distribution is used where there is not very much available data so that you can only place a minimum and maximum value and, as used in the IRA, this interval of $(0, 10^{-6})$ applying to what we would say are quite rare events, almost by definition, there won't be a lot of information available about rare events, and accordingly the IRA team considered a uniform distribution was appropriate. Would you agree that the use of these distributions is very much going to depend on the amount of information that you have and if you don't have information that would allow you to identify a most likely value, then you would come back to a uniform distribution in appropriate circumstances?

Dr Sgrillo
199. Yes, I would say that the choice of distribution will depend on how much information you have, not how much numerical data only, but how much information, numerical data and other kind of information. If you want to represent an event that is close to zero, it is completely valid, you choose a triangular distribution with the most probable value zero, and then choose the maximum value to give some variability to your generated number.

Australia
200. Could I ask my colleague Dr Barry now to...

201. Dr Sgrillo, in reply to question 133 concerning the probability intervals contained in table 12 of the final IRA report, you stated that the intervals seem to have been arbitrarily chosen and that 5, 7 or 8 categories could have been used instead of 6. In Australia's comments, it noted that your statements appear to suggest that various approaches to setting intervals were available and as such the use of 6 intervals, rather than 5, 7 or 8, was not inherently flawed. Do you have any observations you would like to make on Australia's comments?

Dr Sgrillo
202. Yes, I have. Actually I was trying to find out a solution for the negligible event. I mean that "negligible" means "negligible", when you treat with the population. That is my problem with your choices. This is because the population is high. You have to compensate this with some kind of adjustment in the individual probabilities. Another thing is that you can "linearize", the generation of sampling numbers: one sampling number for the exponent and another sampling for the mantissa. So you will have an even distribution from one to zero. If you linearize that you have an even distribution. Every range will have the same chance to be sampled. Otherwise, you know that you will concentrate your sampling in the upper part of the distribution.

Australia
203. So is there any international standard on the number of categories and the width of the intervals that should be used?

Dr Sgrillo
204. As far as I know, there is no guidance to modelling in general. I mean, quantitative or qualitative. The only guidance is not in the IPPC but in the OIE. The IPPC just refers to the names: there is "quantitative" and "qualitative".

Australia
205. On a new topic, Dr Sgrillo, in your reply to question 134 concerning the use of the interval $(0, 10^{-6})$, to define "negligible" in the final IRA report you stated that "[c]onsidering the importation of 200 [million] fruit per year the category negligible could represent 200 fruits per year. However, an event that 'would almost certainly not occur' should be expected to occur only once in each several years."

Dr Sgrillo
206. Yes, there is no place that this is written, that a "negligible" event, in the phytosanitary context, should represent one event in each several years. This came from my experience as a technical secretary of COSAVE. COSAVE is the plant protection committee of the Southern Cone (Brazil, Chile, Argentina, Uruguay and Paraguay). We had some discussions on that and this came that a rare event will occur one each several years. But this is not recognized. It is not a rule. It is just a consensus of a group.

Australia
207. So I agree that if there were two hundred million fruit that carried a particular pest or pathogen, then applying a one in a million probability of an event occurring, would lead to 200 expected events. Would you agree that if only a proportion of the population was infested, then the expected number of events would be reduced proportionally, i.e., if only one percent of the total population was infested, i.e. only 2 million fruit out of a two hundred million, then the number of expected events would be 2, i.e., one percent of two hundred.

Dr Sgrillo
208. Yes, but look, I am mentioning here, each step, I mean each probability range, apart from other. I am not aggregating probability ranges, I am trying to evaluate the final. If you say that the probability that an apple has a hole is one in one million, I expect that in 200 millions, I will have 200 apples with a hole. But just a single probability range of – considering one event, not considering the effect of one probability, and another, another and another.

Australia
209. I agree with you there. So in the fire blight risk assessment, if we assume that a volume of 150 million apples was in the trade and the expected infestation rate found by the IRA team was 3.9 per cent, would you agree that the relevant population for considering entry, and establishment and spread would be 150 million by 3.9 per cent, i.e., about 5 million 850 thousand apples, rather than the original 150 million?

Dr Sgrillo
210. You are multiplying the 3.9 per cent by the population, so I agree with the numbers that you came up with.

Australia
211. Thank you.

Australia
212. This is just one final question. And this returns to some of the questions posed by the Panel in relation to hypothetical pathways. We were just interested in Dr Schrader's response to this. Dr Schrader, you might recall in your response to question 45 from the Panel in your written questions, you stated "[t]he absence of historical proof of a pathway would not be a scientifically sound reason for excluding a certain risk". I think you have already given us quite a few comments that are related to this point, but I was just wondering – do you remain of this view and could you elaborate on this?

Dr Schrader
213. I remain of this view because even if some event didn't happen in the past it may happen in the future due to change of incidences or due to changes of new trade, or change in trade. So I would not say in any case that you should not rely on the fact that when in historical times there was no risk that it will be for the future in the same way.

Chairman
214. May I just ask one question for clarity. It relates also to your seeming acceptance of... If you had a uniform distribution, Dr Sgrillo, and you did sampling on that, and I think you said that twice today, the result would end up by the events being on the higher side. Why is that?

Dr Sgrillo
215. If you consider a segment of a line with the size of this probability range. So you divide it in tenths. So the segment is from one times ten to the minus seven to one times ten to the minus 6, the next from one times ten to the minus 8 to one times ten to the minus seven, and going closer to zero. Each part in this line, each part, when you approach zero, will be ten times less than the previous part. So that part has ten times less probability to be sampled, than the greater part in the upper.

Australia
216. We didn't pursue that at that moment, but there are some issues that surround sampling from a uniform distribution which, bearing in mind that a Monte Carlo method was used here, so that I think my colleague Dr Barry might be able to usefully provide a little bit more information that may assist Dr Sgrillo and the Panel, because we are talking about the application here of the Monte Carlo method in the sampling.

217. Just to clarify, Dr Sgrillo, the uniform distribution makes all points within the interval equally likely. I am just trying to understand the basis of your argument. The sampling, within the uniform distribution, every point is equally likely. So as you sample from that, if you build a histogram of that, the histogram would be equally likely across the interval.

Dr Sgrillo
218. If you divide this into intervals of tens, of one magnitude, and approaching zero, the greatest probability to sample a number is close to the higher part of the interval. Not in the lower part of the interval. I don't know. Probably you have the... let's see if I can find. I present a table, a small table, with 90 per cent and then nine per cent, and then point nine per cent.

Chairman
219. Just for clarity that is in Question 136. There is a table there.

Dr Sgrillo
220. This is expected, because you are dividing all your range by one magnitude. So, each range close to zero will be much lower. So the probability to sample that range. This is why I said that one thing that you could do is to linearize this interval. Take the log of this interval and then sample the log. Then you translate the log to a decimal number again. So you give more chance... or equal chance for a number to be sampled close to zero as in the upper part of the distribution.

Australia
221. I suppose as a technical clarification based on your question and, I agree and understand that you are talking about the log scale and you want to sample uniformly across the orders of magnitude, but I suppose the question is uniform sampling of the interval, as was done in the IRA, that gives each value in the interval ... if we were using the negligible range $(0, 10^{-6})$, it gives equal value within that range. Your point, just to clarify, if we are interested in having equal values across each order of

magnitude as you go down, then you would look at doing it differently. But in terms of a uniform distribution over the interval, each value is equally likely.

Dr Sgrillo
222. You are right, but this is another try to solve the problem of the term "negligible", trying to generate a number more close to zero than close to the upper part of the "negligible" range.

Chairman
223. May I ask if the experts have anything to add on this point? We might want to explore this also. Okay. Thank you very much. Thank you for the contributions so far. Let us break now. We have finished that section and we will start with fire blight when we get back.

Chairman
224. We will start off with fire blight and I would like to request my colleague Ms Hillman to start off with the questioning in this case. Perhaps, before we start I think what we will try to do is run until 16:30 because the coffee shop closes at 17:00 and we will take a short recess, stretch legs and get coffee, etc. and then start after that. That would be my intention. We will see how it goes but that gives us three hours where we can maybe do two of the main subjects in that period of time.

Kirsten Hillman
225. So just to give you a bit of a taste of what is ahead. We are going to ask you some general questions on fire blight and then we are going to go through different importation steps where we have questions and then ask some other general questions a little later on. My first question to Drs Decker and Paulin is the following. Can you confirm whether in your view Australia has presented in the IRA scientific evidence, from a qualified and respected source, that indicates that mature symptomless apple fruit free from trash can disseminate fire blight. As an additional element to add to that question, this qualified and respected science does not have to reflect the majority view it can be a minority view, as long as it is qualified and respected.

Dr Paulin
226. I think the key word in your question is "can". The problem is that Australia has shown that you can imagine a system in which mature apple fruits can carry the bacteria from New Zealand to Australia. The point is that it is difficult to estimate the value of "can". Is it a rare possibility, a frequent event or something exceptional? This is far more difficult to tell. I think that anything which goes from New Zealand or from a contaminated country to another country "can" carry something including bacteria, including pathogenic bacteria, that is the minimum level. Then you may have specific transport of specific bacteria, e.g., with rootstock material which would be the maximum danger for the installation of the disease in a new country. So, the apple fruit, which is in-between these two limits, cannot be excluded. That that can be considered as a real risk, is the matter in question.

Dr Deckers
227. I would like to add here some aspect that indeed can be very important, which is the survival of the *Erwinia* bacteria in the calyx of the fruits. There is scientific evidence of the presence of *Erwinia* bacteria on this place in mature apple fruit. So, there is indeed scientific-based risk present for the introduction of the bacteria through mature apple fruits.

Kirsten Hillman
228. Before I offer the floor, I have two little follow-up questions on the same topic. Again to both Dr Decker and Dr Paulin, in your replies to question 18 you indicate that the probability that epiphytic

populations of *E amylovora* on mature symptomless apples could be transmitted to a susceptible host and initiate a fire blight infection and that this probability is rather low or that the possible mechanism is questionable. What I understood you to have just said in your previous answer, and I would ask you to confirm, is that you believe that Australia has presented evidence that such transmission is possible. That is the first question: could you confirm that that is how we should interpret your previous answer. The second question is: how does this probability compare to Australia's chosen appropriate level of protection?

Dr Deckers
229. Perhaps I can take part of the answer. Indeed, we can confirm that there is a possibility of epiphytic populations to be transferred with the transport of infected fruits. In this case, maybe something I miss in the whole discussion here, is that fruits coming from orchards where hail appeared could be a higher risk than fruits from non-hailed areas. Because hail in the epidemiology of fire blight increases the risk of this epiphytic populations on apple trees. This is something that maybe should be better specified in the higher risks assessment. Of course, I think this fact that epiphytic populations can be present is sufficiently documented in our risk system, and it is true it is necessary to do.

Dr Paulin
230. Maybe I could add at that moment that the term epiphytic is probably not the best term to be used in the case of *E amylovora*. This is a bacteria which has a poor fitness with plant surfaces. So if you have bacteria on a plant surface, on apple trees surface, this is something which is temporary and not permanent. We tend to qualify as epiphytes bacteria which are able to multiply on plant surfaces without producing symptoms. This, I think everyone would agree, is not the case with *E amylovora* on apple or pear. We may have on apple and fire blight the presence of a bacteria which is sourced from elsewhere, such as ooze or progressive infection in the same tree in the orchard. You may have, sometimes, a bacterial population. If this population is not able to infect the plant tissues, for some reason, then this population will tend to disappear and you will have residual populations like the one that was being evocated in the calyx of the fruits. *E amylovora* is not a true epiphytic bacteria.

Kirsten Hillman
231. If I might re-state. I did have a part of my question was whether this probability, namely the probability that these populations of mature symptomless apples could be transmitted, and how that probability relates to Australia's chosen appropriate level of protection. If you cannot comment, that is fine, but I just wanted to remind you that that was also part of my question. If you have some comments on that I would, of course, welcome them.

Dr Paulin
232. In this case, Australia has to take care of this population. The population of bacteria which may represent some danger for the introducing country is residual bacterial population on healthy fruits, not a permanent multiplying population of bacteria. That means that the risk will tend to decrease with time, with any other elements like that, e.g. temperature. It is quite different from a bacteria which is multiplying in plant tissues so I would say it is an easier case to consider, as far as decreasing the risk is concerned.

Kirsten Hillman
233. One follow-up question in this regard and I will pass the floor over. Again to Dr Deckers and Dr Paulin, do you have any comments and if you do could you offer them on Australia's requirement then to (i) disinfect apples; and (ii) disinfect packing house equipment, based on the answers you have just provided, if you have some elaboration on those measures.

Dr Paulin
234. In my opinion, disinfection of apples is an additional safety. If you have these residual populations I was speaking of at the moment, then it will disappear with the disinfection process. This applies to fruits. I would say that for the equipment of the packing house, I think disinfection is useless because then you are really speaking of disappearing populations and you have no examples of the presence of bacteria on equipment in the fruit stations. So for fruit, I would agree this is a safety measure that would seem to be reasonable.

Kirsten Hillman
235. Dr Paulin, if I could ask in your response just now, you had said that you felt that this was a useful additional protection measure, but additional to what, if I may ask.

Dr Paulin
236. In my mind, additional to the first safety measure, which would be to be sure that the fruits are originating from orchards without active fire blight symptoms.

Dr Deckers
237. I would like to add also the comment in the necessity of the disinfection of the equipment. First of all, when the fruit has been disinfected the chance that the equipment afterwards gets infected by the *Erwinia* bacteria is very low and even when the bacteria would be there, there is a very low chance that it would survive the struggle between the other micro organisms and bacteria in this environment. I agree with Jean-Pierre that the risk there is really very low.

Kirsten Hillman
238. That is my grouping of questions on this particular issue. So, if I could ask New Zealand if they have anything to add?

New Zealand
239. Dr Paulin, could I refer to the response that you gave to Question 36, please, where you said in relation to the conclusion in the IRA and in relation to the probability of exposure of a susceptible host to *Erwinia amylovora*: "Only some fragments… are supported by scientific evidence. Very often suppositions or speculations are proposed rather than certitudes, just because these problems have never been addressed scientifically (or at least experimentally). As a consequence, I do not see how it is possible to rely objectively on any figure for the likelihood of this 'exposure' step". And, could you please confirm that that is still correct.

Dr Paulin
240. I think it is interesting to remember the discussion that we have heard this morning, because I was actually referring to exactly the same problem. We have no data for most of these steps. No experimental factual data. So, we have to rely on, what I was calling, supposition of speculation but that can be more elaborated, obviously. Anyhow, we lack biological information. For me, because I am not a risk assessment man, I think I have found it difficult to place a figure on these judgements which are just human judgements, not based on biological data. I am a biologist.

New Zealand
241. I wonder if Dr Deckers could please comment on the same point?

Dr Deckers
242. I think there is indeed a very difficult situation because you have no proof available that this transfer can occur. So, it is indeed speculation with, for me, a low level of likelihood to be a reality.

New Zealand
243. Dr Paulin, if I could please draw your attention to your response to Question 19, where you said, in your written responses, "The highest probability I can think of is a pollinating insect taking the few bacterial cells to the hypantium of a flower of a host plant. This remains unlikely because trace bacterial populations (not multiplying) would be hardly grasped by insects (it would be easier in the case of a multiplying population, where cells are embedded in exudate). Finally, the likelihood of successful multiplication on the hypantium and infection would be extremely low. In addition, it would be necessary that such open flower be available when these surface polluted fruits are present. All this cannot be considered to constitute an evidence". I just wondered if you could comment if whether that is still your view.

Dr Paulin
244. Yes, I could. The basis of this statement is that the bacteria in the normal cycle of the disease has a state of multiplication in the plant which produces ooze, which means bacteria are embedded in polysaccharides which are sticky and made of sugar. All these attract insects and explain the role of insects in the transport of the bacteria from bacterial source, which is ooze on the diseased plant, to flowers. In the case of a supposed population which is just decreasing, not multiplying, not producing any external extra cellular polysaccharides, I do not see how an insect can be contaminated with some level of certitude with bacterial cells which are not multiplying, not active and how they can carry this bacteria to a flower. You just miss the stick, glue, between the bacteria and the insect in the case of a bacteria population which is not multiplying. That was the basis of my reservation on this step.

New Zealand
245. Thank you. Could I just sum up your responses to the last questions and ask you whether you agree with the proposition that there is no scientific evidence to support this transfer stage.

Dr Paulin
246. I have seen no evidence of that. Yes.

New Zealand
247. In fairness, I could ask Dr Deckers if he could comment too, in particular on the last point.

Dr Deckers
248. I think it is important here to consider that the transfer of a normal infection in the cycles of *Erwinia amylovora* is the ooze produced by the overwintering canker where the insects come and take some of the bacteria in their mouth or on their legs. It is totally different, the situation, when we would have an infected fruit with the calyx infection at the top of the fruit. It will not be infected as the ooze from the overwintering canker, so the chance of transmission is much lower in reality than it is in the case of transfer from canker to blossom.

Kirsten Hillman
249. I believe New Zealand has no more questions. Australia, do you have any questions?

Australia
250. I just have one quick question to ask before I hand over to my colleague Dr Roberts, who will go into this in a bit more detail. When you talk about direct evidence are you talking about experimental evidence?

Dr Paulin
251. Not only. Because in some cases you have just case history or things like that. But it is true that an experimental evidence is far better if you can have it, but it is not compulsory.

Kirsten Hillman
252. Can I make a quick comment before I give the floor back to Australia, just to remind you that we are asking questions around the grouping of questions that I had asked. Obviously, you will be given the chance at the very end of this section to ask other questions.

Australia
253. In a lot of these questions addressed to Drs Paulin and Deckers, there are a lot of responses on the individual steps in transmission and we seem to have entered this transmission area, given the questions asked by New Zealand. In light of your responses on the individual steps in transmission, I would like to clarify some of your responses to Questions 35, 37 and 43. Dr Paulin, you stated in your response to Question 37 from the panel that you did not see any objective analysis in the IRA that the pathway could be completed either under orchard or experimental conditions. However, you also went on to say in your same response that you did not think it is possible to experiment in this matter and that this does not mean that mature apple fruit may not be a source of fire blight infection. Just turning to Dr Deckers's reply to Question 37, you say something very similar in your response to Question 37 that the step of transfer would be difficult to prove. So, I assume you mean experimentally there. I would like you both to elaborate on your responses. In particular, is it correct to say there is something similar to your response in Question 35 where you state that "The fact that *E amylovora* can be transmitted to a susceptible host via insects feeding on discarded apples is conceivable through an apparently logical succession of events, each of them being questionable but never completely impossible".

Dr Paulin
254. It's again a matter of frequency. I think you can imagine, because it is logical. If bacteria were in the soil then you cannot imagine a way from the soil to a blossom. When it is on the plant surface you can imagine something. It doesn't mean that it happened, but you have to consider that it can happen. I think for most of these questions that is the basis of the answer. You can conceive that it may exist sometimes and because you cannot experiment you have to accept this possibility, even if it is very low, but you have to accept it.

Dr Deckers
255. Maybe I can add here that the most likely transfer for *Erwinia amylovora* is pollinating insects, flying from flower to flower. Of course it is not excluded that honey bees feeding on rotten fruit on the ground that is infected get infected by *Erwinia* bacteria when they are feeding in the neighbourhood of the calyx with the bacteria in it. It is a possibility which can never be excluded 100 per cent. That's what we are trying to say.

Australia
256. Sorry for our deliberation here. We have got another question which we weren't intending to ask now, but it really is in direct response to some matters that New Zealand raised in response to your questions. We are prepared to hold off if you prefer to proceed with yours, but we are just flagging we would like to come back to an issue of multiplication of bacteria, which New Zealand raised.

Kirsten Hillman
257. I know we have gotten slightly off track here and would appreciate the opportunity to get back on track. We will be getting back to that and we have questions on that regard ourselves. Taking us back to a more general question, Australia argues that while the experts expressed some doubts about the overall probability of importation of *E amylovora*, this conclusion should be weighed against the specific support for individual importation steps, that is support from the experts. In Australia's view, there is sufficient support for the detail of the IRA team's reasoning to suggest that "any purported exaggeration of the probability range is not a serious flaw". So we would like the

experts to comment on this statement by Australia and if it adequately reflects your view on the matter.

Dr Paulin
258. Again, it's difficult to estimate the exaggeration and the seriousness of the flaw. It is true if you have several exaggerations, you can have a serious flaw. Nobody can tell actually against what you are estimating exaggeration. We are just lacking data. I think there is no objective answer to this question, I am sorry to say.

Dr Deckers
259. As far as I have understood in this area, I don't feel that there was an exaggeration of the estimation there in the importation steps. I think there is a real risk present that should be estimated as good as possible. For me it was not an exaggerated situation here. I think you are right to take the estimation in this way.

Kirsten Hillman
260. I would like to go back and follow-up an answer that Dr Paulin provided a little while ago when I had asked him to comment on Australia's requirement to disinfect apples or packing house equipment and you said at that time that if you disinfect fruit then fire blight would disappear. Could you please explain to us what you mean by disappear.

Dr Paulin
261. I am not sure I used the term "disappear". I mean that if you disinfect the fruit, then you enhance in a very large proportion the quantity of bacteria that will disappear due to the disinfection. You have very good chemicals that are able to disinfect fruits. This means that if you begin the disinfection with a low bacteria population, you will end up with fruits which will be clean, without bacteria.

Kirsten Hillman
262. I would like to move on to a question on Importation Step No. 2. I am sorry… Did New Zealand have a comment?

New Zealand
263. I was wondering if Dr Sgrillo could be given an opportunity to comment on your question in relation to the purported exaggeration of the probability range, whether that is a serious flaw in the IRA?

Kirsten Hillman
264. Dr Sgrillo, would you like me to re-read the question?

Australia
265. Sorry, just to come in on that question, Australia regards purported exaggerations at any particular steps in the biological pathway to be questions of biology. So, if possible, we would ask that any methodological aspect of that question be put appropriately.

Kirsten Hillman
266. We take note of your comment and refer to the Chairman's opening remarks and the manner in which he dealt with your earlier observation on the same point. So, we will have Dr Sgrillo answer the question and then we will consider or not in due course.

Dr Sgrillo
267. What is the number of the question, please?

Kirsten Hillman
268. It is a question from the Panel, so not a question that has been already answered. The question relates to a comment made by Australia in characterising the views of the experts, specifically Dr Deckers and Dr Paulin. Australia argues that while the experts express some doubts about overall probability of the importation of the *E amylovora*, this conclusion should be weighted against the specific support for individual importation steps that they expressed. In Australia's view there is sufficient support with the detail of the IRA team's reasoning to suggest that "any purported exaggeration of the probability range is not a serious flaw". The question I had asked is whether the experts believe that this characterisation adequately reflects their views.

Dr Sgrillo
269. This is the final result of one part of the importation risk analysis, the expected percentage of apple fruits that would be infected when you import. A mathematical model is a hypothesis, is a general hypothesis. This number is a hypothesis. And this hypothesis is composed of partial hypotheses, each of these partial hypotheses is an importation step that has a number. If you validate each of the partial hypotheses, then the final hypothesis is validated. But this was not the case with this model. So, if you don't have the partial hypotheses validated, I mean the probability ranges validated, there is no way to evaluate what is the meaning of this final result of 3.9 per cent, because it is based in numbers that were not validated. So it is a result that could be completely wrong or could be completely right. The result is from many numbers that were not validated.

Kirsten Hillman
270. I will move on to my question on Importation Step No. 2. This is a question to Dr Paulin. In your response to the Question 6(c), you indicated that "[t]he reasoning established by the IRA seems… coherent and usually based on available evidence, although it may tend to exaggerate the risks of *E amylovora* associated with fruits". You may feel you have answered this already, but I am going to give you an opportunity once again, could you explain to us in this particular importation step what you mean by exaggerate?

Dr Paulin
271. I must say I don't remember exactly the figure. This step 2, which deals with epiphytically or surface-infested fruit, seems to me of quite low danger, because this population is low and easy to remove. So that is why I think this step could be considered as low risk.

Kirsten Hillman
272. Again with respect to importation step No. 2, in its comments on experts responses to Question 24 about the probability range and distribution patterns for importation step No. 2, Australia indicates that a number of issues may have been overlooked by the experts in arriving at the conclusion that the probability range ascribed to this step is not scientifically supported. Would any of the experts care to comment on this comment by Australia? And, would anyone wish to add or modify their response in light of the information that was provided by Australia in its comments? Would it be helpful if we reminded you of the issues in relation to these comments? In particular, the issues relate to the relevance of endophytic infection, epiphytic infestation of mature apples, the weighting of studies derived from orchard showing fire blight symptoms and the weighting of other scientific evidence.

Dr Paulin
273. I am sorry to say I do not see the point. Exactly to which question do you refer?

Kirsten Hillman
274. The question was Question 24 which I can read to you: "Are conclusions in Australia's IRA as to the probability range and distribution patterns for importation step 2 sufficiently supported by the available scientific evidence?" You provided us with some responses to which Australia came

back with fairly lengthy comments. The gist of those comments was that there were a number of issues that may have been overlooked by the experts at arriving at their conclusions, that the probability range ascribed in this step is not scientifically supported. They went on to provide further information on the issues I just mentioned: the relevance of endophytic infection, epiphytic infestation of mature apples, the weighting of studies derived from orchard showing fire blight symptoms and the weighting of other scientific evidence. You don't have to comment. We are offering you an opportunity based on these fairly lengthy comments back from Australia to the effect that you had perhaps overlooked some information, whether or not in the light of that further information that was provided by Australia you may wish to add something or if, in fact, it doesn't change your views and answers on this.

Dr Paulin
275. I think I do not change my opinion on this point. For me, epiphytic surface bacteria is important, endophytic bacteria is less important.

Dr Deckers
276. I would like to add here the situation that fire blight infection occurs in orchards and many of the trials that have been made in this area are follow-up on a infection artificially made in an orchard and then the surrounding of this infection around. But here in the case of the importation we start from orchards where fire blight would not be present, orchards where fire blight have been found and active fire blight has been found, are excluded. On the second part of the answer, even when you have a disinfection of the fruits coming from the orchards without active fire blight recent infections, I think the chance is really lower than is mentioned here. But, it is difficult to prove. This is my comment on this.

Kirsten Hillman
277. Those are my only questions on importation step 2. I would be happy to provide the floor to New Zealand and Australia, if you have questions related to the specific questions I have asked. If you have other questions on importation step 2, please ask them later.

Australia
278. It is really a factual clarification. This is in some of our comments to the experts replies. Imp step 2 is assessing unrestricted risk. I gathered from Dr Deckers reply to you just a moment ago that he was talking about restricted risk where risk management was in place, and that is some of the clarification we tried put in our comments. Just a factual clarification to us about unrestricted risk with no risk management measures present.

Kirsten Hillman
279. I have one question on importation step 3. Importation step 3 is the likelihood that clean fruit is contaminated by the *E amylovora* during picking and transport to the packing house. This is a question to Dr Sgrillo. In your response to Question 26 you state that Australia's evaluation related to importation step 3 is objective but that available evidence is not sufficient to support it. We are trying to understand how those two statements work together. Specifically, are you implying that in your view the evidence is insufficient or are you implying that the available evidence does not support the conclusion, or something else. Could you clarify?

Dr Sgrillo
280. It is Question 26? The values for the distribution used were taken from two papers. One says that "Only 3 of 72 uninoculated and non-disinfested fruit developed blight symptoms – all through injury in the puncture treatment". So, from these figures the import risk assessment team calculated 4 per cent of the fruit damaged would be contaminated by fire blight. Four per cent came from 3 fruits divided by 72 uninoculated fruits. I mean, the figure came from one single paper. The other paper is just an abstract of four paragraphs. There is no specification of how the samples were collected, what

methodology, no statistical analysis and the result shows that, at the time of the first check, the percentage of damaged apples was lowest for the careful picking treatment, about 8 per cent, compared with 37 per cent in the whole container in the packing house. The IRA used 4 per cent multiplied by 37 per cent to indicate the magnitude of the most probable value of the triangular distribution. There is evidence, but these numbers are based on papers with no specification of the methodology. If they ought to repeat the same experiment and make the sample again, there is no guarantee that the numbers that come will be equal to these ones. I mean there is no way to evaluate the reliability of these numbers. So, it is not contradictory. There is not enough data.

Kirsten Hillman
281. That is the only question that we have on importation step 3. Does anyone want to comment? New Zealand do you want to comment on the answer provided? Or, not comment, ask related questions?

Chairman
282. I am going to ask a few questions and wondering if we can be a little bit speedy. ould I get a sense from the two Parties on whether you have many questions? This is a qualitative thing – we have eleven more on this issue. We would like to finish our questions in 10 minutes, so we are in a difficult position here. Could I get a sense of how many questions you have around on this area of fire blight.

Australia
283. I think perhaps not a large number but we do have several which are quite focussed, so I don't think they will require a long response.

New Zealand
284. We will also have only a few questions.

Chairman
285. What I would like to do is ask my first question and then ask five questions in a row without you responding, because they will be going to different experts and then you can think about your response. The first one is on Importation Step 4 and it relates to Question 28, which deals with the IRA's conclusions as to the probability range and distribution patterns for Importation Step 4. The likelihood that *E amylovora* would survive routine processing procedures in the packing house. It seems that some of the experts for some of the responses assumed that fruit would be disinfected, stating that in this case they considered the probability range to be too high. May I ask what your response would be if the fruit were not disinfected, in particular, Dr Sgrillo, Dr Deckers and possibly Dr Paulin might wish to comment on this question.

Dr Paulin
286. In my opinion, the fruit will get out of the station as it entered with or without disinfection, maybe with a little less bacterial population but nothing significant.

Dr Sgrillo
287. The only comment that I have and I don't know if it was asked directly, is that the concept of cleaning the fruit that is used if you are thinking of the fruit leaving without any bacteria is one thing. Probably you will never get there. It is a logarithm exponential decay. You reduce the population to the half, to the half, to the half, but always you will have some population. So completely clean, you will never have. However, what I have tried to say is that there is a threshold of bacteria population below which it is almost the same thing as no bacteria, because it needs a greater number to initiate an infection and that necessarily there is no need to completely clean the fruit of bacteria. But, if you remove the population up to a level, that it will be the same thing as a clean fruit.

Dr Deckers
288. Just a very short comment, when we propose disinfection of the fruit, I think we should be aware that we never take away 100 per cent of the bacteria present. It is very difficult to disinfect for 100 per cent in the calyx area. I think we should be aware of this reality.

Chairman
289. I am going to do, more or less, what my colleague Mr Ehlers did and I am not going to ask specifically the Parties whether they would like to comment. I will give a quick look and if you don't seem to be interested, then we can move on to the next question. I see no specific need to intervene at this point, so I will move on to a set of questions which relate to proximity and exposure.

290. The first question relates to Question 27, Australia's reasoning regarding the ability of the number of *E amylovora* found on mature apple fruit to spread to a susceptible host and initiate an infection under natural conditions. The question would be, please explain whether you consider Australia's reasoning in the IRA to be objective and credible? Is it based on respected and qualified scientific sources? That is the first question. Let me ask a few and then give you the opportunity of responding to these.

291. The second question: in its comments on the experts responses to the Panel's questions on epidemiologically significant populations, Australia argues that the experts responses illustrate a state of scientific uncertainty, rather than evidence of significant scientific deficiency in the IRA. I would be interested in your comments on this comment from Australia.

292. And then, if I may ask a question to Dr Paulin: in Australia's view your response to Question 35 is consistent with the IRA team's reasoning, that "the most likely mechanism of transfer of bacteria from discarded apples to a receptive site in a susceptible host is by browsing insects". Could we request whether this statement adequately reflects your views on the matter? Maybe I am stretching it a bit by asking too many. I will stop there. On the first two questions, have the experts any comments? Or, Dr Paulin on the third question.

Dr Paulin
293. In my mind, I don't see anything else than browsing insects which can carry the bacteria from these two places, so I would support this.

Chairman
294. Any responses for the first questions?

Dr Deckers
295. Maybe here we should mention also the coincidence of the host plant in the susceptible stage. So that even when the transfer possibility is there I think another important point is that you have host plants in a susceptible stage that can be infected. I don't know when the seasons are exactly when the discarded fruit would be in the open air, which should be taken into account before a real infection can take place.

Chairman
296. The issue of scientific uncertainty versus evidence of significant scientific deficiency. Anybody want to comment on that? I am going to move on to another question. Dr Deckers: in your response to Question 35 you state that "The chance that the epiphytic bacteria will be transmitted to the susceptible organs of a host plant on the appropriate moment to realise an infection is rather small". How does this compare to the conclusion in the IRA which states that the exposure value for an individual apple should be in the range from 0 to 1 in a million?

Dr Deckers
297. This value between 0 and ten to the sixth is also very low, so I think this is true. But maybe we should add here something, when we see an evolution of fire blight in a country that has the fire blight for the first time, you see that often it is not on the fruit trees it is found in the first place, there are other more susceptible plants that show it earlier before you can find it on the fruit trees. Maybe this is also a fact that should be included in the reasoning of this first infection. It is not always on the fruit tree – mostly not.

Chairman
298. In its comments on the expert replies to Question 36 regarding overall probability of exposure, Australia indicates that the experts conclusion that the overall exposure value in the IRA was not supported by scientific evidence appears at odds with the support provided by the experts for individual steps. I know we have had a question quite similar. I wonder whether that has been exhausted. If you want to make a comment on that, you are welcome to do so. My colleagues have just reminded me that this is the exposure proximity element. I see no further comment on this.

William Ehlers
299. Question 11 that we had put to the experts said: "Do you consider the IRA to be objective and credible when qualifying the biological and economic consequences of fire blight as 'high'?" There were plenty of answers to that, but I would like to look at part of Dr Paulin's answer where you state that: if each of the three factors namely, climate, cultivar susceptibility, and cultivar receptivity "can be evaluated… the combination of the three to give a reasonable prediction [on the severity of the disease] is non-realistic". To facilitate our understanding of what you mean here, could you explain a little more, please.

Dr Paulin
300. Yes. You know that if you want a disease to develop on a plant you need the presence of bacteria on the plant, under suitable general conditions. You can know in advance what is the susceptibility of the plant. You can know whether it is receptive to the disease or not. You can know if the bacteria are present or not. It is far more difficult to predict what will be the severity of the disease, in terms of damage which is expected. For example, in Europe we have been quite surprised to see that fire blight is a very serious disease of apple in Germany, where it was expected to be a serious disease of pear in the South of France, if you just look at the climatic conditions and the susceptibility of the cultivars. That means that there are some elements which are just missing and you can predict for sure that the disease will be able to develop, but it is far more difficult to quantify this development in terms of economic loss.

William Ehlers
301. Let me move on to something else. Concerning the alternative measures. New Zealand has proposed two alternative measures in respect of fire blight: namely, restricting apple fruit imports to fruit that has been cold-stored; or limiting imports to apples that are retail-ready packaged fruit. Would, in your view, one of these alternative measures meet Australia's appropriate level of protection?

Dr Deckers
302. I think the alternative measure here mentioned, fruit storage in a cold environment, would not reduce directly the number of bacteria on the fruits. Bacteria normally can support very well the cold storage conditions. I think this is not really a measure to reduce the risk of infection by this supplementary measure. The retail-ready fruit, as we discussed this morning, is indeed something where you can say, okay when you prepare this in this way, you can be quite sure that the fruits are free of trash. In contrast to when you prepare the fruits in bins, where there is much more availability for contamination.

Dr Paulin
303. I fully support this answer.

William Ehlers
304. As there are no other requests, let's move on. Drs Deckers and Paulin, in its comments on the responses to Question 47 regarding the IRA's requirement that a packing house provide details of the layout of the premises, Australia has stated that: "The replies of the various experts to this question indicate that they may not have understood the reason behind Australia's imposition of this requirement… The measure is intended to facilitate the verification of packing-house procedures by AQIS officers and thus make such on-site verification more efficient and swift, and therefore less costly to New Zealand exporters". Would anybody like to make a comment on the basis of this clarification?

Dr Paulin
305. On this point I am not in my field of expertise and I don't know well enough how things are organized. So I cannot answer.

William Ehlers
306. Can you identify specific points where in your view Australia's IRA, through its design and methodology or with respect to the consideration of specific steps, may have underestimated the likelihood of importation, establishment and spread of fire blight or the associated potential biological and economic consequences, in relation to imports of apples from New Zealand. I stress underestimated, because a lot of answer seem to imply that there has been over estimation in some cases. We are trying to see if there are any other instances you have seen where this might have been underestimated.

Dr Deckers
307. Maybe one point that I don't find in the IRA system is that the importance of hail is not mentioned sufficiently. This is epidemiologically one of the very important factors that can allow an inoculum build up very shortly after this damage. Of course, healthy mature fruit can come from an orchard where hail occurred and this may be an important factor where you should be more aware than the system today.

William Ehlers
308. No one else is requesting the floor. Next question – Assuming that the semi-quantitative method could be considered a legitimate methodology, do you believe that the way the IRA has applied this risk assessment methodology for fire blight is objectively justifiable?

Dr Sgrillo
309. I think that this was commented on here before. I think that the quantitative part of the methodology has not enough numerical data to generate credible results.

Australia
310. Just in relation to Dr Sgrillo's comment, could I go back to the question that we asked this morning and you indicated that you thought the model itself is good, or okay, I don't want to put words into your mouth, but there were issues about inputs. This semi-quantitative model was okay and you expressed these concerns about data and we heard other comments about data as well from other experts. The situation we are talking about is, at this point, the issue of data and not the actual model itself, as it is being applied.

Dr Sgrillo
311. The model is okay. But the data generates the problems.

William Ehlers
312. I realize there might have been some redundancy in the question, but sometimes it is good to go over again just to make sure we have it good and clear. So, if nobody else would like the floor then I will hand back to the Chair.

Chairman
313. We are not running too badly. We are a little bit behind time, but I would like to offer New Zealand to pose questions on the whole gamut of fire blight.

New Zealand
314. If I could refer Dr Paulin to Question 22 of his written responses where Dr Paulin stated that: "If the probability of 1 means that all orchards are contaminated by *E Amylovora* each year, it is probably a mere exaggeration… The chance for apples to be sourced from orchards harbouring *E Amylovora* should be significantly less than one". Do you still agree with that?

Dr Paulin
315. Yes, sure. I think there is a problem of vocabulary. I think what I was meaning is that, due to the historical situation of fire blight in New Zealand, you cannot be sure in advance that any orchard is free of fire blight. The history of the disease in the country is very long. It's the same situation that we have in France now. That doesn't mean at all that each orchard presents symptoms of fire blight each year – these are things which are completely different. The supposed risk represented in each orchard in New Zealand can be evaluated each year in place after a survey. You cannot decide in advance this orchard is full of fire blight. It is not true.

New Zealand
316. If I could just clarify, when you answered the written response you were referring to the pathogen *Erwinia amylovora*, you are now talking about fire blight. Do you still stand by your written answer that the chance for apples to be sourced from orchards harbouring *Erwinia amylovora* should be significantly less than one?

Dr Paulin
317. Yes. Absolutely.

New Zealand
318. If I could address a question to Dr Sgrillo in relation to your answer to Question 22 in which in your written response you said the probability of one for importation step 1 "means that it is absolutely true that fire blight is present and will always be present in all of New Zealand orchards. The scientific evidence presented in IRA does not guarantee that this is true". Can I ask you to confirm whether that is still your view; and secondly, whether that conclusion also applies to the pathogen *Erwinia amylovora* and not just the presence of fire blight, the disease.

Dr Sgrillo

319. Actually, I was not thinking in the idea of "free from", as defined in international standards. When I wrote down that there is no support for the probability of one, this is implicit that in some times and in some place you might have orchards without *Erwinia amylovora*. That is the sense of it.

New Zealand
320. If I could move to Importation Step 2 and ask Dr Paulin a question in relation to your response to Question 24, the likelihood that mature apples are infested or infected with *Erwinia amylovora* and you said that Australia's evaluation "is not scientifically based, cannot be objective and… is just not credible as a whole". Is that still your view?

Dr Paulin
321. Yes. I think so.

New Zealand
322. Dr Deckers, in your response to Question 24, the same question, you wrote that the IRA's assessment "doesn't take into account the sporadic character of the fire blight disease". Is that still correct in your view?

Dr Deckers
323. I think it corresponds to what we said in the question before. Fire blight in an area where fire blight is present, you cannot find orchards, or very rarely, orchards that have no fire blight during the last years. This is typical of fire blight that in some years you will see the problem more on pear than on apple or on other host plants. This sporadic presence of the disease makes it very difficult to make a calculation and risk estimation in advance. I think it is linked with the disease. I think Australia should be really careful looking to how exactly they define the risk situation because the risk can be from most of the orchards, apple or pear or even from other host plants in the neighbourhood.

New Zealand
324. Also in response to that question, you said that the value of 3 x 10 to the negative 2 "seems to be a quite high rate of picked fruit being infected" with *Erwinia amylovora*. Does that sum up the comment that you just made?

Dr Deckers
325. Yes, because this value of 3 x 10 to the negative 2 is the situation where you have I think you can obtain this relatively high amount of bacteria in orchards where active fire blight history is present. Where there is no new infection found during one season, I think you will never get this level of bacteria.

New Zealand
326. Dr Paulin, I believe that you said in your responses that it would be possible to experiment and to obtain data to find out the likelihood of whether mature apples are infested. Is that correct?

Dr Paulin
327. Yes, if the figure provided by the IRA were true, I think they are amenable to experiment, because they are quite high. So, these figures can be checked experimentally, not so easily, but it can be done, I think.

New Zealand
328. In your view, does the IRA actually present such data?

Dr Paulin
329. The figure produced which is 3 x 10 to the negative 2 is a figure produced by the IRA, proposed by the IRA. It is this figure I have in mind when telling that such a high rate could mean that you can experiment, because it is not beyond reach of experimentation.

New Zealand
330. Are there experiments in the IRA that would, in your view, lead to that conclusion?

Dr Paulin
331. No, I don't think so.

New Zealand
332. If I could move now to Importation Step 3, the likelihood that clean fruit is contaminated by *Erwinia amylovora* during picking and transport to the packing house. Dr Paulin, your response to Question 26 that "the evaluation of risk for this step seems too high, for mature symptomless fruits". Is that your view?

Dr Paulin
333. Yes.

New Zealand
334. Dr Deckers, in your response to the same question, you wrote that "the overall chance of 1 per cent [in the IRA] seems to be rather high when the fire blight infections are only sporadically present in an orchard". Could you confirm whether that is your view?

Dr Deckers
335. That is indeed exactly what I think, because fire blight will not be uniformly distributed when present in an orchard. So the one per cent overall value I think will not be the reality.

New Zealand
336. Moving to Importation Step 4, the likelihood that *Erwinia amylovora* survives routine processing procedures in the packing house. Dr Paulin, in your answer to Question 28 you said that "it will never be possible to extend results obtained with artificial infestation of fruits to actual bacterial population naturally placed on certain sites on the fruits". Could you confirm whether that is still your view?

Dr Paulin
337. Yes, this is still my view because when you experiment with bacteria and fruits you technically place the bacteria cultivated in the lab on the surface of the fruit and this is very artificial as compared to bacteria which, for example, result from an earlier infection and which remain alive on the surface and which are embedded in polysaccharide on the surface of the fruits, e.g., in the calyx. The interaction you may have between the bacteria and the plant tissues are basically different. That means that experimentally when you dip fruit, for example, in bacterial suspension you can get tendencies and information about the decrease of the bacterial population due to the antiseptic solution. But you cannot have a real picture of what is happening in the field, under natural conditions.

New Zealand
338. A couple of questions in relation to Importation Step 5, the likelihood that clean fruit is contaminated by *Erwinia amylovora* during processing in the packing house. Dr Paulin, your written response to Question 47 was that: "Very few scientific data, if any, support the risks of contamination of fruits by *Erwinia amylovora* in the packing houses". Could you confirm whether that is still your view?

Dr Paulin
339. I have never found such data and I do not see that, in a normal storage station where poor quality (with symptoms) fruits are discarded before processing, I cannot see that you may have mutual contamination between fruit during processing.

New Zealand
340. Just a point in relation to your response to Question 30, it relates to the same importation step – contamination during processing in the packing house and you said "the probability suggested in the IRA seems to be strongly exaggerated". Is that your conclusion?

Dr Paulin
341. Yes it is.

New Zealand
342. Thank you Mr Chairman, that concludes our questions on this point.

Chairman
343. Thank you, New Zealand. You went through those with great speed. I was wondering whether there were any comments from either my colleagues here or from Australia on the points that you have raised. If you wish to proceed. If not, Australia you have the opportunity to ask your own questions.

Australia
344. Just following on from our New Zealand colleagues, what we would propose to do is to first address a couple of new steps. We will address Importation Steps 2, 3 and 5 and then Dr Roberts will address some of the issues on transmission. We will have a few additional questions in light of what our New Zealand colleagues have said before, but we will try to present as quickly as possible.

345. Dr Deckers and Dr Paulin, thank you for your responses. If I could just take you to Importation Step 2, I don't want to dwell too much on the actual probability ranges but what I would like to talk about is some of the biological issues underpinning the analysis at this point. Dr Paulin, if I could turn to you first, in your response to Question 6 you stated that the IRA team's reasoning regarding epiphytic infestation is "coherent" and "based on available evidence". Do you still hold that view?

Dr Paulin
346. Yes.

Australia
347. This question is for Dr Deckers and for Dr Paulin, in your responses to Question 8 and 22 you mentioned that infestation could arise, if there were infected hosts in the surrounding areas. Could you elaborate on this response, please?

Dr Deckers
348. I think it is important to consider other host plants in the neighbourhood of the orchards area. We have the experience in Europe, in Belgium and other countries around us, that it is sometimes very important to consider other host plants, like crataegus or sorbus that can be host plants that produce continuously during season, fresh bacterial inoculum. So, it is not only the orchard itself that should be taken into account when considering the contamination risk.

Dr Paulin
349. I support this view, but that means on the contrary that if you have no active symptoms in an orchard or in the surrounding of the orchard you cannot expect to have an epiphytic or surface population on fruits.

Australia
350. Dr Paulin, in your response to Question 22 you have stated that late infection in the symptomless orchard could occur if there was contamination of hosts or wind-driven rain, or insects or some other vector. Could you elaborate on this response, please.

Dr Paulin
351. Yes. This is more or less in relation with the hail storm infection that was referred to previously. Normally, in the normal disease cycle of the disease most infection takes place during the

blossom period or shoot growth period which take place both in spring or early summer. But you may have in certain conditions, e.g., strong hail or secondary blossoms, you may have late infections and these infections may be active and progressing late in the season and not too far from the cropping (pick up) date, which can be important in your case.

Australia
352. One final question on Importation Step 2. As you know that experiments on this issue of infestation have very variable detection limits on how much bacteria they can detect. In your view, what conclusions can we draw from experiments whose detection limits cannot detect bacteria below 10,000 cells? And, in particular, do you think that this is reliable evidence that *Erwinia* would be absent?

Dr Paulin
353. This problem of the detection limit is a serious problem. But I think that most of the epidemiology that we know at present relies upon techniques which were detecting 10 to the 3 bacteria per mil. and they proved to be good data, I would say. Now, we have techniques which allow us to go far below that threshold, but as far as I see they do not provide very useful information in terms of technical data or epidemiological data. They can tell you that you can have some remaining population of some cells, but as far as I know nobody is able to tell what is epidemiological significance of these populations. So, 10 to the 3 is a quite high number, but it seems to reflect what is of interest in the disease cycle.

Australia
354. I might just follow-up on the answer to that last question. Most of the epidemiological work that has been done, not surprisingly, has been done in the situation where the disease is widespread in an established fire blight area. So, the question is would you be concerned about experiments that do not detect levels of around about 10,000 cells per risk unit in relationship to the spread of the disease into new areas?

Dr Paulin
355. I think if the bacteria is able to multiply, it will very soon reach a quite high level, I mean more than 10 to the 10, or something like that. If it is active, it will very soon reach a high level which will be detectable. I am not sure I answered your question.

Australia
356. I might leave that for now and turn back to some of our questions on transmission. I have interrupted my colleague.

357. I have two very short questions on Importation Step 3 and 5, then I can pass over to Dr Roberts. In relation to Importation Step 3, Dr Deckers and Dr Paulin you gave me a response to Question 26 and you talked a lot there about contamination events such as rain and hail. Do you still consider these to be potential sources of contamination during harvest and transport, and endangering processing if, of course, there is trash present, or rain, or hail, or those sort of things.

Dr Deckers
358. Yes, I think this is an important aspect, even at the end of the season. Wind-driven populations of *Erwinia amylovora* is an important epidemiological point.

Australia
359. Just one final question in relation to Importation Step 5, which is somewhat related to this point: Dr Paulin if I could refer to your response to Question 30 and I can quote back the text. You stated that: "if decaying apples… or large amounts of infected trashes, were present, the dilution

effect [that is in apple dump tank], in a non-disinfected medium could lead to a significant amount of bacterial cells on fruit surfaces". Just to confirm, do you remain of this view?

Dr Paulin
360. Exactly.

Australia
361. And, Dr Deckers, do you have any comment on that?

Dr Deckers
362. Well, it is typical for *Erwinia amylovora* that it can go from low bacterial inoculum to a very high one in a very short time. So it is possible.

Australia
363. Dr Paulin, in response to Question 21 you stated that: "it has been proven that low bacterial populations (Hale et al, 1990) sometimes as VBNC (Ordax et al, 2008) may be present for some times in the calyx of fruits. If these fruits were discarded in the open and exposed to the elements, the decaying fruit could constitute a suitable medium for a multiplication of these low bacterial populations. They could multiply, or resuscitate from the VBNC status, and therefore constitute a potential inoculum for near-by host plants". Do you still remain of that view in response to Question 21?

Dr Paulin
364. Yes. Exactly.

Australia
365. Do you consider that it is feasible to assume that conditions for bacterial multiplication may occur in the orchard in rare cases?

Dr Paulin
366. Why not?

Chairman
367. Thank you, Dr Paulin

Australia
368. Turning to exhibit NZ-23, Taylor et al., this is the paper we distributed earlier this morning. Please, if you look at the paper, in particular table 1 is particularly relevant. Would you agree that the results of this experiment conducted in the orchard which appear in the first few columns of that table, labelled "MARC orchard cv. Royal Gala", would you agree that the results of the experiment conducted in this orchard show that inoculum concentrations of 10 colony forming units multiplied to 100 million colony forming units can cause symptoms in apple flowers and I will ask Dr Paulin and Dr Deckers to have a look at that and answer please.

Dr Paulin
369. Yes I have seen this table. The results of the first column for Royal Gala in MARC orchard are, I would say, surprising because if the number of bacteria indicated in the inoculum concentration were to be really ten to the one, you would expect frequency of success of the infection less than 100 per cent, because at so low an inoculum level you cannot expect to have 100 per cent of your droplets containing at least one cell. So my conclusion from this table is that probably, and this is quite frequent, the number of bacteria in the inoculums was higher than expected by the experimentator and so that means probably down to ten to the three it's OK. I would be more sceptical about ten to the two and ten to the one. This is just a technical consideration. And that is my opinion. So probably

one cell, maybe one cell, can actually infect one flower, but this is not demonstrated in this table. I would not be confident for this result just on this table. You know, that is my opinion.

Australia
370. Before Dr Decker's answers, can I just perhaps ask a follow-up question in the light of your reply. As far as we can understand from the paper, and I might add that this paper was put forward by New Zealand and they haven't made any commentary about the methodology, I mean this suggests though that very low numbers of bacteria should be of concern in terms of their potential to start fire blight disease under ideal conditions. Would you agree with that statement?

Dr Paulin
371. Not entirely. I would say, for me, that means that if you want to have a reasonable chance of success in your inoculation I would not go down to less than ten to the three cells per flower and this seems to be generally accepted in a number of other papers. When you are counting bacteria with these techniques that were used here, you have a quite large range of error and I think in this case they were too high. Honestly, if you have one cell you will have to inoculate maybe ten thousand flowers to get one infection. I think the probability of success of infection decreases very rapidly and maybe from ten to the three up then you tend to go to the 100 per cent success. It is not indicated in this paper, but it is my expectation from what I see from other results.

Australia
372. I am sorry, Dr Deckers, I will continue with this theme and we will get to you in a minute or I hope to get to you in a minute. What you are saying, you are suggesting that the reliable threshold for this type of experiment is around 10,000 cells, reliable in terms of producing symptoms in flowers?

Dr Paulin
373. One thousand, I would say, would be the threshold if you want to have at least, say, from ten per cent to eighty per cent success in your infection. It will depend on the variety, on the period, the age of the flower, and so and so. This is just a broad indication.

Australia
374. Yes. But doesn't that suggest that the experiments that have got detection limits of around 10,000 cells really don't necessarily tell you very much about what is really going on at epidemiologically significant numbers?

Dr Paulin
375. Well, it tells you what is probable, but doesn't tell you what can happen exceptionally.

Australia
376. Thank you. I might ask Dr Deckers if he would respond on this particular issue, please.

Dr Deckers
377. I would like to add our own experiences with this artificial inoculation test for apples, as it is mentioned here. For me, the ten to the third with a 100 per cent infection and even ten to the second, ten to the one, this is very exceptional. When you want to have infection successfully, surely not on apple, maybe on another more susceptible host plant, you can go lower in the inoculum but not on an apple. This is surprising me, this publication. My second point is that, okay, here they are talking about apple blossom, but for me when you have an introduction somewhere in a new area, it is not mostly on apple that the bacterium starts. It starts on more susceptible host plants.

Australia
378. Thank you for that comment. We have heard quite a lot on fire blight this afternoon particularly in the light of New Zealand's questions about some concerns you have got about

probability values that have been put on certain steps. But we have also heard I think both experts on fire blight make statements about our measures and the risks and the need to manage those risks. So, in the light of all of that consideration, do you think that the IRA reaches a rational logical inference that the probability of transmission by trade in apple fruit is not zero? In other words, on the basis of what evidence we have, is it rational to conclude that transmission by apple fruit could occur, albeit as a rare event? So that question is directed to both Dr Deckers and Dr Paulin.

Dr Deckers
379. Here I would say yes, there is indeed a risk for the importation of infected fruits. It is clear. But, on the other hand, I must say that in other situations where countries try to keep out fire blight, they are not talking in the first place about fruits, they are talking more about plant material and potential infections on plant material, root stock or variety materials. So that is maybe an important point.

Dr Paulin
380. In my view, the importation of bacteria with apple is probably possible. The further step from this imported bacterial population to a new plant in Australia is probably even less likely. And I think that the total process, the risk represented by the total process, is probably of the same order of magnitude as the transport of contaminated insects by natural way from New Zealand to Australia by air jet or things like that. So that is my personal view, that there is a possibility which level of risk is not far higher than the natural spreading possibility of the bacteria to go from place to another with something else, I would say, which has no connection with trade of apples.

Australia
381. Okay, I have got one final question, both to Dr Paulin and to Dr Deckers. You have noted in your responses to Question 43 that the experiments of Hale et al. (1996) and Taylor et al. (2003a), which are relevant to this issue, do not have an experimental design that would detect rare events. In your view, is it scientifically sound to conclude on the basis of this evidence that rare events can never occur, that in quantitative terms the probability of transmission should be represented as zero based on those type of experiments?

Dr Paulin
382. No, I don't think so, obviously.

Dr Deckers
383. No, I don't think so either.

Australia
384. We have finished our questions on fire blight.

Chairman
385. Would New Zealand like to respond to some of these questions?

New Zealand
386. I do have two or three follow-up questions. First is for Dr Paulin, and it relates to the issue of VBNC. Specifically, Dr Paulin, your response to question 42, in which you referred to the complete absence of data on the occurrence of VBNC for *Erwinia amylovora* in natural conditions. I wanted to ask whether that is still your position.

Dr Paulin
387. Yes, sure. VBNC are quite difficult to manipulate, as you know, and to demonstrate. You can demonstrate that in the lab in very special conditions. If *E amylovora* has been shown to be able to go to the VBNC status, that shows that these bacteria has the genes and the systems and so it is able

to do so. We don't know if the conditions which are conducive to such state exist in the orchard. It was the meaning of my answer.

New Zealand
388. And, just to follow on from that, is there any scientific evidence that you were aware of, that demonstrates that VBNC has significance in epidemiological terms?

Dr Paulin
389. Maybe it's not useful to focus on VBNC. Actually we are speaking of long-term conservation of bacteria in a steady state. So we know for long that in some cases, say in dried ooze, for example, the bacteria is able to survive for months, in certain conditions. So VBNC just adds something to this possibility, but it is not a completely new element in the epidemiology of *E amylovora*.

New Zealand
390. If I could just turn to the issue of the number of hosts that could be susceptible to *Erwinia amylovora*. In your written responses in Question 10 you said that the IRA "maximises the real risks in considering too high number of host plants". I just wanted to ask whether that is still your view.

Dr Paulin
391. Yes, sure. This is quite common in countries without fire blight. They consider that any apple or any pear varieties are equally susceptible to the disease and will be host of the disease, which is scientifically sound and reasonable, but when you are in a country with fire blight you know well that actually this is not true and only a quite weak, fortunately, weak percentage of the cultivars are actually common hosts of the disease and do show a real damage. So that is why the term of host has a different scientific and practical meaning: any cultivar of pear, for example, is a host for fire blight, but economically only few cultivars are to be considered as such, in such a case.

New Zealand
392. You made a comment earlier about *Erwinia amylovora* multiplying on fruit. Are you aware of any scientific papers that report that this has occurred?

Dr Paulin
393. Do you mean on decaying fruit, or rotting fruit, or something like that, or fruit on the tree?

New Zealand
394. Mature fruit or decaying fruit.

Dr Paulin
395. Decaying fruit, as far as the chemical composition of a decaying fruit, can be a suitable medium for the multiplication of *Erwinia amylovora*, although this remains to be demonstrated. But you may have some multiplication, probably. On the surface of a mature clean fruit without wounding I don't know of any data showing multiplication on such a surface and I would not expect *Erwinia amylovora* to multiply on such a surface.

New Zealand
396. One final question for Dr Paulin and Dr Deckers. It is in relation to the paper that Australia has handed around this afternoon, which is exhibit NZ-23, the paper by Taylor et al. In light of the methodology employed in that paper, is it a fair comment to say that, under the conditions of the experiment, it was designed to ensure that if an infection could occur it would occur?

Dr Paulin
397. Yes, exact, because special care was taken to protect the infected flowers with some chambers and this during at least 24 hours and this provided conditions which probably facilitate the infection in a very large proportion.

New Zealand
398. Dr Deckers, do you have anything to add?

Dr Deckers
399. I think indeed it is a very artificial situation that they created there. This is not the reality of the infections and the orchard condition. So maybe this could also give the explanation of the high percentage of infections that they had on this apple variety, even with low infection doses. I would like to add maybe the last comment on the VBNC status of the bacteria, what we were discussing about. And maybe it's more important than we realize, because surely when the VBNC status is temporary and it comes back to a viable status afterwards, then we know that copper is in use in the VBNC status. Now copper is used a lot in orchards, and as well on apple as on pear, and they could induce indeed this type of status of the bacteria and I think it's not so exceptional as we described it.

New Zealand

400. I am sorry that I didn't ask you to comment on that question earlier, but perhaps I might just ask the same follow-up question as I asked Dr Paulin. Are you aware of any scientific evidence on the occurrence of VBNC for *Erwinia amylovora* in natural conditions?

Dr Deckers
401. Yes, there are indications that there is VBNC status present under orchard conditions.

New Zealand
402. Could you please advise which papers you are referring to?

Dr Deckers
403. I just discussed this matter, during a European cost meeting in Valencia in Spain but it is not published yet. It was only two weeks ago and there we discussed this matter of the VBNC status of the bacteria with Dr López of Spain.

Chairman
404. Australia?

Australia
405. I've just got one question which goes to one of New Zealand's questions about hosts. It is really, we have heard a lot of discussion about experimental work done in orchards and I note that Dr Deckers in particular has mentioned the importance of other hosts. Now, New Zealand apples, if they are imported or when they are imported into Australia, would be largely consumed in our major cities, which are very low density with backyards with large numbers of fire blight hosts such as cotoneaster, crataegus, apples for that matter, and various pear varieties. So, perhaps I am just asking Dr Deckers to provide any additional thoughts he has on the risks posed by hosts in the urban environment for the establishment and spread of fire blight.

Dr Deckers
406. I can tell you there that we monitored the development of the fire blight introduction in Belgium by cotoneaster salicifolius floccosus plants. They were host plant that we introduced in areas where fire blight was not found, just to know when the bacteria really appeared they were seen first on this host plant. And then of course you can monitor the spread of the disease when present. This was with these host plants, indicating where the infection was and was not.

Australia
407. Can I just follow that up. I don't want to prolong us too much, but I think some of your questions before actually implied, and this answer to this particular question implied, that some of these other hosts can be much more susceptible to fire blight disease, at least under some conditions. Is that correct, or is that your view of the biology of the disease?

Dr Deckers
408. Yes, absolutely. It is correct.

Chairman
409. It seems as though we've to a large extent exhausted the questions. But let me ask the experts on this area, is there anything that you would like to add, that might not have been subject to a question and that you think might be relevant for the Panel? Thank you then to all the experts that participated in that section. I think we then turn to European canker and I would like to ask Ms Hillman to lead this section first, thank you.

Kirsten Hillman
410. Once again we are going to proceed in the same way with a few general questions, actually quite a few general questions and then some questions on the specific importation steps and other areas. So starting off with a very general question to those experts who have expressed views in this area. Can you confirm that in your view Australia has presented scientific evidence in the IRA that indicates that mature symptomless apple fruit can disseminate European canker and can you confirm whether this scientific evidence, if you feel that it is present, is from a qualified and respected source once again reminding you as we did earlier with fire blight that that respected source need not represent the majority view of science. So can you confirm if such evidence exists that it supports the analysis regarding the likelihood of the importation, establishment and spread of European canker through the importation of mature symptomless apple fruit. Dr Swinburne?

Dr Swinburne
411. I think there is in fact no evidence in the literature that infected apples which rot with this fungus are responsible subsequently for the dissemination of the disease to other hosts, to further hosts. Much of the evidence that has been presented concerns observations made in the 1920s by Dillon-Western concerning a variety known as Worcester, which is an extremely early variety, which does rot on the tree and produces mummified fruits. In the 40-odd years that I have worked on this, I never have had the privilege of seeing such a thing and I have attempted in the past to produce perithecia and ascospores from rotted apples for experimental purposes and failed. Fruit which rot with *Nectria* as I will call it, does produce conidia if it is incubated under moist humid conditions to a limited extent in the centre of the lesion. It is not a prolific source of spores for subsequent release and I fear that nobody has actually done the experiment of determining whether a rotted apple can release spores into a new orchard situation and bring about disease. It is amenable to experimentation, along the lines of an experiment which we made in East Malling not too long ago with discarded canker wood which we pulverized and placed under potted trees of the most susceptible variety that I know, namely Spartan, and we failed to induce a single canker as a result of pulverizing several, maybe 50 kilos, of cankers underneath them.

Kirsten Hillman
412. Dr Latorre.

Dr Latorre
413. Yes, I would like to also say that I have no information indicating that European canker can develop from rotten fruit. In my experience, rotten fruit is very rare, almost never occurs in our conditions and sometimes we do see it after several months or weeks of cold storage in a very low proportion and never sporulated on the surface of the fruit. If this fruit are not sporulating it is

impossible to admit that it can spread at least easily to the clean fruit. Well, I think the observation about perithecia on the fruit is something that has to be reviewed. We have never seen perithecia form on the fruit. I don't have any good paper that can really demonstrate that in the recent years they have seen perithecia in very susceptible varieties, but not in today's apple varieties. So the most important inoculum could be conidia, and conidia are only formed under very wet conditions in the orchard, mainly in the cankers and almost never, or never at least in my experience, on the fruit.

Kirsten Hillman
414. I think that Australia would like to make a comment on this question.

Australia
415. We just actually had two follow-up questions. One for Dr Latorre and one for Dr Swinburne. We should say at the outset that Australia has a series of questions on climate, but rather than engage in those issues now it might be easiest to deal with them together, because that involves some audiovisual presentations. The question is for Dr Latorre. In your written responses to the Panel's questions, particularly your comment on guideline (g) and Question 65 you stated that and I quote: there is a risk of the entrance of *N galligena* associated with asymptomatic (symptomless) fruit carrying latent infection, as such fruit cannot be differentiated from healthy fruits at harvest. Could you please confirm that this is still your view?

Dr Latorre
416. I agree. This is my view.

Australia
417. I will handle over to my colleague Mr Heinrich from Plant Biosecurity Australia, he has a question for Dr Swinburne.

418. Professor Swinburne, in response to Question 64 you stated that "there is no data that confirms or refutes that rotted fruit forms a pathway for the long-distance transport of infection with *Neonectria galligena*… Virtually all research publications emanate from countries in which the disease is essentially endemic…, so it is not surprising that the fact that the focus has been on more obvious routes, such as orchard to orchard, and hedge row to hedge row". Could you please elaborate on this response?

Dr Swinburne
419. I suppose in my first response I did elaborate on it in a way. The production of conidia from an apple rotted with *Nectria* discarded in an Australian orchard and thinking in terms of its ability to initiate a new series of infections in an area which had never had it. As I have said, there is no information concerning the possibility that that would happen. The point that I want to emphasize is that fruit has to produce conidia in order to become an infectious unit. It may be infected, but it is not necessarily infectious and even if it does become infectious, being presumably on the ground and not deliberately placed somewhere up in a tree, the dissemination of those conidia which depends on splash dispersal is actually going to be over a very limited area but I wouldn't want to overemphasize that particular possibility but there is no information which tells us that it cannot happen and as biologists one can never say it would never happen.

Kirsten Hillman
420. Thank you.

Australia
421. Just a very short follow-up question, if that's okay. So just to clarify, do you take it that you agree that the possibility of infected fruit acting as a pathway for the long distance spread of European canker cannot be ruled out?

Dr Swinburne
422. No, of course it cannot be ruled out, but it probably has a vanishingly small probability.

Kirsten Hillman
423. In fact, you have covered a question that we had. However, we had intended to also pose the question to Dr Latorre. So if I could just refer to the answer that was just provided by Dr Swinburne, that while this possibility that the movement of mature apple fruit could be a pathway for the long-distance spread of European canker cannot be ruled out, that you maybe, I won't put words in your mouth, whether you agree with the characterization that Dr Swinburne has put, it can't be ruled out, but it's very, based on your earlier answer to our question, it is a very limited possibility?

Dr Latorre
424. I agree with Dr Swinburne. The first step would be to have an infected fruit, and this is possible, without symptoms. You can take this fruit and carry it from New Zealand, perhaps to Australia. This is also possible, in my opinion. But another question is that this fruit is going to be rotten in Australian conditions and, secondly, another situation will be that that rotten fruit will produce spores, conidia, on the surface and, thirdly, that those conidia can be spread nearby, primarily by rain splashing, which is the main dissemination way. So, in general, we agree. The difference is, in my earlier response, is that it was only referring to the possibility that mature fruit can hold the pathogen. And those fruits can certainly be moved from one place to the other.

Kirsten Hillman
425. In our Question 63 we had asked the experts to comment on whether the alternative measure proposed by New Zealand, in respect of European canker, namely to require that New Zealand export to Australia only mature symptomless apples, would achieve Australia's appropriate level of phytosanitary protection. Could we ask both Dr Latorre and Dr Swinburne to clarify whether your reply to Question 63 somehow implies that limiting exports to mature symptomless apples would not achieve the appropriate level of protection. We struggled a little bit with your answers here, so if you could perhaps explain your position on this issue.

Dr Swinburne
426. The assumption behind this particular measure is that fruit which become infected during the growing season will all show symptoms of rotting, at or before they are mature harvestable fruit, because of the nature of the varieties. And the point that was made in some of the presentations was that the resistance mechanism, which is expressed by cooking apples which restricts the fungus for some period of time, wasn't present in modern dessert varieties. That is not the European experience. In Europe, and particularly in United Kingdom, we know that fruit of the same sort of varieties as we are discussing here, can and do become infected during the growing season are harvested at the appropriate moment but still symptomless and are then placed in store, and this is the important thing is what happens to them in store, because the conditions the gas and temperature conditions within those stores is critical to the development of subsequent rotting, but they can therefore develop rots some weeks perhaps months after they have been picked in a mature symptomless condition. So it is incorrect to assume that that particular measure would satisfy the requirement.

Dr Latorre
427. Well, I agree with Dr Swinburne has mentioned. Since it is impossible to differentiate symptomless fruit from those having the internal infection, I think this measure would be very difficult to satisfy the requirement, in my opinion.

Australia
428. We just have, if it is okay, two questions to follow up again for Dr Swinburne and one for Dr Latorre in relation to measures. Dr Latorre, in relation to Australia's requirement that apples be sourced from export orchards or blocks free of European canker, you made the following statement in

your response to the Panel's questions. And that was on guideline G and I quote "assuming that there is a risk (perhaps negligible, but different from zero) of entrance, establishment and spread of *N. galligena* in Australia on mature asymptomatic apples imported from New Zealand, this phytosanitary measure appears reasonable for mitigating the risk of entrance". Could you please confirm that this is still your view?

Dr Latorre
429. Yes, I agree with those comments.

Australia
430. I will now just hand over to my colleague from Biosecurity Australia again.

431. I would like to ask Dr Swinburne. In your written responses, you stated that Australia's insistence on receiving only fruit from inspected orchards certified as free from canker would eliminate virtually all risks of fruit being latently infected. Could you please elaborate on this statement.

Dr Swinburne
432. Well, for fruit to become infected it generally requires to be grown on a tree which is itself infected. I doubt if many fruit are infected on a tree to tree basis, so we are talking about fruit being infected from its mother tree, from its own particular tree. So if you pick fruit from trees which have no canker, the likelihood of them having latent infection is vanishingly small. And as I gather, 95 per cent of New Zealand's orchards are largely canker free, from the exhibits presented.

Kirsten Hillman
433. Okay, so our next question. Dr Deckers, Dr Latorre and Dr Swinburne all replied to question 68, which was regarding the survival of spores on mature apples without continued moisture. But the responses do differ from each other somewhat. So we would like to offer you an opportunity to comment on those differences, if you so desire.

Dr Swinburne
434. Shall I begin? In the discussion concerning fire blight *Erwinia*, we were pointing out that *Erwinia* is not an epiphytic organism and neither is Cylindrocarpon heteronema, which is the asexual stage of *Nectria*. The conidia are relatively short lived in any event, particularly in a dry climate, and they cannot survive on the unbroken surface of an apple. They do require an entry point in order to infect. Conidia merely contaminating the surface of fruit at harvest will not play a part in any future latent infection. Those infections will already have taken place, as is known in the European context, either through the calyx end, stem end or under very rare circumstances through open lenticels, because in very wet climates the lenticels on the fruit are actually open, enabling spores to enter.

Dr Latorre
435. Well, on my experience conidia cannot multiply on the surface of the fruit, and they can only survive maybe for a very short period of time, depending on weather conditions of the environment where they are. So in this terms I agree completely with what Dr Swinburne has expressed right now. There is no data showing that conidia can really survive for a long period of time outside the host, on the surface of the host, in this case on the fruit, nor that they can multiply on clean fruit.

Australia
436. We actually have a follow-up question to that. It actually involves a comment by Dr Swinburne, but we are happy for Dr Latorre to comment on it as well. To make asking you the question most efficiently, it might be useful if I just ask my colleague to distribute a document, which is an extract from our rebuttal submission, just two paragraphs, and we can give it to the Panel as well.

Kirsten Hillman
437. Perhaps while we are waiting for that document to be distributed, we can offer Dr Deckers an opportunity to comment if he has any comments he would like to make.

Dr Deckers
438. I would like to add here one comment on the infection of the apples. We see in some varieties in our production condition, that use of gibberelins is interfering with the open calyx end of the fruits, which sometimes allow the fungal infection of the *Nectria* in these fruits. So there is some interference sometimes from the use of some plant growth regulator PGR treatment.

Australia
439. So, apologies for that short delay. Just thought it might be more efficient if the experts had a hard copy of the relevant bits of their submission before them. And since it is a methodological question, I will hand over to my colleague Dr Barry from the CSIRO.

440. Professor Swinburne, in your responses you state that at several of the importation steps the IRA emphasizes the risk of superficial contamination, but omits reference to any opportunities for these to convert to infections. Paragraphs 497 and 498 of Australia's rebuttal submission addresses this apparent misunderstanding of the methodology and that's what is being distributed now. Are you able to offer any clarification in relation to your initial response, in light of this material?

Dr Swinburne
441. The majority of fruit infections in the European context are to be found either at the stem end or the calyx end, and generally speaking it means that the core has been infected. The time at which that infection occurs is currently being investigated. We have had a shot at it in the past, as well. But it is surprisingly early on in the fruits development. It isn't at the time of harvest, it isn't in the dump tank. That core rot is taking place at some stage when either the calyx or the stem end is enabling an entry point for conidia which are washing down over the surface of the tree and either accumulating it in the stem well or going around and entering the calyx. I don't have any information which would ever suggest that infestation of apples at harvest, and I stress that point, or even after harvest leads to any significant level of rotting in commercial conditions.

William Ehlers
442. The discussion just now sort of reminded me of a statement by Dr Paulin, concerning fire blight, when he said more or less that spread by fruit was no higher than by any other natural means. Does that apply in this case also?

Dr Swinburne
443. Could you elaborate just a little bit.

William Ehlers
444. What I understood from the statement by Dr Paulin was that, if you are trying to decide whether the pest has spread through one way or another, the fact that it was by fruit or by any other natural means was virtually indistinguishable. So I was asking if that kind of situation applies here, too, by what you were saying where in the mature fruit there was no infestation and it only comes from the mother tree to the fruit and not to another fruit from another tree and that sort of thing.

Dr Swinburne
445. Yes, the fruit will be infected from the mother tree by cankers which are on that tree and probably by no other route, so that the quiescent infection with which it carries into store is carried from the orchard.

Australia
446. Apologies. I actually neglected to ask Dr Latorre for his response to the previous question which Dr Swinburne had answered.

Kirsten Hillman
447. Dr Latorre, would you like to answer Australia's question?

Dr Latorre
448. The question that was raised just raised before?

Australia
449. That's right. The infestation leading to infection question.

Dr Latorre
450. Infestation of the fruit? Excuse me.

Australia
451. This relates to those paragraphs 497 and 498 of our rebuttal submission.

Dr Latorre
452. Well, infestation of the fruit means that the inoculum arrives from the fruit. But in order for the fruit to be infected, I mean really colonized by the pathogens, you have to have some other conditions at the same time. There are some weather requirements that are very important, but more important than that, you have to have a susceptible host and you have to have injuries. Otherwise, the infestation means nothing, that is the end of the inoculum. It will not survive for a very long time.

Kirsten Hillman
453. If I may follow up then back to the document that Australia has circulated to us. In paragraph 498, Australia has stated about four lines down, that at each of the steps (picking, transport to the packing house, processing and transport to Australia), at each of these steps, there is the potential for the surface infestation to lead to infection. Any such infection may remain latent for some time. And just to make sure I understand correctly did you say, Dr Latorre, that in your opinion in fact the infection doesn't last for very long?

Dr Latorre
454. What I have said is that the infestation doesn't last very long. Then you move to infection, which means that you already have a relationship between the pathogen and the host, it could last for very long if the pathogen is already inside the fruit.

Kirsten Hillman
455. Dr Swinburne?

Dr Swinburne
456. Can I follow up on that same paragraph? It says that "it is well known that infestation/infection of fruit in the dump tank…" I should add, that is not necessarily *Nectria* that we are talking about. It is well known for a number of other pathogens that cause rot, notably *Penicillium expansum*, the blue mould rot. When that gets into the dump tank, you do have trouble.

Dr Latorre
457. One brief comment I want to make in this sense is that *Nectria* moves from one place to the other, long distance movement, primarily by nurseries stock, in our opinion, and fruit rarely. I doubt if somebody has proof that really infected fruit can be the cause of the development or new focus of European canker in a new area.

Kirsten Hillman
458. Let me move on, if I may. One final question, before I am finished for now. This is in relation to the responses to Questions 69 and 70, regarding the relevance of perithecia on rotting mummified apples to the spread of European canker. Here Dr Deckers, Dr Latorre and Dr Swinburne have all replied to these questions, but the responses seem to differ from each other, at least in part. We would appreciate if you could, if you care to, comment on any of these differences between your responses.

Dr Latorre
459. The only comment that I can make is that, in my experience perithecia only occur under certain conditions and most often they are not important from the epidemiologic point of view. The development of the disease is associated to the production of the asexual spores, namely conidia, but not to the production of the sexual part of the fungi, which is the so-called production of perithecia and ascospores.

Kirsten Hillman
460. Dr Swinburne, do you care to comment?

Dr Swinburne
461. As I have said, perithecia; I have never seen them on rotted fruit. I would dearly love to have done so, because for experimental reasons to have been able to obtain single ascospore cultures at will would have been great fun. So I don't think the ascospores would ever form an important part if apples rotted with *Nectria* in a foreign country. I have already mentioned about conidia. Conidia can form on a rotted fruit. Quite often when a fruit rots, particularly if it happens to be on the cheek of the apple from the lenticel, the peel will crack and become almost paperlike and through that small crack you will get the formation of conidia. You have to remember the fungus is in, it has got to get out, and it's only when the only actual surface of the fruit breaks that the conidia will form and they form in relatively small numbers under such circumstances, that's on the fruit.

Dr Deckers
462. I would like to add just a comment from the climatological side of this type of possible infection. Maybe climatological conditions for this further development of the disease by conidiospores is rather unlikely in most areas of Australian conditions.

Chairman
463. I will ask a few questions in this area as well. In its rebuttal submission New Zealand argues that Chinese Taipei has a domestic apple industry, individual apple trees and many of the alternative hosts for European canker, that the climatic conditions in the central mountainous regions mean that Chinese Taipei is at relatively high risk for European canker establishment and spread, and that they are comparatively more favourable to the development of European canker than mainland Australia. Please indicate whether this affects your responses to Question 44 on the relevance of New Zealand's experience with apple exports to Chinese Taipei. I notice that Dr Deckers and Dr Paulin responded to Question 44.

Dr Paulin
464. Yes, my answer to Question 44 was referring to fire blight exclusively, so I was just underlining some important differences between apple growing conditions in Taipei and other regions which make the risk of introduction of fire blight far less dangerous in Taipei. I think that these reasons are mainly the scarcity of host plants, even if you have some indeed, and the remoteness of the orchards, which are all in the mountains, that means in the centre of the island and far from any commercial establishment which could process apples from abroad.

Dr Swinburne
465. I find it very difficult to answer questions like this, because you are being asked why did something not happen. There can be a huge range of reasons why something like this did not happen, starting with the volume, starting with the possibility that within that volume there were no infected apples, ending with the possibility that a few infected apples arrived in the wrong place and couldn't possibly present the infection. Otherwise, it is really rather an unanswerable question.

Chairman
466. We can take that to a couple of the other questions we are trying to deal with here, but thank you for that Dr Swinburne. Any further comments on that. If not I am going to move on to another question to Dr Latorre and Dr Sgrillo. In your reply to Question 84 you indicate that in your view the IRA's conclusion on the mean infection infestation rate of apples has not been validated for this pest. Please explain what validation means in this context?

Dr Sgrillo
467. Well, validate in this context would mean to sample fruits from New Zealand that were processed with the same processing that would be done with the fruits to export and to check out what infection rate they would have. If they find something that's around this percentage, it is okay, so you have to make a plan and to sample enough number of fruits with this statistics: means, confidence level and things like that. And this is because again, as in the model for *Erwinia*, the parameters of each hypothesis were not validated, so the general one, I mean the final number, could not be validated.

Dr Latorre
468. Validated means to estimate whether your model is really explaining the real situation, at least it is trying to estimate whether the model explained was in reality what happens. That's it.

Chairman
469. I am going to ask one last question and I think then what we are going to do after this last question is give you an early coffee break, for an objectively justifiable reason. My next question would be that Australia indicates that the relevant CABI data sheet explicitly states that one of the plant parts liable to carry the pest in trade transport is fruits. Please explain the relevance of such a datasheet for a risk assessment and can such a datasheet be considered scientific evidence, specifically the CABI datasheet. Anyone want to comment on that?

Dr Swinburne
470. I wish someone was here from CABI to answer.

Dr Latorre
471. Well, I just want to say that this is a general statement that does not necessarily apply to each pathogen or pest. In this case, in my opinion, it does not apply to *Nectria galligena*. It is really true for other pathogens, *Penicillium, Botrytis* and several others, but this is not the case for European canker.

Dr Swinburne
472. I did write the CAB documentation on *Nectria* that is in the latest publication. I didn't write the leaflet.

Chairman
473. I detect a distinction in your response. Any further comments on this particular question, perhaps. Australia.

Australia
474. If I could I might just get my colleague from Biosecurity Australia, just to clarify a point with respect to that CABI datasheet, which was specifically for *Neonectria galligena*.

475. The CABI datasheet that we are referring to does specifically say that apple is a means of… and I am just wondering whether either of you could clarify whether that is wrong.

Dr Swinburne
476. If I had a nasty tendency, I could bring an apple which is infected by *Nectria* from New Zealand into Australia and I would know how to inoculate it. So I suppose to that extent an apple can carry an infection, but whether it is a natural pathway is an entirely different story. There is no evidence anywhere in the literature, except in the stray remark apparently in the CABI datasheet, that fruit is a pathway.

Chairman
477. I suggest we break now and according to my watch it is twenty past four. Let's break for 20 minutes, if that's alright and we will see you back here in 20 minutes.

Chairman
478. I think we can continue, if you are ready. I am going to ask Mr. Ehlers to take four last questions from us on this matter.

William Ehlers
479. This might have been answered in part, but I think it bears asking anyway. Australia argues that surface infestation is the necessary first step leading to fruit infection with *Nectria* and that thus Importation Steps 3, 5 and 7 deal with infestation and infection. Would you care to comment and indicate if your relevant response would be any different in light of this argument?

Dr Swinburne
480. I do beg your pardon. I am afraid other matters were happening when you were asking the question. Could you repeat that?

William Ehlers
481. Australia argues that surface infestation is the necessary first step leading to fruit infection with *Nectria* and that thus Importation Steps 3, 5 and 7 deal with infestation and infection. Do you have any comment? Would this argument impact your answers in any way?

Dr Swinburne

482. I think this point was perhaps covered in earlier questions. Obviously, for fruit to become infected the inoculum – the conidia – has to wash down over the surface, which might be described as infestation. But it does not, I believe, apply post-harvest, I am talking about pre-harvest events.

Dr Latorre
483. I understand. This comment doesn't change my opinion. I think infestation is the early step, but the most important step is penetration, colonisation and so on, which means infection, whether this occurs in the Step 3, 5 and so on, is another question.

Mr William Ehlers
484. I don't see anyone else asking the floor on this. New Zealand.

New Zealand
485. Dr Latorre, if I might draw your attention to a quote that you made, in response to Panel Question 77, where you said that "the likelihood that these spores might contaminate fruits superficially is extremely rare, and the probability that spores contaminating the surfaces of mature fruits will cause infection is negligible. In conclusion, fruit contamination with spores of *N. galligena* during picking and transport to the packing house should be disregarded. There is no scientific evidence on this subject to strongly support this hypothesis". My question is, would this still be your view?

Dr Latorre
486. Yes, certainly.

Dr Swinburne
487. Inevitably, fruit harvested from orchards with no or very low levels of infection will be most unlikely be infected themselves and therefore it would be an appropriate measure to be taken.

William Ehlers
488. Let us move on to the next one. This is perhaps a repetition of one that I asked earlier on, but I am asking it in the three pests, just to make sure we get it right or at least get answers for all three. Are there any points where in your view, Australia's IRA, through its design and methodology, or with respect to consideration on specific steps, may have underestimated the likelihood of importation, establishment and spread of European canker through the importation of apples.

Dr Latorre
489. I did not see any situation where they underestimated the situation.

Dr Swinburne
490. This is still my view.

William Ehlers
491. Anything else? Okay. Let us move to the issue of alternative measures. New Zealand has proposed two alternative measures in respect of European canker, namely: restricting imports to apples sourced from pest-free places of production, to be determined by a single inspection of each exporting orchard and maintained through controls on the subsequent movement of nursery stock; limiting imports to apples sourced from areas of low-pest prevalence, to be determined by inspection of sample of orchards. Please comment on whether these alternative measures would achieve Australia's appropriate level of protection?

Dr Latorre
492. In my opinion these are appropriate measures that can mitigate the possibility that infected fruit can carry the pathogen from New Zealand to Australia.

493. I agree. I didn't find anywhere where there was underestimation.

William Ehlers
494. If I don't see any other parties wishing to take the floor then I will hand back to the Chair.

Chairman
495. We had a question, which we had dropped thinking that it had been answered, but it might be a question simply of typographical error. I will read it in full. It is to Dr Latorre. In your response to Question 49, in the last paragraph it says: "To my understanding, the challenged requirements imposed by Australia were based mainly on the possible occurrence of latent fruit infection, but not on endophytic infections. The possibility that clean fruits may be infected from inocula

contaminating epiphytically mature fruits in dump water in packing houses… is negligible and irrelevant". So the question is really to that sentence where you talk about latent fruit infection, but not endophytic infection. Is there a difference? What are the differences between those two terms?

Dr Latorre
496. In my opinion, there would a difference in the way of how the fruit gets the pathogen inside.

Chairman
497. Was the quote correct? In other words, the comparison was between latent fruit infection, but not on endophytic. That was a correct quote and a correct... Yes, okay.

Dr Swinburne
498. Endophytes are usually regarded as being carried symptomlessly and that there is no interaction between the organism and the host in which it is endophytically spreading. In quiescent infection, there is damage done to a small number of cells invisible to the naked eye, but in which the tiny lesion, in which the fungus is actually arrested, it is stopped from growing. That is what happens in a quiescent infection.

Chairman
499. I think we have exhausted our questions for the moment on European canker and would like to offer New Zealand the opportunity of asking any questions they might have.

New Zealand
500. No questions for the moment.

Chairman
501. Australia.

Australia
502. We do have a number of questions, some questions relating to the probability of entry, establishment and spread and then, as I said, a series of questions on climate. So we can just kick those off now, if it is convenient. A short question for Dr Swinburne and Dr Latorre, could you please confirm in light of your written responses that you accept that *N galligena* occasionally causes latent fruit infection in New Zealand.

Dr Swinborne
503. Inevitably, I have absolutely no first-hand knowledge that would lead me to give you a proper answer to that question. There would appear to have been a number of reports, and it is reported in hearsay, and I would be surprised if it never happened, in those localities in which canker is said to be prevalent. But, having said that, I don't even know how prevalent it is in the areas in which it is present.

Dr Latorre
504. Canker on Gala could be possible. However, weather conditions are absolutely needed in order to have infected fruits. If that doesn't happen, the possibilities are zero, or very, very low, negligible. Maybe it never happens.

Australia
505. I will just hand over to my colleague Mr Heinrich, from Biosecurity Australia.

506. Dr Latorre, in your written response to Question 76, you made the following statement: "The reasoning in Australia's IRA with respect to the possibility that latent infections of *N galligena* may occur in mature New Zealand apple fruit and not become apparent until after storage is based on

published information… [I]t is possible to assume that latent infection may occur if fruit rot caused by *N galligena* were to occur. There is always a concern that fruit rot can further develop in cold stores. It is true that IRA relies on scientific research about latent fruit infection in the UK and Nothern Europe, but differences between New Zealand and Northern Europea can only be expected in relation to the likelihood of this event". Can you please elaborate on this response.

Dr Latorre
507. I was only trying to say there is a possibility there. Fruit can be infected if conditions are adequate. However, most of the information that I remember from the statement made by the IRA related this with research being done in Northern Europe, where weather conditions are completely different and by the time this experiments were done other varieties were prevalent. So there is also a chance that varieties susceptibility may play a role here. That was the idea, the main idea in this paragraph.

Australia
508. We have a follow-up question for you, if we may. In your written response to Question 65 you stated that: "Considering that fruit rots occur in New Zealand… and despite the fact that this is a very rare event, latent infection in mature fruits cannot be ruled out. It is possible that asymptomatic but infected matured apples could develop symptoms and eventually sporulate during transit and commercialization in Australia". Could you please confirm that this is still your view?

Dr Latorre
509. The possibility cannot be ruled out. The question is how frequently this is going to happen. In those terms, I still have the same opinion.

Australia
510. We just have one further question for Dr Latorre. In your written response to Question 69 you stated that: "Based on published scientific information, Australia assumes that fungal growth and fruit rot resume when fruit is removed from cold storage, sold to consumers and stored at room temperature. Therefore, rotted fruits discarded near susceptible hosts could be potentially (but not necessarily) a source of inoculum (mainly conidia) for infections in new areas. This conclusion is acceptable..." Could you please elaborate on that?

Dr Latorre
511. It is impossible to negate the possibility that an infected fruit from New Zealand to Australia carrying the pathogen cannot develop rot symptoms. Eventually, it can probably develop spores on the surface, if weather conditions are adequate. o, this possibility cannot be ruled out. However, I insist, another situation is how frequently or which is the probability that that biological event could happen under the conditions in Australia.

Australia
512. That concludes our questions on the probability of entry, establishment and spread. We would like to now, as we said, ask them questions in relation to climate.

Chairman
513. I note that New Zealand would like to make an intervention.

New Zealand
514. Just before we move on to this new area relating to European canker, we have a question we would like to ask relating to what we have been discussing. Dr Latorre, I just wanted to go back over some of the quotes that you have been commenting on. I can't recall the order, but if we start with Question 65 where you confirm that it is possible that asymptomatic but infected mature apples could develop symptoms and eventually sporulate during transit and commercialization in Australia.

"However", and this is to continue the quote, "I would consider the probability of this event as extremely low to negligible". I was just wondering if that is still your view.

Dr Latorre
515. Yes, I haven't changed opinion.

Chairman
516. Australia, would you like to continue on the climate?

Australia
517. One of the common threads running through New Zealand's case on European canker is the alleged unsuitability of Australia's climatic conditions for development of this fungal pathogen. Australia now has a series of questions on this issue, which it would like to explore with you. These questions draw on the climate analysis that was undertaken by the Bureau of Rural Sciences, which are annexed to both our first written submission and our rebuttal submission; Annex 2 in both submissions. As this is quite a technical area and involves some climate charts, we would like to use the audio visual equipment, and my colleague, Dr Barry, when he is not writing chapters on Monte Carlo methods, he leads a team as was mentioned at the CSIRO researching new approaches to environmental monitoring and modelling and over the last decade he has published a variety of papers involving climate analysis in biological invasion literature. As a result, I thought it might be useful if we get him to run through some of this climate analysis.

Chairman
518. May I just enquire? You are talking about material which has already been made available. Your intent is to provide it in a visual format for us leading to a question or to questions.

Australia
519. Absolutely right. There is no new material here, it is all contained in our written submission. It is just a matter of talking through the charts, so it is easier for people to follow the discussion.

Chairman
520. It sounds alright from the Panel's point of view.? Technically, how do we go about this now?

Australia
521. The first couple of questions don't require audio visual, so if you like we can run with those. We have brought paper copies if our PowerPoint dies on us. Dr Swinburne and Dr Latorre, could you please advise the Panel whether there is any one universally accepted epidemiological model for predicting the world locations where European canker would establish?

Dr Swinburne
522. The complete model that would enable a climate to be assessed has yet to be developed. It is inevitably rather more complicated than, for example, a climate model for wheat rust or some such airborne pathogen. Around the world, in Chile and East Malling and elsewhere some of the parameters of that model have been evaluated, but it has not yet been put together in a joined-up form that would enable a new climate to be fully assessed. But one can make certain judgements, perhaps, based on the various components that we already know.

Dr Latorre
523. I have to say there are only two situations, one in Chile and another in the UK, where the biology of this pathogen has been expressed in a model. With the primary idea of estimating the infection conditions, or the conditions for the fungus to infect the host, in this case.

Australia
524. As a follow-up question to both, you are saying that the parameters can potentially change from location to location in terms of what can predict canker development?

Dr Latorre
525. In the case of the model proposed in Chile, it is based on the conditions needed by the conidia to infect the twigs or the stems of the apples, as primarily based on weather conditions, humidity and temperature conditions.

Dr Swinburne
526. There are two main differences in the locations in which European canker occurs. The differences are based mainly on water and, in particular, on leaf wetness and the frequency of leaf wetness. I use the word "leaf wetness" when I don't really mean leaves, but we tend to talk about the surface of a plant remaining wet for periods of time and we use the term "leaf-wetness periods" just to cover that. In a location such as Northern Ireland, which represents an extreme environment in which apples are produced, there are no dry periods at all. Rain falls fairly evenly throughout the year and in some locations on every day of the year. Models used to predict, for example, the other disease of apples, namely apple scab, fail to work properly in Northern Ireland because normally in Kent and in Europe, for example, an event of 8 hours leaf wetness duration of a certain temperature would trigger an alarm which would send out the fungicide sprayer, but in Northern Ireland such a Mills period might last for seven weeks. As a consequence of that, in Northern Ireland inoculum of Nectria for infection is available constantly. In other locations, and I am thinking here of California in particular where we have had some data, and to a more limited extent to Kent, perhaps more extremely to New Zealand, you have very pronounced dry periods and very few, relative to Northern Ireland, days of leaf wetness. It is that difference which makes a difference in the pulse of the disease. The availability of the inoculum in relation to those leaf-wetness periods is crucial. They are virtually absent as a problem in Northern Ireland because the inoculum is always there. But in a location such as California, and I am assuming also New Zealand, that pulse is interrupted by pronounced dry periods, particularly during the summer months. So, the cycle of events would differ between those two locations.

Dr Latorre
527. A brief comment, in addition to what Dr Swinburne has said. Weather conditions are also very, very important during summer time for inoculum production and then for inoculum dissemination, infestation (as mentioned before), and finally infection. In some places of the world where apples are produced, like Chile, California and New Zealand, summers are very dry, no rain at all. If it ever happens, there are very short rainy periods and not enough to fulfil perhaps inoculum production.

Australia
528. The next question we have here relates to the two-page extract from the BRS analysis attached to Annex 2 in our rebuttal submission and the question is reasonably long, so we have given a hard copy of the question in order to assist you and my colleague Dr Barry will address it too.

529. This is addressed to Dr Swinburne and Dr Latorre. Annex 2 of Australia's rebuttal submission contains a further detailed climate analysis undertaken by the Bureau of Rural Sciences (referred to as BRS from here on in). As part of this analysis BRS asserts that there is some debate about the exact temperatures and rainfall for conditions required for infection and spread of European canker. BRS illustrates this by outlining differences between the papers: Grove (1990); Lolas and Latorre (1996); Latorre et al. (2002); and New Zealand's Beresford and Kim model. BRS concludes that the different findings in these studies indicate that successful European canker infection occurs over a range of temperature and associated rainfall, with lower instances of infection associated with, but not prevented by, sub-optimal conditions. BRS also notes that the Beresford and Kim criteria of

30 per cent rain days and temperatures of between 11 and 16 degrees Celsius for more than eight hours represent the more restrictive end of requirements in the debate about the temperature and rainfall combination required for infection. Are you able to offer any general observations on the research findings that successful European infection occurs over a range of temperatures and rainfall, with lower instances of infection associated with, but not prevented by, sub-optimal conditions?

Dr Swinburne

530. Can I begin by trying to answer that by drawing attention to the piece that you sent around before and in particular to draw your attention to the second paragraph, which contains a particularly important error? On the last sentence, "On developed cankers, ascospores and conidia are present throughout the year". That is incorrect. The key feature, which you have omitted from your attempts to identify the appropriate climate, is that you have not allowed for inoculum production. Without rainfall, you will get no spores, even in Northern Ireland, if you have about 10 or 15 dry days, the existing perithecia and sporodochia releasing the conidia take some time to get underway to start releasing again. It is a period of time. But, for the benefit for the Panel, I should point out, a point which is not totally understood. The sexual stage, the ascospores are produced in little flasks. These grow superficially on the surface of the canker. The asexual spores, the conidia, are produced on little cushions that are also superficial on the surface of the canker. For the production of both of these structures, you need surface water. Water coming from the inside of the tree doesn't seem to count. You need wetness for both of those structures to form. So, it is incorrect simply to look at leaf wetness temperatures concerning how long it takes to get a leaf scar infected by the inoculum, you have to get the inoculum in the first place. And, if you have a long, dry summer you will not get the inoculum formed. We know from California that normally they do not produce perithecia on their cankers because the period of leaf wetness is too short, even though they have a high volume of rain but in a relatively short period of time. This is why the regional differences in the models need to be taken into consideration. Leaf scar infection data alone cannot describe the model. Volume of rain would not describe the model either. It is the duration of leaf wetness and the only approach we have to that, is the number of rain days in various locations. That is what was in the Beresford and Kim model, which was rather closer to what one would expect to happen in nature.

Dr Latorre

531. I would like to indicate that perithecia are produced only under certain conditions, apparently. And, those conditions are correlated with rains. In our observations we need very frequent rains, maybe over 2,000 millimetres a year, in order to be able to find some perithecia formation. However, conidia are produced in areas with less rain falls a year.

Australia

532. If I could ask my colleague to bring the slide of figure 10 from Australia's first written submission, the BRS. Dr Swinburne, in light of your written response to Question 72, could you please confirm that you accept on the basis of New Zealand's Beresford and Kim model that "the climate of the coastal cities of Melbourne and Sydney could marginally support infection in spring and autumn". In terms of the pictures that are here, the bottom is the percentage of days with rain and I think there is daily temperature ranges in the Beresford and Kim model in that order.

Dr Swinburne

533. Given that the area in the immediate neighbourhood of Melbourne has 125 rain days a year, relative to a 175 in Western Tasmania and 125 in the Albany area of Western Australia, I would say that the immediate environs of Melbourne could, if it was raining as it is today in Melbourne, allow canker to develop, but not if you had that very prolonged drought that Melbourne has suffered prior to today's date.

Australia
534. Dr Latorre, you also appear to accept on the basis of New Zealand's Beresford and Kim's model that "areas potentially conducive to European canker establishment were detected in Australia". Could you please confirm this?

Dr Latorre
535. Based on the information you have presented, it is potentially possible in the autumn, winter, just by looking at the rainy conditions of this area. I have to say that the other parameters have to be taken into consideration before you're sure of a real risk of infection. Summer time appears to be unsuitable.

Australia
536. Could I just ask my colleague to bring up the slide of figure 6 from Australia's rebuttal submission, Annex 2 BRS. Dr Swinburne and Dr Latorre, Annex 2 of Australia's rebuttal submission contains a further detailed climate analysis undertaken by the Bureau of Rural Sciences. One of the main findings of this analysis was that there are Australian commercial apple-growing regions, i.e. the Adelaide Hills region, and Australian port cities, Melbourne and Sydney, that meet the infection requirements for European canker specified in New Zealand's Beresford and Kim model. Are you able to offer any observation in relation to this aspect of BRS's climate analysis? Before you answer, I might just add: there are four different charts related to this, so we might just cycle through all of them so you can see the relevant data from the chart. This one relates to winter. If we start off with spring you can see this top right-hand quadrant, M = Melbourne; L = Landswood which is an area in Adelaide Hills. In summer there is a Landswood data point in that top right-hand quadrant and if we move through to autumn, you have Melbourne and Landswood again and Sydney on the margins. Finally move through to winter, you can see Sydney, Landswood and Melbourne on the margins. I must emphasize that we are using New Zealand's Beresford and Kim analysis.

Dr Swinburne
537. I don't know how much of the fruit growing area that actually represents. Assuming that you have applied the Kim-Beresford model, I would suggest that there is a possibility for canker becoming established, perhaps not through fruit, but through some other route, like infected planting material, which is the most likely route that you will ever get canker.

Dr Latorre
538. It is difficult to provide a good answer by just a first look at this information. I would be suspicious that some potential for European canker exists in some years and locations where you can fulfil conditions for infection, provided inoculum is present and the host is susceptible, which not necessarily may occur in winter time, for instance, when you have the most canker development. Most of the good weather conditions that occur in some locations, occur in winter time, which is not the best time of the year for canker development.

Australia
539. Dr Latorre, just to confirm in that in the top quadrant autumn, which is the highest risk of infection, that there are areas of Australia that meet the Beresford and Kim requirements in autumn?

Dr Latorre
540. Sure, you have some places where conditions appear to be potentially favourable.

Australia
541. My colleague, Dr Barry has a further question for both Dr Latorre and Dr Swinburne.

542. A 1999 paper reports on significant fruit infection during summer in Southern England, East Malling in Kent. However, New Zealand's Beresford and Kim model suggest that the summer

climatic conditions in East Malling in Southern England are not suitable for European canker and that the fruit rot phase is only important for Northern Ireland. What conclusions can be drawn about New Zealand's climate analysis from this failure to predict that East Malling's summer climatic conditions are suitable for fruit infection? Up on the screen is figure 3B from New Zealand's first written submission, Beresford and Kim Annex. To explain, you will notice from there that East Malling is in the bottom quadrant, not the top quadrant which is the Beresford and Kim acceptable parameters quadrant.

Dr Swinburne

543. Since we are talking about East Malling, I better respond to that, I suppose. We are enjoying a good summer this year. The last previous two years have been very wet and we do have alternating periods of time when it's conducive to fruit infection in summer and not conducive to infection in summer. In other words, East Malling does have a marginal problem with fruit rotting and if it's bad it's in the order of 5 per cent of the fruit becomes infected. The contrast, of course, is Northern Ireland, where the very first stores that I visited in the '60s to determine what the problem was, the volume of rotting was 85, because the rain had been going continuously. I need to add, of course, something which needs to be taken into consideration. The Northern Ireland situation was caused by canker brought about by the introduction of refrigerated, sealed, controlled-atmosphere, stores. Fruits stored in barns never rotted with *Nectria*.

Dr Latorre

544. Perhaps the only comments I can make from these charts now, is the situation in the Chilean condition, called Talca, which appears there being favourable in summer time for infection. That could be true, because in some years they get some rainy periods in the summer time. However, I have never seen any apple been rotting in any orchard that I know in this area. Maybe this is because inoculum is not being produced. Weather condition for inoculum production may not be fulfilled with this conditions immediately. Or, maybe they are fulfilled to produce one conidia, but that conidia of one hectare of apples means nothing. Perhaps that explains why people producing apples in this region, that region in Chile, and are never concerned about the possibility of apples being rotting with *Nectria*. Not even when they store apples for a very long period of time, maybe six months or more. However, if you move perhaps to a more rainy season area in the Southern part of Chile, eventually you may have, after maybe a couple of rains in summer time some infections. Why? Because, during the rest of the year, inoculum is being produced, being active. You don't have only one canker per hectare, but maybe several per hectares, where the inoculum can be produced. It is very difficult to make a judgement in a minute here about what could be the conditions of *Nectria* in Australia or any other place, just by looking at this information alone. I would be very willing to make other comments later on if its needed, if I have the time to review some other information.

Australia

545. New Zealand's climate analysis also fails to predict that Sonoma County in California is suitable for infection in summer, yet according to McCartney (1967), high fruit infection occurred in the 1965 outbreak. Could you please comment on this failure of New Zealand's model?

Dr Latorre

546. I am sorry, but I don't have any comments on Sonoma. I know the area, which is quite dry in the summer time, and they do have canker. But to my knowledge they are not concerned about apple rotting in California. This doesn't mean that eventually they may have one fruit in the whole production of the year, maybe.

Dr Swinburne

547. Models are based on means and averages. You are talking here about singular events and I think this is not something any model can actually cope with.

Australia
548. That is the end of our questions about climate. We have a related consequence question which we can ask now or wait for our New Zealand colleagues to respond to that and ask later. We will be guided by you.

Chairman
549. New Zealand, do you have anything on this climate issue first?

New Zealand
550. We do have a couple of questions and I will ask my colleague to pose those.

551. This is to both Dr Latorre and Dr Swinburne, about whether it is scientifically justifiable in your views to use weather data from particular years selected in a non-objective manner and using criteria in the case of the earlier charts, using Beresford and Kim versus various locations in all seasons, selecting weather data from wettest and driest years, which bear no correlation to the variables that are being tested which are moderate temperatures or percentage rain days as against hours between 11 and 16 degrees. Is that an appropriate method?

Dr Swinburne
552. I didn't quite follow the nature of your question. What is it you are trying to get a comment on?

New Zealand
553. The question is: Whether it is scientifically justifiable to predict risk using weather data from particular years. In this instance, selected according to criteria, wettest and driest years, which bear no correlation to the variables against which it is being tested, and whether you have a view on that?

Dr Swinburne
554. If that data relates to a particular year as opposed to prolonged multi-year means, then that wouldn't be justifiable. But that is multi-year means, isn't it? I need to ask that question myself, before I can answer. Are those multi-years means or are they taken from particular years?

Australia
555. They are taken from particular years – the wettest and driest years.

Dr Swinburne
556. One would like to see them done for minimum of 10-year means.

Dr Latorre
557. I was almost at the point of saying the same thing. This is an exercise to begin the analysis, but certainly if you want to raise a conclusion out of this, it would be very dangerous. You have to review a fair number of years before you make a conclusion.

Australia
558. If I could add that this may be an issue that New Zealand and ourselves may wish to discuss in the next two days.

New Zealand
559. We have no other questions related to climate, though when we come perhaps to the conclusion of the discussion of European canker we may come back with a question or two, but not on climate.

Australia
560. Dr Swinburne, in light of the discussion we have had about climate and noting your written response to Question 60, could you please confirm that you could accept that the IRA team's assessment of consequences of European canker is objective and coherent, if it were demonstrated that there are areas of Australia, including major apple-growing regions like Landswood and Adelaide Hills, with climatic conditions conducive to European canker.

Dr Swinburne
561. Based on the observation of where your fruit growing regions are, taken from your submission, and noting that it lies within the band with less than a 100 days rainfall on average per year and perhaps nearer to 50. I think the consequences of an infection event within your fruit industry would not have as large a damaging effect as you had claimed. That is essentially what I said then and I reiterate it now.

Australia
562. We have a few extra questions on consequences, which we can ask now or wait for our colleagues from New Zealand? We can go ahead? This question to both Dr Latorre and Dr Swinburne, Australia's appropriate level of protection is expressed as providing a high level of sanitary or phytosanitary protection aimed at reducing risks to a very low level, but not to zero. As an island continent, Australia is free from many of the world's serious agricultural pests and diseases and it takes a very robust approach to post-border biosecurity to maintain this favourable pest and disease status. This is evident by the rigorous eradication programme that we put in place following the 1950 Spreyton disease outbreak in Tasmania, as well as the 2004 outbreak of citrus canker in Queensland. Such programmes typically involve measures such as extensive surveys throughout Australia, application of chemical treatments, disruption of transport arrangements, removal of significant number of commercial orchard trees, removal of native plants. Do you accept that Australia's particular circumstances in this regard were a relevant consideration for the IRA team in arriving at a conclusion that consequence of European canker would be moderate? We note that this can be contrasted by the high rating assigned to fire blight.

Dr Latorre
563. Certainly, you have some experience that this information is useful for Australian people and to estimate the consequences. But based on the general overall information in the world, in my opinion European canker is an important disease, but it is not a limiting factor for apple production, nor is it a limiting factor for commercialization outside Australia. At least this hasn't been the situation that I can speak of, in the Chilean production. We do have this problem in part of the country, that farmers can really address, and so far as I know, it has never been a real problem or been a reason for not to commercialize the fruit properly, adequately, in most of the international or local markets. In this regard, I tend to disagree with the moderate conclusion on consequence that you have arrived at. However, you may have different data that maybe can support this conclusion.

Dr Swinburne
564. I am really trying to think this one through. The Tasmanian outbreak, which you referred to, with the amount of effort that you went to, to remove infection, has caused me subsequent to writing my submission, to think about that particular instance quite a lot. I note that it didn't spread to native plants, but I did note that you had an enormous difficulty in getting rid of it, in spite of constant pruning and cutting, and so forth. Even though you had no inoculum, there was no spore production. What I should have said in my original submission, which only dawned on me subsequently, is that that infection was imported with the trees that you planted on that occasion. Those trees were almost certainly infected when they put were in the ground. The desperate difficulty that you had in getting rid of it from those individual trees was because it was semi-systemic infection. In fact, the infection was moving through the tree inside. Although you cut off a canker there, you were astonished to find another one appearing on a branch over there. But it had not spread from there to there directly, it had

gone the other way, it had gone, as we now recognize, semi-systemic infection and that clean planting material is crucial to the prevention of canker. So long as you keep Australia's quarantine system going for new cultivars or new root stocks and make sure that the nurseries that produce the commercial trees are screened absolutely, I don't think Australia will have a particular problem from canker spreading by the airborne route. A question which has also haunted me since I have put the pen down, is why don't you have it? Why isn't European canker there already? Because you have had apple trees for a very long time and you must have had canker delivered to your doorstep in the form of trees that you had brought on to the continent. It disappeared. That is a question which has also engaged me.

Australia
565. Thank you for bearing with us through this afternoon. We have one final question. My next question is to Dr Latorre and Dr Swinburne. Annex 3 of Australia's rebuttal submission compiles a range of statements from New Zealand, Europe, North and South America, attesting to the fact that European canker has serious consequences. Are you able to offer any comment in relation to this material. We have just handed out a copy of that Annex for a reference to everyone.

Dr Latorre
566. Is this a question related to the last information, Annex 3? For those who are not plant pathologists in this room, I have to say that there is a big problem with plant pathologists. In the sense that they tend to consider their work as the most important and most serious of anything in the world. In doing so, we tend to over emphasize the importance of some diseases. I think I make myself that mistake too, sometimes. In reality, diseases are very important, this is true. The question is, is this a limiting factor by which the farmers or the country cannot produce a certain commodity? Speaking on European canker, this is not the case. It has not been the case in any place in the world where this disease has arrived, as far as I know. I would like to take the opportunity to make a short comment about Dr Swinburne's last answer. Just to emphasise that the only way that I can see how Australia can get European canker, really, is by the commercialization, international transportation of nursery plant material. Perhaps this has been the way how this disease has been moved around the world. I have seen no possibility that in reality fruit can really be the cause or be the introduction, establishment and spread of the disease in a new area. You can introduce *Nectria* on apples, but the other question is whether this disease is going to be established in a location from the inoculum arriving on fruits. So, I am not surprised about the comments in Annex 3. If you look in the literature, normally it is called "a major disease of apple".

Dr Swinburne
567. There is one area that I know particularly well where canker does re-shape the apple industry, is of course Northern Ireland. There they grow this cooking variety, called Bramley Seedling, because it has a measure of resistance and in fact it's one variety in which the pathogen never goes systemic. It is a very localized branch-to-branch distribution. It can be very damaging. It can follow certain events, as has happened in Sweden. If you get very severe frost damage where bark splits, you have got a huge area available for infection. An infection on a massive scale can take place. But, I think I also have to agree with Dr Latorre, we do in writing our grant applications point out that the disease we are working on could end civilization as we know it.

Australia
568. I would like to thank you both for their patience in going through that material with us this afternoon.

New Zealand
569. I am aware of the need to move on, but we just have one or two final questions. The first is to follow up to the discussion we have been having about consequences, we would like to ask both Dr Latorre and Dr Swinburne, whether it is relevant or appropriate to compare consequences in one

part of the world, e.g. Northern Ireland, with climatic conditions extremely conducive to the disease, but which are not necessarily the same as in Australia?

Dr Latorre
570. If I understand correctly, yes. The effect of the disease may vary from place to place depending on weather conditions, for instance.

Dr Swinburne
571. I think most of the fruit growers in Northern Ireland would give their right arm for an orchard in either New Zealand or Australia.

New Zealand
572. Just one final question directed to Dr Swinburne, in the first instance. Your answers with respect to New Zealand's proposed alternative measure, which is to ensure that apples are limited to mature export grade apples, appear to be directed at the risk of the arrival of latently-infected fruit *per se*. The measure New Zealand is proposing as an alternative, that measure is aimed at the risk of apples providing a pathway of entry, establishment and spread. As I understand from your responses today, such risk is vanishingly small. Could you confirm that view?

Dr Swinburne
573. There is an absence of what I would call "survey data" on the nature of the various pathogens that may or may not be responsible for rotting fruit in New Zealand. But from what little there is, it would appear that the frequency of infection with *Nectria* is quite low. But there is some confusion as to whether or not this is becoming apparent before or after harvest. I gather from some of the reports that rotting has been seen before harvest. It does surprise me and it would be of interest today whether that would ever actually lead to a progressive infection, or whether it is just a dry eye rot, which can sometimes take place, but which never develops further. I have no data. There has been no data presented on either side, relating to what is coming out of the controlled-atmosphere stores three or so months after harvest. That is the one I would really like to see. I would imagine the infection frequency would be very low, but I wouldn't say it would be non-existent. There could well be infections which develop post-harvest two or three months down the line of the store distribution chain.

New Zealand
574. Just a follow-up question related to the second and third elements, looking particularly at establishment and spread, and factoring in your comments earlier today on establishment and spread, in particular related to exposure and transfer to a susceptible host, whether you have any additional comments?

Dr Swinburne
575. I don't know that I do have any additional comments. A hypothesis: if one had 100 infected apples that arrived in Australia healthy, but became rotted subsequently, what would happen to those 100 infected apples? Perhaps 70 or 80 of them would never develop spores anyway, so it could be disregarded. Then you have to consider where the others might end up in relation to the dispersal of inoculum. In other words, all the processes are attenuating all the time down the chain and that is what I don't see coming through in the IRA at all. The longer the apples are stored in New Zealand, the less is the likelihood that an apple infected with *Nectria* would be sent to Australia, because it would be culled out before it went into the retail-ready pack and went on the train. So, the attenuation process after harvest would be continuous throughout the transport chain which is not terribly well reflected in the IRA.

Chairman
576. I think what we have done today, and particularly in this last session, is to quite thoroughly go through questions that we wish to ask the experts and certainly the Panel would not like to treat this last pest area with any less importance. We could continue for another hour, but the number of questions we have will not be concluded in the next hour. It would seem to me that it might be better not to split up the ALCM discussion into halves. One option is to continue until 19:00. Even if we do continue, I don't believe we will finish within the hour and I am sure you might have questions yourselves. So the question is do we start now or simply start tomorrow on ALCM. The experts, I know their answer. What would the Parties preference be? Let me say my preference would be not to split it, but we can continue for another hour and then carry on tomorrow morning, but I am not sure whether that is best way to deal with it. On the other hand, you might want to do some preparation tomorrow morning.

New Zealand
577. We are very much in your hands and I am conscious of the burden that the long hours have been on the Panel, the experts and the Secretariat. We would be open to continuing on now, but we are in your hands.

Australia
578. We do have a slight preference for continuing now, even if only for another hour because there is a lot to get through. If you feel strongly, we can review.

Chairman
579. That's fine. We are available until 19:00. I know Dr Cross has been sitting patiently all day, so I am sure he is also ready to respond. I would like to ask Ms Hillman to start off with our questions.

Kirsten Hillman
580. These are some general questions and where we have specific questions on importation steps we will ask those and then move on to consequences and alternative measures much as we have done with the other pests. The first question we have for you refers to Question 94. If you recall Question 94 is a fairly lengthy question where we ask for your view as to whether or not the evaluation in Australia's IRA for each of those issues mentioned in that question was objective and credible and relied on respected and qualified scientific sources and was based on available, credible data. Again, as I said a few times today, recognizing that those scientific sources don't have to have a majority of opinion, it can be a minority, but our concern is whether or not they are respected and qualified sources. So, I don't know if you want me to go through for you the issues that were covered in that. There is quite a list of them. But, specifically we had asked, with respect to each of those, whether or not you felt in Australia's IRA the evaluation there was based on credible sources. We would like offer you an opportunity to add anything there that you might like to add in terms of a more definitive conclusion.

Dr Cross
581. I think I don't have any specific requirement to alter any of the things I have said here at this stage, but there may be things that come out in specific questions relating to the different parameters. There is quite a lot of material here.

Kirsten Hillman
582. If we looked at Question 94(i) on the condition of adult emergence of the ALCM, New Zealand submits that its "use of 13-18 days for the timing of adult emergence… was in respect to the minimum time required for adult ALCM emergence, not the range". New Zealand concludes that its "position on the maximum length of time required for adult emergence is consistent with Prof. Cross's: the scientific evidence indicates that adult emergence could in some cases take a

considerable length of time, possibly more than one year depending on conditions". So, we are wondering if you have any comments on New Zealand's comment on your response.

Dr Cross

583. I think New Zealand's position is that a period of at least 13 days would be necessary before any adult leaf-curling midge could emerge. And, actually in their recent submission had provided some more experimental data which seem to support this. But that data is relating to fruit that has been stored for many weeks. I think that if apples had not been stored for a long time, they could have pupae on them which could emerge quite quickly when the apples arrived in Australia. There could be live pupae on those fruits that were near to emergence and if they were exposed to the correct conditions they could emerge very quickly, maybe in a space of a few hours or a day or two. On the other hand, there will be individuals which require more than one diapause-breaking period in order to emerge. It is conceivable that there would be some individuals that might not emerge for a year. I think that the emergence period of the midges could be very protracted depending on the conditions which the fruit has been stored in, and the conditions when it is released into the environment in Australia in some way, those conditions make quite a big difference. It would depend on the time of year, the temperatures, and what have you. The period does seem to me to be quite a protracted one, of many weeks. There are published papers which show intervals of eight weeks for emergence of adults in spring and even the New Zealand data just submitted on 2009 experiments of Sandanayaka and Rogers show a 2 to 3 week interval over which emergence can occur. I think it is not possible to be very definitive over the interval, but we do know it is quite a long time. I don't think threshold temperatures for development have necessarily been worked out for adult emergence, but at the time of release into the environment there would have to be conditions suitable for emergence and flight of the midge, at some stage.

Kirsten Hillman

584. I will just keep moving on, unless I see signs that either Party would like to make a comment. Following-up, in your replies to a number of questions you indicated that the existing data or evidence was inadequate to resolve certain issues. For example, regarding conditions of adult emergence, flight range, adult life span, etc. Australia argues that in such conditions it used expert judgement to determine the probability ranges for certain steps. In your view was this expert judgement exercised in an objective and credible way?

Dr Cross
585. I think we need to look at each of these factors one at a time, maybe taking the flight range of the midge. There clearly isn't very much evidence as to how far the midge can fly. There is one study I did myself on males that showed they could fly 50 metres towards a pheromone source over a period of 24 hours. But there is another new Zealand study that shows that the midge can move in an infested plantation in a range of 30 metres a year. There is no study that shows how far the midge can fly. There is no definitive flight range. So, it's rather difficult to establish what the flight range should be. Obviously the midge is a weak flier. In my view, it is unlikely to be able to fly long distances. There is the question of whether it could be distributed by wind over long distances, but there is no evidence to support that is the case. I felt that the Australian IRA in respect to this, which suggested that a flight range of 200 metres was not unreasonable and that orchard surrounding the wholesale pack houses were often less than these distances, seemed to be quite reasonable. Yes, another area of where there are not sufficient data or only one study was this viability of the insect inside occupied cocoons. Clearly we have this one study by Rogers et al, which has been much quoted, where we only have one study which is open to some interpretational difficulties. I felt that that viability question had not been adequately addressed in the Australian IRA. It didn't seem the viability itself did not seem to be taken into account. So, there are several different factors. Maybe some of these things will come up more in individual questions.

New Zealand
586. We have one question. You stated in your response to Question 94(iii), on page 3, that "The distance in Australia's IRA of 200 metres is… not supported by evidence". Can you confirm that this is still your position.

Dr Cross
587. Yes, I don't think there is any study showing how far this midge is able to fly. In the study I did, the maximum distance investigated was fifty metres. I did not look at distances of 200 metres, and I only looked at males attracted to a pheromone source, which is not the same situation as a female being attracted to a host plant in the absence of other surrounding host plants. As far as I know, there is no study that has investigated that scenario, where the mated female midge has to fly to find its host. All we know are some things about those types of midges in general. They are rather weak flyers. This particular species spends a lot its time close to the ground and only flies up into the tree to oviposit in the shoots when the wind conditions are rather slight. So, it seems unlikely that it would have a very long range of dispersal. I think it would be unlikely that it could fly many hundreds of metres. But I felt that the 200 metres distance was not totally unreasonable. It doesn't seem to me to be an impossible distance for this midge to fly.

Kirsten Hillman
588. Still on Question 94, in paragraph 4 regarding climatic factors in respect to ALCM spread, you conclude that "Australia's IRA was objective and credible, but did not draw sufficiently on available information and did not conduct a sufficiently detailed analysis". Could you elaborate a little bit on that.

Dr Cross
589. This is the climatic conditions. I don't know very much about the climate of Australia, but clearly there are certain climatic requirements for the midge's survival and we do know that it doesn't occur everywhere in the world and there are parts of the world where apples are grown and the apple-leaf curling midge doesn't exist. In Europe, where it seems to have reached its equilibrium position, it doesn't occur in the Southern European areas of Southern Spain, in Italy South of Naples. Similarly, over in the United States it doesn't occur in the more southerly areas. I think the thing is that it needs cool temperatures, a sufficient accumulation of cool temperatures in winter to break diapause. Unless diapause is broken, the emergence in spring is not synchronized properly. In areas that don't have a sufficient period of cool in winter the midge cannot exist. I am questioning whether those limits have

been established in Australia. If you look at a world map of where the midge exists, it doesn't seem to occur in latitudes much less than about 38°. I haven't investigated this thoroughly, but clearly in these warmer areas there are not sufficient cool temperatures in winter and that limit has not been established in Australia by a climatological analysis. Similarly, the midge also needs reasonably regular summer rainfall. Although it can clearly survive droughty periods for a year or two, drought conditions in summer do not favour it all and it needs reasonably regular summer rainfall in order for it to be a successful species and it doesn't occur in areas of the world that are dry. A great example of this is Washington State where West of the Cascade Mountains, where there is quite a lot of rainfall, the apple-leaf curling midge exists. But to the East of that mountain range, there is an arid area where there is little summer rainfall in the central summer months, and the midge doesn't exist. Presumably, because there is insufficient moisture for it to complete its development. So, I am suggesting that a study needs to be done of these factors and it needs to be established where in Australia the climatological conditions exist for the midge based on its known distribution throughout the world.

Kirsten Hillman
590. If we could ask for a follow-up response to Question 95. The question is this: In your view, are the probability values chosen for the potential entry and establishment of the ALCM in the IRA sufficiently supported by scientific evidence?

Dr Cross
591. I think we are fortunate there is some data on the incidence of the apple-leaf curling midge cocoons that are occupied by larvae or pupae. I think we do have some reasonably good data on which to base that part of the analysis. I felt that the August 2005 data provided by New Zealand, which showed the incidence of the midge on four and a half million apples over a period of four years, gave some quite good indication of the sort of likely infestation level. Beyond that, there are the questions of what percentage of those cocoons contain viable larvae or pupae. It must be stressed that I believe that those are occupied cocoons and that data does not relate to empty cocoons. Beyond that, the viability of those cocoons needs to be taken into account and parasitism, and an important factor is the rather protracted emergence of the midge in relation to its very short life-span. If the midge is only able to survive for a couple of days in the natural world and the midges are emerging over a period of 3, 4, 5, 6 weeks or maybe up to a year, then the chances of the establishment of the infestation are very significantly reduced. In my opinion, those three factors were not really adequately taken into account in the analysis.

Kirsten Hillman
592. In its comments on expert responses New Zealand notes that you may have confused export bulk bins and harvest bins in your replies to Question 98 and Question 101. Did you have any comment on this? Just for clarification.

Dr Cross
593. Yes, I may have done that. I was unsure whether bins that are used at harvest would ever be used and sent to Australia. I considered that possibly the fruit might be harvested in New Zealand and that the harvest bins could be sent there before re-packing. So there may have been some confusion there. This is an unlikely practice possibly. Perhaps the Parties could say what they think.

Kirsten Hillman
594. We'll have lots of time to chat with the Parties tomorrow. Next question relates to Question 120. In your reply to Question 120 you state that: the "unrestricted risk estimation" in the IRA "needs to be re-calculated for several reasons". Does this imply that in your view the conclusion in the IRA that the inspection of a 600 fruit sample from each import lot would not achieve Australia's accepted level of protection?

Dr Cross
595. I am unable to do the risk assessment... Well, perhaps if I tried, I might have been able to do it, but I didn't attempt to do the risk assessment calculation. I just note that some of these important factors had not been taken into account in the IRA. Once we have been through the viability, the parasitism, the protracted emergence and the number of apples that have to be disposed of within the range of female flight of apple trees, and the mode of trade. All those things are very important factors and they haven't been taken into account in an adequate way in the IRA in my view. If the IRA is re-done, it might be found that the unrestricted risk assessment comes out to below the ALOP, but I don't know whether that is the case or not. The exercise needs to be done. If the risk assessment shows there is a risk above the ALOP, then a sampling procedure needs to be, the size of the sample needs to be, calculated to reach the ALOP. It should not be calculated to demonstrate that New Zealand apples have got apple-leaf midge. It should start at the point of preventing the risk. I didn't go through that exercise myself because I think that is something that Australia needs to do.

Australia
596. We should just like to ask a question following-up on your remarks, and in respect of your written replies where you did indicate that the probability of entry, establishment of spread for ALCM may be reduced if fruit waste was enclosed so that adult midges could not escape and if all apples were held in cool chain conditions until sold to consumers. Our understanding is that your comments about the reduction in probability were conditional on whether waste would be so enclosed and whether apples would not be distributed to orchard packing houses. It would be useful if you could clarify that point. And, also, you would be aware that Australia and New Zealand disagree about whether apples would be distributed to orchard packing houses and also as to how apple waste is managed in Australia. Could you expand on your written comment that the potential risks would be higher if large numbers of fruit were held in ambient temperatures in the vicinity of an apple orchard.

Dr Cross
597. Yes, I think that this is sort of self-evident, exactly that. If large volumes of apples are packed or graded and there is waste fruit from that process and it is disposed of or held in the vicinity of apple orchards, within less than 200 metres of the site that this is occurring, clearly those apple fruits could potentially have a low incidence of infested or occupied cocoons on them and a small number of midges may emerge and they would have a higher chance then of being able to make it to the nearby apple orchard. Clearly, the way the fruit is handled in Australia seems to me to have a very large effect on the risk. If that happened, I think the risk as demonstrated in the IRA would be probably quite substantial. If large numbers of fruit were disposed or held quite close to orchards and not in refrigerated conditions, then I think there would be a substantive risk. But if that doesn't occur and fruit is retail-ready and sold in small quantities to individual consumers, then the risk is, in my view, greatly reduced because it is unlikely that large numbers of apples would be disposed of or held close to an apple tree.

Chairman
598. Could I follow-up with a question more on transparency, perhaps. In commenting on your responses to Question 113,. Australia submits that "pursuant to the recommendations of the Final IRA Report, there will be no 600-unit inspection by AQIS *on arrival* in Australia, because the consignments are to be *pre-cleared* in New Zealand". In your view is that fact clear from Australia's IRA?

Dr Cross
599. I was uncertain as to whether a second inspection would occur. Clearly, that's an important point. That's not something that I can really determine myself.

Chairman
600. But, from the IRA, it was not clear to you whether there was…

Dr Cross
601. I was in doubt as to whether or not it would occur, or not, and how effectively it would be done; whether it would just be a cursory inspection or whether it would be a thorough 600 randomly drawn sample, that it's supposed to be, if it's a proper inspection…

Chairman
602. Maybe just a related question. In your view, is the IRA clear on the relationship between pre-clearance and the procedures to be applied to New Zealand apple fruit?

Dr Cross
603. Sorry, say the last bit again.

Chairman
604. Is the IRA clear on the relationship between pre-clearance and generally the procedures to be applied to New Zealand apple fruit?

Dr Cross
605. It may be clear, but I may not have understood it fully.

Chairman
606. Okay. Let me move on to a question related to importation step 2. According to Australia, cocoon viability is taken into account through its use of a triangular distribution for importation step 2. In its rebuttal submission, New Zealand argues that taking viability into account should have shifted the entire distribution. Would you have any comment on this statement?

Dr Cross
607. Yes. I believe that's the case. I think the most likely value for the importation step 2 should perhaps be the one that is given as the general mean in the August 2005 data, which is .16 per cent. So we do have this quite good estimate for this central value. Maybe the minimum value is very small, perhaps negligible. And perhaps the maximum value should be the greatest level of infestation that has been detected in lots in recent years. I think there's some data from New Zealand exports to California, which shows some values of about 5 per cent infestation in some of those lots. I'd have to check the data. That would appear to be the sort of range of data values that should be used, in my view. Clearly, if you had a viability of 50 per cent, it would shift the whole lot, you know, one way. It wouldn't just alter the upper value or the lower. It would shift the whole lot. And similarly with parasitism. And I don't think that that choice of values has reflected that properly.

Chairman
608. Let me move on to another question, in the light of your response to question 111. And this is more relating to step 3. Is it your view that the IRA over-estimates the probability that apples are contaminated during picking and transport to the packing house?

Dr Cross
609. I think we don't know what proportion of the contamination of the fruit arises at different stages in the growing process. I personally believe that the amount of contamination that would occur during the harvest process would be a rather small proportion of the amount that would arise during the earlier part of the whole growing season. But there is clearly a low probability that larvae could, when the fruit is picked, that they could fall onto the fruit as the fruit is picked, and that would slightly increase the risks. But I don't know how you would quantify those values and the values chosen, I don't know on what basis. Here, (10^{-3}, 5 x 10^{-2}) uniform, I don't know how those were chosen, those values, or whether there's any study that shows what would be appropriate values. But all this is in a way taken into account, because of the end point inspections data actually includes both components anyway.

William Ehlers
610. There is something that is baffling me a little bit. That's probably because I don't understand. So I'd like to just quickly go back to one of the earlier questions, which is the range of flight. Because, if I understood correctly, even though there is very little study that can really confirm all of this, at least one study that you have done had a limit of about 50 metres for males being attracted by pheromones.

Dr Cross
611. It wasn't a limit. It showed that the midges could fly 50 metres, but that was the longest distance investigated. And that was attracted by a pheromone source, which is different from the female being attracted to its host plant. And we don't know whether males and females have a similar flight range.

William Ehlers
612. And which is the stronger of the two, do we know?

Dr Cross
613. Well, I think it's a powerful life force, the female finding where to lay its eggs is pretty important to it. You know, I don't know that we have any evidence to say that the flight range of the females is shorter or longer than the males. We don't have information on that. They have a bigger body weight, but then again they are stronger insects. They are slightly larger, stronger insects.

William Ehlers
614. So these factors, then, help me to understand better why your estimation of 200 metres is reasonable, because of the stronger attraction to finding a place to leave the eggs, and because they are stronger animals…

Dr Cross
615. No, not necessarily a stronger attraction, it's that we don't know how far the females can fly. There is no good data that gives you some sort of statistical distribution of how far a midge can fly in this particular instance. We have very little data. All we can do is sort of observe the midge's habits, and the way it lives, and think that probably long range dispersal of this midge is rather unlikely. It's a weak flyer. But the 200 metres assumption of the Australian IRA seemed to me to be reasonable… It wasn't out of court. It might be wrong, of course. It might be wrong by a factor of several fold. It could be that they can fly 500 metres, or something, but we don't have any better data.

William Ehlers
616. I was trying to contrast, you know, the 200 with the 50 and the fact that they stay on the ground a lot and rise according to how the wind helps them and that. But now I understand better, with what you've said. So let me go back to our list of questions. In the light of your response to question 112, is it your view that the IRA's analysis at importation step 3 appropriately takes into account the potential availability of sufficient flushes of leaf growth suitable for ALCM infestation during harvest in New Zealand, given that later growth flushes may be stimulated by irrigation or wet seasonal conditions?

Dr Cross
617. Well, I think that, clearly, apple trees can be growing at harvest. It depends on the weather conditions, how much rainfall there has been, how vigorous the orchard, how many shoots there are. But apple leaf midge infestations can go on beyond harvest. And it tends to drop off because, as the fruit gets bigger, the shoot growth of the trees tends to shut down. So, the probability decreases, but it most certainly can happen that there is shoot growth in apple trees at harvest, which is attacked by apple leafcurling midge, which is reaching its mature state larva stage, ready to go, fall from the tree,

and then infest the apple. And that's certainly the case. I don't think either Party disputes that. So what is the angle that you're trying to elucidate?

William Ehlers
618. I think you've covered it with your answer. In your reply to question 101, you indicate that the basis for the values given in the IRA at infestation step 3 for the likelihood that clean fruit contaminated by ALCM during the picking and transport to the packing house, is not clear.

Dr Cross
619. So this is the risk that clean fruit maybe then subsequently infested from leaf material attached to that fruit at harvest?

William Ehlers
620. Yes, if you… The question originally said please comment on whether the consideration in Australia's IRA of the likelihood of entry and establishment of ALCM, through mature apple fruit from New Zealand, was objective and credible, relying on respected and qualified scientific sources with respect to the possibility that clean fruit could be contaminated by ALCM during picking and transport through any leaf material.

Dr Cross
621. I think there is a rather small risk that this could occur. Most of the leaf material attached to fruits would not be growing shoots that harbour the leaf galls. They would probably be too old and would be unlikely to harbour apple leaf midge. But you can't exclude the possibility that there might be some young shoot material amongst that at harvest. So there is a risk, but I would think the risk was rather small. But particular values have been chosen here, (10^{-3} to 5×10^{-2}), so particular values have been chosen, and I was just questioning what was the basis for those values and how they were chosen. I don't understand how they were chosen.

William Ehlers
622. We were following exactly that line. So would that mean that, in your view, the analysis is not sufficiently supported by the scientific evidence?

Dr Cross
623. Well, the choice of values, I would say, is not supported by scientific evidence, because the basis for the choice is not clear. There has been no study of what the likelihood is going to be. So these values appear to have been picked to help making the calculation as a whole. I don't know what basis on which they've been picked, the particular values. All I can say is that I believe that the risk will be rather low from this step.

William Ehlers
624. Okay. I don't see anybody asking for the floor, so I'll move on. Does – and this refers to importation step 6, that's the likelihood that ALCM will survive palettisation, quality inspection, containerization and transportation – does the paper by Rogers et al (2006) support New Zealand's contention that only 15 per cent of ALCM cocoons contain live pupae?

Dr Cross
625. Why is this particularly relevant to importation step 6?

William Ehlers
626. Yes. The reference was to Australia's second submission, paragraph 622, so I'd have to look for that.

Dr Cross
627. This is back to the question of the viability of the larvae, in the cocoons. We have this one study of Rogers et al, which shows – I have to get the exact figures out – I think it's 25 per cent of occupied cocoons contained viable larvae. So this particular study showed that three-quarters of the larvae were not viable. So we have this one snap shot of viability, and we have very little data to go on. But I believe it is a very significant factor, looking at the different lots of apples in the Rogers paper, there was quite a range of values given for the different lots. And the highest viability among those different samples was 50 per cent. In the absence of any other information on this factor, one would think that perhaps a conservative estimate of 50 per cent would be a sort of reasonable compromise. But we need more data. I notice that in the recent papers from New Zealand, again there is some information on this Sandanayaka and Rogers paper of 2009, which does again seem to support this rather low viability percentage of… If you look at the data itself, I'd have to go back to it, but I think only about 20 per cent of the larvae in cocoons, having been stored for at least ten weeks, I have to say, in cold store, were actually viable. So we do have perhaps two or three sources of data that show that not all the larvae in these, all the insects in these cocoons, are viable, and that a substantial proportion of them are not. And clearly that needs to be factored in.

William Ehlers
628. Okay. But I notice that all the numbers you are using are above the 15 per cent.

Dr Cross
629. Yes. I have to go back. There is a difference between the percentage of the whole sample and the percentage of the ones that were occupied. I'd have to get out the paper by Rogers et al., but it is clear in there that, I think I've got it the right way round, that the 25 per cent is the viability of the occupied cocoons. And it's occupied cocoons that are the – the incidence of occupied cocoons – that occurs in the August 2005 data. So perhaps we should just think about the viability in occupied cocoons.

William Ehlers
630. Okay. Let's move on. Question 94(i). Regarding the conditions for adult emergence of ALCM, you wrote: "Unless evidence to the contrary is produced, Australia's IRA relating to this issue was objective and credible and relied on limited scientific evidence available. However, an important point is that longer period of adult emergence would substantially reduce the likelihood of small numbers of individuals in a consignment emerging within a few days of each other, and being able to mate and lay eggs to start an infestation." So does this last sentence imply that the IRA has overestimated the risk, as New Zealand implies in its rebuttals submission?

Dr Cross
631. Yes, it does. Clearly, that long protracted period of emergence with a short life span substantially reduces the risks. If they all emerged on one day, there would be a much higher chance of them being able to find each other and mate and go off and find their host plant. But a male emerging on day one is most unlikely to meet a female emerging on day seven, say. So, no calculation has been done. I believe it would be possible to make a reasonable statistical calculation of the factor by which this risk is reduced. But I believe that this prolonged emergence period does reduce the risk several fold. A simple consideration of a three day life span and a three week emergence period would clearly reduce the likelihood of small numbers of males and females being able to meet for the purposes of mating, it would be substantially reduced several-fold, even in that rather narrow consideration of just a three week emergence period.

William Ehlers
632. All right. Getting back to climatic conditions. In question 94(v), you wrote that "Climatic conditions in SE Australia, which have been exceptionally hot and dry, have been quite unsuitable for ALCM survival".

Dr Cross
633. Yes. I understand there's been a period of drought in the South-East part of Australia, maybe for up to a decade. And there have been long periods without regular summer rainfall. The apple leaf curling midge does need summer rainfall to survive, to thrive. We know that in my country that we get dry periods, the population of the midge reduces greatly. I think the same experience has been found in New Zealand, that the midge is favoured by wet weather. It's most troublesome in areas where there is a wet climate. Northern Ireland is one of its worst epicentres. And the places where it is truly a serious pest problem tend to be wetter areas. So I question whether an area where there has been a prolonged period of drought could support, or the apple leaf midge could survive in those areas. But maybe that's in the last ten years. And then there's the longer term considerations over what period should we consider the risk. If you looked at the long-term data, for 25 years or something, it may be that there is adequate rainfall in many of these areas.

William Ehlers
634. Yes. Well, in that context, New Zealand says in its rebuttal submission that the only area in Australia that is prone to the establishment of apple leaf curling midge is Tasmania. Would you agree with that?

Dr Cross
635. Well, I haven't done the climatic analysis and I think it is important that a proper climatic analysis is done to look at maybe the ten year average, which is the drought period, and the longer term. Who knows what the future climate is going to be like, but that has a profound bearing on the risk. I haven't done a climatic analysis, and a climatic analysis hasn't been done of other areas of Australia, in the South, where there may be adequate rainfall, and cooler conditions, where it could survive. Maybe in the southerly areas. So, yes, my limited knowledge of the climate of Australia would lead me to believe it could survive in Tasmania. I think an analysis needs to be done to see, to demonstrate whether it could, what other areas it could occur in. I think, overall, the vast bulk of the territory of Australia has an unsuitable climate for apple leaf curling midge. And it's only in these very southerly areas where there's adequate rainfall, that it poses a risk. And that clearly should have quite a big effect on the risk assessment.

William Ehlers
636. I think Ms Hillman will take it up now.

Chairman
637. May I ask of the Parties, because we are now at 7 p.m. Do you have a number of questions to Dr Cross? We have another four. One question from New Zealand. Would you have many questions? I am just wondering whether we could spend a little bit more time and just finish it. The alternative is to come round tomorrow morning. I understand New Zealand has one question. We have another four or five questions.

Australia
638. In that case, Mr Chair, we may well be able to finish in another half an hour. I don't think we have very many on our side.

Chairman
639. Are you willing to continue for another half an hour? Seems so. Are we happy to continue? I should just also note, remember there is a final section, called "Other". Do you have other questions, beyond this area, because we'd just have to check whether… We don't, but you might. And New Zealand says no.

Australia
640. We may have one or two, but that would be the maximum.

Chairman
641. Shall we give it a shot?

Australia
642. I'd like to, if that's all right with New Zealand.

Chairman
643. Dr Cross, are you satisfied if we continue for another half an hour?

Kirsten Hillman
644. This is a question regarding Question 115. This question addresses the IRA's consideration of the likelihood of entry and establishment of ALCM with respect to whether adult emergence from diapause may take place in seasons other than spring. Now, Australia has interpreted your response to that question as largely confirming – and that's a quote from Australia's comments – its position "in respect to suitable environmental conditions and emergence". Do you agree that your answer to that question largely confirms Australia's position?

Dr Cross
645. Clearly, if apples are brought into an environment, apples which have cocoons in them, that contain post-diapausing larvae, are brought into warm conditions, indoors, in a supermarket, in a house, or whatever, in winter, or at times when apple trees are not growing out of doors, clearly the apple leaf curling midge could emerge fairly quickly. If apples were introduced into Australia in the winter in the outdoor environment, the conditions wouldn't be suitable for emergence at that time, but they would be subsequently, when the conditions warmed up, the cocoons would still be there subsequently. So can you just repeat exactly what you want from me?

Kirsten Hillman
646. I think you may have… et's just have a look at Australia's comments… Australia's comments actually don't go into very much more detail than that, but the question had to do with the likelihood of entry and establishment of ALCM through mature apple fruit in seasons other than spring. For example, if the cool chain is broken and day length and temperatures replicate spring, such as in a controlled environment like a supermarket or a packing house, etc.

Dr Cross
647. Yes. I agree that that would occur if, you know, that could definitely occur in winter, the conditions for emergence could occur. Or the fruit may be disposed of, and they would occur later, if the fruit was disposed of out of doors, then when spring came along the cocoons would still be there and they could emerge then.

Kirsten Hillman
648. They could emerge, okay.

Dr Cross
649. They don't necessarily die after a short period. They can hang on for a long time in cool conditions.

Kirsten Hillman
650. That answers our question. Thank you. The next question would be in respect of your response to question 117, where you stated that Australia's IRA was objective and credible in its treatment of climatic conditions for the spread of ALCM in Australia, but then you identified some shortcomings. Both Parties argued in their comments that your reply supported their respective positions. So we'd be interested in your views on your answer.

Dr Cross
651. I think I've already sort of answered this. Obviously, apple leaf curling midge will only spread and cause a problem in areas of Australia where the climate is suitable for its existence. And it's only going to be successful where the climate is well suited. I mean, there may be marginal areas where it can sort of hang on, but not do very well. And I think that those areas have not clearly been established, these geographic and climatic boundary conditions haven't been established, of where exactly in Australia this could occur. As I said, this business of latitudes less than 38 degrees in Europe, the climate is generally too warm for it. That needs to be taken into account in the analysis. And I think that that's what I gave in my reply to 117. And I think I spelt out earlier.

Kirsten Hillman
652. Thank you, Dr Cross. I think that's actually very helpful.

Australia
653. Sorry. Ms Hillman, would you mind if Australia asked a question on that point.

Kirsten Hillman
654. Not at all. Please, Australia.

Australia
655. Professor Cross, you have indicated that there may have been some shortcomings in the IRA in respect of climate and spread, but in your answers you did indicate that Australia's IRA on this point was objective and credible, and you concluded that the overall assessment is correct. Is that still your position?

Dr Cross
656. I may have … Yes, there's a difference between agreeing with the conclusions based on the methodology used, and thinking that the methodology itself was correct. I wasn't, perhaps, sufficiently aware; I didn't comment or I didn't consider, I think there's some instances where using the methodology used, the conclusions seem to be reasonable, but I question whether the actual methodology was correct overall. And I think in one or two cases I may have come to the conclusion that, using the methodology, the conclusions drawn were correct, but maybe the methodology used was not really itself quite correct. Perhaps I ought to come to an example of this. A good example would be the impact of apple leaf curling midge, where I think the IRA considered that the impact would be severe, or not severe, highly significant locally, and this led to a certain score being drawn, a C-score if I remember correctly, yet if the conclusion was different and a D-score was drawn, the overall conclusion reached would be the same because the methodology used didn't allow for, didn't reflect differences adequately in, the methodology didn't reflect these differences very well. I haven't explained myself very well. But there could be instances where the methodology used – I concluded that using that methodology, the conclusion was good, but maybe I didn't sufficiently draw that there were areas where the methodology perhaps wasn't taking in the factors into consideration properly.

Chairman
657. Okay. Dr Cross, in your response to Question 96, in the penultimate paragraph you write that the effects of ALCM infestation on skin finish or fruit quality are rare, and that the type of damage reported from New Zealand is extraordinary. Could you explain the word "extraordinary"? We weren't quite sure what you meant by that.

Dr Cross
658. I have seen photographs – I think quite old photographs of damage – where very heavy infestations of apple leaf midge that occurred during blossom led to some distortion or effects on the fruit quality. I've never seen or heard of that occurring anywhere else. And I think it's a highly unusual event. Certainly it does not normally occur in Europe, where the apple midge has been

around for a long time. So I think that effects of the apple leaf midge on fruit quality would be pretty unusual. And I don't know whether it's been recorded in New Zealand in recent years at all. It may be varietal as well.

Chairman
659. So, the sense you are using it in, it's unusual, extraordinary in the sense of unusual, rare to occur.

Dr Cross
660. Yes.

Chairman
661. Well, I'd like to come to the Question 120. There is a disagreement among the Parties as to the correct interpretation of your response to Question 120 on the inspection of a 600 versus a 3,000 fruit sample from each import lot. There are two questions here. One, does your response imply that in your view Australia could achieve its appropriate level of protection through another measure that would be less trade restrictive than requiring a 3,000 fruit sample from each lot, with a detection resulting in mandatory treatment or rejection for export? And, secondly, does, in your view, the alternative measure, i.e. the inspection of a 600 fruit sample from each import lot achieve Australia's appropriate level of protection, either by itself or only in combination with other measures?

Dr Cross
662. Without recalculating the risk assessment incorporating these additional factors, it is very difficult to know what sort of size of population would lead to a risk, but exceeds the ALOP. So I think, in order to answer that question, the IRA needs to be sort of recalculated to determine what size of population, what instance of occupied cocoons would pose a risk above the ALOP and then the sample size should be adjusted to address that risk.

663. So, I mean, if a 600-fruit sample showed that there is a 95 per cent chance of the 0.5 per cent of fruit being infested, if half of those fruit are non-viable, another 30 per cent parasitized and, in any case, there is only a 1 in 5 chance that the male and female would mate and meet each other in their life, then that effective infestation level is reduced by a factor of 10, 20, 30 times, whatever the factors are that are determined by doing this risk assessment and then that needs to be applied to the sample sizes that are needed. And I find it difficult to prejudge what those sample sizes should be. Clearly one factor that I believe would make quite a big difference to the risk is the mode of trade question. If the apples were retail-ready, ready-packed in smaller packs, that were not handled at the seven wholesalers, that would greatly reduce the risk, maybe that would overcome the risk all together, but until that calculation is redone, it's difficult to decide what the sample size should be. 3,000 fruits, to inspect 3,000 fruits is a big requirement and would be, I think, quite a restriction on the possible trade. It would be very difficult to do, in my view – expensive to do, if it was done for every orchard.

Chairman
664. Let me ask you our ultimate question. And maybe it has been responded to, but in a more general sense. Assuming that the semi-quantitative method could be considered a legitimate methodology, and you have had some comments about that in one or two of your questions, do you believe that the way that the IRA applied this risk assessment methodology to ALCM is objectively justified?

Dr Cross
665. Could you repeat the question again, so that I can just think about it a bit more.

Chairman
666. Sure. More in a sort of overall sense, if one assumes that the semi-quantitative method can be considered a legitimate methodology. Okay. If that's the case, do you believe that the way in which it was applied is, as it has been applied to ALCM, is objectively justifiable?

Dr Cross
667. I think the general methodology seems to be okay, with some problems in it. And if it's applied, including the different factors that haven't been included properly, I believe it will give a better and more objective assessment of the risks and should then lead to a good consideration of what measures would be needed to reduce or mitigate those risks.

Chairman
668. Fair enough. That exhausts our questions. Not the Panel, but the questions from the Panel. I saw no one wanting to specifically latch onto any of our questions, but certainly New Zealand if you have any questions, please you are welcome to pose them.

New Zealand
669. We just have one question and it does actually relate to one of the earlier questions put, it's a follow on.

670. We just have one question, which relates to your response to 94(v), where you state that "A weakness in the IRA is that Australia failed to quantify (or at least delimit) the geographic range and range of conditions which are necessary for establishment and spread of ALCM, both in terms of temperature and rainfall and their seasonal occurrence. The geographic and climatic limits were not established." Can you confirm that is still your position?

Dr Cross
671. Yes. I haven't yet seen a climatological analysis in relation to where apple leaf curling midge could exist in southern Australia. I don't think the delimiting parameters have been established. What is the winter chill requirement? It could be something like days or hours below 5 degrees centigrade. I don't think that has been established and I don't think the number of days of rainfall, or maybe even something like monthly seasonal totals of rainfall, I don't think that those parameters have been established. And then, based on that, where those necessary conditions occur for apple leaf curling midge to exist in Australia. I realize that any assessment is probably going to be a crude one, based on where the midge occurs elsewhere in the world. But I haven't seen that analysis, an attempt done at that analysis. And I think that that's quite an important thing to do for the risk assessment.

Chairman
672. Satisfied, New Zealand? Australia.

Australia
673. Australia just has four questions on ALCM if everybody wouldn't mind staying a little bit longer. My colleague, Mr Heinrich, will ask the first question.

674. Dr Cross, in your written response on ALCM, you commented that there is significant lack of data in respect of a number of key matters. Do you think it's fair to stay that there have been relatively few studies and reported reliable data on the ALCM generally, in comparison to many other pests of phytosanitary concern?

Dr Cross
675. It is surprising that this creature is being the subject of this concern between the Parties and that more good quality studies haven't been done. For instance, in the 2005 data it would have been very good if we'd had some data on the viability of those larvae. We've only had the Rogers study on

viability. We have no study on the flight range of females. There's opportunity to do a great deal more good quality studies to elucidate these factors. And a lot of this would help take some of the guesswork out of the analysis. So yes, I agree. I mean, some insects have been quite extensively studied, in depth. Apple leaf curling midge is not one of those. And it's probably been less well studied than some of the other similar members of the same genus, such as the Brassica pod midge or some of the other species.

Australia
676. I have a question in relation to the IRA's evaluation of importation steps 1 to 8 and the August 2005 data. Do you agree that the IRA team effectively arrived at two entirely separate conclusions as to the unrestricted risk on the basis of its two estimates for probability of importation? That is, one based on the analysis of importation steps 1 to 8. And the other based on the August 2005 data provided by New Zealand. And if you do agree, therefore, that it made no difference to the overall outcome, that the IRA team chose to retain its analysis of the older published data set out in importation steps 1 to 8, given that Australia's 3,000 unit measure is ultimately based on the August 2005 data conclusion.

Dr Cross
677. I do appreciate that the IRA did consider those two different data sets. I find it slightly surprising, or rather surprising, that the conclusions reached were broadly the same given the fact that the parameters, I think, varied by a factor of 30-fold in the central infestation level in the two considerations. So yes, I appreciate that was done. Could you repeat the latter part of your statement/question?

Australia
678. I think you've mostly answered the question, but my question related to the fact that the measure that was ultimately selected by the IRA team, well, its decision that a 600-unit inspection would not be sufficient, was based on its analysis of the August 2005 data and the infestation level that that data provided, as opposed to the infestation level that they estimated on the basis of importation steps 1 to 8.

Dr Cross
679. Yes, I appreciate that. But neither analysis took into account adequately the viability, the parasitism, or the extended period of emergence relative to the short life span, and some other factors as well.

Australia
680. Now my colleague, Dr Barry, has some questions for Dr Sgrillo.

681. Dr Sgrillo. I'd like to ask you a question about determining an appropriate inspection rate for a consignment in circumstances where it has been determined that the unrestricted risk is above the appropriate level of protection, and therefore that risk management measures are required. Would you agree that the relevant factor for determining the appropriate rate of inspection for ALCM is the underlying level of infestation?

Dr Sgrillo
682. Yes, I agree. The infestation will tell you how much you have to decrease this infestation to reach your appropriate level of risk. And also how sensitive has to be your inspection system to catch any infestation above this level.

Australia
683. Sorry, one more question, for Dr Sgrillo again. Would you agree that in order to have confidence in detecting a pest within a consignment that has low levels of underlying infestation, a

higher rate of inspection may be required, compared to a consignment which had a higher level infestation, if so could you explain why?

Dr Sgrillo
684. Usually, considering the same population size, if the infestation is higher you have to sample less, because … I mean, if you have more units infested in the population, this will require a smaller number of samples to catch this infestation. However, if you have a very low number, a very low per cent of infestation, you have to increase your sample size to catch it.

Australia
685. And when you're doing the sampling, what is the standard statistical level of confidence you would consider appropriate when selecting an inspection rate? Is there a typical level of confidence that is typically used?

Dr Sgrillo
686. I'm not sure if in the IPPC Standard on Inspection Methodology it is specified. I would say that the majority of service that use, I mean a plant sampling technique, will use 95 per cent of confidence level. But I'm not sure if this is an international standard or not.

Australia
687. That's all of Australia's questions on ALCM.

Chairman
688. New Zealand.

New Zealand
689. Just got one final question, and this is a question for Dr Cross. In your response to Question 120, on page 22, you state that, in determining the appropriate measure for ALCM, "the sample size should not be adjusted to fit the infestation rate which appears to be the case in the current analysis. It should be set to meet Australia's ALOP." Can you confirm that this is still your position?

Dr Cross
690. Yes, I think it is. Logically, to me, it seems that Australia wants to ensure that the amount of apple leaf curling midge on the lots of apples that are coming from New Zealand have lower than its ALOP. And so the sampling should be devised to meet that requirement and not devised to detect the pest on the majority of New Zealand apple samples, which is a different process.

Chairman
691. All right. Done with ALCM? Well done. "Other". I think Australia… or have you included your other already in that batch of questions?

Australia
692. We have one question. And if I can just go briefly back to the risk assessment process. And this is just a question for Dr Schrader. Just wanted to ask, would it be fair to say – and this is taking into account ISPM No. 11 – would it be fair to say that, as part of the risk assessment process, there is no need to take into account or assess the risk for a pest or disease that does not occur in the geographical area for which the risk assessment has been conducted?

Dr Schrader
693. So you mean, whether you should not assess the risk of an organism which is not present in the area you are assessing? No, of course not, because there is no need to do that.

Australia
694. We have no other questions on this.

Chairman
695. It would seem to me that we have reached the end of today's meeting. And I think what certainly – certainly from the Panel's perspective, and I'm sure also from the Parties – today has been very enlightening and has assisted us, I believe, greatly. And it remains, therefore, I think, from the Panel's side, just to thank the experts for the work that they have done up until this point and also today engaging with us. I think it has been certainly very, very useful. And we'd like to thank you for being here. And for the Secretariat and the Parties for continuing until now. I think it has been useful to actually conclude this session today. It will allow us a little bit of preparation for what happens tomorrow. And if I understand it correctly we'll be in this room tomorrow at 3 p.m. for the second meeting with the Parties. With that, the meeting is adjourned.

Annex 1

Corrections Suggested by Experts

Paragraph 103, Dr. Schrader:

"Pest free area" should be replaced by "pest free place of production", as this would be the correct term.

Paragraph 581, second sentence, Dr. Cross:

This is a mistake. Actually the Sandanayaka and Rogers (2009) data appended in NZ 2nd submission do not show that cold storage kills pupae. The apples were artificially infested with mature larvae then exposed to 10 weeks + cold storage. There were no pupae. It shows that adult emergence is delayed by at least 13 days after cold storage.

Paragraph 589, Dr. Cross:

The first sentence should read: "I think we are fortunate there is some data on the incidence of apples that are infested with the apple-leaf curling midge cocoons that are occupied by larvae or pupae." Otherwise the sentence and the whole section don't really make sense.

Paragraph 654, last sentence, Dr. Cross:

I did not explain this at all well and my answer is perhaps confusing. What I was trying to explain is that I assumed that the overall methodology was a recognized /generally accepted one, and the conclusions drawn were reasonable. But there were clearly problems with the overall methodology.

Paragraph 688, Dr. Cross:

The last sentence should read: "And so the sampling should be devised to meet that requirement and not devised to detect the pest on the majority of New Zealand apple samples, which is not what is required."

Annex 2

Slides Shown by Australia at the Panel's Meeting with the Experts

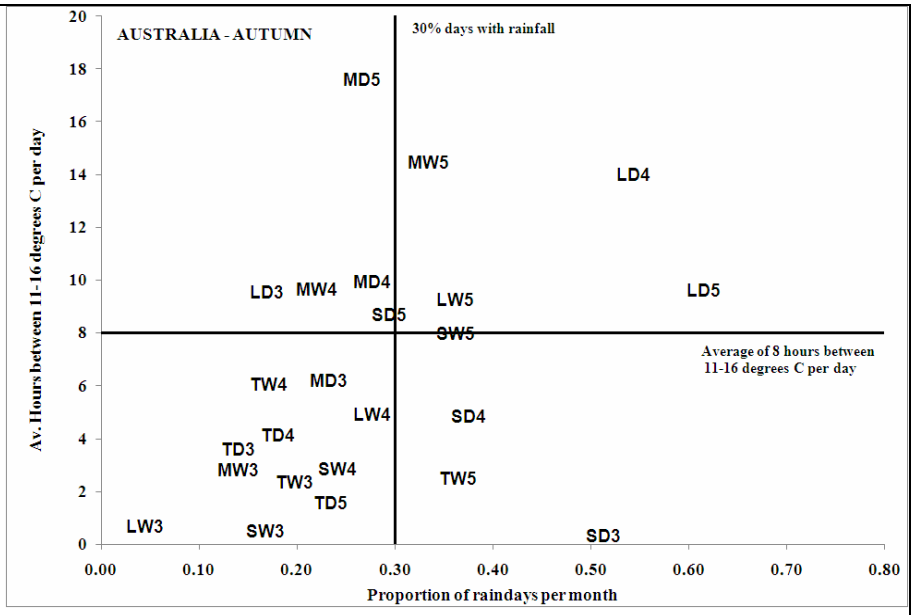


Figure 6 Australian autumn (March, April, May) data for wettest and driest years between 1998 and 2007.

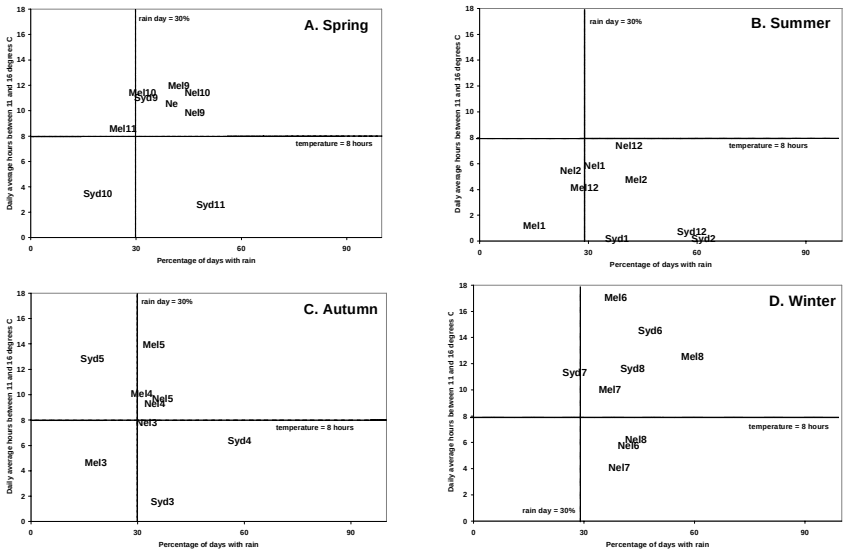


Figure 10. Climatic suitability for European canker establishment in Melbourne, Sydney and Nelson using the rainfall and temperature criteria cited by Beresford and Kim (derived from Dubin and English 1975). The first three letters of each plotted point represent the locality name (Mel = Melbourne, Syd = Sydney, Nel = Nelson) and the numeric postfix represents the month of the year (where January is '1') for: A. Spring; B. Summer; C. Autumn; and D. Winter.

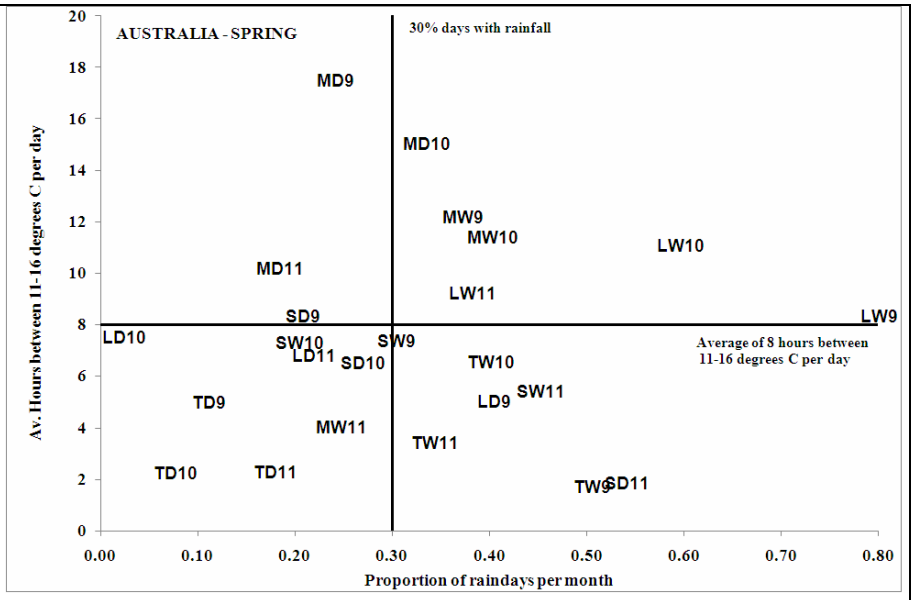


Figure 8 **Australian spring (September, October, November) data for wettest and driest years between 1998 and 2007**

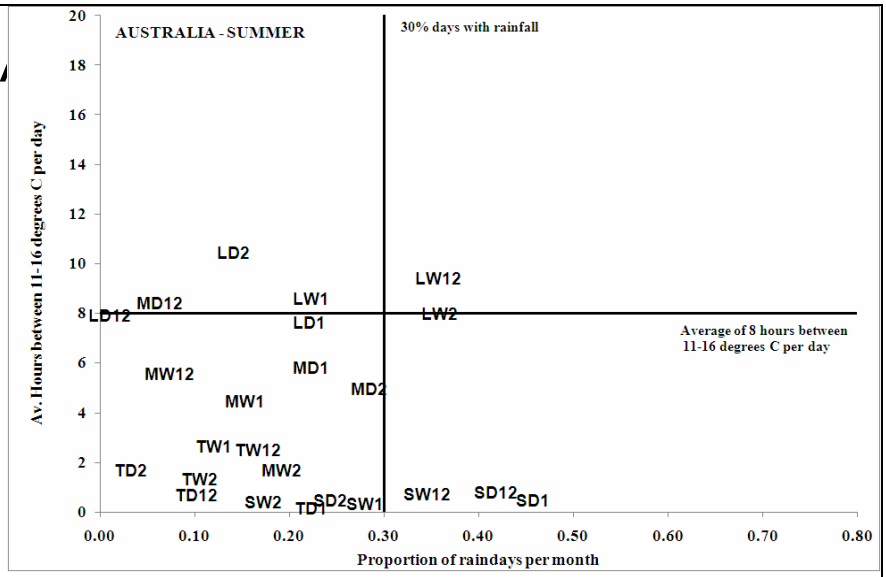


Figure 10 **Australian summer (December, January, February) data for wettest and driest years between 1998 and 2007**

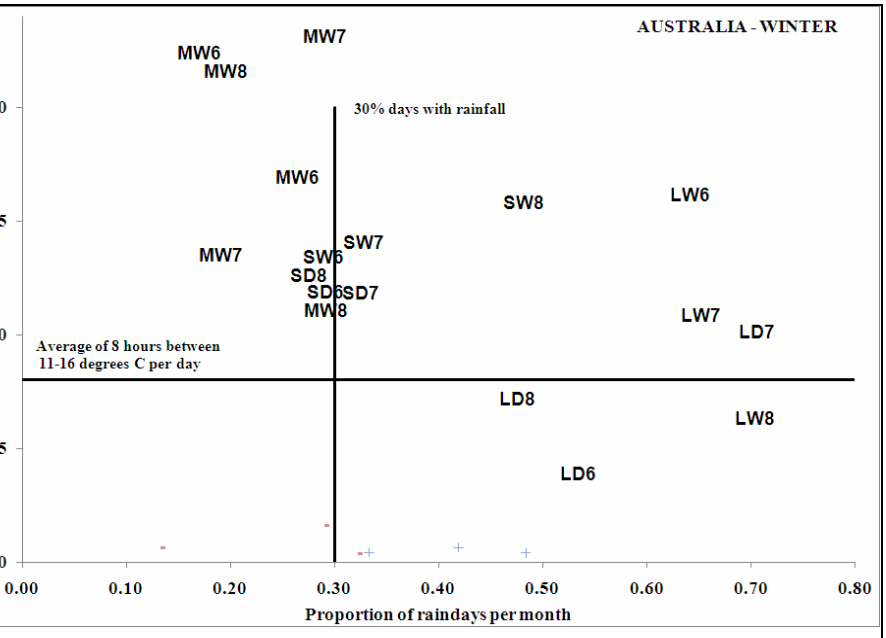
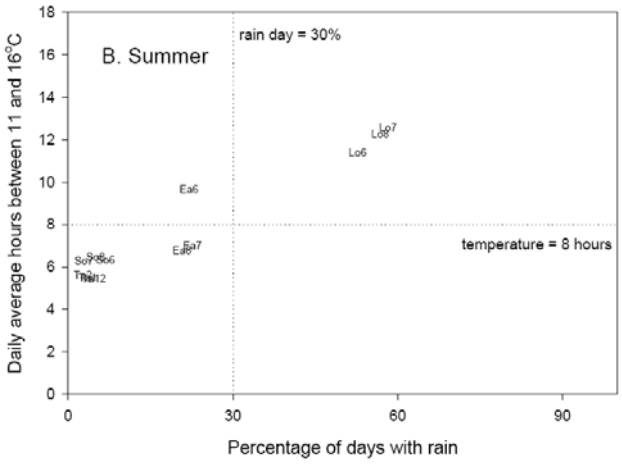


Figure 12 **Australian winter (June, July, august) data for wettest and driest years between 1998 and 2007**

Figure 3. Climatic suitability for European canker development in Chilean, United Kingdom and Californian localities shown in Appendix 2 using the rainfall and temperature criteria derived from Dubin and English (1975). The first two letters of each plotted point represent the locality name and the numeric postfix represents a month during A) spring, B) summer, C) autumn, and D) winter. Analysis of published studies showed that simultaneous occurrence of >30% of days with rain and >8 hours per day between 11°C and 16°C represent high risk of European canker infection. Ea = East Malling, Lo = Loughgall, So = Sonoma, Ta = Talca



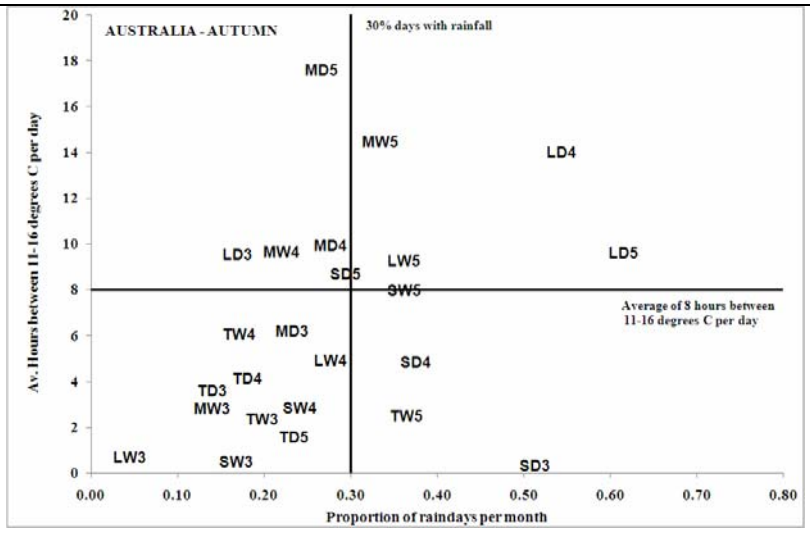


Figure 6 Australian autumn (March, April, May) data for wettest and driest years between 1998 and 2007 for two major trade centres (Sydney and Melbourne) and two major commercial pome fruit regions (Lenswood and Tumbarumba, near Batlow)). Sydney wettest = SW, Sydney driest = SD, Melbourne wettest = MW, Melbourne driest = MD, Lenswood wettest = LW, Lenswood driest = LD, Tumbarumba wettest = TW, Tumbarumba driest = TD; month of the year for each location indicated by numeric postfix (eg. Tumbaruma's driest March between 1988 and 2007 is 'TD3').