WORLD TRADE ORGANIZATION

WT/DS367/11
9 August 2010

(10-4164)

Original: English

AUSTRALIA – MEASURES AFFECTING THE IMPORTATION OF APPLES FROM NEW ZEALAND

Replies from the scientific experts to questions posed by the Panel[*]

In accordance with the Procedures for the Panel's meeting with the experts and Parties and the Panel's second substantive meeting, adopted by the Panel on 11 June 2009, the list of replies from the scientific experts to questions posed by the Panel will only be available in electronic format.

[*] The List of replies from the scientific experts to questions posed by the Panel is initially available in its original language. A translation to French and Spanish will follow when resources become available

ANNEX B-1

I. GUIDELINES FOR EXPERTS

Guideline (a)

You may familiarise yourself with the case by getting an overview of the issues at hand and the scientific arguments and materials provided by the Parties. Relevant background documents include the Parties' submissions, statements and responses to the questions posed by the Panel, as well as the exhibits attached to these. To get an overview of the scientific materials provided by the Parties, please check the summary exhibit tables in the relevant submissions. Australia's Final Import Risk Analysis Report for Apples from New Zealand is available in Exhibits AUS-1, AUS-2 and AUS-3, and in part in Exhibit NZ-1. A good understanding of all of these materials is essential. To facilitate your work, the Panel has included, at the end of every question, an indicative list of references to relevant parts of Parties' submissions, replies and exhibits. This list is not exhaustive and the Parties may have included other information in their submissions, which they consider relevant. If you have any questions regarding the background documents, please contact the Panel Secretary, Serra Ayral (serra.ayral@wto.org).

Guideline (b)

As explained in the "request for experts" letter sent to you by the WTO, the questions relate to the following four issues:
Erwinia amylovora (fire blight), including its potential spread through trade in apples and the phytosanitary measures to be applied to control its spread;
Neonectria galligena (European canker), including its potential spread through trade in apples, the climatic conditions for its establishment, and the phytosanitary measures to be applied to control its spread;
Dasineura mali (apple leafcurling midge), including its potential spread through trade in apples and the phytosanitary measures to be applied to control its spread; and
Pest risk assessment, including the use of semi-quantitative methodologies.

Guideline (c)

In drafting your replies, please answer only those questions that you feel competent to answer. Please provide citations and references to the scientific evidence and literature on which you base your answers.

Guideline (d)

The list of questions starts with some general, introductory questions. The other questions are organized according to the relevant pests (i.e., fire blight, European canker and apple leafcurling midge). Some questions on risk assessment are separated into a special part at the end of this document; some other questions also related to risk assessment are listed under the relevant pests and marked with an asterisk.

Guideline (e)

The questions are numbered consecutively throughout this document. Please identify the number of the relevant question in your response.

Guideline (f)

Please remember that the three panellists serving on the case have limited background in the specific scientific issues raised in this dispute, and need your help to digest the extensive scientific material submitted by the Parties. Therefore, please provide your answers, to the extent possible, in terms that are concise and may be understood by non-experts, in order to better clarify the issues at hand and assist the Panel in reaching its legal findings.

Guideline (g)

New Zealand contests the conclusions of various steps in Australia's Final Import Risk Analysis Report for Apples from New Zealand, as indicated or referenced below. The Panel will need the experts' assistance in order to:
Identify and elaborate on the scientific basis upon which the challenged measures contained in Australia's IRA were adopted. When referring to scientific sources that are not contained in the IRA, please explain how do those relate to the scientific sources that are contained in the IRA or to the measures identified in the IRA;
Determine whether the relevant analysis in Australia's IRA is based on respected and qualified scientific sources, in terms of both the author(s) of, and the arguments made in, such sources (Please note that the scientific basis relied upon by the IRA need not reflect the majority view within the scientific community but may reflect divergent or minority views as long as it has the necessary scientific and methodological rigour to be considered reputable science.);
Determine whether the reasoning articulated by the IRA on the basis of such scientific evidence, including the methodologies applied, is objective and coherent, and whether the particular conclusions in the IRA find sufficient support in the scientific evidence relied upon; and
Determine whether the results of the IRA's assessment sufficiently warrant the challenged measures.

Dr Latorre:

1. Australia's measures contained in the Import Risk Analysis (IRA were based on the biology of *N. Galligena* and on current knowledge of the epidemiology of European canker on apple. The scientific information is discussed in AUS-2BA (p.117). According to the Australian government, there is a risk that *N. Galligena* will enter the country on asymptomatic mature fruits. These fruits may be infected in the orchard, remaining asymptomatic (latently infected fruits) for several weeks in cold storage. This conclusion has been based on by previous reports from France and the United Kingdom (Bondoux and Bulit, 1959; Swinburne, 1975; McDonnell, 1970), indicating that a proportion of apparently healthy fruits develop symptoms after cold storage. The phenomenon has also been observed in other apple-producing countries, in fruits from infected orchards in areas with frequent summer rains.

2. Therefore, there is a risk of the entrance of *N. Galligena* associated with asymptomatic (= symptomless) fruits carrying latent infection. However, latent infections would be extremely unlikely in apples from orchards free of European canker in the absence of summer rains. Under these circumstances, the risk of latent infection is close to zero (in practice zero). Fruits with latent infection cannot be differentiated from healthy fruits at harvest.

3. The IRA conducted by Australia was performed in accordance with today's concepts and knowledge of plant diseases. As stated in AUS-2 BA (p. 40), risk is a function of the likelihoods of an event occurring (entrance, establishment and spread) and the consequences or impact resulting from the occurrence of such biological events. Therefore, there is no criticism, in qualitative terms, with regard to the Risk Estimation Matrix presented in Tables 1 and 11 (AUS-2 BA, p.4 and p.41, respectively), where the appropriate level of phytosanitary protection (ALOP) was set at **very low risk** (but not zero).

4. However, there is no scientific evidence substantiating the probability intervals and midpoints used for the semiquantitative analysis described in Table 12 (AUS-2 BA, p. 43). Comment: (i) Using midpoints (averages of wide likelihood ranges) tends to overestimate the occurrence of a given biological event. (ii) It is difficult to accept that a midpoint probability of 0.175 would be equal to low risk (Table 12); indeed, this is a very high probability for any biological event associated with *N. Galligena*. (iii) If negligible is defined as a probability varying between 0 and 10^{-6}, using the midpoint 5×10^{-7} appears to overestimate the likelihood of all biological events approaching zero, particularly if such an event has a remote possibility of occurring.

5. Therefore the overall probability of entrance, establishment and spread of *N. Galligena* was 7.0×10^{-2}, which was rated as low (Table 37, AUS-2 BA p. 150). Indeed, this a very high, rather than low, probability for any biological event associated with *N. Galligena*. If this likelihood value is true, and assuming that market penetration in Australia is equal to 50,000,000 apples annually (AUS-2 BA, p. 19), *N. Galligena* should be present in 3,500,000 apples (7%) annually, which is non-credible. Therefore, the overall probability (7.0×10^{-2}) should be validated before acceptance. Data validating the probability values given in Table 12 were not presented.

6. Nevertheless, 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, the following phytosanitary measure appears reasonable for mitigating the risk of entrance:

> "The requirement that apples be sourced from export orchards/blocks free of European canker (pest-free places of production)."

7. Other proposed measures appear to be auxiliary (ancillary) or unnecessary.

Guideline (h)

Wherever applicable, please formulate your response with regard to "mature apple fruit free of trash, either packed or sorted and graded bulk from New Zealand" (IRA, Part B, p. 9), and indicate whether your response would be any different in the context of (i) "mature, symptomless apples" from New Zealand (New Zealand's First Written Submission, para. 3.44) or (ii) "apples imported from New Zealand" to Australia in general (WT/DS367/5). For questions relating to apples from a specific WTO Member other than New Zealand, please similarly provide separate responses with regard to (i) mature apple fruit free of trash, either packed or sorted and graded bulk; (ii) mature, symptomless apples; and (iii) apples in general.

Guideline (i)

Where applicable, please specify whether there has been any development in the relevant science subsequent to the time of adoption of the IRA in November 2006, and if so, please explain.

Guideline (j)

The following abbreviations are used in this document:
ALCM – apple leafcurling midge;
ALOP – appropriate level of sanitary or phytosanitary protection;
AQIS – Australian Quarantine and Inspection Service;
FWS – First Written Submission to the Panel of a Party to the dispute;
IRA – Australia's Final Import Risk Analysis Report for Apples from New Zealand (November 2006);
ISPM – International Standards for Phytosanitary Measures;
Opening statement – opening statement at the Panel's first substantive meeting;

R – response by Parties to questions posed by the Panel.

Guideline (k)

The deadline for providing your replies is Monday, 23 February 2009.[1]

I. INTRODUCTORY QUESTIONS

Question 1
Is there a commonly accepted definition or criterion (biological, physiological, commercial, etc.) for determining if an apple fruit is mature? Is there a distinction between physiological and commercial maturity?

Dr Deckers:

8. Yes, the final picking date is determined by the evolution of different fruit maturity factors like starch degradation in the fruits, fruit firmness, fruit colour (red colour formation or the green background colour development) and the soluble solids (Brix). These different parameters are related to each other and should reach at the beginning of harvest specific and variety dependent values. These values can be put into a formula in order to calculate the so called maturity index or Streif index where the formula is F/(R*S). For each variety there is a maturity index on which the harvest would start and a maturity index at which the harvest would end. Fruits are harvested in the preclimacteric phase and in fact fruits are mostly harvested as immature fruits that still have a good storage capacity. The fruits should be ripe at the time of the final consumption with a full development of the flavour at fruit maturity.

Dr Latorre:

9. Physiologically, apple fruits are mature (physiological maturity) at the stage of development when fruits will continue ontogeny even if detached. Commercial maturity indicates the stage when fruits have developed all their qualities (attributes) and are ready to eat. Therefore physiological and commercial maturity are not synonyms.

10. Yes, there is an accepted criterion to determine apple maturity, which can be determined on the basis of starch content, firmness, juice sugar and acid content, seed color, flesh color, background color, and internal ethylene concentration. For the best storage results, apples must be harvested at their physiological maturity, just before the onset of the climacteric rise (commercial maturity). Although the best way to determine the optimal harvest period is by monitoring the respiration rate, in practice, this is estimated by the starch content (iodine index), background color, solid solubles, and/or firmness. These maturity indices have been adjusted to reflect the particularities of the main apple cultivars. In addition, days after full bloom can be used as a general guide to estimate fruit maturity.

Dr Paulin:

11. There are several accepted definitions and criteria for determining if an apple is mature. Several simple tests exist (such as starch hydrolysis, as detected on halves of fruit by the iodine

[1] As a result of the amendments to the timetable made by the Panel on 2 February 2009, the deadline given to experts to provide their written replies to questions was extended to 9 March.

reaction, which allows the determination of a maturity stage). Maturity itself can be defined in relation with cropping-time (to determine when a fruit is best picked up), or with trade (when the fruit can be best shipped and sold), organoleptic (when the fruits are at their optimal eating quality). All these correspond to different stages of physiological maturity. Usually, for apple, the commercial maturity occurs before the optimal physiological maturity (stage where the fruit is at its best flavour and taste quality, before decaying).

Dr Schrader:

12. A commonly accepted definition or criterion for determining if an apple fruit is mature is the Streifindex. According to this method developed by Dr Streif, the degradation of starch (determined with Lugol's solution), the firmness of fruit flesh and the content of sugar is combined ([firmness/ (percentage soluble solids concentration x starch index)]. The practical utility of the Streifindex method lies in the ease with which apple fruit maturity at harvest can be evaluated for its suitability for long-term storage (e.g. Delong, J. M., Prange, R. K., Harrison, P. A., Schofield, R. A., Deell, J. R, 1999, "Using the Streif Index as a final harvest window for controlled-atmosphere storage of apples", *Hort Science* 34 (7), pp. 1171-1191 (26 ref.), pp. 1251-1255).

13. The table below lists values of firmness, sugar content, degradation of starch and the Streifindex for the orientation for harvest for different apple varieties (table from Höhn, E., Dätwyler, D., Gasser, F., Jampen, M. (1999) "Streifindex und optimaler Pflückzeitpunkt von Tafelkernobst, Schweiz" Z. *Obst-Weinbau* (18/99), pp. 443 – 446).

Ernterichtwerte für die Fruchtfleischfestigkeit, den Zuckergehalt, den Stärkewert und den Reifeindex nach Streif für Tafelkernobst.

Sorte	Fleischfestigkeit Penetrometerwert (kg/cm^2)	Zuckergehalt Refraktometerwert (°Brix)	Stärkeabbau-wert Jodtest (1–10)	Reifeindex (nach Streif)
Äpfel				
Arlet	7,0 – 8,0	12,0 – 13,0	5 – 6	0,11 – 0,13
Boskoop	8,0 – 9,0	11,0 – 12,0	4 – 5	0,15 – 0,20
Braeburn	8,5 – 10,0	10,0 – 11,0	4 – 5	0,16 – 0,22
Cox Orange	8,5 – 10,0	11,5 – 12,5	4 – 5	0,18 – 0,24
Elstar	6,5 – 8,0	11,0 – 12,5	3 – 4	0,17 – 0,30
Florina	7,0 – 8,5	11,5 – 13,0	7 – 8	0,06 – 0,08
Gala	8,5 – 10,0	10,0 – 12,0	5 – 6	0,14 – 0,20
Glockenapfel	9,0 – 10,0	11,0 – 12,0	4 – 6	0,14 – 0,16
Gloster	8,0 – 9,0	11,0 – 12,0	2 – 4	0,24 – 0,40
Golden Delicious	7,0 – 8,0	11,5 – 13,0	6 – 7	0,09 – 0,12
Gravensteiner	8,0 – 9,0	11,5 – 12,5	8 – 9	0,10 – 0,14
Idared	7,5 – 8,5	11,0 – 12,0	2 – 4	0,25 – 0,35
Jonagold	6,5 – 7,5	11,5 - 13,0	7 – 8	0,07 – 0,08
Jonagored	6,5 – 7,5	11,5 - 13,0	7 – 8	0,07 – 0,08
Maigold	8,0 – 10,0	11,5 – 13,0	3 – 4	0,16 – 0,22
Rubinette	7,0 – 8,0	12,0 – 13,0	4 – 5	0,10 – 0,13
Birnen				
Comice	4,5 – 5,5	13,5 – 14,5	7 – 8	0,04 – 0,06
Conférence	6,0 – 7,0	11,5 – 13,0	4 – 6	0,10 – 0,13

Dr Swinburne:

14. The term "mature fruit" used in the context of both FWS appears to refer to the stage at which fruit is ready to be picked, not to any subsequent ripening process.

15. The physiological processes involved in the ripening of fruit such as apple are well understood and consist of a number of stages, beginning with the appropriate time to harvest. The optimum time of picking is generally judged to be when the fruit have achieved maximum colour and can be readily detached (abscission layer formation), perhaps augmented by using an iodine test to ascertain when starch has largely disappeared (converted to sugar). Fruit at this stage can be referred to as "mature", however with many cultivars this would not be commensurate with optimum eating quality as the ripening process would still be incomplete. Moreover, fruit of many cultivars at this stage retain a measure of resistance to fungi responsible for rotting.

16. Apples, along with other fruit such as banana, undergo major changes post-harvest, characterised by the onset of an increase in respiration (the climacteric), triggered by the production of the hormone ethylene in the form of a diffusible gas. At this time cell walls start to become softened, the levels of soluble sugars increases whilst those of acids decrease. Volatile compounds

associated with flavour are at their highest levels following the climacteric. This is the optimum stage for eating (ripe), both for flavour and texture, but perhaps rarely achieved in commerce. The onset of the climacteric can also herald an increase in susceptibility to rotting by a number of fungal pathogens, including *N. Galligena*.

17. In the following stage respiration rates steadily diminish and wall softening continues leading to loss of texture. Acids and volatiles also decline, adversely affecting flavour and the fruits lapse into senescence (over-ripe) and become unmarketable. Fruit frequently reaches this later stage in the domestic environment (i.e. after retail sale).

18. Post harvest technologies are designed to extend the marketing period of fruit. Of these the most obvious is cold storage, which reduces respiration rates at all stages. More sophisticated methods have in addition the aim of reducing the availability of oxygen within the store, either by simply sealing the chamber to exhaust supply, or by flushing with nitrogen. These affect both pre- and post climacteric changes. Scrubbing the atmosphere of ethylene delays the onset of the climacteric. The degree of sophistication applied to store conditions is usually in accordance with plans for marketing, the most complex being reserved for the longest held.

19. In commerce fruit is usually sent to market in a continuous stream following harvest, subject to quality controls imposed by either the buyer (e.g. supermarket chains), and/or the producer. This will involve storage periods of varying length. Thus it is to be expected that fruit shipped from New Zealand to Australia would span the physiological stages outlined above from harvestable maturity up to the point where the eating quality drops below the demands of the consumer.

Question 2
Do the requirements established by Pipfruit New Zealand (Exhibit NZ-93) in regard to maturity and absence of damage correspond to a commonly accepted definition or criterion? (IRA, Part B, pp. 63 and 315)

Dr Deckers:

20. Yes, this is a standard method of maturity and fruit quality determination.

Dr Latorre:

21. Yes, the requirements established by Pipfruit New Zealand with regard to maturity and absence of fruit damage are acceptable.

Dr Paulin:

22. Yes. This requirement corresponds to a high standard of quality. The specifications for absence of damage are of a proper level of precision to guarantee "symptomless fruits" (black spots, rots...: nil allowance).

Dr Schrader:

23. With regard to the requirements for maturity, the inclusion of background colour and ethylene content as established by Pipfruit New Zealand goes beyond the requirements asked by the Streifindex. Regarding absence of damage, class 1 as defined in Exhibit NZ-93 is more detailed than e.g. quality standards required by the European Union, where trade class 1 refers to good quality, slight shape and development defects, slight colouring defects, only slightest bruises, sufficient firmness.

Dr Swinburne:

24. Pipfruit New Zealand exhibit NZ-93 outlines the measures normally taken to ensure the maintenance of quality standards in apple cultivars generally. One would normally expect to see more detailed information with respect to individual cultivars, perhaps these are in the appendices etc. These would refer to tolerance limits for size, shape, colour and other attributes such as bruising and even the frequency of lenticels.

Question 3
Based on the parties' arguments, is there any difference between on the one hand, "class 1 export quality apples" and, on the other hand, "mature apple fruit free of trash" or "mature, symptomless apples"? Even if these three notions correspond to each other, is there a risk that "class 1 export quality apples" exported from New Zealand might not always be mature, symptomless or free of trash? (R 1-6 by the Parties)

Dr Deckers:

25. Class 1 export quality apples are mature apple fruit free of trash and the notion mature symptomless apples corresponds in fact to the same. In the frame of this discussion I think it is important to stick on one definition like "mature apple fruit free of trash". It is important that the fruits are free of trash, because the trash could harbour disease like *Erwinia* bacteria from infected orchards. Fruits packed retail-ready can be considered automatically free of trash, but apple fruits prepared bulk in bins are not always free of trash.

Dr Latorre:

26. Class 1 export-quality apples = Mature apple fruits, harvested at physiological maturity, that comply with the specification established for Class 1, e.g., as indicated in Exhibit NZ-93.

27. Mature apple fruit, free of trash = apples harvested at physiological maturity, free of leaves, stems, other plant organs or junk materials that eventually can contaminate fruits at harvest.

28. Mature symptomless apples = apples that are visually (externally) healthy (asymptomatic) and have been harvested at physiological maturity, estimated by the use of one or more maturity indices.

29. Following 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.

Dr Paulin:

30. The category "class 1 export quality apple", as defined in the NZ 93 includes in its definition "mature apple fruits free of trash". Trashes are not specifically mentioned in NZ description: it is considered standard that, at this stage, no trashes are to be found associated with shipped apples. Again, "mature symptomless apples", are included in the category "class 1 export quality apples" which describes safely apples for export to Australia: it excludes fruits with symptoms (whatever they are), immature fruits, and trashes. These last items are not supposed to be exported under this category.

Dr Swinburne:

31. It is difficult to discern any meaningful difference between the various terms applied to the quality of fruit that would be exported from New Zealand. The criteria for "maturity" quoted are

seemingly used to determine harvest dates; thus fruit harvested within the acceptable limits of those dates would be "mature" (see Q1). Class 1 fruit, which seems to be synonymous with export quality, must then refer to "mature" harvested apples which have been subject to inspection and grading for blemishes (including rot symptoms) and other factors such as size and colour to the standard required. Trash is usually removed, and indeed eliminated at this stage.

Question 4
Based on the relevant parts of Australia's IRA, and Parties' arguments, what is your understanding as to the exact level of involvement of AQIS in audits and inspections required by Australia in regard to fire blight, European canker and ALCM? (IRA, Part B, pp. 314-315; R 45-52 by the Parties)

Dr Deckers:

32. The involvement of AQIS in audits and inspections should be based on mutual respect of the existing quality assurance procedures in New Zealand in the different fields like in the production at orchard level, the preparation of the fruits in the packing house and the exportation of the fruits. This counts for the 3 diseases EA,NG and ALCM.

Dr Latorre:

33. In agreement with the plant quarantine authorities of New Zealand, the Australian Quarantine and Inspection Service (AQIS) has the right to inspect and/or audit protocols and phytosanitary procedures to certify a pest-free area, pest-free place of production or pest-free production site, with regard to European canker, fire blight and ALCM. Inspection or auditing should focus on those aspects concerning the abovementioned pest and diseases. There is no reason to inspect and/or audit other steps of the apple production in New Zealand unless they are directly related to these sanitation problems.

Dr Paulin:

34. According to IRA it seems that Australia wants that AQIS agents be directly involved in audits of procedures used in the field as well as in the packing houses, with respect with the three pests. My understanding is that there is a request from Australia that AQIS staff be involved in these audits. But direct inspection by AQIS agents is not on the agenda.

35. The pre-clearance steps, on the contrary, according to IRA, must imply an active inspection of orchards by AQIS agents, as well as of fruits in the packing houses, in addition to audits of other arrangements. I could not decide from the documents whether involvement of AQIS agents is meant for AQIS agents alone, or in cooperation with New Zealand officers.

36. May be I could just add that it is not exaggerated from Australia, at least in the first trade, to be willing to obtain the best possible view on the way the proposed measures are applied at the field and packinghouse levels. Even if as I understand, AQIS usually behaves in a more confident way towards other imports from New Zealand.

Question 5
Based on the relevant parts of Australia's IRA, and Parties' arguments, what is your understanding as to whether Australia requires a systems audit of 100% of survey teams in the field in the first year and 100% of all packing houses, as New Zealand argues in R 46? How would this requirement compare with those applied by other Members facing similar risks and situations? (R 45-46 and 52 by the Parties)

Dr Deckers:

37. A systems audit should not mean an audit for 100% survey of the teams in the field in the first year and for 100% of all the packing houses involved. Australia should clarify its exact intentions more transparently in this field. An audit of the different existing quality systems at the different levels by AQIS looks normal.

Dr Latorre:

38. I am not in a position to give a definitive answer to this question, except to say that both countries must develop a consensus protocol and that, at least during the first year, the level of inspection and/or audit should be in accordance with the level of disease prevalence and severity existing in each apple production area.

Dr Paulin:

39. My understanding is that Australia intends to audit with standard audit techniques each survey team and all the packing houses, on the first year of trade.

40. I just do not know how this compares with requirements of other members in similar situations.

Dr Schrader:

41. In accordance with Australia's reply to question 52, a 100% audit of survey teams suggests that auditors will apply auditing techniques, "questioning, listening, observation, documentation", to each and every member of each of the survey teams. By contrast, the statement that "[t]he audit would include 100% of survey teams" signals Australia's intention to audit each survey team by applying audit techniques, "questioning, listening, observation, documentation", to sufficient members of each team to satisfy the auditors that the team is meeting the requirements outlined in the Final IRA Report. This clarifies that Australia intends to audit each survey team, but not each member in each survey team. However, the amount of members of the survey team being audited is not quantified – Australia only states that "sufficient members of each team…" will be audited. Therefore, in an extreme scenario, Australia could conclude, that the team has to be audited in total as it would otherwise not be sufficient. With other words, no limits for members of survey teams to be audited are set; "sufficient" is an arbitrary term and quite vague.

42. In addition, I see a discrepancy in the clarification of 100% audits of survey teams and the explanation, what 100 % of packing houses means. The explanation given by Australia, that "a '100% audit of packing houses' means that each packing house will be audited by AQIS officers while they are present undertaking fruit inspections for pre-clearance" is not in line with the explanation given regarding the contrast between a "100% audit of survey teams" and "the audit would include 100% of survey teams". It should be clarified, whether this formulation should be in line with the formulation for survey teams, i.e. "the audit would include 100% of packing houses."

Dr Swinburne (Response to questions 4 and 5):

43. Australia's IRA (Part A, p.17; Part B, p. 314-315) seems to imply that to initiate trade in apples with New Zealand AQIS officers would be involved directly in all aspects of the risk management systems they wish to see adopted, during a period referred to as "Preclearance". The IRA (Part B, p. 313) recognises that MAFNZ would be the competent authority in the registration of orchards and issue of phytosanitary certificates, so it is presumably the intention that during this "Preclearance" phase AQIS officers would work alongside their MAFNZ counterparts, and be directly

involved in all inspections. Australia's responses (R 45-52 by the Parties) suggest that after gaining this initial experience the role of AQIS officers would be reduced to that of systems auditors. What are not clear are Australia's requirements for allowing the role AQIS officers to evolve from direct, hands-on inspectors of all orchards and pack-houses to that of systems auditors, or how many seasons it might take for the transition to be effected.

II. FIRE BLIGHT

Question 6
Please comment on whether an apple fruit may be naturally (i.e., through means other than artificial inoculation) epiphytically infested or endophytically infected with fire blight and still develop into a healthy-looking mature fruit. Please comment also whether any of the challenged requirements imposed by Australia with respect to fire blight are based on a finding in Australia's IRA that such situation is possible. If so:
a. What is the scientific basis contained in the IRA for such a finding?

Dr Paulin:

44. Epiphytically infested fruits: Fruits may be surface infested and still develop into a healthy-looking mature fruit, in an orchard where the disease is active (that is when symptoms are developing on the trees). Fruits on the trees showing active symptoms may be surface contaminated by ooze produced from near-by symptoms, or by direct contact with diseased parts of the tree (shoots, other fruits, late blossoms). In such an orchard with active symptoms it is possible also that wind-driven rain carries bacteria from the surface of symptoms to fruits, or that rain washes exudate from symptoms situated in the upper part of the tree down onto the fruits. In this case the bacterial population is transient (Thomson, 2000). Without wounding, the infested fruit will not be infected, and will develop into a normal healthy fruit. If wounded (hailstorm...) symptoms would develop on immature fruits.

45. Endophytically infested fruits: I do not know of any description of internally infected symptomless mature fruits. On an infected tree it can be supposed that few bacterial cells in the xylem may migrate up to the fruits, without being active, and therefore without producing symptoms. As far as I know, nobody has ever detected such endophytic populations in fruits. Anyhow an endophytic detection in mature fruits would suppose that the tree bearing these fruits has had a history of fire blight the previous weeks or the previous years.

46. (a) The scientific basis in IRA for the finding that *Erwinia amylovora* may be present on and in fruits are a number of results obtained in diverse conditions, in New Zealand and the United States, showing that bacteria in small amount may be present in the calyx-end of fruits issued from severely infected orchard (Hale et al., 1987). Results may vary according to detection techniques used (more or less sensitive or specific, some of them unable to separate dead from living cells). But the general picture is that, in an area showing active symptoms of fire blight, a possibility exists that limited population of surviving bacterial cells may be present on fruits. These populations tend to decrease with time. The existence of these surviving populations (external or internal) is due to the fact that, like a number of other bacteria, *E. amylovora* has developed a number of tools for its survival in "adverse" conditions (starvation,.. etc). Among them, the IRA reports on EPS, quorum sensing, VBNC, general regulator of metabolism like sigma factors.. etc. All these allow the bacteria to face periods of time where it cannot multiply, and therefore to survive for some times.

b. Is the finding in the IRA in this regard based on respected and qualified scientific sources?

Dr Paulin:

47. Not all the papers included in the IRA with respect to survival of *E. amylovora* associated with fruits provide perfectly established results. As already mentioned by a stakeholder, some of them are very difficult to understand, due to the complexity of the design of the paper (van der Zwet *et al*., 1990). Very often it is not easy to know what is precisely intended by "severe fire blight" in the orchard (or in the vicinity), and this makes the results difficult to evaluate, or to extend to the general case. Nevertheless the possibility of the presence of a limited surviving population of bacteria on the surface of mature fruits issued from severely infected environment, given by the whole data presented, seems to be acceptable.

48. Internal contamination of fruits, on the contrary is not well established in the IRA: the experiments reporting positive isolation from internal tissue of fruits are not convincing (van der Zwet *et al*., 2000), or obtained in artificial conditions which do not represent the conditions of mature fruits in the orchard. (Dueck, 1974). The paper supposed to show that *E. amylovora* is associated with ovules and seeds is just not credible, due to the lack of appropriate determination procedure of bacteria, and to the presentation of data, which does not allow the association of a precise bacterial species with a corresponding seed or ovule. It remains that *E. amylovora* has repeatedly been supposed (rather than shown) to be trapped in xylem vessels (Vanneste and Eden Green, 2000), and, if this were really the case, this could result in the presence of *E. amylovora* in mature symptomless fruits. To my knowledge, this has never been proven.

49. Conversely to the above "field" data, the scientific data concerning the diverse means for the bacteria to survive are well established, in laboratory conditions. The diverse mechanisms described for survival are potential tools for bacteria in general. They work in *E. amylovora* as well. The assessment of the exact role of each of these mechanisms is difficult. It remains that *E. amylovora* is not considered as an ubiquist bacteria (like *Listeria* for example) which would have a strong potential for surviving and multiplying in diverse ecological niches.

c. Is the reasoning articulated by the IRA on the basis of such scientific evidence, including the methodologies applied, objective and coherent and do the particular conclusions drawn in the IRA find sufficient support in the available scientific evidence?

Dr Paulin:

50. The reasoning established by the IRA seems consequently coherent and usually based on available evidence, although it may tend to exaggerate the risks of *E. amylovora* associated with fruits (mature symptomless).

d. Do the results of the IRA's assessment in this regard sufficiently warrant the challenged requirements relating to fire blight?

Dr Paulin:

51. The results of the assessment therefore support sufficiently the need for information on the orchard previous infection, for inspection of orchards before export, as well as for the disinfection of fruits in the packing houses. May be the fact that we are dealing with low populations of surviving (not able to multiply) bacteria, and likely to be present only when active symptoms of the disease are present in the close vicinity or in the orchard where the apples are sourced could be better taken into account, with request of less strict prescriptions.

Dr Deckers (Response to whole question):

52. In the biological cycle of EA mature apples are not included as an important way of spreading the fire blight disease. In contrast to the absence of specific measures on export on fruits, specific measures are imposed in Europe in the countries with fire blight around the fruit tree nurseries with the aim to prevent export of contaminated trees from infected countries to countries free of fire blight. The trade of apple fruits between the different countries is not subjected to special measures. This means that the spread of the fire blight disease by fruit tree nursery material is considered to be much more important than the risk for spread by the export of contaminated apple fruits.

53. Apple fruit can be infected epiphytically and naturally and can still develop into a healthy-looking mature fruit. Epiphytical presence of the disease in the calyx end of the fruits has been sufficiently documented. There are circumstances that can increase the epiphytical presence of the EA bacteria on the apple fruits. This can happen when there is an important hail damage on the immature fruits in an orchard which can result in an important increase of the bacterial inoculum in the orchard with ooze formation on the immature fruits on the hail wounds and the build up of an important epiphytical population of *Erwinia* bacteria on healthy looking mature fruits at the end of the season. Bacterial ooze formation does not occur on mature apple fruits, because the amylum which is present in the immature fruits allows a rapid multiplication of the EA bacteria. The mature fruits don't contain amylum because the amylum is transferred into sugars during the maturation process.

54. The chance to find an endophytically infected fruit that develops to a healthy looking mature fruit is not sufficiently scientifically documented in the fire blight epidemiology. Whenever an endophytically infected fruit would be present on the tree, there is a great chance that such fruits will fall prematurely through internal ethylene formation and will not reach the maturity stage at which the fruits are normally harvested.

Question 7
Please comment whether the challenged requirements imposed by Australia with respect to fire blight are based on a finding in Australia's IRA that mature apple fruit can be infected with fire blight and that mature apple fruit can disseminate E. amylovora or serve as a source of new infection/infestation in orchards. If so:
a. What is the scientific basis contained in the IRA for such a finding?

Dr Paulin:

55. The infection of mature fruits implies that the bacteria are allowed to enter the mature fruit and then that they are able to multiply in the fruit tissue in order to produce symptoms.

56. (a) No scientific basis is presented in the IRA which could support this proposal. In my opinion the papers presented in this respect are based on experiments that are too far from natural conditions to be of value in the case of orchard conditions (Azegami *et al* 2006, Tsukamoto *et al* 2005).

b. Does the scientific evidence support the contention that an apple fruit may be naturally infected with fire blight (i.e., infected through means other than artificial inoculation) and still develop into a healthy-looking mature fruit?

Dr Paulin:

57. True infection is not reported: it would need a penetration of the bacteria in the mature fruit, followed by multiplication of the bacteria in the fruit tissues. This multiplication would produce

symptoms, therefore the fruit would no longer be symptomless. Surface infestation, even if this should be rare, could occur, but only in orchards (or areas) where there is an available inoculum (ooze), produced nearby by active symptoms of fire blight (see above).

c. Please comment on the scientific basis for claiming that mature, symptomless apples could harbour endophytic (internal) populations of E. amylovora?

Dr Paulin:

58. See above for the supposed xylem invasion by bacteria. In the xylem, the bacteria are starving, because the content of the xylem constitutes a very poor medium as far as nutrients for the bacteria are concerned. Then it is expected that *E. amylovora* will not multiply in vessels. Nevertheless it could survive for some times, possibly at the VBNC stage, before disappearing.

d. Is the finding in the IRA based on respected and qualified scientific sources?

Dr Paulin:

59. See (a).

e. Is the reasoning articulated by the IRA on the basis of such scientific evidence, including the methodologies applied, objective and coherent?

Dr Paulin:

60. In my view, there is no basis here for a specific reasoning for prevention of fire blight spread in the case of mature apple fruits free of trash.

f. Do the results of the IRA's assessment in this regard sufficiently warrant the challenged requirements related to fire blight?

Dr Paulin:

61. No specific requirements are needed to prevent the risk associated with infected apples.

Dr Deckers (Response to whole question):

62. Mature apple fruits can harbour EA bacteria epiphytically on the fruit skin or in the calyx of the fruit. But it is generally accepted that the fire blight bacterium is not surviving well as an epiphytical bacterium. On the fruit skin the EA bacteria will dry out easily and die while in the calyx end they will be able to survive for a longer period. But multiplication of the epiphytical EA bacteria in the calyx end of the fruits will not occur; multiplication of the bacteria will only occur on a medium rich in sugar or in amylum. This means that the level of epiphytic populations of the EA bacteria on the apple fruits will remain low. This will make it difficult to detect these low number of bacteria on the fruit skin. The chance that this epiphytic population of EA serves as a new source of infestation in the orchard is very small is not described in the biological cyclus of an EA infection under orchard condition. The calyx of the fruit is not a place where the EA bacteria can multiply ; in the best case the bacteria can survive for a period on that place.

63. There is not sufficient qualified research available that indicate the importance of endophytic populations of *Erwinia amylovora* in apple fruits.

Question 8
Based on Australia's IRA, do the challenged requirements imposed by Australia with respect to fire blight provide for any tolerance of the presence of E. amylovora in or around areas from which New Zealand apples would be sourced? Please comment on whether the following terms express different concepts: "area freedom", "pest free places of production", "freedom of the disease" and "freedom of the visible symptoms of the disease". How relevant is a distinction between "area freedom" and "low pest prevalence" in the context of Australia's measures for fire blight? In light of Australia's IRA and Australia's R 36, how do these various terms relate to relevant ISPMs, in particular ISPMs Nos. 4, 5, 10 and 22? (Paras. 4.443 and 4.447 of New Zealand's FWS; paras. 156-160 of Australia's FWS)

Dr Deckers:

64. Freedom of the visible symptoms of the disease does not mean that the disease is not present on the fruit or that there is a pest free place of production. It only describes the situation of fire blight in the orchard and indicates that there is a reduced risk for the presence of fire blight bacteria on the fruits because there are no active fire blight infections in the considered orchards. It is important that not only the orchards should be fire blight free but also the immediate environment around the orchard should be fire blight free. The sporadic appearance of the fire blight disease in an orchard or on another host plant is one of the typical characteristics of this bacterial disease under orchard conditions. This means that the disease will not be present every year on the same place and in the same intensity.

Dr Paulin:

65. The IRA indicates that no area can be proved to be without fire blight in apple production zones in New Zealand. If this is correct, the requirements are made on the assumption that there is not any orchard from which apples are exported which is (or has ever been in the 50 last years) without fire blight: therefore the IRA provides a certain level of tolerance.

66. – "Area freedom": Area without fire bight, the disease is regularly surveyed and has never been seen , or reported (for example: South Africa) ISPM 4.

67. – "Pest-free places of production": Places without fire blight (likely orchards or nurseries) within an area where the pest (fire bight) is present, (for example, it would be the case of Corsica, which is surveyed without fire blight, in France where fire blight is present) ISPM10.

68. – "Freedom of disease": No symptom ever seen. The trees are supposed to have never been infected by *E. amylovora.* Therefore should be used only in "area freedom" or "pest free place of production".

69. – "Freedom of the visible symptoms of the disease": No symptom seen (during this growing season), or symptoms immediately removed.

70. – "Area freedom": Should be an area without symptoms.

71. – "Low pest prevalence" ISPM 22: A zone where the disease is poorly active and therefore the symptoms are rare (low number of diseased plants, and limited development of symptoms). This is basically different from ISPM 4, which is area freedom. In a low pest prevalence area some symptoms are likely to be present, at least from time to time.

Question 9
What is the scientific basis in Australia's IRA for the requirement relating to the suspension of exports on the basis of evidence of pruning? Is the reasoning in the IRA with regard to the use of pruning as a possible means to remove or hide symptoms of fire blight objective and coherent? Is such reasoning based on respected and qualified scientific sources? Do the results of the IRA's assessment sufficiently warrant the challenged requirement relating to the suspension of exports on the basis of evidence of pruning as a possible means to remove or hide symptoms of fire blight?

Dr Deckers:

72. Pruning out the fire blight infections during growing season is a control measure that is taken in many countries to keep the fire blight situation under control in an orchard. It should not be regarded as a way to hide the fire blight infections because this pruning out of the fire blight infections during season can easily be distinguished from the standard pruning measures that are made during dormant season. It seems to be logic that Australia takes into account this fire blight control measure when making the evaluation of the fire blight situation in an orchard. Suspension of the export when recent fire blight pruning in an orchard has been observed seems to be a logic measure in the IRA.

Dr Paulin:

73. The scientific basis of this requirement is that symptoms of fire blight develop in spring and early summer, associated with the presence of blossoms and then actively growing shoots. The main infection period is the blossom period. If an orchard is infected in spring or early summer, the development of symptoms will stop in summer and autumn. If the trees are trimmed for suppression of symptoms, it could well be that the orchard looks symptomless when actually it has shown activity of the disease (and hence production of ooze with bacteria), which could have consequences on fruit infestation. In addition, it remains possible that a renewal of activity of the disease takes place after the inspection, but before cropping. That would be a situation resulting in a risk (even if weak, see question 6) of surface infested fruits.

74. The reasoning of IRA is therefore coherent and objective (as soon as it limits the ban of pruning, for export orchards to the pruning which takes place after the beginning of the blossom period).

75. This assessment of the suspension of export is soundly based on the evidence of this late pruning (obviously winter-pruning is a standard procedure in tree management).

Question 10
Part of the analysis of exposure and establishment with respect to fire blight in Australia's IRA proceeds on the basis that host plants in Australia are susceptible to being naturally infected with E. amylovora (i.e., infected through means other than artificial inoculation). (IRA, Part C, p. 105; R 66 by the Parties)
a. What is the scientific basis contained in the IRA for such a finding, including the scientific basis for assuming that such hosts would be susceptible to being naturally infected during the same periods in which apples from New Zealand would be imported by Australia?

Dr Paulin:

76. As a preliminary comment it could be indicated that the 150 species of host plant described in the literature do not represent the number of species actually exposed to the disease in natural condition (as underlined in the IRA). It seems more realistic to consider only the 10 "true" host species that are cited in the IRA as hosts of primary and secondary economic and ecological

importance. Very likely, the other ones would play no role in the installation and spread of the disease. In addition, in each of these host species, not all the cultivars are susceptible to fire blight. That means that among these 134 groups of plants theoretically exposed to develop the disease, only a restricted proportion is actually at risk: some cultivars only among 10 of these 134. But these cultivars are economically important, of course, and they can be expected to be as susceptible in Australia as they are in other parts of the world.

77. (a) Host plants of fire blight, when susceptible, do not show a constant receptivity to the disease along the growing season. This receptivity to natural infection may be totally absent: this is the case in dormant period (winter) and when the shoots are not growing, that is generally (it depends on the host species) in late spring and summer. Conversely, the receptivity to infection is high during blossom period, and during shoot growth. Classically, for most host plants of fire blight, shoot growth takes place after blossoming, and stops in early summer. Obviously this timing will depends on local conditions (water supply, nutrition of the plant, temperature, pruning...). In addition, shoot growth in some cases (hawthorn) takes place before bloom (early spring). For most host plants natural infection is easier during bloom. Shoot infections are often related to summer hailstorms. This applies to each host of fire blight. For each host the blossom period and the shoot growth period are specific in their date of appearance and in their duration. Theoretically, it is possible that at least some of the host plants are in bloom or shoot growth nearly all the year round: for example *Chaenomeles* blooms in winter, *Cotoneaster* in early summer. To be more realistic, I think that the risk is high, in case of introduction, only in spring and early summer, with a possible additional dangerous period in summer and late summer if some hosts such as certain cultivars of apple and mainly of pears produce secondary blossoms (blossoms opening after the normal blossom period, usually associated with some physiological disorder). In addition, the probability of infection will be associated with the number of hosts at the suitable stage at the moment of introduction. In this respect, the maximum period of susceptibility to an introduction of *E. amylovora* will be the full bloom period of pears, and then of apples.

b. Is the finding in the IRA in this regard based on respected and qualified scientific sources?

Dr Paulin:

78. Against this background it is difficult to tell when an introduction of *E. amylovora* would be most prone to turn into a natural infection. But it can be assumed that most of the time during the year most host plants should not be at a receptive stage when the import of apple would take place (peak in winter).

c. Is the reasoning articulated by the IRA on the basis of such scientific evidence, including the methodologies applied, objective and coherent, and do the particular conclusions drawn in the IRA find sufficient support in the available scientific evidence?

Dr Paulin:

79. The reasoning of IRA is coherent, but maximises the real risks in considering too high number of host plants, and in taking no account of the discontinuity in susceptible host plants receptivity during the year.

d. Do the results of the IRA's assessment in this regard sufficiently warrant the challenged SPS measures related to fire blight?

Dr Deckers (Response to whole question):

80. For the realisation of a fire blight infection there must be a co-incidence of 3 important factors:

81. (1) The host plant must be in a susceptible stage of development e.g. flowering time is a susceptible stage.

82. (2) The climatological conditions for an EA infection should be sufficient with appropriate temperatures and relative humidity for bacterial multiplication followed by an infection.

83. (3) The EA bacteria must multiply on a susceptible organ (e.g. stigma on the style of a flower) before the infection can take place. Rain is often an important factor to transmit the bacteria from the multiplication site to the infection site. This means that drought can limit the spread of the EA bacteria.

84. For each factor an evaluation can be made for the infection conditions under Australian conditions at the moment of apple fruit import. During dormant season, there is no infection possibility of the fire blight host plants when the host plants don't have the susceptible organs or when the climatological conditions are not allowing bacterial multiplication.

Question 11
Based on the relevant parts of Australia's IRA, do you consider the IRA to be objective and credible when qualifying the biological and economic consequences of fire blight as "high"? Can you comment on the specific meaning given in the IRA for the impact score for "high" and, in particular, how it compares with what is considered to be a "high" impact with respect to other risks? (IRA, Part B, p. 104; paras. 493-523 of Australia's FWS; paras. 4.253-4.264 of New Zealand 's FWS; R 67-68 by New Zealand)

Dr Deckers:

85. The biological and economical consequences of a fire blight introduction can indeed be classified as "high".

86. The biological and economical consequences of a possible fire blight infection in Australia depends of the success of the installation and the spread of a new infection on a possible host plant. The different regions in Australia will not have all the appropriate climatological conditions for an optimal EA infection and development. Further, there will be differences in disease development from one year to another: this is typical for an infection of EA that the disease incidence is not the same every year.

Dr Paulin:

(a) Direct impact

87. *Economical consequences of introduction of fire bight in Australia:* I am not sure that fire blight is the most serious disease of apple everywhere. Apple scab would be probably considered as more costly in many areas. But fire blight is undoubtedly the most serious bacterial disease of apple and pear. It is impossible to predict the economical consequences of the introduction of fire blight in a new area. The severity of the disease (the importance of damages) is the result of the combination of three factors (at least) at the local level: climate, cultivar susceptibility (genetic), cultivar receptivity. If each can be evaluated (at least for trends) the combination of the three to give a reasonable prediction is non realistic. Nevertheless it explains that it is just not possible, and it has

never been seen, that fire blight is devastating to the same degree at any place and on any plant as soon as introduced in a new area. In addition the spread of the disease, even if not eradicated, is relatively slow, and the invasion, if any, should be progressive. The damage to crops is difficult to estimate. In France, as far as I can tell, I have not seen a large number of bearing trees of apple killed by fire blight, in spite of the high susceptibility of some of our varieties. This is not true for pear varieties, but the disappearance of entire trees due to fire blight remains limited to few varieties only. The overall production of fruits in a whole country has never been seriously decreased, even by a severe fire blight epidemy, even if damages can be very costly at the local level, in certain years for certain varieties. Therefore the impact score of "F" could be exaggerated.

88. *Biological consequences of introduction of fire bight in Australia:* Up to now, in the different areas invaded by fire blight in the world, the introduction of the disease has had limited biological (ecological) consequences (at worse serious limitation in certain zones of specific cultivars of fruit trees (pears) and ornamentals). To my knowledge it has not induced the disappearance of any grown or spontaneous species of plant. I do not see for which reason it would be very different in Australia.

89. In this case, the score of "A" seems correct.

(b) Indirect impact

90. Conversely, the introduction of fire bight in Australia would be a very serious issue. Actually, even if the destruction of plants by the disease itself was limited, the simple introduction of the disease in a "new" country is costly, due to the fact that *E. amylovora* is a quarantine disease for many countries, and that the status of "without fire blight", for a country, implies a number of commercial advantages for export of plant material (*to say nothing of the fruits?*). The attempts at eradication would be very costly. Comparatively, the cost of additional sprays requested by the risk or presence of fire blight seems almost negligible. But the lost of the "without" status with respect to quarantine could justify a "F".

91. *Domestic trade:* Given the likely low impact at the national level of the disease on overall production, the losses for domestic trade or industry look exaggerated and unrealistic.

92. *International trade:* The impact could be strong on nursery exports due to quarantine restrictions, but this is not suggested in the IRA. The impact on fruit trade will be limited, especially if the eradication methods are effective (it will not be possible to consider any apple orchard of Australia as infected by fire blight). Again the rating of "D" seems too high.

93. *Communities:* This is related to the loss of production. As seen earlier it is not likely that the loss is perceptible at the national level. An impact, if any, on job availability would concern only a very local level, not national.

94. The qualification of "high" for the impact of fire blight is to my eyes appropriate, based on the possible international consequences of this introduction. Although it can be argued that *E. amylovora* has already been found in Australia.

95. I have no idea on other threats to apple and pear production in Australia.

Dr Schrader:

96. The Australian IRA Guidelines do not give examples for ratings. This is a general shortcoming in many risk assessment guidelines. However, for example, the Canadian Risk Assessment Scheme includes guidelines for rating. There, an economic impact is rated as "high" when "the pest has a severe impact on the standing crop with significant host mortality" (Plant Health

Risk Assessment, Commodity Risk Assessment, Canadian Food Inspection Authority, Plant Health Risk Assessment Unit, Science Advice Division).

97. In the case of fire blight, the rating of high for economic consequences would be adequate, as this bacterium can cause severe damage to the crop as well as to the plants themselves. After favourable weather conditions during blooming, yield is considerably reduced and in it is possible that there is no or nearly no yield at all. In the following year, productivity is also significantly affected due to the destruction of fruiting spurs. In susceptible hosts the infection spreads so quickly through the plant that, once infected, trees cannot be saved, even by drastic and immediate surgery, and die in a short time after the first visual signs of infection. (e.g. Van der Zwet, T., Keil, H.L., 1979: Fireblight: a bacterial disease of rosaceous plants. USDA Agriculture Handbook No. 510).

Dr Sgrillo:

98. IRA is objective in qualifying the economic consequences. However IRA makes very few references to actual losses caused by *E.amylovora* elsewhere.

99. Although fire blight is distributed in more than 50 countries[2], IRA cites only three cases of losses (direct impact in plant health) by *E.amylovora*: New Zealand in 1998, USA in 1976/77 and USA/Michigan in 2000.[3]

100. The economic losses of New Zealand in1998 were estimated in 2.8% of the country production.[4]

101. The losses of USA in 1976/1977 were estimated in US$ 2-5,000,000[5]. The values of apples production in 1976/1977 were US$ 566,102,000 and 620,979,000[6] respectively. Therefore, the losses caused by fire blight correspond to 0.35% and 0.80% of the country production respectively.

102. The outbreak that has occurred in Michigan, USA in 2000 is analyzed by Longstroth (2001).[7] This author describes losses of 15% of the trees, 9% of the area and 37.5% of the production in southwest Michigan, which contains 29% of the apples acreage of the State of Michigan. The losses for the Michigan state were then 4.4% of the trees, 2.6% of the area and 10.8% of the production.

103. Fire blight is a serious disease of pome fruit trees. However, based in the figures above, the consequences considered by IRA[8] (country loss of 50% and 20% for pear and apple respectively) seem to be overestimated. Should be considered also that the perfect fire blight conditions, unusually warm, humid and wet weather, are expected to occur only once in each 10 years.[9] To clarify these points would be necessary additional information on losses in countries where the *E. amylovora* occurs.

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[2] (*footnote original*) Exhibit NZ-6 European and Mediterranean Plant Protection Organization (EPPO), (1997) Data Sheet on Quarantine Pests: Erwinia amylovora, prepared for the European Union.

[3] (*footnote original*) IRA pp. 98-99.

[4] (*footnote original*) New Zealand 's FWS para. 4.261.

[5] (*footnote original*) IRA p. 98.

[6] (*footnote original*) Reference 09: Crop Values 1975 1976 1977 – Season Average Prices Received by Farmers and Value of Production. USDA Economic, Statistic, and Market Information System.

[7] (*footnote original*) Exhibit AUS-44: Longstroth M. (2001) "The 2000 fire blight epidemic in southwest Michigan apple orchards", The Compact Fruit Tree 34 (1), pp. 16-19, p. 16.

[8] (*footnote original*) IRA p. 99.

[9] (*footnote original*) New Zealand 's FWS paras. 4.256, 4.260.

104. The IRA categories[10] to assess potential impact in the production may be summarized as:

"Unlikely to be discernible": No production losses
"Minor significance: Minor production losses; reversible effects
"Significant": Moderate production losses; effects reversible or not
"Highly significant": Large production losses; irreversibly effects

105. The impact score for *moderate production losses* in national level (significant) or *large production losses* in regional, district or local level (highly significant) corresponds to category *F*, according to IRA's Table 10.[11] The overall consequences are considered to be high if the consequences of the pest with respect to a single criterion are *F*.

106. The *high* impact score is compatible with the losses caused by fire blight described in the literature, as reported in EPPO data sheet:

> "The fire blight pathogen causes considerable damage to susceptible hosts. It is not only destructive to the current year's crop but also extremely dangerous to the plants themselves. After favorable weather conditions during blooming, yield is considerably reduced and in some cases nullified. The next year's productivity is also significantly affected because of the destruction of fruiting spurs. In susceptible hosts the infection spreads so rapidly through the tree that, once infected, trees cannot be saved, even by drastic and immediate surgery, and die in a short time after the first visual sign of infection. In some states of the USA, pear cultivation has been largely abandoned because of the disease."[12]

Question 12
What is your understanding as to how the likelihood of entry, establishment and spread into Australia of Japanese Erwinia associated with Japanese nashi pears could be compared to that of Erwinia amylovora associated with New Zealand apples? Is there a sound scientific basis for New Zealand's argument that there are comparable risk profiles at issue in the case of Japanese Erwinia and Erwinia amylovora, considering also the potential biological and economic consequences? (Paras. 4.436-4.439 of New Zealand's FWS; paras. 986-987 of Australia's FWS; and R 101 by Australia)

Dr Deckers:

107. There is a great similarity between the Japanes *Erwinia* associated with nashi pears and *Erwinia amylovora* on apples from New Zealand. In both cases it concerns a bacterial disease on fruits, the one Japanese *Erwinia* on pear and the other fire blight one on apple and pear. This distinction will of course have consequences for the global risk evaluation. On the other hand the risk for epiphytical presence of the bacteria on the surface of the fruits is comparable for both bacteria and should ask for a comparable strategy to avoid entrance of the disease.

Dr Paulin:

108. The key-difference between the two situations, as far as risk associated with fruits is concerned, is that, according to Australia (following Japan statement), this disease caused by *Erwinia sp.* on Nashis is present only in the Hokkaido Island. This allows Australia to import fruits

[10] (*footnote original*) IRA p. 38.
[11] (*footnote original*) IRA p. 39.
[12] (*footnote original*) Exhibit NZ-6 European and Mediterranean Plant Protection Organization (EPPO), (1997) Data Sheet on Quarantine Pests: Erwinia amylovora, prepared for the European Union.

from an other area, remote from Hokkaido. This is a clear objective difference between *Erwinia*/Japan, and *E. amylovora*/New Zealand situations.

109. Otherwise it seems that the bacteria (*Erwinia*-Japan and *E. amylovora*) are very similar but not identical, and the symptoms seem similar as well, but *Erwinia* from Nashis shows a narrow range of host plants, which could account for lower risks (limited to pears?). To be honest it is important to recognise that little is known in the literature about this Nashi disease.

110. In addition it is not certain that this particular *Erwinia* from Japan be considered as a quarantine pathogen. Therefore the limitations in international trade could not be a consequence of an introduction. Again, too little knowledge is available on this Nashi disease to be sure.

Question 13
What is your understanding as to how the relevant measures identified in Australia's IRA compare to the domestic measures imposed by Australia in reaction to the fire blight incursion at the Melbourne Royal Botanic Gardens in 1997? What factors would be relevant in assessing whether any differences or similarities between the two sets of measures applied in these two situations could be used to determine the coherence of the reasoning contained in Australia's IRA? (Para. 4.262 of New Zealand FWS; paras. 77, 356 514 of Australia's FWS; and R 69 by the Parties)

Dr Deckers:

111. The domestic measures imposed by the Australian government in reaction to the Melbourne outbreak included also a restriction in apple and pear movement in the whole state of Victoria. This is compatible with the risk estimation in the IRA of the apples imported from New Zealand. There was also a restriction on the movement of the fire blight host plants in the Melbourne area and all the host plants in the botanical garden were removed. This is in contrast with the measures that should be taken in the orchards in New Zealand: also in the orchards the infections should be removed and burned as soon as possible to avoid a wide spread of the fire blight disease.

Dr Paulin:

112. The relevant measures in IRA should compare in the eradication, destruction of diseased and healthy plants in the surroundings, surveys and diagnostics, which were undertaken: such measures should be taken in case of an introduction with fruits. The survey was probably far more difficult to organize in the case of the Botanical Garden, due to the absence of credible hypothesis on the origin of inoculum (site and date). If an introduction associated with fruit import was experienced, the survey would be far simpler, because the exact place and date of the introduction would be probably soon traced out. In addition, it should be underlined that in no country, even in recently infected countries with only scattered sites of infection (such as Spain, for example), the restriction of trade of mature fruits inside the country has never been suggested or implemented to limit the risks of spread of further of the disease.

113. The diverse levels of action should be the same in the two cases. Nevertheless, as far as I know, the Botanical Garden case was discovered on a relatively isolated plant (paucity of potential host plants in the immediate surroundings). The domestic measures could be more difficult (more plants to be destroyed in the surroundings of the first blighted tree) if the symptom was detected first in an orchard, surrounded by hundred of trees of the same variety, and therefore the same susceptibility at the same stage of receptivity.

Question 14
Please comment on the alternative measures proposed by New Zealand in respect of fire blight, namely (i) restricting apple fruit imports to fruit that has been cold stored, or (ii) limiting imports to

apples that are "retail-ready packaged fruit". How do these measures compare to the relevant measures imposed by Australia in terms of risk mitigation? (IRA, Part B, pp. 4-5 and 105-115; para. 4.490 of New Zealand's FWS; and para. 1087 of Australia's FWS)

Dr Deckers:

114. A cold storage period can reduce the epiphytical EA populations on the fruit surfaces but will not be able to eliminate these populations completely. Limiting the imports to the apples that are retail ready is not excluding apples that could host some fire blight bacteria in their calyx end of the fruits. This means that both measures proposed by New Zealand will not reduce the risk sufficiently.

Dr Paulin:

115. (a) *Import of fruits which have been cold-stored.* Results from (Hale and Taylor, 1999, and others) show that the bacterial population harboured on fruits decreases over time during cold storage. Nevertheless it cannot be expected that this procedure will for sure allow to obtain to disappearance of all living cells. Cold temperature is known to prevent bacterial multiplication (*E. amylovora* will not multiply under 3-5°C (Paulin, 2000)). But on the other hand, low temperature (4°C) is the condition under which bacteria are kept alive in the laboratories for longer periods than at room temperature. Therefore it is not possible to rely on cold storage condition to ascertain that not a single bacterial cell will remain alive. It is reasonable though to assume that along with time, bacterial populations (unable to multiply) will decrease. Among the options evaluated by Australia (source apple from orchard without symptoms, chlorine disinfection) cold storage is probably the less effective.

116. (b) *Retail-ready packaged fruits.* In my understanding of this qualification of these fruits, the apple exported will be symptomless mature apple, in boxes. This means that an additional check of fruit aspect is performed when apples are placed in boxes, and adds a safety degree with respect of both "symptomless" and "mature". But the main interest of this additional step (placing the fruits in boxes) is that it will eliminate absolutely the risk of carrying trashes (from orchards) in association with fruits. In my view trashes may be twigs, dried pieces of leaves, plant debris, which could have been infected by *E. amylovora* before being transformed into trashes, and which could be equivalent of "cankers", which are the natural form of conservation of the disease in the field (Thomson, 2000). Such trashes could serve as vectors because if they result from infection, they may harbour internal population of bacteria. But they are visible and completely eliminated in the normal process of packaging of the fruits. The mitigation of risk through this way of packaging would be drastic.

Question 15
Please comment on whether the alternative measure proposed by New Zealand in respect of fire blight to create a mandatory requirement that apple exports to Australia be limited to "mature, symptomless apples" would achieve Australia's ALOP. How does this measure compare to Australia's measures in terms of risk mitigation? (IRA, Part B, pp. 4-5; paras. 4.492-4.512 of New Zealand's FWS; and paras. 1082-1086 of Australia's FWS)

Dr Deckers:

117. The limitation of apple exports to mature symptomless apples is not enough to achieve Australia's ALOP. Traceability of the fruits to the level of orchard where the apples have been produced is necessary for the risk evaluation in Australia. Fruits from heavy infected orchards or from orchards with hail damage can harbour the bacteria in the calyx end of the fruits.

Dr Paulin:

118. "Mature symptomless apples"

119. *Mature*: Indicates that fruits have completed their development on trees, and therefore that they were not infected at an early stage (otherwise they would not achieve this development up to the mature stage). Mature fruits are recognized as resistant to infection: they do not develop symptoms if inoculated**,** because they do not allow the multiplication of bacteria.

120. *Symptomless* means that they show no fire blight (or other disease) symptoms: this eliminates infected fruits issued from early infections.

121. Both these measures are actually necessary to eliminate the more evident risks of transfer of the bacteria with fruit. It decreases the risk drastically in eliminating the opportunities of carrying high population of *E. amylovora* which are associated with tissues harbouring progressive infection, for example in immature fruits. Nevertheless it cannot be considered as eliminating absolutely the risk of introduction of low (external) bacterial populations associated with fruits. It can then be considered to decrease the likelihood for entry of the bacteria with fruits from very low to extremely low, in the ALOP.

122. The restriction of export to mature symptomless apples would make even safer the different measures taken by Australia (disinfection, storage...), but could not replace any of them.

Question 16
Please comment on whether there is a sound scientific basis for claiming that recent fire blight incursions in territories different from those of Australia and New Zealand can be attributed solely to the introduction of nursery stock from infected areas? Is there any indication that trade in apple fruit had any role in these incursions? (Para. 3.53 of New Zealand's FWS; para. 80 of Australia's FWS; R 61 by New Zealand)

Dr Deckers:

123. Between the different countries in the European union, the risk for fire blight introduction in new countries by infected plant material (variety and or rootstock) is estimated much more important than the introduction possibility by infected apple fruits. In Spain there is a strong indication that some of the fire blight infections was related to the import of infected host plants coming from Belgium. Therefore there is a European legislation regulating the control measures in and around the fruit tree nurseries. The risk for introduction of the disease by infected fruits is estimated much lower and no special measures for the export of fruits are undertaken between the different countries in Europe.

Dr Paulin:

124. Let us consider some "recent" introductions in new countries (introductions which took place from 1962 up to now), these where mainly Egypt 1962, then UK 1966, then continental Europe: Poland, The Netherlands, France and Belgium 1972.

125. First of all it is necessary to keep in mind that the first assessment of the disease in any new place is NOT the first discovery, and this is worse for the first published assessment. Nevertheless our knowledge relies usually on the first official (published) assessment. That means that in most of the cases we lack the exact information that we would need to discuss accurately on the actual cause of introduction in each case. The information that we get is usually the situation when the disease is obvious to local people (who in most cases have not specific training to detect this disease, which is new to them). This is to say that places, plants, and dates which are given in the relevant literature as the actual data of the first introduction in a new zone are probably never the real data.

126. In this context it just impossible to ascertain or to deny on the sole basis of the characteristics of the first assessment of fire blight in a new site the real origin of this introduction. At best some evidences can be assessed:

127. – Most first assessments in Europe where not in or near-by nurseries (UK, France, The Netherlands, Denmark...). Although it is likely that in any given place where orchards are planted at least some nurseries can be found, it seems that at least the zones of first assessment (Egypt, UK, France, Belgium…) do not coincide with zones of active nurseries.

128. – In the cases on which I can personally testify (Samson and Paulin 1972), first discoveries in France where in an area with no pear and apple orchards, and no nursery. The first symptoms were found on *Crateagu*s constituting hedges around meadows for cattle breeding. The same situation was for Denmark and western coast of Germany (Schlesvig-Holstein) some years before. In these cases at least, the introduction through nursery stock seems very unlikely, and birds, or wind-driven rain (or insects) carrying bacteria from actively oozing orchards in South East UK were proposed as vectors- but this was impossible to prove.

129. – The first introduction, in any part of the world, has never been associated (Van de Zwet and Bonn, 2000) with the vicinity of fruit storage or import premises. Many examples may be found of introductions that are not fully explained, but I have never heard in these introductions of the hypothesis or even the likelihood of an introduction with import of fruits.

130. – In my personal experience with fire blight that I have had the opportunity to visit soon after its supposed first introduction (UK, Denmark, France, Belgium, Israël, Lebanon, Syria…), the most common suggested cause of introduction was "wind driven bacteria" rather than nursery stocks.

131. – It remains that the introduction with nursery stock is not only conceivable, but also easy to demonstrate experimentally. It has been described convincingly in several cases. But it is evident that it is not the sole mode of dispersion of the disease, and probably not even the most frequent.

Question 17
Paragraphs 357-359 of Australia's FWS refer to a paper by Billing and Berrie (Exhibit AUS-26), which appears to conclude that plant materials or fruit bins contaminated with E. amylovora may have been responsible for the introduction of fire blight into the United Kingdom. Are you aware of any scientific evidence confirming or experimentally testing this hypothesis? Does this paper provide reliable scientific evidence that the spread of fire blight may be related to the movement of mature apple fruit? How does any such evidence relate to the relevant evidence relied upon by Australia's IRA?

Dr Deckers:

132. The paper of Billing and Berrie formulates a hypothesis of possible fire blight introduction into the Kent area in UK but does not prove the way of introduction by mature fruit sufficiently scientifically. I had the opportunity to discuss this matter with Eve Billing in a personal discussion and she considers more the bacterial ooze as a factor of spread of the disease in the contaminated fruit boxes. Bacterial ooze can be present on fruits coming from heavily infected orchards.

Dr Paulin:

133. The paper from Billing and Berrie which is referred to by Australia is a scientific paper which is devoted to looking at any epidemiologically plausible explanation for the introduction of fire blight in south east UK which would be coherent with the activity of the disease as related to climate. It is by no means a work that intends to provide "scientific evidence". At best it gives indications on what

is "not impossible". In this context, it is normal that the discovery of crates originating from New Zealand found at that time in newly contaminated orchard is cited. As far as I know, the actual presence of bacteria on these bins has not been checked, and neither has been the transportation of this putative inoculum to receptive host plants. Therefore this fact does not add any element to the dispute. In addition this paper suggests "rotten pears", as possible vehicles for the bacteria, but never "symptomless apples". This could mean that these authors were not in the opinion that apple - or healthy looking fruits- could play a role in this spread? Keck et al (1995) demonstrated that on crates (wooden and plastic) artificially polluted with suspensions of bacteria, *E. amylovora* may survive up to 4 months at 4°C. That can be considered as a possible first step for this way of dissemination. But much would remain to be found to confirm that the complete succession of steps is likely to occur to reach the infection of a new host plant. May be it is relevant to indicate that the paper from Berrie and Billing ends with the following quotation: "Scientific knowledge is a body of statements of varying degrees of certainty, some most unsure, some nearly sure, none absolutely certain". It seems to me that this shows that the authors themselves were well aware that they were not providing any "scientific evidence".

134. This cannot be considered to support the evidence relied upon by Australia.

Question 18
Please comment on whether there is a sound scientific basis for claiming that mature, symptomless apples could harbour epiphytic (external) populations of E. amylovora and that a transfer mechanism exists that would be capable of transmitting E. amylovora from such apples to a susceptible host such that a fire blight infection would be initiated under natural (as opposed to laboratory) conditions? (Paras. 4.13 and 4.15 of New Zealand's FWS; paras. 250-262 of Australia's FWS; R 61 and 62 by New Zealand)

Dr Deckers:

135. Mature symptomless apple fruits coming from heavily infected orchards can harbour an epiphytic population of EA bacteria in their calyx end or as bacterial ooze sticked on the fruit skin and dried out, but the chance that this bacterial population will be capable to start a new infection on a susceptible host plant under natural conditions is rather low.

136. When the fruits are harvested from orchard without active fire blight symptoms in and around the orchard, without the presence of hail and when the fruits have been disinfected during preparation in the packing house, the chance for an initiation and establishment of the disease under natural conditions in another area is considered to be extremely low.

Dr Paulin:

137. As explained earlier (question 6), mature symptomless apple may harbour some surface cells of *E. amylovora,* at least if collected in an orchard showing active symptoms before cropping time. The only "mechanisms" I can think of for the transfer of such bacteria to infection site on a living susceptible host plant at the proper stage of receptivity are:

- Insects (whatever they are, pollinating or not), which could take bacterial cells to open blossoms, or to wounds on growing shoots,

- Wind driven rain.

138. These two "mechanisms" suppose a close proximity between these fruits and the infection sites. Both are questionable, due to the expected low or very low level of the bacterial population present on these fruits, and its localisation (calyx), which makes the accessibility of cells rather

difficult. In addition, bacterial cells on fruit are probably not embedded in ooze (as they are when actively multiplying from active lesions) and therefore not well protected from adverse conditions, and, which is more, probably have not the adhesive capacity, which is said to be a facilitating factor for transportation by insects**.**

Question 19
Please comment on whether there is sound scientific evidence that epiphytic infestations by E. amylovora exist on mature apple fruit in quantities that are under natural conditions capable of reproduction; being transferred to a host plant; and ultimately initiating an infection in that host plant? (Paras. 4.14-4.30 of New Zealand's FWS; paras. 250-262 of Australia's FWS)

Dr Deckers:

139. There will be no multiplication of the epiphytic bacterial population on the fruit surface or in the calyx tissue. The fire blight bacteria are not surviving well as an epiphytic bacterial population. The bacteria should be transferred first to a susceptible organ of a fire blight host plant like a stigma of a flower where the bacteria can multiply and start a new infection. The chance for such a successful transfer and multiplication of bacteria will be rather exceptional.

Dr Paulin:

140. We have already commented on the probability of the presence of residual population of bacteria on fruit surface. Some people prefer to use the word "epiphytic" exclusively for microorganisms that are able to multiply, and therefore to reach high level of population on leaves or plant surfaces, without producing symptoms. This is typically the case of some phytopathogenic bacteria called *Pseudomonas syringae* (some pathovars of this species). Such a capacity does not exist in the case of *E. amylovora* (Thomson 2000), except on stigma in flowers. Therefore the bacterial cells eventually present on the fruit surface could not multiply on the same site. They should be first carried to a suitable site for multiplication (i.e. an infection site, presumably an open flower on a host plant).

141. The transfer to an host plant could be performed most likely by insects or wind driven rain, but the probability of successful localization at the right place should be very low, and the probability of infection even lower. The highest probability I can think of is a pollinating insect taking the few bacterial cells to the hypantium of a flower of an host plant. This remains unlikely because trace bacterial populations (not multiplying) will 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.

Question 20
Please comment on whether the requirements identified in Australia's IRA, regarding disinfection treatment of apples in the packing house and the disinfecting of packing house equipment before each Australian packing run, are scientifically justified and reasonable? Are you aware of any reliable scientific evidence of E. amylovora from harvested apples contaminating packing house equipment? If so, would such bacteria survive the packing house process? Are you aware of any reliable scientific evidence of mechanical transfer in a natural environment of E. amylovora from workers' hands to susceptible hosts? (Paras. 4.43-4.47, 4.147, 4.386 of New Zealand's FWS; paras. 855-859 of Australia's FWS; para. 37 of the United States' Third Party submission; R 40 by Australia)

Dr Deckers:

142. Disinfection of the apples in the packing house can reduce potential epiphytic populations of EA on the fruits considerably. Disinfection of the packing house equipment before each Australian packing run has not sufficient scientific base; it is not at all sure that the EA bacteria would be able to survive into the process water within the whole population of other bacteria and fungi. Mechanical transfer of the bacteria in a natural environment from workers hand to a susceptible host seems to be extremely unlikely.

Dr Paulin:

143. The disinfection of apple could be justified when arriving in the packinghouse, if these are sourced from orchards showing fire blight symptoms. In this particular case, external pollution by exudate oozing from active symptoms of the disease is possible on near-by fruits. In addition, it cannot be ascertain that fruits with symptoms, as well as trashes infected by the bacteria are not mixed with healthy mature fruits during cropping in the orchard. These trashes may carry an internal inoculum which could "leak" into the water used to wash the fruits, and consequently contaminate the fruits to be exported. I see no scientific evidence that the bacterial population will not survive the packinghouse process for some times. Again this is only in the case where fruits are sourced from orchards having shown, or still showing, active symptoms of fire bight.

144. Mechanical transfer in a natural environment of phytopathogenic bacteria by worker's hands to susceptible host has never been published, or observed. The only case I know of for a bacterial disease is for *Clavibacter michiganensis* pv *michiganensis*, a tomato pathogen, which is transferred from plant to plant on a row, while the plants are hand-trimmed. In this case the biology of the bacteria is completely different (it actively multiplies in the vessels). Nothing similar for *E. amylovora*.

Question 21
Please comment on whether the consideration in Australia's IRA of the risk associated with the practice of packing houses leaving orchard wholesaler waste uncovered and exposed to the elements on the premises or in landfills is objective and credible, taking into account the likelihood of this situation occurring in packing houses in Australia. (IRA, Part B, p. 82 (fire blight); paras. 4.130 and 4.419-4.421 of New Zealand's FWS; paras. 784-785 and 898-900 of Australia's FWS; and R 100 by Australia)

Dr Deckers:

145. The packinghouses involved in the export of apples from New Zealand should not leave waste uncovered and exposed because this fruit waste can form a risk of contamination for the environment. But this requirement should be included in the standard operating procedures for each packing house.

Dr Paulin:

146. I have personally no idea on the likelihood of the possibility that wastes from fruit packing houses be uncovered and exposed to elements, although I imagine it is low as it should be in any well organized country. This being said, 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. It is important to add that *E. amylovora* cannot constitute the only bacterial population

on these fruits and, being a poor competitor, *E. amylovora* has good chances to disappear, due to trophic competition with other microorganisms (see question 30 below) on decaying fruits, or to antibiosis.

Question 22
The likelihood assigned to importation step 1 in Australia's IRA is based on the finding that the E. amylovora organism can be present in orchards even if disease symptoms are not detected, or the orchard is surrounded by infected alternative hosts. (IRA, Part B, pp. 53-54; para. 4.210 of New Zealand's FWS; paras. 379-382 of Australia's FWS)

a. What is the scientific basis contained in the IRA for this finding?

Dr Paulin:

147. The assumption that *E. amylovora* can be present in orchards even if disease symptoms are not detected (I suppose the same year) is based on the fact that the bacterium, once it has infected a plant, may migrate inside the tree, sometimes in the xylem, or in peripheric tissues (inner bark). At the end of the growing season, the downward progression stops, and a canker is formed, which may be "determinate" or "undeterminate" (in other word visible, or invisible). The bacteria will remain alive in these cankers in winter, and sometimes (but not always) show a new multiplication in the tissues and a production of ooze, providing inoculum for new infections in spring. This cycle is well known (Thomson, 2000): it may result in the presence of undetected bacterial population in the trunks and branches. This population though are unlikely to pollute the fruit surface, being internal into the tree.

148. Another source of bacteria in a healthy orchard could be through wind-driven rain or insects or birds carried bacteria constituted by ooze from near-by (but possibly unseen) "alternative" host plants such as wild hawthorn, which may be difficult to survey, and which presence is sometimes not even acknowledged. These bacteria will disappear soon, because *E. amylovora* is not a good epiphyte, in most of the cases, but they may cause late infection which, progressing slowly if weather conditions are not conducive to symptom expression, may constitute a bacterial population in a symptomless orchard. These two possibilities are probably more likely in the case of New Zealand, where the history of fire blight is very long on the same sites. (The European concept of "protected zone" is probably not applicable in this case.)

Dr Sgrillo:

149. The detection of fire blight in New Zealand both from orchards with fire blight symptoms and those without symptoms.[13]

b. Is this finding based on respected and qualified scientific sources?

Dr Paulin:

150. These two possibilities are well documented. It is more difficult to assess the frequency of such (provisionally) hidden bacterial populations, and of their real importance and role.

[13] (*footnote original*) IRA p. 53.

Dr Sgrillo:

151. Yes, the findings are based in respected and qualified scientific source (Clark et al., 1993).[14] However, the objective of this paper was to test a DNA approach to identify *E.amylovora*. The objectives have not included a survey of the fire blight populations in New Zealand.

c. Please comment on whether the reasoning articulated by the IRA on the basis of such scientific evidence, including the methodologies applied, is objective and coherent, and whether the particular conclusions drawn in the IRA find sufficient support in the available scientific evidence?

Dr Paulin:

152. The reasoning seems objective and coherent. Each stage is based on scientific evidence.

Dr Sgrillo:

153. The reasoning is mainly based on the paper of Clark et al. (1993)[15] who presents results from evaluations of several seasons. The 1987 results are described by the author (p. 62):

> "In 5 orchards (A-E) where fire blight symptoms were seen during inspections at flowering, *Erwinia amylovora* was detected in the immature fruit samples..."
>
> "In 2 orchards (F and G) with no fire blight symptoms at flowering *Erwinia amylovora* was also detected in calyxes of immature fruit. Further inspections at the immature fruit stage revealed the presence of infected alternative host in close proximity to these orchards. In 4 other orchards (H-K) with no fire blight symptoms at any stage during the season *Erwinia amylovora* was not detected using DNA hybridization testing method."[16]

154. The 1988 results of the sampling are (p.62):

> "In 1988 Erwinia amylovora was detected in calyx of immature fruit from 3 orchards ... and further detailed inspection revealed symptoms of fire blight in the orchard"

155. The 1989/91 results were not considered here because the natural environment around the orchards were artificially changed (the alternative host in the surroundings were eliminated) (p. 62).

156. The results indicate that *Erwinia amylovora* is not present in orchards that have no symptoms and are distant from other hosts.

d. Do the results of the IRA's assessment in this regard sufficiently warrant the challenged requirements related to fire blight?

Dr Paulin:

157. Yes.

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[14] (*footnote original*) Exhibit NZ-53: Clark, R.G., Hale, C.N. and Harte, D. (1993) A DNA approach to *Erwinia amylovora* detection in large scale apple testing and in epidemiological studies. *Acta Horticulturae* **338**: 59-66.

[15] Ibidem.

[16] Ibidem.

Dr Sgrillo:

158. The IRA's assessment is not sufficiently supported. Clark et al. (1993)[17] have sampled around 10 orchards, each year, from 1987 to 1991. However the representativeness of the sample in time and space can not be evaluated. The sampling methodology was not appropriate to survey fire blight populations in New Zealand. Therefore there are limitations to use the results with this purpose.

e. In your view, was it methodologically sound for the IRA Team not to assess any apple producing areas of New Zealand that would be free of E. amylovora?

Dr Paulin:

159. The conclusion that no orchard in New Zealand can be considered free of *E. amylovora* seems soundly based.

Dr Sgrillo:

160. No, the results show that it would be possible to find orchards free from *E. amylovora* (see item *c* of this response).

f. Please comment on the probability of 1 contained in the IRA for the presence of E. amylovora in the source orchards for importation step 1. Does this probability fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA?

Dr Paulin:

161. If the probability of 1 means that all orchards are contaminated by *E. amylovora* each year, it is probably a mere exaggeration. For example the fate of bacterial population in canker is either to disappear (Beer, 1978) or to multiply and produce symptoms. In this later case, the orchard is no longer symptomless. Therefore I would say that each apple orchard symptom-free in New Zealand may be temporarily contaminated by *E. amylovora*, not permanently. Therefore the chance for apples to be sourced from orchards harbouring *E. amylovora* should be significantly less than one.

Dr Schrader:

162. The assumption, that orchards in New Zealand are 100% infested with *E. amylovora* lacks sufficient scientific evidence.

Dr Sgrillo:

163. Probability of 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.

[17] (*footnote original*) Exhibit NZ-53: Clark, R.G., Hale, C.N. and Harte, D. (1993) A DNA approach to *Erwinia amylovora* detection in large scale apple testing and in epidemiological studies. *Acta Horticulturae* **338**: 59-66.

Dr Deckers (Response to whole question):

164. Epiphytic contamination of the apple fruits is possible, even when there is no EA present in the orchard itself, but when active EA infections are present on a host plant in the surroundings. In the PhD work of Schouten (Studies on fire blight, 1991, Landbouw universiteit Wagenigen, promoter Dr J.C. Zadoks) the role of *Crataegus* hedges was discussed as a possible infection source around pear orchards. When there is really no EA infection present in the orchard or in the immediate surroundings, it will not be possible to isolate EA bacteria from the fruits. The bufferzones around the orchard that should be free of EA host plants are comparable with the bufferzones around the fruit tree nurseries for the fire blight control.

Question 23
What is the relevance of importation step 1 (presence of E. amylovora in the source orchard) for the risk assessment contained in Australia's IRA in light of the fact that importation step 2 aims to assess the likelihood of picked fruit being infested/infected with E. amylovora?

Dr Deckers:

165. The presence of a fire blight in the orchard increases the risk of epiphytically contaminated fruits in the immediate neighbourhood of the infection. One fire blight infection in an orchard does not mean a complete contamination of all the fruits of the orchard.

Dr Paulin:

166. In my view, relevance of step 1 is major for the risk assessment. Step 2 assesses likelihood of contamination of fruits. This likelihood is directly related to the history of fire blight the year of cropping (presence or not of active symptoms producing inoculum) and the years before (possibility of internal presence of *E .amylovora* in the xylem. Even if this possibility has very little chance to result in internal fruit contamination (especially in mature symptomless apples), these chances are nil if fire blight has not been seen in the orchard for years (or ever).

Dr Sgrillo:

167. IRA does not present results of the sensitivity analysis of the model. Thus the relative relevance of each step in estimating the probability of importation can not be assessed.

168. If the probability of step 1 is set to 1, then this step is unnecessary and could be removed from the model. However the model was developed to be applied by different pests that may not have probability 1 in this step, such as *N. Galligena.*[18]

169. Also, step 1 could be included in step 2. The probability of the fruit picked be infested could be assessed including the probability of the pest be present in the orchard. In this case, however, some transparency would be lost.

Question 24
Are the conclusions in Australia's IRA as to the probability range and distribution patterns (triangular distribution with a minimum value of 1 x 10E-3, a maximum value of 5 x 10E-2 and a most likely value of 3 x 10E-2) for importation step 2 (the likelihood of picked fruit being infested/infected with E. amylovora) sufficiently supported by the available scientific evidence?

[18] (*footnote original*) IRA p. 118.

Dr Sgrillo:

170. The figures available are contradictory and variable[19]. The IRA (pg 55) recognizes the contradictory nature of the available data:

> "The available literature in the context of this assessment is divided; some data supports the presence of *E. amylovora* as infestation (external) or infection (internal), while other data supports its absence from fruit".

171. The IRA Team has chosen a triangular distribution with the most probable value corresponding to 3% infestation. The scientific evidence presented in IRA does not guarantee that this is true. By selecting different set of results it would be possible justify different values for the parameters of the distribution.

Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources?

Dr Sgrillo:

172. The papers cited by IRA seem to be qualified. However, possibly due to the methodology used by each author and to the cultivar, place and season where the samples were extracted, etc., the results are not comparable.

Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, p. 55-65; paras. 4.213-4.220 of New Zealand's FWS; and para. 391-411 of Australia's FWS)

Dr Deckers (Response to whole question):

173. The value of 3 X 10 E-2 seems to be a quite high rate of picked fruit being infected with EA. I think there will be more a gradual distribution of presence of the fire blight bacteria on the fruits around the EA infections present in the orchards.

Dr Paulin:

174. I consider that it is just not possible to provide a probability range from the scientific data taken into account in this discussion. Each paper deals with its own type of fruit (mature or not, sometimes not precisely indicated), its own technique of detection of the bacteria, etc... No general feature for the presence of *E. amylovora* on/in mature apple fruit can be seriously based on these results. The range of frequencies indicated on the table 4 from AUS FWS (from <1% to 75%) just shows that, in these papers, different things were analysed differently. Consequently, it could be not valid to aggregate these technical data. Therefore, I am in the opinion that this evaluation is not scientifically based, cannot be objective and, as shown on table 4, is just not credible as a whole.

175. Most of the scientific sources are respected and credible. Nevertheless, the van der Zwet et al.,1990, paper which is criticized by its own first author should have been removed from the list. Other data, such as in the paper from Sholberg et al, 1988, seems to be very specific, and could represent but a case history, which was probably very different from a normal situation, that would be an orchard devoted to export of apples. The paper on bacteria in seeds and ovule (Mundt and Hinkle,1976) does not provide any proof that *E. amylovora* is present in seeds of apple; which is worse it does not provide clues as to the identity of isolated bacteria with *E. amylovora*. On the other

[19] (*footnote original*) IRA pp. 55-64.

hand the mechanisms described for *E. amylovora* conservation (EPS, VBNC, quorum sensing, sigma factors) do show accurately that *E. amylovora* cells may survive in adverse conditions, including on apples sourced from contaminated orchards. But this information does not allow a quantitative evaluation. It indicates that these surviving populations are of low level, that their capacity of resuscitation (VBNC) is possible but not demonstrated in natural conditions. Anyhow these surviving cells can be expected to be less if the apples are sourced from orchards without active symptoms, if the apples are symptomless, if no trashes are mixed with mature symptomless apples.

Question 25
Please comment on the following statement in Australia's IRA: "Given the widespread distribution of fire blight in New Zealand, the IRA team concluded that more weight should be given to those studies on apples sourced from orchards that were showing symptoms of fire blight disease". Is this statement based on an objective and coherent reasoning? (IRA, Part B, p. 65; para. 385 of Australia's FWS; and para. 65 of the United States' Third Party submission)

Dr Deckers:

176. This statement doesn't take into account the sporadic character of the fire blight disease in an infected orchard; this means that one year with EA infection will alternate with years with a much lower fire blight incidence; there will also be years without EA symptoms even in an infected orchard. It is important to mention here that also the other host plants should be observed for a possible presence of EA infections.

Dr Paulin:

177. It is coherent to assume that no orchard in New Zealand is or has been permanently free of fire blight. That does not mean that all orchards permanently show active symptoms. Therefore it seems that orchards in New Zealand should be considered as a patchwork of orchards with symptoms, and orchards showing no symptom. Then I do not see why more weight should be given to one category (with symptom) only. All the studies, weighted according to their own scientific value should be considered with the same level of interest. The exact fire blight situation of these orchards analysed in these papers and the relevance of the techniques used should be the criteria, if some of them were to be selected as more important than others.

Dr Sgrillo:

178. More weight should be given to studies with more reliable methodology and with more representative sampling. A sample of 30,000 fruits from several orchards is more reliable than a sample of 100 fruits from a single orchard, artificially inoculated, regardless of their results.

Question 26
Are the conclusions in Australia's IRA as to the probability range and distribution patterns (triangular distribution with a minimum value of 1 x 10E-3, a maximum value of 3 x 10E-2, and a most likely value of 1 x 10E-2) for importation step 3 (the likelihood of contamination by E. amylovora during picking and transportation to a packing house described in importation step 3) sufficiently supported by the available scientific evidence?

Dr Sgrillo:

179. The scientific evidence is scarce, coming mainly from two papers. One by van der Zwet et al. (1990)[20] says that:

> "Only 3 of 72 uninoculated and non-disinfested fruit developed blight symptoms - all through injury in the puncture treatment".

180. The IRA Team calculated that 4% (3/72) of the fruits damaged would be contaminated by fire blight[21]. The reliability of this result is low because the sample size was small, the variability was not assessed and the results are valid only for artificially injured fruits.

181. The other paper by Hetzroni et al. (2004)[22] is a four paragraph abstract that says:

> "The results show that at the time of the first check, the percentage of damaged apples was the lowest in the careful picking treatment: about 8% compared with 37% in the whole container in the packing house".

182. This abstract is also poor in details about the methodology and analysis of the results.

183. The 4% (0.04) figure was then multiplied by 37% (0.37) to obtain an indication of the magnitude of the most probable value of the triangular distribution used.[23]

184. The conclusions of the IRA are not well supported. Considering the small sample size used in the first paper and the lack of information on the methodology and on the details of the results of the second paper, the soundness of the scientific support is not well defined.

(ii) Is this evaluation objective and credible on the basis of the available scientific evidence?

Dr Sgrillo:

185. The evaluation is objective, but the available scientific evidence is not sufficient to support it.

Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, pp. 65-71; paras. 4.221-4.224 of New Zealand's FWS; and paras. 412-413 and 439 of Australia's FWS)

Dr Deckers (Response to whole question):

186. The likelihood of contamination by EA during picking and transportation is possible when the harvest takes place in a heavy infected orchard during rainy circumstances, but the overall chance of 1% seems to be rather high when the fire blight infections are only sporadically present in an orchard.

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[20] (*footnote original*) Exhibit AUS-31: van der Zwet T, Thomson SV, Covey RP and Bonn WG (1990) "Population of Erwinia amylovora on external and internal apple fruit tissues", Plant Disease 74(9): 711-716 (p. 713).

[21] (*footnote original*) IRA p. 70.

[22] (*footnote original*) Hetzroni, A., A. Bechar, I. Nir, A. Antler, S. Yosef, N. Shamir 2004. Mechanical injuries in Apples. Retrieved 24 January 2009, from http://old.agri.gov.il/AGEN/Reports/hetzroni003.html.

[23] (*footnote original*) IRA p. 70.

Dr Paulin (Response to whole question):

187. The available scientific evidence shows that *E. amylovora* is not a true "epiphyte", hence it cannot multiply, but only survive, with decreasing population on contaminated surfaces. It could multiply, and then maintain a high level of population, only if it were able to infect the plant. On a mature fruit this possibility of infection does not exist. Mature fruits are then concerned only at best with transient populations, which are likely to be soon disappearing. In addition, these transient populations would be present in the case where active, ooze producing fire blight lesions are present in the orchard at, or just before, picking time. Such a condition seems easy to avoid.

188. Therefore mature symptomless fruit will not bring in a packing house significant population of *E. amylovora* on their surface. Consequently, the evaluation of risk for this step seems too high, for mature symptomless fruits. Decaying fruits and trashes would represent a higher risk.

Question 27
Please comment on whether the reasoning in Australia's IRA regarding the number of E. amylovora bacteria isolated from, and reported on, mature apple fruit that would be sufficient to spread to a susceptible host and initiate an infection under natural (as opposed to laboratory) conditions. Is this evaluation objective and credible? Is it based on respected and qualified scientific sources? (IRA, Part B, p. 69-70; para. 4.224 of New Zealand's FWS; paras. 361-362 and 418 of Australia's FWS)

Dr Deckers:

189. The chance that the epiphytic bacteria that can be present on mature apple fruits initiate an infection on an other host plant will be very low. The critical point will be the transfer of viable EA bacteria to susceptible organs of an other host plant where the bacteria can multiply before a new infection can take place.

Dr Paulin:

190. The number of 38 cells needed to initiate an infection on apple shoot, as obtained by Crosse et al., 1972, seems a soundly established basis for the minimal number of cells able to initiate an infection, when introduced artificially at the proper site of the suitable plant, in optimal conditions for the disease. Unfortunately it gives very few useful indications for the description of events taking place in natural conditions. It has to be noted that the 38 cells reported by Crosse et al. were issued from a culture on artificial medium: they were actively growing cells, not dormant cells at the stationary stage as would be bacterial cells surviving on apple surface. It has to be reminded that *E. amylovora* is not able to multiply on plant surface (except for a short time, on the hypantium of stigmates in flowers). It is difficult to imagine conditions conducive to actively growing cells in natural conditions on the surface of a symptomless apple.

191. The spread of surface population from fruit to infection sites is similarly hard to imagine, especially because these non-multiplying cells are not embedded in exudate, and therefore not attractive to insects or other vectors. In artificial inoculations, bacterial populations at low level need to be placed very precisely at the right site of infection, to successfully infect its host plant (Crosse et al.). This is probably a difficulty impossible for the bacteria to tackle in natural conditions.

Question 28
Are the conclusions in Australia's IRA as to the probability range and distribution patterns (triangular distribution with a minimum value of 0.3, a maximum value of 0.7, and a most likely value of 0.65) for importation step 4 (the likelihood that E. amylovora would survive routine processing procedures in the packing house) sufficiently supported by the available scientific evidence?

Dr Sgrillo:

192. Australia's IRA states that "None of the processes [routine processing procedures in the packing house] undertaken at this stage would have a large influence on the survival of *E. amylovora* on apple fruit".[24]

193. However this statement is not compatible with papers cited by IRA, as follows.

> "Maas Geesteranus and de Vries (1984) refer to reduced survival in pre-cooling."[25]
>
> "Roberts and Reymond (1989) and Goodman (1983) showed the efficacy of washing in removing infestation of *E.amylovora*".[26]
>
> "Toivonen et al. (2001) showed that sodium hypochlorite (100 µg per liter) or peroxyacetic acid at 80 ppm was fully effective in eliminating micro-organisms from the surface of apples".[27]
>
> "Janisiewicz and van der Zwet (1988) reported that 12 mg per L of sodium hypochlorite in vitro totally eradicated *E. amylovora* in 5 minutes".[28]
>
> "Roberts and Reymond (1989) artificially inoculated mature apple fruit with E. amylovora aerosol solutions (8.0×10^6 to 1.3×10^8 per fruit) and immersed fruit in different concentrations (250, 300, 400, 500 ppm) of sodium hypochlorite or 1.0 M acetic acid. The reduction in the *E. amylovora* population averaged 6 to 7 log units less than the number applied to the fruit, but significant differences between treatments were not observed".[29]
>
> "Sholberg et al. (1988) inoculated fruit by swabbing calyces of apples with an average of 107 cfu per mL of *E. amylovora*. These authors demonstrated that the initial population decreased to an undetectable level after 6 months in cold storage".[30]

194. Also IRA refers that "Sorting and grading to remove visibly damaged fruit would reduce the number of apples potentially carrying infections".[31]

195. IRA mentions that 90% of the crop in New Zealand is submitted to pre-cooling treatment routinely and that 37% of packing houses use chlorine in the dump tank with the concentration that varies between 5 and 50%. Also 16% of packing houses uses peroxyacetic acid (Tsunami®), and bromo-chloro-dimethylhydantoin (Nylate ®), which totalize 53% of packing houses being disinfected.[32]

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[24] (*footnote original*) IRA p. 76.
[25] (*footnote original*) IRA p. 71.
[26] Ibidem.
[27] (*footnote original*) IRA p. 72.
[28] (*footnote original*) IRA p. 71.
[29] (*footnote original*) IRA p. 73.
[30] (*footnote original*) IRA p. 75.
[31] (*footnote original*) IRA p 74.
[32] (*footnote original*) IRA pp. 71-72.

196. If it is accepted that the epiphytic population of *E. amylovora* is reduced/removed by pre-cooling[33] and disinfection treatment then the survival in the packing houses would be 0.047 [(1-0.9) * (1-0.53)] that is much less than the minimum value of 0.3.

197. Australia states that "Given the risk scenario addressed by the IRA Team, *E. amylovora* will be taken to have survived this step even if only one bacterium survives routine pack house procedures on any given apple".[34]

198. It is implicit in the above statement the acceptance of the hypothesis that *fruit with 1, 10 or 1 million bacteria has exactly the same probability of initiating an infection*.

199. However, for the majority of plant pathogens, this is not true. Usually a number of conditions have to occur simultaneously to allow infection. The probability that an infected fruit, with one bacterium, starts an infection is different from the probability of a fruit that is infested with 10,000 bacteria. It is known that the probability of establishment is a function of the initial population size.[35]

200. The dose-response curve may present a threshold for the inoculums concentration, below which no infection will occur. We can read in IRA:

> "Van der Zwet et al. (1994) showed that five bacteria were sufficient to cause fire blight symptoms in apple flowers in one season, but in another season a minimum of 5000 bacteria per blossom were required for infection to occur".[36]
>
> …
>
> "Experiments were conducted in New Zealand (Hale et al., 1996) to determine the number of *E. amylovora* cells required to infect apple and cotoneaster flowers. These authors reported that when flowers were inoculated with 1 to 10^4 cfu per flower, there were no disease symptoms and *E. amylovora* was not detected. Fire blight symptoms were only observed when the inoculum dose of *E. amylovora* exceeded 10^6 cfu (Taylor et al., 2003b). Such populations may exist in fruit from heavily infected orchards, but not in fruit from lightly infected or symptomless orchards (Hale and Taylor, 1999). Similar observations were made by Beer and Norelli (1975), who reported that infections are likely to occur when epiphytic populations of *E. amylovora* reached 10^6 to 10^7 cfu under high relative humidity and not at 10^2 cfu"[37].

201. If it is accepted that there is a relationship between the dose of inoculum and the probability of starting a new infection then the decrease of quantity of inoculum may be enough to break the pathogen cycle, having the same effect as the total elimination of the inoculum.

202. It can be concluded that the scientific evidence available does not fully support the values 0.3, 0.65 and 0.7 chosen as parameters for the triangular distribution describing the survival of *E. amylovora* during the routine processing procedures.

Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be

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[33] (*footnote original*) New Zealand's FWS para. 4.226.
[34] (*footnote original*) Australia's FWS para. 421.
[35] (*footnote original*) Reference 01 Liebhold, A.M., W.L. Macdonald, D. Bergdahl, and V.C. Mastro. 1995. Invasion by Exotic Forest Pests: A Threat to Forest Ecosystems. Forest Science Monographs 30. 49 pp.
[36] (*footnote original*) IRA p. 88.
[37] Ibidem.

considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, p. 71-76; para. 4.225 of New Zealand's FWS; and paras. 419-420 of Australia's FWS)

Dr Sgrillo:

203. Part of the evaluation is objective and credible, based on qualified scientific sources and falls within a range that could be considered legitimate. However the values chosen for the parameters are not supported.

Dr Deckers (Response to whole question):

204. The disinfection process during packaging should reduce the risk of survival of the epiphytic population strongly and reduce the distribution pattern substantially. But a total removal of the bacterial population will not be an easy task.

Dr Paulin (Response to whole question):

205. If routine procedure in New Zealand packing houses does not include a disinfectant bath for fruits, I do not see which factor could effectively markedly decrease the supposed bacterial population on fruit surface. I would then consider that processing in the packing house has no influence on the level of bacterial contaminants on fruits. The scientific bases of the IRA conclusions are data on the resistance of *E. amylovora* in adverse conditions, such as low temperature and dessication. It seems that most data tend to show that cool temperature will reduce *E. amylovora* populations over time. Anyhow, 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. It seems certain that, if conditions in the packing house tend to reduce *E. amylovora* population, they will not allow the complete disappearance of these bacteria. The fact that VBNC state has been demonstrated in laboratory conditions for *E. amylovora* adds a potential for survival (whatever the frequency of cells at the VBNC state, and the chance of resuscitation, which is controversial in natural condition).

206. Conversely, if a disinfectant step is included in the process, the decrease of the level of bacterial population can be expected to be sharp. Nevertheless, it will never be possible to prove that a chemical disinfection procedure of biological material is always 100% safe in natural conditions.

207. Therefore, in the case of fruit disinfection, the probability range and pattern distribution for this step seems too high for this step. This applies for mature symptomless apples, but would not be different for trashes associated with fruits.

Question 29
Australia's IRA considers the impact of cold storage and cold conditions on populations of E. amylovora under importation steps 4 and 6. Please comment on the reasoning in the IRA regarding these matters (including whether cold storage will greatly decrease populations of E. amylovora on mature apple fruit, whether cold temperatures prolong the survival of E. amylovora bacteria on mature apple fruit and whether there is a time limit at which E. amylovora bacteria populations on mature apple fruit decline to undetectable levels under cold storage temperatures). Is this evaluation objective and credible? Is it based on respected and qualified scientific sources? (IRA, Part B, pp. 71-76; para. 424 of Australia's FWS; and para. 67 of the United States' Third Party submission)

Dr Deckers:

208. Cold storage of the fruits will reduce the bacterial population of EA on the fruits but will not eliminate the EA populations on the fruits completely. In principle the EA bacteria can easily survive a cold storage period.

Dr Paulin:

209. Data on preservation of bacterial population on apple surface during cold storage seems to accurately show that there is a decrease in these conditions. It is not possible to know if this decrease is linked with temperature, or only with time: because bacteria are not able to multiply, their population decreases naturally, and this decrease could be faster at room temperature; in the precise case of risk of transport of bacteria with mature symptomless fruits anyhow, this has little influence: the conditions in the packing house will not allow the population to increase, but will not allow the population to disappear within the considered period of time (few days?). Especially because of the possibility of VBNC, a time limit can not be accurately determined for the maximal survival of *E. amylovora.*

Question 30
Are the conclusions in Australia's IRA as to the probability range and distribution patterns (triangular distribution with a minimum value of 1 x 10E-3, a maximum value of 5 x 10E-2, and a most likely value of 2.5 x 10E-2) for importation step 5 (the likelihood that apples entering packing houses free of E. amylovora become contaminated during processing) sufficiently supported by the available scientific evidence?

Dr Sgrillo:

210. IRA states: "Ceroni et al. (2004) immersed pear fruit for 15 min in a suspension of *E. amylovora* of 10^8 cfu/mL and could not detect bacteria on the surface after just a few days, with small numbers remaining for longer periods only in the calyx. These authors concluded that bacterial survival on the fruit surface is very short and has a negligible epidemiological role. If *E. amylovora* gets into the core in the dump tank, one would expect some internal infection to develop but this has never been reported."[38]

211. Considering that 10^8 cfu/mL is 10^6-10^7 folds higher[39] than the expected concentration in the dump tanks then the probability that apples become infested during processing should be negligible. However IRA concludes that 2.5% (most probable value) of the fruits would be contaminated during process.

212. IRA informs in Summary of step 5:

> "This conclusion [shape and parameters of the distribution] was based on the potential for the fruit dump tank to become contaminated by bacteria and the fact that disinfection of the dump tank water is not a routine practice in a significant number of New Zealand packing houses".[40]

213. The scientific evidence presented in IRA does not guarantee the probability range chosen.

[38] (*footnote original*) IRA p. 78.
[39] Ibidem.
[40] Ibidem.

Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources?

Dr Sgrillo:

214. The scientific sources are qualified but do not support the conclusions of IRA.

Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? Does it take into account available scientific evidence of E. amylovora contamination of mature apple fruit by packing shed machinery? (IRA, Part B, p. 77-79; para. 4.228 of New Zealand's FWS; and para. 439 of Australia's FWS)

Dr Deckers (Response to whole question):

215. This step in the IRA is not sufficiently in accordance with the standards of the scientific community and the chance that this contamination of apples entering free of EA happens during processing is neglectible when the water during processing is disinfected.

Dr Paulin (whole question):

216. The liquid medium (in which mature symptomless apple fruits are immersed during the process), even without disinfectant, can not be considered as a culture medium for *E. amylovora*: an artificial medium for such bacteria must content among other elements a rather high level of soluble sugar (0,5g/l is a minimum, 5g/l is the standard for a culture medium for *E. amylovora*). Therefore it is rather a dilution effect that could be expected from this step. In this particular case, the probability suggested in the IRA seems to be strongly exaggerated. Only if decaying apples (supposedly decaying from *E. amylovora* infection-then immature and not "symptomless") or large amount of infected trashes, were present, the dilution effect in a non-disinfectant medium could lead to a significant amount of bacterial cells on fruit surfaces. This seems very unlikely in practical conditions.

217. The scientific evidence is that, in artificial medium *E. amylovora* does not compete very successfully against natural antagonistic bacteria (such as *Pantoea agglomerans* or *Pseudomonas fluorescens,* Vanneste, 2008), which are naturally found in high concentration on plant and on fruit surface, and which would compete with *E. amylovora*, thus preventing a multiplication of *E. amylovora*, if present. Finally I would therefore consider that the risk of contamination of apples by packing shed machinery is negligible.

Question 31
Are the conclusions in Australia's IRA as to the probability range and distribution patterns (triangular distribution with a minimum value of value of 0.7, a maximum value of 1, and a most likely value of 0.8) for importation step 6 sufficiently supported by the available scientific evidence? Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? Are such likelihood values based on a finding in the IRA that E. amylovora could survive in epidemiologically significant numbers after palletisation, quality inspection, containerisation and transportation of apple fruit to Australia? If so, is such a finding based on respected and qualified scientific sources? (IRA, Part B, p. 79; para. 4.229 of New Zealand's FWS; and paras. 361 and 431-432 of Australia's FWS)

Dr Deckers:

218. The survival of the EA bacteria during palletisation, containerisation and transport is considered to be low, surely after the external disinfection of the fruits during the packaging process. The cold storage itself will not be able to eliminate EA populations completely.

Dr Paulin:

219. The supposition made by the IRA relies on the evaluation of the survival of *E. amylovora*, during a short period (10 days), which is expected to provoke but a small decrease in bacterial populations. It seems reasonable to take this period into consideration, because it seems agreed that it corresponds to the minimum of time needed for New Zealand-Australia journey. In practice, the period could be longer, and therefore the expected decrease could be a little more, but this does not need to be considered further. At any rate it cannot be relied upon this period of expected decreasing survival to secure a "sterilisation" of supposedly contaminated fruits. The evaluation IRA for this step seems correct.

Question 32
Are the conclusions in Australia's IRA as to the probability range and distribution patterns (triangular distribution with a minimum value of 0, a maximum value of 1 x 10E-6, and a most likely value of 5 × 10E-7) for importation step 7 (the likelihood of clean fruit being contaminated by E. amylovora during palletisation, quality inspection, containerisation and transportation) sufficiently supported by the available scientific evidence? Is this evaluation objective and credible on the basis of the available scientific evidence?

Dr Sgrillo:

220. The scientific evidence is scarce and the IRA is not fully supported by it.

221. The conclusions of the IRA are based in the paper from van der Zwet et al. (1990).[41]

> "Such fruit (with bacteria oozing) are rarely found (van der Zwet et al., 1990), as rotten fruit is not harvested. If harvested, such fruit is rejected before entering the packing line. If rotten fruit is present after cold storage, it is discarded at quality inspection".[42]

222. From the citation above it can be concluded that the final probability of step 7 is composed mainly by three partial probabilities: the probability of a rotten fruit be harvested; the probability of a rotten fruit entering the pack line and the probability of a rotten fruit pass the quality inspection.

223. If the final probability is taken to be 1E-6 (as the maximum value) then each of the three partial probabilities (if they are assumed to be equal) would have the value of 0.01 (because 0.01^3 = 1E-6). This is equivalent to accept that 1% of the rotten fruit would be harvest and that 1% of the rotten fruit harvested will enter the packing line and that 1% of the rotten fruit will pass the quality inspections. One percent seems to be a very large proportion for events that actually would have a negligible probability of occurrence.

[41] (*footnote original*) Exhibit AUS-31: van der Zwet T, Thomson SV, Covey RP and Bonn WG (1990) "Population of Erwinia amylovora on external and internal apple fruit tissues", Plant Disease 74(9): 711-716.
[42] (*footnote original*) IRA p. 79.

224. However if the three partial probabilities are taken to be negligible (according to IRA definition: 1E-6 as maximum value) then the maximum value for the probability of step 7 would be 1E-18 ($1E\text{-}6^3$) what is practically equal to zero.

(iii) Is it based on respected and qualified scientific sources?

Dr Sgrillo:

225. The scientific source is qualified but is not adequate to support the conclusions in the IRA.

Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, p. 79; para. 4.232 of New Zealand's FWS; and para. 438-439 of Australia's FWS)

Dr Deckers (Response to whole question):

226. This risk is neglectible. There is no scientific data available that demonstrate the risk of contamination during palletisation, quality inspection, containerisation and transportation.

Dr Paulin (Response to whole question):

227. In its analysis of this step, IRA does not provide any scientific evidence that such external pollution can happen, except in the case of oozing fruits. It referred to van der Zwet 1999 paper, which has already been considered as not providing the correct information on the case, which the author recognizes himself. In addition, internally infected fruits immature producing ooze, if any, would have been discarded well before this step.

228. I would consider the probability to be nil in this case, for symptomless mature apples.

Question 33
Please comment on the evaluation in Australia's IRA at importation step 7 of the risk of surface contamination of mature clean fruit on an export packing line through bacteria oozing out from internally infected fruit. Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Please comment in this regard on the conclusions reached in the 1990 study by van der Zwet et al and their relation to mature fruit. (IRA, Part B, p. 79; para. 4.215 of New Zealand's FWS; and para. 70 of the United States' Third Party submission)

Dr Deckers:

229. Internally infected mature fruits will not be able to produce bacterial ooze. These fruits will immediately be invaded by fungal infections. Ooze production occurs only on immature fruits where the starch of the immature fruits is used by the EA bacteria during the multiplication phase.

Dr Paulin:

230. Ooze can only be produced following a progressive invasion of susceptible tissue by the bacteria. Oozing out from mature fruit is not described in fire blight symptoms. It could possibly (?) happen in the case of a delayed evolution originating from an infection of an immature fruit, but as far as I know, this as not been described in the scientific literature. The only scientific basis for oozing on mature fruit (?) is from van der Zwet 1990, which has already been discussed, and which can be considered as irrelevant for the case.

Question 34
When the various likelihoods from importation steps 1-8 are inserted into the IRA's risk simulation model, the probability of importation of E. amylovora is estimated as being 3.9 x 10E-2 (mean), 2.2 x 10E-2 (5th percentile) and 5.6 x 10E-2 (95th percentile). The mean infestation rate for E. amylovora is estimated at 3.9% of all apples imported from New Zealand. Please comment on whether the IRA's conclusion on the probability of importation of E. amylovora is sufficiently supported by the available scientific evidence.

Dr Sgrillo:

231. Scientific evidences on the rates of infestation of apples imported from New Zealand are scarce.

232. There are two papers[43] that evaluate infestation in mature apples in orchards sampled in New Zealand.

233. Hale et al., 1987[44] says:

> "*E. amylovora* was isolated only from 3 of 400 fruits (cv. Gala) harvested from the severely infected orchard. In each instance *E. amylovora* was detected only in washings from the calyx-end. *E. amylovora* was not isolated from 1300 fruit (cv. Gala) harvested from 2 lightly infected orchards and 3 orchards where no fire blight symptoms were seen."

234. Hale and Taylor (1999)[45], who evaluated the infestation of *E.amylovora* in orchards with and without fire blight symptoms, describe:

> Fruit from orchards with fire blight symptoms - *E.amylovora* was detected in 2% of fruit before cool storage but not in any fruit after either cool storage, or cool storage and incubation. *E. amylovora* was not isolates from any fruit tested. Fruit from orchard without fire blight symptoms - *E. amylovora* was neither detected in, nor isolated from any of the fruit tested before or after cool storage or after cool storage and incubation.

235. The results of these studies suggest that the level of infestation generated by the IRA's model (3.9%) is greater than what would occur in reality.

Please also comment whether these overall probability results are correct based on the values given to importation steps 1 to 8. (IRA, Part B, p. 80; para. 4.235 of New Zealand's FWS)

Dr Sgrillo:

236. The overall probability results are correct and based on the values given to the importation steps, according to the model presented in Table 4 of the IRA.[46]

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[43] (*footnote original*) Australia's FWS para. 398 (Table 4).
[44] (*footnote original*) Exhibit NZ-21: Hale, CN, EM McRae and SV Thomson (1987) "Occurrence of Erwinia amylovora on apple fruit in New Zealand" Acta Horticulturae 217, 33-40.
[45] (*footnote original*) Exhibit NZ-24: Hale, CN and RK Taylor (1999) "Effect of cool storage on survival of Erwinia amylovora in apple calyxes" Acta Horticulturae 489,139-143.
[46] (*footnote original*) IRA p. 24.

Dr Deckers (Response to whole question):

237. In the IRA risk simulation, they make an addition of the different risks for each importation step and this gives a mean risk of 3.9%. This is a relative high percentage and could be overestimated.

Dr Paulin (Response to whole question):

238. As explained above, the different steps of import are of diverse influence on the overall probability of importation of *E. amylovora*. It seems to me that, if one can speculate or discuss on the likelihood of any event involved in the possible transport of *E. amylovora* with apples, the quantification of probabilities of each one of these events is just not feasible. This quantification relies on an arbitrary estimation, which, even in the best-documented case, is just hidden behind a "scientific" explanation, which is never completely relevant, if only because the conditions in the laboratory are only partially mimicking natural conditions. This quantification of probability may have a merit in trying to assess the relative risks attached to each step, as compared to each other.

239. The overall figure resulting from the combination of these probabilities is just not credible: if the 3.9% figure had any consistency, it is a figure that could be quite easily checked experimentally (as is, for example, spread through planting material). Such an experiment would have been more convincing than the present efforts by IRA to demonstrate what cannot be really demonstrated.

Question 35
Please comment on the finding in Australia's IRA that E. amylovora can under natural conditions be transmitted to a susceptible host via insects feeding on discarded apples leading to the initiation of a fire blight infection. Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Is it based on respected and qualified scientific sources? When undertaking a risk assessment, how should potential vectors be factored in? In your view, would a rigorous methodology oblige the IRA Team to disregard a potential vector because it has never been shown to "demonstrate" transmission of E. amylovora? Are there respected and qualified scientific sources to support the position that there may be a pathway for the spread of E. amylovora from apples to susceptible hosts by way of "mechanical processes" in a natural environment? (IRA, Part B, pp. 80-90; paras. 4.22-4.26 and 4.243 of New Zealand's FWS; and paras. 418 and 465-476 of Australia's FWS)

Dr Deckers:

240. Feeding of insects on discarded apple fruits is not described in the biological cyclus of EA as a factor for the spread of the disease. Insects that are more important for a possible spread of the disease are the pollinating insects because they can transfer the bacteria to a flower where the infection can take place. An insect feeding on a discarded fruit is not considered to be a normal way of spreading the disease between an infected fruit and an other host plant. 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.

Dr Paulin:

241. 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.

242. Many insects are supposed to be able to transmit the bacteria from a source (ooze) to an host plant. To be of some effectiveness, this should take the bacteria right to the infection site *(i.e.* stigma

in flower, or young tissue of a growing shoot). That reduces the period of time were this spread is effective to full bloom, and to growing periods of shoots: that is only few weeks within a year, even taking account of the blossom periods, and growing periods of the many diverse host plant of fire blight.

243. As far as insects are concerned, I am in the opinion that any insect that is able to travel from a source of inoculum (drop of ooze) to an infection site can be considered as a potential vector. It could be considered more dangerous if it is a pollinating insect, because it goes to the right place, more receptive, on the plant, or if it is a browser of fresh watery tissues. The list of insects involved in *E. amylovora* transport is neither complete nor limitative. It is an assessment of what has been seen, or thought to be vectors of the bacteria. To my knowledge, there is no specificity between any given insect and *E .amylovora*.

244. Apart from these general considerations, I see no scientific source that could account for mechanical transmission of *E. amylovora* from fruits to infection sites.

Question 36
Are the values presented in the section of Australia's IRA headed "Exposure" regarding the likelihood of transfer of E. amylovora from infested or infected mature apples to a susceptible host plant (uniform distribution with a minimum value of 0 and a maximum value of 1 x 10E-6) sufficiently supported by the available scientific evidence? (IRA, Part B, pp. 85-90; paras. 4.249-4.252 of New Zealand's FWS; and paras. 370-371 of Australia's FWS)

Dr Deckers:

245. For this aspect there is no sufficient scientific data available that describes the likelihood of this transfer possibility.

Dr Paulin:

246. In this section only some fragments of events 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.

Dr Sgrillo:

247. The literature review presented by the IRA allowed the development of the hypothesis that *E. amylovora* can be transferred from a mature apple fruit to susceptible hosts, beginning an infection.

248. The transfer of *E. amylovora*, from mature infested/infected fruits to new hosts, would require several successive events to occur, each with its own partial likelihood, as described in the IRA[47].

249. The arguments are logical but nevertheless there are no reported cases of this event. Thus the scientific evidence presented does not support the conclusions because there are no factual data to validate the hypothesis.

250. As an exercise to evaluate the conclusions of the IRA Team it could be given values for the partial likelihood of one of the possible paths, as follows:

(a) Probability that the bacteria survive in discarded waste

[47] (*footnote original*) IRA pp. 85-90.

(b) Probability of the bacteria to retain its viability

(c) Probability that the inoculum dose is adequate

(d) Probability that a vector pick the bacteria in sufficient concentration

(e) Probability that the vector find a host

(f) Probability that the host is in a susceptible stage

(g) Probability that the environmental factor be favorable

251. It can be assigned, as an exercise, the value of 0.01 (1%) as the most probable value of each partial probability. The maximum exposure value would be, then, 1.E-14 what is more than 50 million times lower than 5E-7 (the arithmetic average adopted by the IRA team) and practically equal to zero. This would be more appropriate to represent an event that has never been reported to occur.

Question 37
Does the IRA contain an objective and credible analysis, based on respected and qualified scientific sources, for a proposition that the introduction of fire blight via mature apple fruit has ever occurred or could occur, either experimentally or under natural conditions or that populations of E. amylovora on mature apple fruit could be the source of fire blight infections under natural conditions? Are you aware of any scientific evidence outside of the IRA for such a proposition?

Dr Deckers:

252. As mentioned earlier there is a possibility that mature apple fruit can harbour viable epiphytic EA bacteria. The step of the transfer from these infected fruits to the possible host plant stays the most critical step and will be difficult to prove.

Dr Paulin:

253. No, I do not see any objective analysis for that proposition in the IRA. To be honest , as a scientist, I do not think it is possible to experiment in this matter. I do not know of any scientific evidence in this field. (This does not mean that mature apple fruit may not be a source of fire blight infection, of course).

Question 38
Please comment on the finding in Australia's IRA that one E. amylovora bacterium or a very limited number of such bacteria on the calyx of a mature apple could be spread to a susceptible host and initiate a fire blight infection under natural conditions. Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? (IRA, Part B, p. 88-92; Australia's FWS paras. 362, 449, and 458-459; New Zealand's FWS paras. 4.14 and 4.244; and R 63 by the Parties)

Dr Deckers:

254. To realise an EA infection, there is a need for bacterial multiplication before an infection occurs. This multiplication should take place on a susceptible organ like on the stigma of a flower of a susceptible host plant. The transfer from calyx of the apple to the susceptible organ is the critical step in the infection and this transfer chance is rather low.

Dr Paulin:

255. The question basically at issue is the ability of a very low number of *E. amylovora* cells (even only one), to provoke an infection. First of all, it could be useful to remind that the experimental manipulation of very low level of bacterial populations (say less than 100 cfu/ml) is extremely difficult to perform with a sufficient level of accuracy. In order to be sure to actually use this low number of cells, it is necessary to use particular statistical pattern of experiments, or special kind of experiments. Papers suggesting a minimal concentration threshold for infection by *E. amylovora* are not all really credible in this respect. If Crosse and Goodman's paper (1972) seems to establish clearly that less than 50 cells in experimental condition have the potential to infect a plant, results obtained in the field by van der Zwet (1994) are just technical data, indicating how many bacterial cells are necessary for an inoculum in the field to obtain a reasonable frequency of positive inoculations.

256. Nevertheless the fact that one (or very few) cell(s) of *E. amylovora* may have the potential to infect a plant is important for the biology of this bacteria, but does not imply that any one cell of *E. amylovora* in the open has a chance to provoke fire blight on a host plant. The key-point here is that the few cells inoculated in these scientific papers are cells at their optimum capacity: artificially grown on suitable medium as pure culture, they are collected during their phase of exponential growth. All these conditions, always strictly observed in experiments, maximise the potential of the bacterial population. In natural conditions, the bacterial cells would be placed in far less favourable conditions, and the number of cells needed to succeed in infection could be expected to be far higher. Therefore, between a passage of few cells in the calyx of an imported mature fruit to a suitable infection site followed by infection, a stage of multiplication of the bacteria is necessary. (see above)

257. As a consequence, the probability of bacteria from the calyx of mature apple to infect a plant supposes many steps. One only (infectivity of one or very few cells) is based on scientific evidence, but in condition very different from natural conditions.

Question 39
Please comment on the finding in Australia's IRA that populations of E. amylovora typically found at harvest can survive on apples for periods considerably longer than that needed to import, distribute and sell New Zealand apples in Australia when apples are in cold storage. Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? (IRA, Part B, pp. 75-76; paras. 4.17-4.19, 4.225-4.231 and 4.242 of New Zealand's FWS; paras. 419-426 and 457 of Australia's FWS)

Dr Deckers:

258. The storage of the apples at low temperature can reduce the epiphytical populations of EA but will not eliminate the epiphytic populations of EA in the calyx of the fruits. The period of survival can be longer than the period necessary for import, distribution and selling of the fruits in Australia.

Dr Paulin:

259. This evaluation is based on the fact that cold temperatures are usually good conditions for the conservation of culture of bacteria in the laboratory. Results of Temple et al (2007) which show that bacterial populations keep longer during cold storage (on pears) are therefore not surprising. Results from Hale and Taylor (1999) showing a tendency for bacteria to disappear on apple during cold storage are apparently conflicting with those of Temple et al. But this can be due to different conditions and/or different fruit (apple versus pear). These two scientific results seem both credible and objective.

260. In addition the recent information, that *E. amylovora* is able to turn to the VBNC state under certain conditions, which has been recently checked with *E. amylovora* placed in calyx of apples, demonstrates that the survival of *E. amylovora* (even if the resuscitation in natural conditions is unclear for these VBNC) lasts probably longer than initially assumed. Consequently, this evaluation by the IRA is objective and credible. Even if it remains impossible to quantify the importance of this long conservation.

Question 40
Please comment on the conclusion in Australia's IRA regarding rapid multiplication of bacteria in natural orchard environments. Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? (IRA, Part B, pp. 92-93; para. 4.245 of New Zealand's FWS; and paras. 470-471 of Australia's FWS)

Dr Deckers:

261. The EA bacteria can indeed multiply rapidly but not on the epiphytic surfaces of the fruits. This multiplication can only occur on susceptible organs like immature fruitlets or on the stigma of the flowers and only when the climatological conditions (temperature and relative humidity) are optimal for bacterial growth. The question here is if these circumstances will be present at the time that the fruits from New Zealand arrive in Australia.

Dr Paulin:

262. The rapid multiplication of *E. amylovora* in natural orchard environment can be observed only after infection (or artificial inoculation) of a susceptible host plant. The values indicated in the literature are strictly linked to the conditions in which they are obtained (no nutrient limitation, no water limitation, optimal and constant temperature). They are obtained from credible scientific sources, but need to be considered as the maximum potential for the bacterial multiplication, in absence of any limiting factor. In addition the multiplication rates obtained in the laboratory (for example on sections of immature fruits) follow relatively massive inoculations with young fresh bacterial cultures.

263. The most likely limiting factor for *E. amylovora* in orchard condition is the site where it could multiply. Except in laboratory conditions, no multiplication of *E. amylovora* outside an infection of host plant (the first step being on the hypantium) has ever been described. So a rapid multiplication is possible in an orchard, but only after infection, in the plant tissues.

Question 41
Please comment on whether Roberts and Sawyer (2008) is an objective and credible scientific analysis containing statistical data appropriately to measure the risk of importing E. amylovora on commercial apple fruit. Or does the Roberts and Sawyer (2008) analysis suffer from the three fundamental flaws argued by Australia in paras. 363-376 of its FWS? If your response to the latter question is yes, do these flaws result in seriously underestimating the fire blight risk associated with export apples, as argued by Australia in its response to Question 105 by the Panel? (Paras. 4.26 and 4.251 of New Zealand's FWS; paras. 363-376 of Australia's FWS; paras. 17-27 of the United States' Third Party submission; R 64 by New Zealand and R 105 by Australia)

Dr Deckers:

264. This publication is clearly written in function of the dispute between New Zealand and Australia with the aim to correct some of the data of the earlier publication of the same author.

265. The orchards that are described in the table 1 in the publication of Roberts and Sawyer 2008 can not be considered as a representative sample for the New Zealand orchard situation of fire blight. It is not clearly described in these orchards what control measures were taken in each orchard. The risk of these orchards is indeed a restricted risk because some fire blight control measures have been taken.

266. A second comment is that part of the bacteria can be present as VBNC (viable but not culturable) bacteria. This can be the case when copper treatments have been made in the orchards and this can be a very important aspect because the VBNC status can be temporary and become viable again.

267. A third comment is that the bacteria can be present at a low level and are difficult to detect at these low levels.

Dr Paulin:

268. The Roberts and Sawyers (2008) paper is an interesting piece of serious reasoning about the quantification of the probability of transport of fire blight with fruits. Because I am not a statistician, I will not comment of the figures given for probabilities and intervals.

269. As usual in this type of work, the key-step of the selection of the input data is very difficult to achieve, because it must refer to already published data, which have not been collected for the purpose of the present study: the data set can not completely satisfactory. Therefore a certain level of heterogeneity makes the statistical analysis quite difficult. In this case, the orchards under analysis were very different from each other, (but I do not understand the point made by Australia that only one is from New Zealand?), and the size of the samples (n° of fruits assayed) is highly variable (from 20 to more than 1000). This makes it difficult to analyse the data. In addition, the description of the situation of the orchard with respect to fire blight is probably too simple, and may reflect, under the same qualification, situations of the disease which may be completely different in the field.

270. From these data, the authors did their best to construct a sound reasoning with appropriate calculation. But due to the fact that many assumptions are done, even if I agree that they seem perfectly reasonable, I do not think that this paper may help objectively in the dispute. I am just unable, from the reading of this paper to tell if it over- or under-estimates the fire blight risk associated with fruits. I would say that it is an elaborated estimate of what could be the risk, and that the figures obtained seem credible, when each step is followed, and each assumption accepted.

Question 42
Please comment on the conclusions in Australia's IRA regarding the ability of E. amylovora to enter into a viable but non-culturable (VBNC) state in mature apples. Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Please comment in this regard the conclusions reached on this matter by Roberts and Sawyer (2008). (Para. 405 of Australia's FWS)

Dr Deckers:

271. The VBNC status of the bacteria is a known status that can be present under orchard conditions when Cu treatments are made as a chemical control method. This status can indeed interfere with the detectible level of EA bacteria in the epiphytical populations in the orchard.

Dr Paulin:

272. The ability of *E. amylovora* to enter into the viable but non-cultural state is established according to the standard in that sort of microbiological studies. There is no doubt that VBNC state exists for *E. amylovora* in the conditions used in the laboratory for this demonstration. That this VBNC state can be obtained with bacterial populations (artificially) placed in apple calyx is an additional confirmation of this capacity of *E. amylovora*. This ability is shared by many other bacteria, including *Enterobacteriaceae*, the taxonomic group in which is placed *E. amylovora*. The two open questions are: does VBNC occur in natural conditions from the small population of *E. amylovora* trapped in apple calyx? and, to what extent the resuscitation takes place in natural conditions, and if so, in which quantity? The VBNC state is somewhat a matter of controversy in microbiology, and considered by some as just a "pre-death" stage.

273. Anyhow, even if it must be considered that E. amylovora does have the ability to enter the VBNC state in the calyx of apple, and that resuscitation can occur, in laboratory conditions, the conclusions of Robert and Sawyer (2008) for P2 (Probability of survival of the pathogen during storage and transport) remain probably valid, due to the speculative incidence of this phenomenon, linked to the very low proportion of cells able to survive as VBNC, and to the complete absence of data on the occurrence of VBNC for *E. amylovora* in natural conditions.

Question 43
Please comment on the probability levels that could be reliably derived from the sample sizes reported in Hale et al. (1996) and Taylor et al. (2003a). Were those sample sizes large enough to detect events like the spread of fire blight when apples contaminated with E. amylovora are placed in an orchard? If not, were the sample sizes in these studies nevertheless appropriate? (IRA, Part B, pp. 80-90; paras. 4.22-4.26 and 4.243 of New Zealand's FWS; and paras. 474-475 of Australia's FWS)

Dr Deckers:

274. It is normal that the size of the samples for such a detailed study is limited. But this allows interesting conclusions concerning the survival rate under the different circumstances. Extrapolation of these data to the spread of the disease under different natural conditions on a larger scale under orchard conditions should be considered with prudence.

Dr Paulin:

275. Combining the sampling dates, the size of the number of fruits analysed reaches 683 fruits in Hale et al (1996). This size is probably not enough to detect a very rare event. Nevertheless it provides a valid information on fruit contamination by *E. amylovora* under these experimental conditions: such a contamination, whatever the distance of the fruit sampled from the source of bacteria, is not common. At least it is demonstrated by these results that the fruits picked from a tree showing symptoms are usually not surface contaminated with the bacteria, in these experimental conditions.

276. The size sample is similar (600) in Taylor et al (2002). It is again probably not enough to demonstrate the occurrence of a very rare event. But it has to be underlined that each of these apples were inoculated with a significant level of bacterial population. As such it can be supposed that this sample of 600 infested apples, placed in a blooming orchard maximizes the theoretical risk of introduction of *E. amylovora* with potentially infested apples fruits. It is probably the maximal size of a sample that can be handled properly in an experiment. The study, with these 600 fruits shows accurately that the "leak" of bacteria from fruit to blossoms does not normally take place.

Dr Sgrillo:

277. According the binomial distribution there is a relation (formula 5, appendix 3, ISPM 31)[48] between the proportion of the population infected and the probability of, at least, one infected unit be sampled. This relationship is illustrated in Figure 1 for a sample size of 1,830 units.

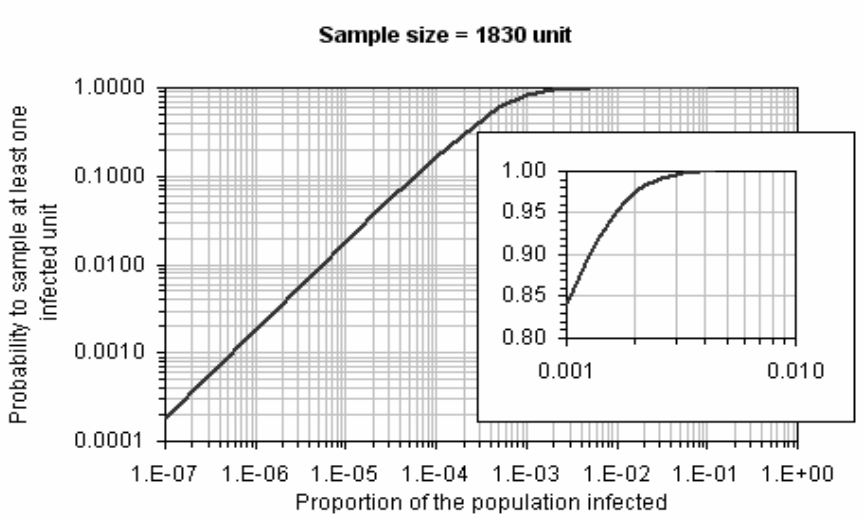

Figure 1. Relationship between the proportion of the population infected and the probability of, at least, one infected unit be sampled (ISPM 31[49]). The detail shows the upper portion of the curve.

278. It can be seen, in the detail of Figure 1, that the higher confidence will only occurs when the infection level in the population is above 0.001.

279. The sample size, reported in the paper, is small to detect very low levels of infection. Hale et al. (1996)[50] have used 30 fruits as source of inoculum to infect the orchard. The expected level of infection of the fruits in the orchard, considering a probability of infection of 1E-6 per fruit, would be 3E-5 (calculated from the Poisson distribution). To detect this level of infection, with 90% confidence level, it would be necessary sample of 76,752 fruits.

280. The confidence level provided by the sample size used (1,830 fruits) is 5%.

Question 44

Please comment on what factors, other than the volume of trade in apples between New Zealand and Chinese Taipei, would need to be taken into account to support a contention that New Zealand's experience in exporting apples to Chinese Taipei may be used to draw conclusions on the potential for fire blight to enter, establish or spread in Australia as a result of imports of New Zealand apples? Are the parameters described in the example mentioned by New Zealand in its first written submission regarding the possibility of transmission of fire blight through trade in apples in the next shipment

[48] (*footnote original*) Exhibit AUS-30: International Plant Protection Convention, International Standard for Phytosanitary Measures No. 31: Methodologies for sampling of consignments, 2008, from Report of the Third Session of the Commission on Phytosanitary Measures, Rome, 7-11 April 2008.

[49] (*footnote original*) Exhibit AUS-30: International Plant Protection Convention, International Standard for Phytosanitary Measures No. 31: Methodologies for sampling of consignments, 2008, from Report of the Third Session of the Commission on Phytosanitary Measures, Rome, 7-11 April 2008.

[50] (*footnote original*) Exhibit NZ-27: Hale CN, Taylor RK and Clark RG (1996) "Ecology and epidemiology of fire blight in New Zealand", Acta Horticulturae 411: 79-85.

from New Zealand or the United States to Chinese Taipei scientifically reasonable and justified? (Paras. 4.183-4.185 of New Zealand's FWS; para. 310 of Australia's FWS)

Dr Deckers:

281. It is not sure that the Taipei situation and the Australian situation is comparable. Is the climate for both export area comparable? Climatological parameters such as temperatures T max. and T min. and amount of rainfall during susceptible period like during the flowering time can be decisive for the EA risk. A second point is the availability of the host plants; are they comparable in both area?

Dr Paulin:

282. The main differences between Australia and Taipei, apart from volume of trade, and as far as introduction of fire blight is concerned, are probably two:

283. – Even if apples are produced in Taipei, apple orchards are not common, they are scattered onto mountains, for climatic reasons, and not concentrated in large production zones in the vicinity the big towns. It is very unlikely that packing houses for import fruits are placed in the vicinity of these orchards.

284. – Even if Rosaceous plants (crab apples, *Stransvaesia*) may be found in Taipei, especially in altitude (1500-2000 m), host plants of fire blight are not common in town plantations, in streets and family or public gardens and parks. They are quite rare, and *Cotoneaster, Pyracantha* or hawthorns are probably almost absent. They are the most common potential host plants of fire blight in many countries, and possibly in certain areas of Australia. Therefore, associated with the paucity of orchards, the number of potential host plants in Taipei for fire blight is far less, and this should be taken into account in the analysis.

285. – In addition, the climate is generally, for most of the years, in most of the places in the island, not favourable to *E. amylovora*, because too hot (maximal daily temperatures of more than 30°C are unfavourable to the disease).

Question 45
Please comment on the relevance, if any, of any absence of historical proof of a pathway for excluding or assessing a certain risk. Please take into account any IPPC standards on the matter. (R 65 by the Parties)

Dr Deckers:

286. Historical data can indicate indeed that a certain pathway of disease introduction is very unlikely and can give interesting indications on the real risk assessment.

Dr Paulin:

287. Generally speaking, absence of historical proof can be relevant to the estimation of a risk. Although it provides just an assumption of future absence, it can be enough in certain cases.

288. For fire blight, it is more difficult to rely on such an absence of historical evidence, for two reasons:

289. – Epidemies of fire blight are impossible to trace, conversely to epidemy of some human viral or bacterial diseases, because the pathogen (*E. amylovora*) shows a low level of variability. Even if

some differences between strains have been observed, they are not enough to characterize a strain found in a new place and to link accurately the new outbreak to one particular source of inoculum. Some attempts have been presented in the literature (Jock et al. 2002) but they remain very limited, and not completely convincing.

290. – Conversely to human diseases, plant diseases, even when carefully surveyed in some places like fire blight are not officially assessed with a rigor and a frequency which would provide definite clues for the introduction of the disease in a new site. (See question 16).

291. Actually in the general situation for fire blight, the origin of inoculum is just a matter of assumptions and suppositions. Nothing is really proven, except in some cases, notably when the recent introduction of contaminated host plants is involved. But very often these plants are illegal imports, and not traceable.

292. Against this background, the absence of historical proof is not *per se* a sufficient reason to exclude a possibility. Given the importance of international trade of apples, between contaminated and non-contaminated countries in the world, the total absence of historical proof (or even suspicion) shows at least that fruits do not constitute a common pathway for introduction of fire bight in a new area.

Dr Schrader:

293. The absence of historical proof of a pathway would not be a scientifically sound reason for excluding a certain risk regarding the entry of a pest, as conditions and situations may change, e.g., the concentration of the pest in the area of origin, the application of treatments in an infested area, the frequency and volume of pathways, etc.

Question 46
Does Australia's IRA provide an objective and coherent assessment of the likelihood and implications of New Zealand apples being repacked at rural packing houses in close proximity to orchards, when assessing the risks related to fire blight, European canker and ALCM? Was such assessment made with proper methodological rigour? (Para. 4.418 of New Zealand's FWS; R 99 by Australia)

Dr Deckers:

294. This assessment is not convincing and seems not to be based on objective criteria.

Dr Paulin:

295. The assessment made by IRA is apparently coherent, but I do not see how are evaluated the relative levels of probability for each situation. Besides, I do not see the point, if apples from New Zealand are imported as ready to sold. This question seems to me of minor importance for fire blight risks. I think that the time of import (whether it takes place during a period of receptivity of host plants or not) is more important in the risk assessment than the site of import and (hypothetical) packaging.

Dr Sgrillo:

296. The Australia's IRA considers two extremes scenarios: a) from 70% to 100% of apples being repacked at rural packing houses and b) from 0.1% to 5% of apples being repacked at rural packing houses.[51]

297. As Australia states that "the probability values derived from the two different P1 values barely differ at all"[52] there are no implications whether the imported apples will be repacked or not.

Question 47
Is the requirement identified in Australia's IRA that a packing house provide details of the layout of the premises, sufficiently justified by the scientific evidence relied upon? (IRA, Part B, pp. 317; para. 4.149 of, and pp. 242 and 247 of Annex 4 to, New Zealand's FWS; and para. 963 of Australia's FWS)

Dr Deckers:

298. I don't see the scientific evidence for that measure.

Dr Paulin:

299. Very few scientific data, if any, support the risks of contamination of fruits by *Erwinia amylovora* in the packing houses. It seems that the requirements of providing details of the layout of the premises is not based on any "scientific evidence".

Question 48
Please comment on whether, from a technical perspective and as described in Australia's IRA, the 17 specific measures that have been challenged by New Zealand can be distinguished as either measures active in risk reduction, or measures designed to implement or support active measures. (R 14-26 by the Parties)

Dr Deckers:

300. I think that all the measures described in Australia's IRA can be considered as measures that reduce the risk for infections actively directly or indirectly. Some of the measures describe the implementation of the measures, but in fact the result for all these measures is always with the intention to reduce the infection risks.

Dr Paulin:

301. I would consider that measures that are active in risk reduction are measures that directly result in decreasing the chance of fruits to carry *E. amylovora*. In this category I would place:

– Apples sourced from areas free from disease symptoms,

– Orchard be suspended in case of pruning suspected to be devoted to cancel symptoms,

– Orchards be suspended in case of fire blight symptoms,

[51] (*footnote original*) IRA Table 21 p. 97.
[52] (*footnote original*) R 99 by Australia.

– Apples be subjected to disinfection,

– Grading and packing equipment be cleaned and disinfected.

302. These measures could be supposed to be supported by the followings:

– Orchards are inspected for fire blight disease symptoms, in order to minimize the available inoculum at the time of picking the fruits,

– An orchard methodology inspection be developed..., *in order to guarantee a level of safety and objectivity to the inspection,*

– Packing houses be registered for export process only fruits sourced from registered orchard, *in order to minimize the risk of pollution of apples to be exported by bacteria possibly contaminating apples for local market,*

– AQIS be involved in inspection for fire blight,

– AQIS be involved in verification of packing houses,

– New Zealand guarantees that export orchard are registered,

– Lay out of the premises of packinghouses is made available.

Dr Schrader:

303. Principally and without going into detail for each of the 17 measures, a distinction between measures active in risk reduction and measures for implementing active measures can be made. E.g. measure No. 3 (The requirement that an orchard/block inspection methodology be developed and approved that addresses issues such as visibility of symptoms in the tops of trees, the inspection time needed and the number of trees to be inspected to meet the efficacy level, and training and certification of inspectors) can be seen as an implementing measure to measure No. 1 (The requirement that apples be sourced from areas free from fire blight disease symptoms). See also comments on systems approaches below (answer to question 140).

III. EUROPEAN CANKER

Question 49

Please comment on whether an apple fruit that is naturally (rather than experimentally) endophytically infected or epiphytically infested with European canker can still develop into a healthy-looking mature fruit. Based on Australia's IRA, please comment also whether any of the challenged requirements imposed by Australia with respect to European canker are based on a finding that such situation is possible. If so:
a. What is the scientific basis contained in the IRA for such a finding?
b. Is the finding in the IRA in this regard based on respected and qualified scientific sources?
c. Is the reasoning articulated by the IRA on the basis of such scientific evidence, including the methodologies applied, objective and coherent?
d. Do the results of the IRA's assessment in this regard sufficiently warrant the challenged requirements related to European canker?

Dr Deckers:

304. It is possible that apple fruit is infected naturally endophytically with NG and still develops to a normal looking fruit at the end of the season and that the NG infection shows up only after a storage period of some months.

305. The epiphytical infection of NG will not be so frequent in comparison with the endophytical infection.

Dr Latorre:

306. Australia's IRA was based mainly on the possibility that mature apples may carry latent infections, which cannot be detected at harvesting or during processing in the packing house. Latent infections may occur in a small proportion of the fruits harvested from cankered trees if frequent summer rainfalls occurred at harvest. Fruit infections are negligible or extremely low in areas with dry climate conditions at harvest. If cankered trees are not prevalent (0% infected trees), I would not expect to observe any latent infections, even under high summer rainfalls. Therefore, the risk of entrance would vary considerably based on climate conditions and disease prevalence and severity.

307. Endophytic *N. Galligena* has been suggested to occur on young apple trees, which may be important because it could explain the development of European canker in young apple orchards, particularly in areas where other sources of primary inocula are nonexistent (Langrell, 2002, Mycol. Res. 106:280-292; McCracken *et al.,* 2003). However, to my knowledge, there is no information demonstrating that *N. Galligena* can be found endophytically on mature apple fruits. Therefore, an endophytically infected fruit is a possibility rather than a real issue, which needs to be proven before admitting this as an important mechanism for long-distance dissemination of *N. Galligena*.

308. There is no information demonstrating that conidia or ascospores of *N. Galligena* can survive epiphytically (as surface contaminant) on mature apple fruits. It is important to consider that conidia survive desiccation for relatively short periods and it would be very unlikely that conidia and ascospores, contaminating apple surfaces, can survive post-harvest fruit management.

309. 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 (Importation step 5) is negligible and irrelevant. Arguments supporting this conclusion have been provided by Australia (AUS-BA Part B, p. 127). Nevertheless, this possibility was analyzed by Australia's IRA, which acknowledged that a small number of spores could contaminate fruits in the packing process. However, there is no experimental information convincingly supporting this conclusion. Therefore, this was a rather arbitrary decision that should be reviewed.

Dr Swinburne:

310. *Epiphytes* are organisms that can not merely survive, but can actually grow (at least to some extent) on the intact surface of plants drawing on nutrients leached through the cuticle. There is no evidence that *N. Galligena* can survive as an epiphyte *per se.* However, it is possible that the surface of an apple fruit could become contaminated by spores washed down from active canker lesions by rain. During the summer season these would be conidia. The data obtained by Dubin & English (1974) implies that conidia on the fruit surface would only survive for a few days if humidity was maintained at 100%. At lower humidity (85%) the half-life of conidia would be a matter of hours. Unlike some other species responsible for apple rots e.g. *Phomopsis mali*, (Ayob & Swinburne, 1970),

conidia of *N.galligena* are not able to initiate infection through the intact cuticle of fruit, consequently conidia adhering to the unbroken surface are unlikely to survive for long periods or contribute to fruit rots.

311. *Endophytes* are organisms which can grow within the tissues (intra-cellular or in the transpiration stream within the xylem) without provoking overt symptoms. There is some evidence (Dewey, Li & Swinburne, 1995) that *N. Galligena* can survive and be transported within the xylem into regions of the wood that are remote from canker lesions. Whilst this may be superficially symptomless, internal staining of affected areas implies that cellular damage is taking place, thus *N. Galligena* can not be classed as an endophyte.

312. A central contention of the IRA is that fruit at the time of harvest can be infected but show no symptoms of rotting. Apple fruit of all cultivars (see Q55) can express resistance to rotting by *N. Galligena* during their early developmental stages, up to and usually including harvestable maturity. Where wood canker lesions are prevalent, and the weather conditions are conducive to conidia production throughout fruit development, it is inevitable that infection (in the strict sense) of fruit can take place at any time. These infections will involve some colonisation of cells in lenticels, around growth cracks in the well at the stem end, or within the open area at the calyx. Cells in the immediate neighbourhood will respond by producing benzoic acid, which is fungitoxic. Further growth of the fungus may be prevented permanently or temporarily. If the latter, then the term most applicable is that a quiescent infection has occurred. As apples ripen two major changes, reduction in the acidity and increase in soluble sugars, reduce the toxicity of benzoic acid, enabling the fungus to resume growth and progressively rot the fruit. For most cultivars in current commercial production this will occur after harvest. Consequently it is possible for infected fruit of all varieties to be harvested with no visible symptoms (see Q55).

313. The IRA makes the statement that dessert apple varieties rot with *N. Galligena* before harvest quoting Swinburne, 1975. This actually refers to the observations made by Dillon-Western (1927), with the cultivar Worcester. This cv. ripens exceptionally early, often before harvest and which can not be held in store for long periods; all reasons why it has lost favour in commerce. It is untypical of modern dessert cultivars, in which most rots are seen post-harvest.

314. The sequence of events outlined above are applicable to regions where wood cankers are frequent and weather conditions favour production of conidia during the summer months (e.g. the U.K., and N. Ireland in particular). The presence of stem lesions alone can not predict the likelihood of fruit infection. It is unfortunate that there is so little data on the causes and extent of rotting of fruit in New Zealand, but what there is suggests that summer weather conditions are not favourable for infections by *N. galligena* and the challenged requirements seem excessive (see Q72).

Question 50

According to respected and qualified scientific sources, can export-quality mature apple fruit carry the causal organism of European canker (either internally or externally)? Please comment on the timeframe for an infected mature apple fruit (taking into account varietal differences) to develop visible symptoms of European canker. Did Australia's IRA take into account relevant evidence in this respect regarding fruit harvested from an orchard?

Dr Deckers:

315. Export quality apple fruit can carry the NG fungal infection internally on susceptible apple varieties. The chance for externally presence of the disease on the fruit skin is not expected to be high because the fungicide treatments made at the end of the growing season against storage diseases can have an effect on the NG that should be present externally on the fruit skin.

Dr Latorre:

316. On the basis of the information discussed in AUS-2 BA, Australia's IRA considered biological and epidemiological evidence currently available with respect to European canker.

317. To my knowledge, the external contamination (epiphytically) of apple fruits with *N. Galligena* has not been documented scientifically; it is very possible that external contamination does not occur or has no epidemiological consequences. Therefore, the probability that mature fruits carry *N. Galligena* externally should be equal to zero, and disregarded in the risk analysis.

318. However, it is feasible that *N. Galligena* can develop as a latent infection at harvest and hence, apparently healthy (asymptomatic) mature apples eventually could carry *N. Galligena* internally. Infected but asymptomatic fruits would be impossible to differentiate from healthy fruits at harvest or during post-harvest processing.

319. The timeframe for an infected mature apple fruit to develop visible symptoms varies from a few (6 to 7) days to several weeks or months (2 to 3 months), primarily depending on apple variety and on ambient temperatures. Highly susceptible varieties become rotted and drop off in the orchard before harvesting. This considerably reduces the risk of entrance of *N. Galligena* on mature apples.

Dr Swinburne:

320. As outlined above (Q49) apples can have quiescent infections at harvest. The time-frame for the subsequent development of overt rots rests on a number of variables which have not been explored experimentally in detail. These include the cultivar, the time of infection and the conditions imposed during the storage period. Low temperature coupled with controlled atmospheres can and will delay rot development in any cultivar. The conditions within the store play a major role in the speed of development of rots (Berrie, Xu &Johnson 2007, and appendix 1). Obviously the longer fruit remain in store the more rots develop.

Question 51
Based on Australia's IRA, do the challenged requirements imposed by Australia with respect to European canker provide for any tolerance of the presence of N. Galligena? Please comment on whether the following terms express different concepts: "area freedom", "pest free places of production", "freedom of the disease" and "freedom of the visible symptoms of the disease". How relevant is a distinction between these concepts in the context of Australia's measures for European canker? In light of Australia's IRA and Australia's R36 to the Panel, how do these various terms relate to relevant ISPMs, in particular ISPMs Nos. 4, 5, 10 and 22? (Paras. 4.443 and 4.447 of New Zealand's FWS; paras. 156-160 of Australia's FWS; R 33-35 by New Zealand)

Dr Deckers:

321. Depending on the different weather conditions , there will be different situations concerning the NG presence and development in orchards. For NG freedom of visible symptoms of NG does not mean that there can not be a hidden presence of the disease with a delayed symptom expression e.g.on fruits with an internal infection of NG. Also infections of NG on young fruit trees coming from infected fruit tree nurseries can be present for some months before the symptom expression becomes visible on the trees.

Dr Latorre:

322. In the challenged requirements with regard to *N. Galligena*, a disease tolerance level was provided by Australia for fruits imported from New Zealand; this disease tolerance level was implicit

in the import risk analysis (IRA) (AUS-2 BA, p. 152 and p. 113). Australia's IRA recognized disease tolerance while establishing an appropriate level of protection (ALOP) in their pest risk analysis (PRA) approach, which was part of the overall IRA. According to Australia, the ALOP reflects the maximal acceptable risk (or expected loss) from a disease incursion in Australia. On the basis of this analysis, a LOW annual probability of entry, establishment and spread (PPEES) was assumed for fruits imported from New Zealand, and MODERATE consequences were established if European canker eventually established and spread in Australia. The interaction of these two factors allowed Australia to conclude that a LOW unrestricted annual risk exists. Therefore, management measures to mitigate this risk were proposed.

323. The PRA, and particularly the ALOP, is the crucial aspect of this dispute with regard to European canker. Australia considers PPEES low. However, there is a general perception that PPEES is extremely low or negligible in other apple-producing countries. Data provided by Australia to support their conclusion appear to be insufficient. For instance, data to validate the probability of *N. Galligena* entrance via asymptomatic fruits has not been provided; similarly, data supporting the probability of establishment and spread were not presented.

324. The long experience of other exporting countries where European canker is present (e.g., Chile, United States) suggests that the probability that asymptomatic fruits carrying latent infection may introduce *N. Galligena* into a new area is negligible (extremely rare), rather than low. This probability would increase if apples were harvested from infected orchards located in areas with high summer rainfalls. Therefore, the risk of long-distance disease spread by infected fruits (fruits with latent infection or visible symptoms of the disease) should be considered extremely low or negligible until sufficient experimental evidence is provided to neglect this conclusion.

325. "Area freedom" was used to denote "pest-free area", and "freedom of the disease" was used to denote "pest freedom," as explained below. "Freedom from visible symptoms of the disease" is a pathological expression meaning asymptomatic (=symptomless) or apparently healthy. Asymptomatic fruits may or may not have latent infections.

326. The following terms have been defined previously by FAO (International Standards for Phytosanitary Measures, ISPM); they are defined and used primarily in the International Standards for Phytosanitary Measures (ISPM) Nºs 4, 5 and 10 and, to my understanding, they are correctly applied by Australia's IRA. ISPM 22 refers to a related subject, "Requirements for the Establishment of Areas of Low Pest Prevalence":

327. **Pest-free area**, an area where it has been scientifically demonstrated that a specific pest (pest or disease) does not occur and in which, where appropriate, this condition is being officially maintained. The objectives of the pest-free area and the pest-free place of production are similar, but are implemented differently. A pest-free area is much larger than a place of production. It may include several places of production and it may extend to a whole country or parts of several countries. A pest-free area may be isolated by a natural barrier or buffer zones. A pest-free area is officially maintained by the National Plant Protection Organization (NPPO) of the exporting country over many years without interruption. This concept is defined in ISPM 5 and the requirements for the establishment of a pest-free area are discussed in ISPM 4.

328. **Pest-free place of production,** a production place in which a specific pest (pest or disease) does not occur, as demonstrated by scientific evidence and in which, where appropriate, this condition is being officially maintained for a defined period (ISPM 10). A pest-free place of production may be situated within an area where the pest concerned is prevalent; it is isolated by buffer zones in its immediate vicinity. It may be maintained for only one or a few growing seasons, and it is managed individually by the producer, under the supervision and responsibility of the NPPO.

329. "It provides a means for an exporting country, if so required by an importing country, to ensure that consignments of plants, plant products or other regulated articles produced on, and/or moved from, the place of production are free from the pest concerned, because it has been shown to be absent from that place over a relevant period of time."

330. **Pest-free production site,** a defined portion of a place of production in which a specific pest (pest or disease) does not occur, as demonstrated by scientific evidence, and in which, where appropriate, this condition is being officially maintained for a defined period. The pest-free production site is managed as a separate unit in the same way as a pest-free place of production (ISPM 5 and 10).

331. **Pest freedom** is a condition established by surveys and/or inspections to prevent the entry of the pest (pest or disease) into the place of production. The operations are supported by appropriate documentation. The concept of "pest freedom" allows exporting countries to provide assurance to importing countries that plants, plant products and other regulated articles are free from a specific pest or pests and meet the phytosanitary requirements of the importing country when imported from a pest-free place of production (ISPM 10 and 22). The establishment of areas of low pest prevalence (ALPP) has been addressed by Australia's IRA as another strategy to minimize the risk of entrance of *N. Galligena* on apple fruits from New Zealand (AUS-2 BA, p. 152).

Dr Swinburne:

332. The concepts encompassed by the various phrases used in the documentation to describe pest/pathogen status of production areas are confusing, and need to be "unpacked". There are three possible conditions: 1) the pest/pathogen is absent, therefore no disease, 2) the pest/pathogen is present, but for some reason the disease is not visible, 3) the pest/pathogen is present and the disease is visible.

333. In contrast to the fire blight pathogen there are no tests for the presence of *N. Galligena* in trees that would make it possible to assess an orchard for the presence of the fungus in the absence of visible symptoms. Consequently the IRA depends upon the inspection of orchards for these symptoms. However, the IRA does not seemingly define or is ambiguous concerning the unit of production which must be disease free. This could be the block, orchard, farm or geographical region. It also has no tolerance limits for areas with low levels of infection.

Question 52
In your view, is the proportion of retail-ready, compared with bulk, apple imports from New Zealand relevant for the assessment of risks in relation to European canker? If yes, how? (IRA, Part B, p. 9; Paras. 4.74 of New Zealand's FWS; R 8-10 by the Parties)

Dr Deckers:

334. I think there is no substantial difference between retail ready apples compared with bulk apple preparation in relation to European canker. The moment of preparation of the fruits will determine whether hidden infections will come out later or not.

Dr Latorre:

335. As "retail-ready" apples are more commonly imported than bulk apples, one would expect to lower the chances of symptom development because "retail-ready" apples are commercialized and usually consumed more rapidly than bulk apples. Additionally, it is possible that bulk apples would be packed near apple orchards, increasing the probability of spread and establishment in the case that infected and sporulating fruits were discharged near apple trees.

Dr Swinburne:

336. The location of the final grading operation could have an impact on the probability of fruit rotted by *N. Galligena* entering Australia from New Zealand, linked with the time elapsed since harvest (see Q50). Symptomless fruit harvested, held in New Zealand in bulk bins and then shipped to Australia could have time develop rots, which would then be graded out only after arrival. By contrast, fruit graded into retail ready packs in New Zealand would remove any rots and would greatly reduce the time available for further rots to develop before consumption (see Q91).

Question 53
Based on Australia's IRA, what is your understanding as to what might be the "appropriate cultural practices and fungicide sprays used to minimise the likelihood of [European] canker infections"? In practice, could this encompass no treatment at all where nursery stock has been sourced from areas verified as free from European canker?

Dr Deckers:

337. There are important periods for fungicide applications with the aim to reduce NG infections: such a period is the leaf fall period at the end of the season because the leaf scars form a preferred infection pathway for a NG infection. When a NG infection occurs on the branches via leaf scar, it is important to prune the infection out and to remove it from the orchard and burn the infection. A second important period for fruit infection by NG is the blossom period during which an internal infection in the core of the fruit can develop. Also here the application of specific fungicides can reduce the infection level substantially.

338. Exclude the treatments completely when the nursery stock has been sourced from areas verified free from European canker can be risky.

Dr Latorre:

339. Cultural practices (sanitation) should include (i) canker removal (cankered stems) and (ii) removal of rotting fruits from trees and orchard floors. These cultural practices should be integrated with chemical control using specific fungicides, preventively sprayed (pre-infection) in late November or early December to reduce inoculum production and fruit infection.

340. The program would involve no treatment where summer rainfalls are non-existent and where disease trees (cankered trees) have not been detected. However, treatments should be considered in areas highly prone to disease development.

Dr Swinburne:

341. Appropriate cultural methods specifically for the control of European canker are obviously required only where the pathogen is present. These would include the rigorous removal of infected branches or trees, and the application of fungicides at vulnerable periods such as leaf fall or bud burst. However the application of protectant and/or eradicant fungicides for the control of other diseases, notably apple scab (*Venturia inaequalis*) have a profound impact on the severity and spread of canker (Swinburne, 1975) as has been confirmed by Cooke (1999) and Lolas & Latorre (1996). It is reassuring to see that newer scab fungicides replacing those that have been withdrawn also control canker.

342. As the "Millennium Trial" (McCracken et al, 2003) indicated, sourcing trees for new orchards from disease-free nurseries is an important first step in the prevention of European canker. There have been a number of instances in the UK where early problems with this disease in new orchards

were probably attributable to the use of maiden trees that carried the disease from the nursery (personal observations).

Question 54
Please comment on whether Australia's IRA assessment regarding the transmission of European canker depends on mature apple fruit being either latently infected or infested with the causal organism of European canker. Please comment on whether Braithwaite (1996) is reliable and relevant in this context. (Paras. 4.63 and 4.273 of New Zealand's FWS; para. 545 of Australia's FWS; and R 70-71 by the Parties)

Dr Deckers:

343. The transmission of European canker can be made aswell by fruits latently infected as by fruits infested via the lenticels with spores of NG. The rotten fruits can bring the NG into an orchard and spread the disease. The experience of Braithwaite is considered to be relevant in this context.

Dr Latorre:

344. Australia's IRA assessment was based mainly on the possibility that mature fruits can be infected before harvest without developing symptoms at harvesting or during processing in the packing house (latently infected fruits). Symptoms will develop after weeks or months of cold storage. Braithwaite (1996) (Exhibit NZ-34) published a brief review on the currently available knowledge regarding European canker, based on studies of the disease's development in the United Kingdom and Northern Europe, without examining conditions in New Zealand. No new objective data is reported in this paper. Therefore, I agree that it is not a reliable and relevant reference to support the hypothesis that latent infections may also occur in mature apple in New Zealand.

Question 55
Based on the relevant parts of Australia's IRA, please comment on whether the IRA addressed the risk associated with latent infection with the causal organism for European canker in regard to dessert apples with the necessary scientific and methodological rigour. (Paras. 4.62 and 4.284 of New Zealand's FWS; R 72 by Australia)

Dr Deckers:

345. In Belgium we have the experience that dessert apples like Jonagold or Gloster can be internally latenly infected by NG and these infections can develop later during storage to fungal rot on the apples.

Dr Latorre:

346. It is true that Australia's IRA based their risk assessment on the information already published from studies in Northern Ireland (Swinburne, 1964, 1975, Exhibits NZ-11 and NZ-9, respectively). These results were obtained on apple varieties quite different from those produced today in New Zealand and under environmental conditions that appear to be far more conducive to fruit infection (in Northern Ireland) than those in New Zealand. Although this does not invalidate the risk assessment analysis, and it does not reject the hypothesis that latent infections may occur in mature fruits in New Zealand, it is a factor that should be taken into consideration by Australia's IRA. Latent infection on mature fruits should not be under discussion, but the probability of latent infection in many apple cultivars produced under different environmental conditions in New Zealand is of utmost interest.

Dr Swinburne (Response to questions 54 and 55):

347. There are no reports which imply that rotted apples are in any way involved in the transfer of infection with *N. Galligena* to "clean" orchards.

348. In Europe dessert cultivars of apples frequently develop post harvest rots by *N. Galligena* and the phenomenon of quiescent (latent) infection is by no means confined to the cooking variety Bramley's Seedling as the NZ FWS states (Berrie, 1989 and appendix 2).

349. Braithwaite (1996) contains an unconfirmed report that fruit rotting with this pathogen has been detected in NZ, and it seems to be accepted by both parties that this does occur occasionally, although it is by no means clear if these reports refer to pre- or postharvest. Braithwaite then goes on to speculate that rotted fruit can transmit infection, basing his argument on European observations on the formation of ascospores on mummified fruit. This is a very rare occurrence, and most unlikely to be found in the climates of NZ or Australia (see Q56 7 Q66). For these reasons this aspect of the paper can be disregarded.

Question 56
When considering the relevant factors for the likelihood of establishment and spread of European canker, taking into account respected and qualified scientific sources, please comment on the relevance of both temperature and rainfall frequency. In order to accurately assess the likelihood of establishment of European canker, what relevance would the annual average amount of rainfall have by itself? (IRA, Part B, pp. 137, 140 and 146; paras. 4.87-4.91 and 4.315 of New Zealand's FWS; Para. 84 of Australia's FWS)

Dr Deckers:

350. The average amount of rainfall on itself is not the most important factor; also the time of rainfall during a susceptible period like the leaf fall period or during the blossom time is more important.

Dr Latorre:

351. As stated by Third Parties, three key factors are necessary for the infection of apple fruit with European canker: (i) conducive climatic conditions; (ii) the presence of a susceptible host; and (iii) sufficient inoculum concentration. The co-occurrence of these three factors is necessary for fruit infection. It has been demonstrated that humid (wet) conditions are necessary for inoculum production and liberation. Frequent rains are essential for conidia and ascospore dissemination from cankered lesions to fruits within infected trees. Therefore, mature fruits would only carry latent infection in cool and rainy summer climates. The likelihood of establishment and spread of European canker after entrance in a new area would be highly dependent on the occurrence of these factors as well.

352. Annual rainfall provides a general indication of areas with climates conducive to European canker. It has been postulated that annual rainfalls higher than 1000 mm are indicative of climate conditions highly conducive to the development of European canker. However, European canker occurs in areas with less annual rainfall. Rainfall is important for infection during two critical periods: (i) during leaf fall, because infection may occur through leaf scars, resulting in twig and stem cankers that appear during the next growing season, and (ii) during harvest, because rainfall favours fruit infection and eventually latent infection in mature fruits. Knowledge of the rainfall distribution, during the growing season, is important for understanding the epidemiology European canker on apples.

Dr Swinburne:

353. Rainfall impacts at every stage of the infection cycle of *N. Galligena,* beginning with the production of spores from existing lesions. This is particularly evident in regions that have distinct "rainy seasons". For example, on the western sea-board of the U.S.A. Zeller (1921) noted that perithecia did not appear in Oregon until some months into the wet rainy winters, and Wilson (1966) made similar observations for conidia in California. However, it is not the absolute volume of rain that correlates with the numbers of spores released (both ascospores and conidia) but the duration of "leaf wetness", (i.e. the presence of free water on the plant surface) (Swinburne, 1971). Thus a short storm in which several centimetres of rain falls in an hour would be much less conducive to spore release than when the same volume falls over a period measured in days. Likewise, even after the arrival of viable spores in the infection court (e.g. leaf scars) a continuing period of leaf wetness is required for successful infection. Dubin & English (1974) working in California found that no infections developed unless leaf-wetness was maintained for at least 6 hours. More recently Latorre *et al* (2002) in Chile found that this could be as little as 2 hours at the optimum temperature, and demonstrated the interaction between temperature and wetness. The number of days with rain will give a much more accurate indicator of the likelihood of infection, especially when examined in terms of the seasonal frequency of rain days.

354. There is no information on the effect of temperature on spore formation or discharge, only for infection. Latorre *et al* (2002) demonstrated an interaction between temperature and the hours of leaf wetness required for the successful infection of leaf scars following artificial inoculation, which forms the basis for predictive model used in Chile. The hours required decreased linearly with increases in temperature between 10 and 20 C, and at 20 C only 2 hours was needed, the shortest time so far recorded. It has to be noted that a predictive model for leaf-scar infection such as that from Chile is based on the presumption (probably valid there) that conidia would be available at all times. In regions of intermittent rainfall this would be incorrect (Wilson 1966), and therefore using for example the number of rain days with suitable temperatures for leaf-scar infection without allowing for spore production could overestimate the likelihood of infection.

355. For all these reasons mean annual rainfall/temperature data alone will be misleading in predicting the possibility that *N. Galligena* could become established in any new region.

Question 57
On the basis of scientific evidence, is the reasoning articulated in Australia's IRA objective and coherent in relation to the following points: (i) whether N. Galligena may cause fruit rots in New Zealand dessert apple fruit; (ii) whether latent infections may occur on mature New Zealand apple fruit; and (iii) the likelihood of a pathway via surface-contaminated mature New Zealand apple fruit? Is this evaluation contained in Australia's IRA regarding these points objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? (Paras. 4.57-4.68 and 4.266-4.300 of New Zealand's FWS; para. 604 of Australia's FWS; and R 74 by New Zealand)

Dr Deckers:

356. I don't know the situation of the NG infections in fruit rot in New Zealand but under European situation this would be possible for the three cases mentioned.

Dr Latorre:

357. On the basis of scientific evidence, *N. Galligena* may potentially cause fruit rots in New Zealand dessert apple fruits in areas or years with frequent summer rainfalls at harvest. The prevalence and severity of fruit infection can vary considerably, according to weather conditions.

Frequent summer rainfalls are necessary for inoculum production, dissemination and infection. If summer rainfalls are frequent, it would be reasonable to assume that some of the infected fruits may develop symptoms on the tree, and other fruits may be latently infected, developing symptoms after several weeks or even months in cold storage.

358. After reviewing Figure 1 (Paras. 4.58 NZ FWS), which explains the probability that summer conditions are conducive for European canker at sites in the United Kingdom (Loughgall, East Malling), United States (Sonoma), New Zealand and Chile (Talca), it appears that summer conditions in New Zealand are very unfavourable for the development of European canker, and that fruit infection would be an extremely rare event. Therefore, the likelihood of latent infection on mature apple fruits would be extremely low or negligible.

359. The likelihood of a pathway via surface-contaminated mature New Zealand apple fruit is unknown. Based on the scientific information acquired thus far, surface contamination (ascospores and conidia epiphytically contaminating fruit surfaces) appears to be non-existent. This possibility should be disregarded from the risk analysis.

Dr Swinburne:

360. Fruit of the cultivars of apple comparable to those grown in New Zealand can and do become infected in the UK during the growing season with *N. Galligena*, and these infections generally remain quiescent until after harvest, becoming visible progressive rots only after a period in store. This is the general experience in many fruit growing regions in Europe. Where this is common the distribution of rainfall in the summer months allows (a) the production of spores (conidia) on active stem cankers, (b) the dissemination of those spores in run-off from cankers onto the developing fruit and (c) a sufficient period of leaf-wetness to allow the deposited spore to germinate and colonise limited areas within the calyx or lenticels. (See Q56)

361. The limited information available in both FWS documents suggests that rots attributable to *N. Galligena* in fruit grown in New Zealand are by no means as common as they are in Europe, and (of course) are seemingly confined to regions of NZ where tree cankers are present. The weather data presented in Annex2 of the NZ FWS would accord with a low incidence of fruit infection, and, based on Wilson's (1966) observations in California, even conidial production from stem cankers may be sparse during summer. It is perhaps significant that in what was described as an epidemic of canker in Auckland that Brooke& Bailey (1965) only found occasional fruit rots. Unfortunately that paper does not record whether the rots were found before or after harvest.

362. It is extremely unlikely that in the event that spores deposited on the open surface of fruit at or before harvest would play any part in an entry pathway (see Q49).

Question 58
Please comment on whether the conclusions in Australia's IRA as to the establishment and spread of European canker are objective and credible on the basis of the available scientific evidence? Are they based on respected and qualified scientific sources? (IRA, Part B, pp. 129-145; paras. 4.87-4.95 and 4.301-4.325 of New Zealand's FWS; paras. 645-672 of Australia's FWS; and R 75 by New Zealand)

Dr Deckers:

363. Comparable with the situation of fire blight, the chance that an infected dessert apple fruit can establish a new disease in an unaffected orchard and spread the disease is considered to be rather low.

Dr Latorre:

364. The conclusion of Australia's IRA as to the establishment and spread of European canker is based on the available scientific evidence. However, the information provided is not entirely convincing because: (i) Analysis of the climate conditions in the potential entrance areas is discussed only briefly. It should not be assumed that any area where the rainfalls are close to, or exceed 1000 mm annually, are necessarily prone to European canker development. Temperatures and rainfalls during the entrance periods (fruit-importing periods) should be provided, considering that they will affect the likelihood of establishment and spread of *N. Galligena* after entrance. Weather information for the entrance periods would allow experts to assess the probability that mature fruit carrying latent infections will develop symptoms, sporulate, liberate the inoculum and spread it to nearby hosts. (ii) Injuries, leaf scars, pruning wounds, or other damages are necessary for infection, but leaf scars in the autumn are the most common sites of infection. Fruit importation (and inoculum availability) could occur when leaf scars are not present, reducing the probability of establishment and spread to zero.

Dr Swinburne:

365. The initial establishment of an epidemic incited by *N. Galligena* via imported infected fruit has not been demonstrated, as the submissions by both parties acknowledge. Consequently the issue can only be addressed from a theoretical standpoint. For such a pathway to exist fruit would not only have to develop visible rot, but also to form viable spores which can be distributed to new hosts. The formation of perithecia on fruit has been observed very rarely (Dillon-Western, 1927), and does not feature in any subsequent epidemiological study (Swinburne, 1975; CAB 2001). It is therefore most unlikely that ascospores would be formed or released from rotted fruit. The formation of conidia on the surface of lesions does occur (Swinburne, 1975) generally in the centre of the rotted area where the cuticle has split. This is most obvious in fruit taken from stores in which the humidity has been maintained at c. 100%. Fruit which develop rots later within the retail chain in conditions with lower RH do not usually produce spores (personal observation) which conforms with the observations (e.g. Wilson, 1966) for wood infections, that a period of 'leaf-wetness' is required for conidia formation.

366. Conidia are dispersed by rain splash, over relatively short distances, especially so from ground level. Thus to successfully transfer infection fruit would have to be very close to a susceptible host and have the appropriate weather conditions.

367. Subsequent spread of infection from such an entry point would be dependent on suitable weather conditions, [and possibly the genus of the new host (Flack & Swinburne, 1976)]. The first requirement for long distance dispersal from the initial host would be the formation of perithecia and the forcible discharge of ascospores; processes which are heavily dependent on rainfall (see Q57). The dispute between the parties regarding the suitability of the climate in the fruit growing regions of Australia for the establishment and spread of European canker in apple is difficult to resolve on the basis of the data available. However, the fact that canker has only been seen in Tasmania and that western Tasmania has a higher number of days of rainfall (> 1mm) than mainland Australia is striking. Moreover, it may also be significant that even in Tasmania perithecia were not observed. Thus it is difficult to escape the conclusion that the climate of fruit growing regions of mainland Australia are not conducive to the development of an epidemic of this disease (see Q72).

Question 59

Please comment on the relevance, if any, of data concerning the volume of apples moving out of the Spreyton area during the European canker outbreak for assessing the risk of apples from New Zealand being a vector for European canker into Australia? (Para. 4.94 of New Zealand's FWS; R 78 by New Zealand; Exhibits NZ-109 and NZ-110)

Dr Deckers:

368. This export of high volumes of apples from a NG infected area confirms the very small chance that apple fruits infected with NG can establish the disease in non infected regions or spread the disease in a new area.

Dr Latorre:

369. On the basis of the biology of *N. Galligena* and considering the epidemiological characteristics of European canker, if *N. Galligena* were to enter a new area, I would expect a relatively slow dissemination of the pathogen and subsequent spread of European canker in this new area. The information provided in relation to Spreyton supports the hypothesis of a very slow spread occurred that would make it possible to eradicate *N. Galligena*.

Dr Swinburne:

370. Whatever the volume of apples shipped from the Spreyton area to mainland Australia, the absence of information on the percentage of apples infected with *N. Galligena,* if any, makes it impossible to use the events as a predictor of the likelihood of the disease becoming established through this pathway from similar imports from NZ. Thus it is impossible deduce why these Tasmanian fruit shipments did not lead to canker establishment.

Question 60
Please comment on whether Australia's IRA provides an objective and coherent analysis of the potential consequences of European canker. Are the IRA's conclusions in this regard, in particular the overall consequence rating of "E" (moderate) designated by the IRA, objective and credible on the basis of the available scientific evidence? (IRA, Part B, pp. 145-150; paras. 4.326-4.332 of New Zealand's FWS; paras. 673-718 of Australia's FWS; and R 79 by New Zealand)

Dr Deckers:

371. It is important to distinguish the situation of a NG outbreak in the different regions of Australia and the general estimate the overall consequence rating of "E" (moderate) seems to be high when there are regions where the disease will not be able to develop due to unfavourable climatological conditions like the presence of drought.

Dr Latorre:

372. On the basis of reports in the literature and the experience of other apple-producing countries, the conclusion arrived at by IRA with regard to the overall consequence rating (E, moderate) is overestimated. (i) European canker has been considered as a major disease of apples, proving economically important in Chile (Latorre et al., 2002), primarily because 2-3 fungicide applications are necessary each year to prevent infections through leaf scars. European canker has never limited the Chilean commercial production, although yields can be reduced and production cost increased. (ii) "Climatic conditions in approximately 40% of Australian commercial fruit-growing areas are conducive to infection." This conclusion was only based on annual rainfalls, without any analysis of the climatic conditions during the critical period (e.g., leaf fall in autumn) with regard to the host trees for infection. (iii) "The main economic impact of the disease results from destruction and removal of individual trees or whole orchards because of girdling of branches, which can significantly reduce crop production yields …" Removal of whole orchards of bearing trees is extremely rare, if it ever happens. Removal of some young trees may occur. (iv) "Fruit rot generally develops in the field or before harvest, although storage losses of 10–60% of the stored fruit crop have been reported in various parts of the world." In rainy areas at harvest, storage losses are commonly below

approximately 2%. In areas free of summer rains at harvest, storage losses are 0%. Storage losses of 10-60% may occur in highly susceptible apple varieties that are inadequately managed (without fungicide treatments under poor cold-storage conditions), a phenomenon that has been observed only in areas with extremely favourable environments and under high inoculum pressure.

Dr Swinburne:

373. The overall consequence rating of 'E' (moderate) can only be justified if the assumption that climatic conditions in the fruit producing regions of mainland Australia are conducive to the rapid spread of canker from a point source (discarded rotted apples) across a district. As discussed in Q58 & Q66, and in the light of the limited spread experienced in Tasmania, it seems unlikely that this could occur.

374. There is one, albeit unlikely, scenario for the rapid dissemination of canker across all apple growing regions: that *N. Galligena* somehow enters the production system of new trees, and be unwittingly sent out to different regions (McCracken *et al* 2001). However, as nursery-tree production is apparently carried out under quarantine conditions (Aust. FWS) this seems unlikely.

375. Discarding the nursery pathway leads to the conclusion that a consequence rating "C" would be more appropriate for the impact on plant health. This would be the worst case scenario.

Question 61
Can the likelihood of entry, establishment and spread of brown rot associated with Japanese nashi pears be compared to that of European canker associated with New Zealand apples? Are there respected and qualified scientific sources to support New Zealand's argument in para. 4.443 of its FWS that there is a higher risk profile for Japanese nashi pears in the context of brown rot than for New Zealand apples in the context of European canker? Please comment on whether the economic and biological impact for the Australian agriculture industry may be different if M. fructigena (brown rot) were to establish in Australia than if European canker were to establish in Australia. (Paras. 4.439-4.442 of New Zealand's FWS; paras. 992-993 and 1001-1004 of Australia's FWS; R 101 by Australia)

Dr Deckers:

376. Brown rot and NG are both important fungal diseases in fruit growing. The introduction of brown rot on pear would surely create a large problem of rotting phenomena on different fruit species. But the introduction of European canker should not only create rotten fruit problems but also problems of NG infections on banches or on the rootstock and can even kill fruit trees completely . The impact of NG on apple is thus more important than the impact of brown rot on pear.

Dr Latorre:

377. In my opinion, there is not enough published information to allow an adequate comparison of the risk of entrance, establishment and spread between brown rot and European canker.

378. The likelihood of entry of brown rot (*Monilinia fructigena*) on Japanese nashi pears and European canker (*N. Galligena*) on apple may be similar, although these diseases are quite different in biology and epidemiology. However, Australia claims that pears are imported only from pest-free areas ("areas of freedom"), which I assume was demonstrated previously. If so, the likelihood of the entrance of *M. fructigena* drops down considerably, to negligible.

379. The biological impact of both diseases would be highly dependent on weather conditions during fruit maturity. Among other factors, the severity of both diseases depends on the presence of

frequent rains during harvest. If this is accepted, the economic and biological impact on Australian agriculture, particularly for apple and pear production, would be similar.

380. The arguments provided by Australia (FWS 1003 and 1004) are rather weak: (i) Brown rot rarely causes economical losses, unless frequent summer rains occur, it is weather dependant. The same is true for European canker. (ii) Several of the fungicides used to prevent apple scab (*V. inaequalis*) can also control European canker.

Dr Swinburne:

381. The risk posed by brown rot incited by *M. fructigena* differs from that associated with *N. Galligena* in a number of important respects. For example, it can spread from fruit to fruit in bulk bins leading to "nesting", and thus inoculum enhancement, which is not found with *N. Galligena*. Rotted fruit almost invariably produce prolific numbers of conidia on sporodochia which form in concentric circles across the surface of the rotted area. The conidia are dispersed by wind alone and are thus not reliant on rain-fall. This contrasts with *N. Galligena* in which spore production is relatively low and the spores are dispersed by rain splash (Byrde & Willetts, 1977; Swinburne 1975).

382. As stated in the NZ FWS, the host range of *M. fructigena*, including as it does fruit types of importance to Australia, also suggests that it poses a greater risk to commerce than *N. Galligena*. But if the Japanese pears are indeed coming from localities verifiably free of the disease then perhaps the risk is small.

Question 62
Please comment on the alternative measures proposed by New Zealand in respect of European canker, namely (i) 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, or (ii) limiting imports to apples sourced from areas of "low pest prevalence", to be determined by inspection of a sample of orchards. How do these alternative measures compare to the relevant requirements imposed by Australia in terms of risk mitigation? (IRA, Part B, pp. 4-5 and 150-155; para. 4.491 of New Zealand's FWS; and paras. 1087-1088 of Australia's FWS)

Dr Deckers:

383. A pest free place of production or an area of low pest prevalence will not be easy to guarantee because NG is often spread by the importation of young apple trees where aswell the apple variety as the rootstock can be infected by the NG disease. Restricting the imports of apples to the pest free places or to the areas with low pest prevalence, together with the application of a fungicide schema during bloom will seriously reduce the risk of importation of internally infected fruits.

Dr Latorre:

384. Both alternative measures, "pest-free places of production" and "low pest prevalence," can mitigate the risk of *N. Galligena* entrance via asymptomatic fruits. These are acceptable methods which should be implemented according to FAO (ISPM 4, 10, 22). The New Zealand proposition is less restrictive, economically feasible, and should not affect the application of phytosanitary procedures to mitigate the risk of *N. Galligena* entrance via the importation of mature apples.

Question 63
Please 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 (ALOP). How does

this alternative measure compare to Australia's measures in terms of risk mitigation? (IRA, Part B, pp. 4-5 and 150-155; paras. 4.492-4.512 of New Zealand's FWS; and paras. 1082-1086 of Australia's FWS)

Dr Deckers:

385. Mature symptomless apple fruits can be internally infected by NG and will not be able to fulfil Australia's ALOP.

Dr Latorre:

386. Exporting only "mature asymptomatic apples" from New Zealand would disregard the fact that latent infection may occur on a mature apple fruit, the main issue of this dispute. Latent infection (fruits that are infected but asymptomatic at harvest) may be extremely rare in New Zealand, considering that weather conditions at harvest are not very favourable for European canker in New Zealand apple-producing areas, as previously discussed. However, it has to be admitted that the likelihood of such an occurrence is close but not equal to zero; at least, until objective results prove otherwise.

Dr Swinburne (Response to questions 62 and 63):

387. The difficulty of responding to these questions arises from the presence of flaws in the arguments from both parties.

388. New Zealand relies on the contention that if fruit of the varieties to be traded were infected during development the disease would become visible at or before they reach harvestable-maturity and that consequently there would never be quiescent (latent) infections that could not be eliminated before shipment. Experience elsewhere indicates that this is not correct (see Q55/6). That is not to say that NZ fruit will develop post harvest rots, as this will depend on (a) the presence of the disease in the trees harvested and (b) the climate during the summer season. Given that some 95% of NZ orchards are either disease free or have very low levels of infection, coupled with a climate that is not well suited to summer fruit infection, it necessarily follows that the probability of there being post harvest rots is very low indeed. Exclusion of exports from the remaining 5% of orchards would reduce the risk to insignificance.

389. Australia's insistence on receiving only fruit from inspected orchards certified as free from canker would eliminate virtually all risk of fruit being infected. Moreover, as there is no evidence to support Australia's concerns that cross contamination might occur in the pack house during grading etc. no further measures would be necessary.

Question 64
Please explain with reference to respected and qualified scientific sources, the potential pathways for long distance spread of European canker. How are the planting of infected nursery stock and the movement of apple fruit relevant to long distance spread of European canker? (Paras. 3.65 and 4.95 of New Zealand's FWS)

Dr Deckers:

390. The movement of infected nursery stock is the main way for a long distance spread of the NG disease and will directly infect the newly planted orchards.

391. The infected fruits can be exported as mature desert apple fruits and spread also over long distances but need to meet a host plant in a susceptible stage before an infection can take place and this last step has a rather low likelihood to occur.

Dr Latorre:

392. On the basis of current information, the main acceptable pathway for long-distance (e.g., between countries) dissemination of European canker is the movement of infected plant material (nursery stocks) (AUS-2BA p. 142). Recently, a study conducted in the UK concluded that about 6% of the infection in new orchards could be associated with infected nursery stocks (McCraken et al., 2003b). There is no scientific evidence demonstrating that long-distance spread of European canker is due to the movement of fruits. Eventually, conidia and ascospores can develop in rotted fruits and contribute to local spread (tree-to-tree movement). Therefore, long-distance spread along with mature apple fruits should be regarded as a hypothesis rather than a true fact.

Dr Swinburne:

393. There is no data that confirms or refutes that rotted fruit forms a pathway for the long distance transport of infection with *N. Galligena*, (see Q54). Virtually all research publications emanate from countries in which the disease is essentially endemic (CAB, 2001), so it is not surprising that the focus has been on more obvious routes, such as orchard to orchard, and hedge-row to orchard. However, the possibility that disease could be transmitted asymptomatically in maiden or two year old trees from the nursery has been confirmed (McCracken *et al* 2001). In terms of long distance transport this could be very significant, especially for regions in which the apple itself is a recent introduction, and where by definition the pathogen is/was not endemic.

Question 65
Please comment on whether there are respected and qualified scientific sources to support the position in Australia's IRA that latent infection with N. Galligena can occur in mature, dessert varieties of apples, including in New Zealand. Please comment on whether there is any scientific evidence of latent infections occurring in mature apple fruit, including in New Zealand. (Paras. 4.61-4.68 and 4.272-4.274 of New Zealand's FWS; para. 613 of Australia's FWS)

Dr Deckers:

394. In Europe the latent infection of NG can occur in mature dessert apple varieties; this does not mean that the same situation will occur in certain area of New Zealand apple production under comparable climatological situations.

Dr Latorre:

395. Considering that fruit rots occur in New Zealand (NZ FWS, Paras. 4.60) and despite the fact that this a very rare event, latent infection in mature fruits cannot be ruled out. It is possible that asymptomatic but infected mature apples could develop symptoms and eventually sporulate during transit and commercialization in Australia. However, I would consider the probability of this event as extremely low to negligible. Please notice that *N. Galligena* was not found in a small apple sample (53 apples) from New Zealand, intercepted by Australia's authorities between 1988 and 2003 (AUS-2 BA p. 123). Nevertheless a large apple sample should be studied before reaching a final conclusion.

Dr Swinburne:

396. See Q55.

Question 66
Please comment on whether research relating to European canker in Europe and North America relied upon by Australia's IRA is relevant to the climatic conditions for the entry, establishment or spread of the disease in Australia. Are the climatic conditions regarding European canker in Australia comparable to those in the countries in which the research has been carried out? How relevant is this research to evaluating the risks of N. Galligena associated with apples from New Zealand? (Paras. 2.13, 4.63, 4.68, 4.73, 4.87-4.91, 4.315 and 4.321 of New Zealand's FWS; paras. 570-574 and 627 of Australia's FWS)

Dr Deckers:

397. The specific conditions for entry, establishment and spread can be substantially different between the European climatic conditions and the conditions in Australia. These differences can be decisive for the behaviour of a fungal disease in a country. The amount of rain is an important factor but also the distribution of this rain throughout the season is even more important for the development of the NG fungal infection in an area.

Dr Latorre:

398. This information is a key point in assessing the risk of establishment and spread of *N. Galligena* associated with asymptomatic apples from New Zealand. Climatic conditions in Australia's apple-producing regions must be suitable to disease establishment and spread, otherwise the likelihood of establishment and spread would be zero and the risk analysis should end at this point. Additionally, favourable climatic conditions are compulsory for sporulation (inoculum production, mainly conidia), dissemination and survival of the inoculum. The environmental information provided by New Zealand suggests that temperatures and rainfalls are relatively unfavourable for *N. Galligena* during summer and early fall in Australia, which may be the most critical period for infection (Figure 2, NZ FWS paras. 4.91). If this is the situation, the likelihood of establishment and spread should be extremely low to negligible.

Dr Swinburne:

399. Data from Northern Europe, California and Chile on the basic weather conditions for infection (in the strict sense) have been determined from artificial inoculation experiments (summarised in Swinburne, 1975; CAB 2001, and Latorre et al 2001). This data will be relevant to all apple growing regions, but as they refer to just one aspect of the cycle of events, can not be used alone to predict the suitability of any region for the disease. The essential weakness of the approach in the IRA is that it assumes that inoculum (spores) for infection is always available, and all that is required is a suitable period (hours of leaf wetness within given temperature limits) for infection to occur. The major flaw in this argument is the assumption that regions can be compared on the basis of annual rainfall, without regard to rainfall patterns. Even in regions such as N. Ireland (Loughgall) with rain in all seasons, more than 5hrs of leaf wetness was required following a few dry days before ascospore discharge resumed (Swinburne, 1971b). The situation in regions with a pronounced dry season, such as California and the Pacific Northwest in the USA, spore formation does not even begin until some time (as yet undetermined) into the rainy period (Zeller, 1926, Wilson, 1966/8). For such an area data relating only to simple "infection periods" would greatly overestimate the risk of disease establishment. (see Q72 for an appraisal of weather data in both FWS).

Question 67
Please comment on whether Australia's IRA provides an objective and coherent analysis with respect to conidia and ascospore production and dispersal in light of climatic conditions in New Zealand's apple-growing regions during harvest time. Are there respected and qualified scientific sources to support the view that mature apple fruit can be infested (surface contaminated) with spores at harvest, including via wind currents? Please comment on whether these spores could later infect the apple? What is the impact, if any, of climatic conditions in this regard. Is the evaluation contained in Australia's IRA regarding these points objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? (IRA, Part B, pp. 122 and 124-125; paras. 4.65-4.68 and 4.278-4.281 of New Zealand's FWS; and paras. 542-552 and 635-636 of Australia's FWS)

Dr Deckers:

400. Infection of the apple fruits by spores at harvest time should be only with the conidiospores while there are no ascospores available at that time. The conidiospores will spread more locally and can infect the fruits by the stalk end of the fruits where water can be present for a longer time during the preharvest period during a rainy period. Distribution of these conidiospores over a long distance by wind is rather unlikely.

Dr Latorre:

401. Australia does not provide objective data regarding spore (conidia, ascospore) production and release under the environmental conditions of New Zealand. There are not qualified scientific sources to support the view that mature apple fruit can be infested (surface-contaminated) with spores at harvest, including via wind currents. If this event would happen it would be extremely rare and would not necessarily result in infected fruits, unless enough spores land on damaged mature fruits. Conidia are not wind dispersed; they are dispersed by rains and rain-splash. Ascospores can be dispersed by wind currents to rather short distances (metres from the inoculum source). However, it would be possible that rain-splashes containing spores may be carried several metres by winds. In my opinion this analysis overestimates the risk of inoculum dispersal.

Question 68
Please comment on whether Australia's IRA took into account respected and qualified scientific sources in arriving to the view that, if spores were to be dispersed by rain onto the surface of a mature apple immediately prior to or during harvest, they could survive without continued moisture. Is this conclusion objective and credible on the basis of the available scientific evidence? (IRA, Part B, pp. 124-125; paras. 4.67 of New Zealand's FWS; and paras. 554-557 of Australia's FWS)

Dr Deckers:

402. Conidiospore production can take place around sporulating cankers in the orchard and rain is necessary to spread the spores from the cankers to the fruits. Once on the fruits the spores can be accumulated on the fruits in a natural way in the cavity around the stalk or around the calyx and realise later on an infection.

Dr Latorre:

403. Based on the available scientific reports, this conclusion has no credibility. Puia et al. (2004) (AUS-56) is the only published report that addresses superficial contamination. "…When dessert apple varieties were monitored from the beginning of storage in November until the end of storage in April in Romania, *N. Galligena* was isolated from the surface, inside and even the locules of the

fruit." However, the Materials and Methods used by Puia et al. (2004) (AUS-56) do not allow the authors to conclude that *N. Galligena* was on the surface of the fruits.

Dr Swinburne (Response to questions 67 and 68):

404. The contention that fruit could become contaminated by spores at harvest depends on their presence on tree cankers during any period of rain at that time. It is extremely unlikely that these would be ascospores as even in the wetter summers of Europe perithecia are usually in produced in winter to spring (Swinburne 1975). The production of conidia in summer (including harvest) is dependent on rainfall (see Q66). That some conidia production during summer does occur is indicated by the detection of fruit rots in NZ, albeit rarely. These spores would be deposited in rain-off within the affected tree (Swinburne, 1971), or less likely from a neighbouring tree by splash dispersal (Munson 1939). Wind dispersal releases so few viable conidia (Swinburne, 1971) that these can be discounted.

405. As discussed in Q49 it is most unlikely that conidia which simply contaminate the surface of fruit would play any part in an infection pathway.

Question 69
Please comment on whether Australia's IRA took into account respected and qualified scientific sources in arriving to the view that mature symptomless apple fruit, infested or latently infected with the causal organism for European canker, could produce conidia or ascospores in Australian conditions. Is this conclusion objective and credible on the basis of the available scientific evidence? (IRA, Part B, pp. 123-124, 127 and 134-135; paras. 4.78-4.84 and 4.305-4.308 of New Zealand's FWS; and para. 614 of Australia's FWS)

Dr Deckers:

406. The chance for a successful introduction of NG into Australia by the importation of infected fruit is much lower than for the importation of an infected fruit tree. Depending on the climatic conditions after introduction and on the presence of a susceptible host the transfer of the NG from an infected fruit to the susceptible host will be the critical point.

Dr Latorre:

407. The available information demonstrating that mature asymptomatic apples (infected or latently infected) can readily sporulate under the Australian environment is not provided. Based on published scientific information, Australia assumes that fungal growth and fruit rot resume when fruit is removed from cool 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 and likelihood values for establishment and spread in Australia have been assigned (Table 34, AUS-2BA p.144). However, the likelihood assigned seems to be high and these values have not been validated locally. Based on the general information available, I would assume that these events have a likelihood of occurring different from zero, but still extremely low.

Dr Swinburne:

408. As discussed in Q58 rotted fruit incubated under conditions of high humidity can produce conidia, but it is extremely unlikely that they would produce perithecia, still less that ascospores would be released. The importance of high humidity to conidia production has to be stressed. The surface of fruit held in cold stores is usually moist, and fully developed rots usually produce conidia. Fruits rotting subsequently in retail packs or in a domestic environment at less than 100% RH are not

likely to produce conidia. In the uncertain event that an apple shipped to Australia from New Zealand rotted with *N. Galligena* its ability to act as vector of disease would depend on the handling system on arrival. (See Q52)

Question 70
Please comment on the relevance of perithecia on rotting, mummified apples on the ground to the spread of European canker. Please also comment on whether Australia's IRA took into account evidence from respected and qualified scientific sources regarding whether apple fruit (of either dessert or non-dessert variety) develop perithecia, including in New Zealand. (IRA, Part B, pp. 134-136; paras. 4.83, 4.86 and 4.304-4.317 of New Zealand's FWS; and in paras. 614-626 of Australia's FWS)

Dr Deckers:

409. Swinburne clearly indicates that overwintering mummified fruits can form perithecia that can produce ascospores that can spread over a longer distance than the conidiospores. Critical point here will be the climatic conditions in both countries New Zealand and Australia: will there be a suitable situation for the spores to be produced and will this be followed by an infection scenario.

Dr Latorre:

410. It is possible that perithecia play a minor role in the establishment and dissemination of *N. Galligena* from rotted fruit, if this ever happens. On my opinion, there is not enough scientific evidences supporting the role of perithecia (ascospores), eventually developed on rotted fruits, on the overall epidemiology of European canker.

Dr Swinburne:

411. See Q58.

Question 71
Please comment on whether Australia's IRA took into account respected and qualified scientific sources in arriving to the conclusion that conidia on the surface of mature apple fruit can survive cool storage without drying out, and germinate once the fruit has been warmed to ambient temperatures following removal from cold storage. Is this conclusion objective and credible on the basis of the available scientific evidence? (IRA, Part B, pp. 134-135; paras. 4.84 of New Zealand's FWS; and paras. 619-621 of Australia's FWS)

Dr Deckers:

412. Fruit rot caused by NG can be present in a latent form and come out only after some storage period. Conidia themselves have a limited possibility to survive on the fruit skin surface and will dry out easily.

Dr Latorre:

413. The information provided does not demonstrate that conidia can survive cool storage on the surface of apple fruits. Australia states that cool storage and transport processes would not adversely affect the viability of the fungus. This may be true only for the fungus inside the fruit but not for conidia, mycelia or even ascospores on the surface of the fruit. In other words, mycelia can survive in latently infected fruits; growth may resume after cool storage and eventually the fungus may sporulate on the surface of mummified fruits.

Dr Swinburne:

414. See Q49.

Question 72
Please consider the two annexes provided by the Parties – Annex 3 to New Zealand's FWS and Annex 2 to Australia's FWS – and explain to what extent these documents accurately describe the climatic suitability for establishment of European canker. In particular, please comment on whether Australia's IRA objectively and coherently takes into account whether New Zealand has weather conditions favourable for fruit infections by N. Galligena during the summer (December to February). Please also comment on whether Australia's IRA objectively and coherently takes into account whether the climate in Australian apple production areas is conducive to the establishment and spread of European canker. (Paras. 4.57-4.58, 4.88-4.90, 4.425 and 4.315 of, and Annex 3 to, New Zealand's FWS; paras. 534 and 627-628 of, and Annex 2 to, Australia's FWS; para. 15 of Australia's opening statement; and R 77 by Australia)

Dr Deckers:

415. Weather data should be examined in detail per region and the estimation of NG infections should follow this regional approach for both countries.

Dr Latorre:

416. The models present in both documents (Annex 2 AUS FWS and Annex 3 NZ FWS) can be used for describing and analysing the relative weather suitability of European canker for establishment and development. The weather analysis performed by New Zealand (Beresford and Kim) objectively explains the algorithm used, and provides information regarding the model's validation, using historical weather data obtained in five countries where European canker affects apples, with different prevalence and severity. Therefore, it is an acceptable criterion to assess weather conditions for European canker establishment in Australia, relative to other apple-producing areas in the world where *N. Galligena* is a real problem. This does not discount the possible use of Climex or other models.

417. According to these results, it is possible to conclude that:

(a) Grove (1990) generalization stating that areas where average annual rainfall is greater than 1,000 mm favour establishment of European canker should not be interpreted as a threshold for the establishment of this apple disease. In fact, there are several places in the world characterized by mean annual rainfall far below 1,000 mm where European canker occurs as a major disease. As an example, European canker affects apples to the extent that chemical control measures are needed in orchards located south of Curico-Talca (34º58' S), Chile, where the mean annual rainfall is near 700 mm, but frequent rains occur during leaf fall, favouring disease infection.

(b) Based on rainfall patterns, two critical periods for infection by *N. Galligena* can be defined for apples: a. Autumn infections associated with leaf fall and infection through leaf scars. b. Summer infection associated with fruit infection around the time of harvesting, through the calyx end of the fruits. In some places, rainfalls and temperatures are conducive for autumn infections (e.g., California, Chile), while summer infections are prevalent in other apple-producing areas (e.g., United Kingdom). Based on this weather analysis, weather conditions are relatively less conducive during the summer as compared to autumn for infections in New Zealand.

(c) Following the same analysis, areas potentially conducive to European canker establishment were detected in Australia. Weather conditions were more conducive for infection during autumn as compared to summer. Overall, the climate in Australian apple production areas is relatively less conducive to the establishment and spread of European canker than other producing areas of the world.

Dr Swinburne:

418. In the absence of a fully descriptive epidemiological model for the establishment of canker the approach adopted by Beresford and Kim (Annex3 NZ FWS) is both rational and reasonable, drawing as it does on the climatic factors identified in California and Chile (see Q66). Most importantly it makes allowances for the water requirements for spore formation as well as their dispersal and the infection process itself. In the absence of leaf wetness data, an analysis of the days of rain during critical parts of the season seems to provide a reasonable assessment of infection risk. That this model also enables regions within NZ to be distinguished on the basis of known disease incidence is reassuring.

419. Total annual rainfall is an unsatisfactory measure of infection risk, but is relied upon heavily in the IRA and in the arguments presented in Annex 2 of Australia's FWS (see Q56). To illustrate this point; the mean annual rainfall in Loughgall, Northern Ireland is 800mm, and in Sonoma, California it is slightly higher at 900mm (Table1 Annex 3 NZ FWS). Whilst canker is a sporadic problem in California, in Northern Ireland it is a limiting factor in apple production, constraining the growers to mainly one cultivar, Bramley's Seedling, which has greater tolerance to this disease than dessert types. The difference between these regions can not be accounted for by annual rainfall, but must be related to the season in which it falls. In Loughgall rain is evenly distributed through the year with few prolonged dry periods, in California it falls mainly in winter, when the host is dormant. Spores are produced all year long in Northern Ireland (Swinburne, 1975), but not in California (Wilson 1966).

420. In the absence of a detailed explanation of the CSIRO climate model, or why the results it produced (Annex 2 Aus FWS) differed from the application of the Beresford & Kim model (Annex 3 NZ FWS) it is difficult to provide critical comment on the potential areas of risk claimed (Fig1 Aus FWS Annex 2). However, the use of the Beresford & Kim model does imply that the climate of the coastal cities of Melbourne and Sydney could marginally support infection in spring and autumn (Fig 10 Annex 2 Aus FWS).

421. The scarcity of reports of fruit infection in NZ, even from districts with canker (e.g. Auckland) must reflect the predominant weather conditions in summer, as contended in Annex 3 (NZ FWS). The relatively low number of days of rainfall in the principle fruit growing areas of mainland Australia also suggests that this disease would not spread as aggressively as is contended in the IRA or Annex 2 (Aus FWS).

Question 73
Please comment on whether the reasoning in Australia's IRA with respect to the dispersal range for conidia is objective and coherent. Given weather conditions in the territories of Australia and New Zealand, are the conclusions in this regard objective and credible on the basis of the available scientific evidence? Are they based on respected and qualified scientific sources? (IRA, Part B, pp. 135; paras. 4.85-4.86 and 4.311-4.313 of New Zealand's FWS; and paras. 615 and 635-636 of Australia's FWS)

Dr Deckers:

422. The chance for a NG infection starting from the conidiospores produced by an infected fruit will not be high in both countries.

Dr Latorre:

423. Although the reasoning in Australia's IRA with respect to the dispersal range for conidia was objective and coherent and based on respected and qualified scientific sources, the information provided demonstrates only that conidia (and possibly ascospores) of *N. Galligena* are short-distance disseminated. Conidial dispersal can be expected within the infected tree (rain-splash and runoff), from infected trees to healthy neighbouring trees (splash, wind-splash), and between neighbouring orchards (wind-splash). There is no scientific report demonstrating that conidia can be dispersed between districts, countries or continents.

424. I agree that "dispersal for any significant distance is unlikely to occur when ascopores are produced by perithecia on an apple on the ground where they are less likely to become airborne" (NZ FWS paras. 4.86). However, experimental evidence is necessary in support of this conclusion.

425. "Even if latently infected New Zealand fruit could produce spores in the Australian environment, these spores would need to be transferred to the host plant. Any dispersal of conidia would primarily be by rain splash and would likely only be a few metres from a discarded apple (NZ FWS paras. 4.311)." This is a credible conclusion supported by experimental evidence, published previously. Therefore, the analysis of the probability of establishment and spread of European canker after entering Australia should consider that suitable hosts must be close (a few meters) from the inoculum source. Otherwise, the analysis would be overestimating the risk of establishment and spread.

Dr Swinburne:

426. It is most unlikely that rotted fruit would produce ascospores so they need not be considered further here (see Q58). The dispersal distances for rain splashed conidia quoted in the literature referred to in Australia's IRA are the maximum estimates, and refer to conidia released from tree cankers above ground level. For a fruit rotting on the ground it is reasonable to expect that the distances would be smaller, as argued in the NZ FWS. There are no studies that can accurately guide this judgement, but it is unlikely that this would be more than a few meters, horizontally or vertically. It must be evident that for splash dispersal to operate from a rotted apple on the ground the lesion has to be facing upwards; thus subject to further chance.

427. For such dispersal to lead to an infection any adjoining host has to have sites receptive to conidia. The NZ FWS (4.311-4.313) disputes the claim made in the Aus FWS that leaf scars remain susceptible for weeks after leaf fall, basing there argument on Wilson (1966). All studies made under field conditions conclude that leaf scars are susceptible only for a few hours after leaf fall (reviewed in Swinburne, 1975), leading to the conclusion that the growth cabinets used by Wilson lead to conditions not normally encountered in nature.

Question 74
Please comment on the relevance, if any, of the Tasmanian outbreak of European canker for any conclusions on the existence of pathways and the suitability of Australia's climate for the establishment and spread of European canker. (IRA, Part B, pp. 135; paras. 4.92-4.94, 4.308 and 4.323 of New Zealand's FWS; paras. 627-632 of Australia's FWS; and R 75-76 by the Parties)

Dr Deckers:

428. The fact of the Tasmanian outbreak of NG does not mean that the climate there is suitable for a NG infection. It is even surprising that it was possible to eradicate this infection in the area; this means that the climate condition for NG was surely not so good for an optimal development of the fungal disease.

Dr Latorre:

429. The Tasmanian outbreak demonstrates only that climate conditions are suitable for European canker there. The lack of considerable spread suggests that weather conditions are not favourable for European canker in Tasmania (Spreyton, Tasmania, AUS FWS Annex 2; Beresford and Kim, NZ Annex 3). These relatively unfavourable climatic conditions may imply that prevalence, incidence and severity of European canker remained low and that the disease never spread considerably outside Tasmania. However, these observations do not necessarily support the conclusion that the weather conditions are unsuitable for European canker in the rest of Australia, as indicated by New Zealand (ZN FWS paras. 4.92): "The outbreak of European canker in Tasmania (an area where conditions are marginally favourable to European canker) confirms the unsuitability of the rest of Australia to the disease."

Dr Swinburne:

430. The Tasmanian outbreak of European canker does have some relevance to the suitability of the climate in mainland Australia to support an epidemic. The fruit growing areas in the west of the island "enjoys" more days of rain than Auckland (NZ) and yet the reports suggest that the impact of the disease in terms of severity or spread over a period of years was less in the Spreyton district than now seen in Auckland. The Beresford & Kim model (Annex3 NZ FWS) strongly suggests that this difference is attributable to unfavourable temperatures in Tasmania, and thus providing further support to its use in assessing the disease potential of differing climatic zones.

431. (See Q59 for relevance of fruit shipments from Tasmania to mainland Australia).

Question 75
Please comment on the likelihood values and distribution included in Australia's IRA regarding importation step 2, concerning mature apple being infested or infected with N. Galligena (uniform distribution with a minimum value of 1 x 10E-6, and a maximum value of 1 x 10E-3).

Dr Sgrillo:

432. The conclusions of the IRA are summarized as follow:

> "… IRA Team focused on the fact that under New Zealand conditions fruit is only occasionally attacked and this generally results in rotting of the fruit. Rotted fruit would not be picked. There is some likelihood of fruit getting infected late in the season and remaining latent, but this likelihood would be extremely low."[53]
>
> "... fruit infection is very unlikely to occur."[54]

433. It seems evident that the IRA Team is referring to a number of fruits belonging to a population.

434. The minimum and maximum values that were chosen represent a "per fruit" probability. When the population is viewed the results generated are much greater than "extremely low" and "very unlikely to occur". If a population of 200.000.000 fruits is considered then from 200 to 200.000 fruits would be infested/infected by *N.galligena* what seems to be too high for an "extremely low" category. The parameters chosen should reflect the category concepts also in populational terms.

[53] (*footnote original*) IRA p. 123.
[54] (*footnote original*) IRA p. 122.

Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, pp. 121-123; paras. 4.270-4.275 of New Zealand's FWS; and paras. 529-547 of Australia's FWS)

Dr Sgrillo:

435. The scientific evidence is based in respected and qualified papers; however there is no factual information to support the choice of 1E-6 and 1E-3 as, respectively, the minimum and maximum parameters of the distribution.

Dr Deckers (Response to whole question):

436. This value is an estimation of the risk situation and looks to be high surely when the fruits are coming from orchards in New Zealand where no active NG infections occur.

Dr Latorre (Response to whole question):

437. The Australian IRA's assessment regarding the likelihood of a mature apple being infested/ infected with *N. Galligena* (importation step 2) (AUS-2 p. 121 and 123, Table 12) was considered extremely low-risk. The probability assigned varied from 10^{-6} to 10^{-3}. However, the midpoint ($5x10^{-4}$) was used because a uniform distribution was assumed. On the basis of the information provided, the probability criteria were selected arbitrarily to allow semi-quantitative analysis, but no experimental data supporting these values were presented. Several scientific sources were cited, but none of them report information regarding the frequency of apple infection and latency in New Zealand or elsewhere. Considering the epidemiological characteristics of European canker, the probability intervals given in Table 12 are too conservative for areas with dry summer conditions, although they may be acceptable for areas with frequent summer rains. Therefore, the figures may overestimate the likelihood of each event to occur under dry summer conditions, particularly if fruit infection is extremely close to zero.

Dr Swinburne (Response to whole question):

438. In the absence of definitive data from surveys of fruit rots (post harvest) the assignment of any probability value to likelihood of the presence of quiescent infections in shipped fruit must be arbitrary. Both parties seem to agree that the frequency of fruit rotting is low, given the paucity of positive identifications. That this is so, even from regions with tree cankers (e.g. Auckland) this must be attributable to unfavourable weather conditions, especially the absence of rain, during the summer months.

439. The implication of there being one infected fruit per thousand (max), coupled with the fact that 95% of exported fruit comes from orchards with little or no tree cankers (IRAb p121), is that fruit from infected orchards has in the order of 2% apples that will rot with *N. Galligena* each year. That this would escape the attention of research centres in NZ seems extraordinary, if true. Alternatively, the arbitrary probability maximum set in the IRA is too high.

Question 76

Please comment on whether 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 objective and coherent. Are the conclusions in Australia's IRA in this regard objective and credible on the basis of the available scientific evidence? Are they based on respected

and qualified scientific sources? (Paras. 4.61-4.68 and 4.272-4.274 of New Zealand's FWS; para. 538 of Australia's FWS; and para. 56 of New Zealand's opening statement)

Dr Deckers:

440. I am convinced that latent infections of NG may occur in mature New Zealand apples and not become apparent until after storage. Probably there are significant differences between different apple varieties for these latent infections of NG.

Dr Latorre:

441. 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. Although there is no scientific evidence of latent infections occurring in mature New Zealand apple fruit, it 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 Northern Europe, but differences between New Zealand and Northern Europe can only be expected in relation to the likelihood of this event: very high in Northern Europe and extremely low to negligible in New Zealand. In this regard, New Zealand can be compared with California and Chile, where mature fruit infections are extremely rare or nonexistent because of the unsuitability of the weather conditions during summer months.

Dr Swinburne:

442. (See Q55)

Question 77
Please comment on whether the reasoning in Australia's IRA with respect to the view that latently infected but symptomless mature apple fruit may develop rot and thus generate spores, and the relevance of this fact for the possibility of fruit contamination by N. Galligena during picking and transport to the packing house, is objective and coherent. Are the conclusions in Australia's IRA in this regard objective and credible on the basis of the available scientific evidence? Are they based on respected and qualified scientific sources? (IRA, Part B, pp. 123-127 and 134-135; paras. 4.70-4.72, 4.77-4.84 and 4.277 of New Zealand's FWS; and paras. 538-539, 547-548, 564-565, 578-600, 612-613, 619-620, 629-630, 639-640 and 666-667 of Australia's FWS)

Dr Deckers:

443. This contamination of the fruits by symptomless mature apple fruits during picking and transport to the packing house is very low while at that moment the fungal disease, when present, will be present only in a latent phase. When the rotten apples are visible at harvest, they will not be picked as good quality fruits and will be on the orchard floor.

Dr Latorre:

444. The reasoning in Australia's IRA with respect to the view that latently infected but asymptomatic mature apple fruits may develop rot and thus generate spores of *N. Galligena* during picking and transport to the packing house (although objective) is highly unlikely.

445. Latently infected fruits develop symptoms before producing conidia. Mummified fruits in the autumn may produce ascospores under cool humid ambient conditions the following spring, or after a prolonged period in cold storage. However, this is a rare event (<0.5%). Therefore, conidia and

eventually ascospores may develop after several weeks or months of incubation. The likelihood that these spores may 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.

Question 78
Please comment on the likelihood values and distribution put forth by Australia's IRA for importation step 3 regarding clean fruit being contaminated by N. Galligena during picking and transport to the packing house (triangular distribution between 1 x 10E-6 and 1x 10E-4, with a most likely value of 1 x 10-E5).

Dr Sgrillo:

446. The IRA Team describes hypotheses to explain how a clean fruit could be contaminated by *N. Galligena*. However there is no factual evidence to validate theses hypothesis. There are no records of *N. Galligena* spores being transferred to clean fruit.[55].

447. Considering that, also, there is no factual evidence of a most probable value, the IRA team does not explain why the triangular distribution was chosen instead of a uniform distribution.

448. The IRA Team explains that the chosen values were attributed to the parameters of the distribution because "This range allows for a small number of fruit to be contaminated...".[56]

449. However, the IRA Team does not define "small number of fruit". If 10 is a small number of fruit then the most probable value would have to be 5E-8 (in a 200 million fruits population) which is 200 times less than the number chosen by the IRA team.

Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, pp. 124-125; paras. 4.276-4.281 of New Zealand's FWS; and paras. 548-563 of Australia's FWS)

Dr Sgrillo:

450. There is no scientific evidence to support the choice of a triangular distribution and its respective parameters.

Dr Deckers (Response to whole question):

451. Contamination risk of clean fruit during picking and transport is considered to be very low because the spore survival externally on the fruit skin is limited in time and the spores will dry out easily.

Dr Latorre (Response to whole question):

452. The assumption that *N. Galligena* spores could be transferred to clean fruit should be considered as a hypothesis that needs to be probed. To my knowledge, there is no scientific literature addressing this point. Based on general disease knowledge, it is an extremely unlikely event. It is

[55] (*footnote original*) IRA pp. 124-125.
[56] (*footnote original*) IRA p. 125.

difficult to accept and may be impossible to support the probability values assigned to this step, 1 x 10^{-6} and 1x 10^{-4}. Therefore, this evaluation overestimates the risk at this point on non-objective and credible bases.

Question 79
Please comment on the finding in Australia's IRA, in reference to N. Galligena that "none of the processes in the packing house are likely to substantially reduce infections". Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? If latently infected fruit were to develop visible rot symptoms in storage, is this something that could be detectable at the time of packing in New Zealand, so as to allow removal of such fruit? (IRA, Part B, p. 126; paras. 4.73-4.74 and 4.283-4.285 of New Zealand's FWS; and paras. 568-577 of Australia's FWS)

Dr Deckers:

453. This seems to be logic while most of the NG infections are latent and are situated internally in the core of the fruits or in the stalk or calyx end of the infected fruits. Infected fruits will be easier visible at the end of the storage period when the rotting process makes them more visible.

Dr Latorre:

454. Although there is no relevant scientific evidence, it is acceptable to consider that no aspect of the process in the packing house reduces the number of latently infected fruits. Once the fungus has penetrated mature fruits, the normal post-harvest management including brushing, waxing, sorting and grading, cold storage and even fungicide treatments, will be unable to arrest the fungus inside the fruits. Cold temperature would only be able to retard symptom development by lowering the rate of fungal growth.

455. Latently infected fruits cannot be detected at the time of packing in New Zealand. However, symptoms may appear after several weeks of cold storage. If lots of mature asymptomatic fruits are kept for several weeks in cold storage in New Zealand, it would be possible to remove infected fruits before export to Australia, lowering the risk of entrance.

456. The likelihood that inocula contaminating the surface of the fruits can survive this process, attached to the fruit surface, is negligible or zero and it should be disregarded from the risk analysis.

Dr Swinburne (Response to Questions 77, 78 and 79):

457. The difficulty of analysing the reasoning behind Imp steps 3-7 is that at each stage infection and contamination processes are conflated.

458. Contamination: any conidia deposited on the surface of an apple during harvesting operations would not survive for any length of time (see Q49) and may be discounted from all subsequent calculations. Similar considerations would apply to conidia redistributed from trash. There is, for example, no evidence to support assertions that such as "spores would survive waxing" or "brushing", because these processes are irrelevant to the inherent inability of conidia to survive for long periods.

459. Infection: The European experience indicates, in areas in which summer rainfall is frequent, fruit can become infected during the growing season. Such infections, perhaps invisible at harvest, would not be affected by handling, or washing by water, even with disinfectant present. However, the store conditions and the duration of the holding period will be a factor in any subsequent development

of any quiescent infections that may be present (Berrie, Xu & Johnson 2007 in appendix). For example, if apples held in bulk bins are at a later time graded into retail ready packs those infections which have become visible rots will be removed (irrespective of the causal organism). Consequently there are pack house operations that could reduce the probability of the shipment of infected fruit. (see Q52). Arguably, if any of the rotted apples had produced conidia these could transfer to healthy fruit, but it is probable that these, as mere surface contaminants, would not survive unless, after grading, the fruit was kept wet for several hours, which is commercially undesirable. If a washing process was interposed in this final grading, disinfectant in the water would destroy any conidia thus displaced.

460. The probabilities assigned in the IRA to these steps are all difficult to reconcile with the observations above, especially as they all omit the factor of time. (see Q80/81)

Question 80
Please comment on the likelihood values and distribution put forth by Australia's IRA for importation step 4 regarding N. Galligena surviving routine processing procedures in the packing house (a triangular distribution between 0.7 and 1, with a most likely value of 0.8). Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, pp. 125-126; paras. 4.282 and 4.287 of New Zealand's FWS; and paras. 564-580 of Australia's FWS)

Dr Deckers:

461. Important point in this discussion is the timing of the preparation of the fruits for export: is this done after a longer period of fruit storage, the chance that internal infections of NG develop into rotten fruits is greater and this will reduce the possibility of introduction of the disease into Australia. When the fruit preparation is done after a shorter period of storage, the internal infections of NG will not show up and this will increase the risk for an introduction.

Dr Latorre:

462. There is no scientific literature to support the assumption that the likelihood values for step 4 vary between 0.7 and 1, with 0.8 as the most likely value. This assumption, 80% likelihood that *N. Galligena* will survive routine processing procedures in the packing house, falls within a range that is difficult to legitimize, if this assumption implies that the inoculum must remain on the fruit surface. Rather, it would be possible for the inoculum to be present internally in the fruit.

Dr Sgrillo:

463. The effect of the processing procedures in *N.galligena* depends on whether the fruit is infested or infected, because the pathogen in infested fruits could be removed by washing while the pathogen in infected fruit could not.

464. To correctly assess the survival of *N.galligena* with transparency it would be necessary first to establish what proportion of fruit with the pathogen is expected to be infested and what proportion is expected to be infected. Then, assign a probability distribution (and its parameters) to describe the survival in infested fruits and finally correct the parameters to describe the survival in any fruit.

465. However, considering the washing procedure, IRA recognizes that: "Although there is no specific data to indicate their effectiveness against *N. Galligena*, it is likely these chemicals used at the correct dosage rates (concentration and time) would have varying degrees of effectiveness."[57]

466. Considering that there is no factual evidence of a most probable value, the IRA Team does not explain why the triangular distribution was chosen instead of a uniform distribution.

467. If there is no information on the proportion of infected fruits in relation to the proportion of infested fruits neither on the efficacy of washing to remove pathogen from infested fruits, then there is no scientific evidence to support the choice of the values used in the distribution.

Question 81
Please comment on the likelihood values and distribution put forth by Australia's IRA for importation step 5 (triangular distribution between 1 x 10E–5 and 1 x 10E–4, with a most likely value of 5 x 10E–5) regarding clean fruit being contaminated by N. Galligena during processing in the packing house. Is this evaluation objective and credible on the basis of the available scientific evidence?

Dr Sgrillo:

468. The discussion presented by IRA referring to step 5 supports the conclusion that the probability that a clean fruit is contaminated during processing is negligible. The IRA[58] Team concludes:

> "Given the extremely small likelihood of fruit being infested/infected with *N. Galligena*, the probability of surface spores being present on fruit and contaminating the dump water is similarly extremely small." and "The likelihood of clean fruit getting infected due to twigs at this stage would be extremely low."

469. However the values chosen do not reflect this conclusion. These values shows that for each 200,000,000 fruit passing through the packing house 10.000 could be contaminated by *N. galligena* and this could not be considered extremely low.

470. There is no scientific support to justify the values chosen for the parameters of the distribution.

Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? In answering these questions, please consider the IRA's assessment regarding the potential for clean apple fruit being contaminated by spores in the dump tank water. (IRA, Part B, pp. 126-127; para. 4.288-4.292 of New Zealand's FWS; and paras. 581-582 of Australia's FWS)

Dr Sgrillo:

471. The evaluation is fine, although the maximum, minimum and most probable value does not correctly express the likelihoods when the population is considered.

[57] (*footnote original*) IRA p. 125.
[58] (*footnote original*) IRA p. 127.

Dr Deckers (Response to whole question):

472. This risk seems to be very low because it should be infected by the spores present in the grading water.

Dr Latorre (Response to whole question):

473. There is no scientific literature to support the assumption that the rate of clean fruit contamination with *N. Galligena* would vary between 10^{-4} and 10^{-5} (most likely value of 5 x 10^{-5}) (importation step 5) in the packing house. Based on disease knowledge, it is extremely unlikely to occur under normal fruit management. This should be disregarded from the risk analysis.

Question 82
Please comment on the likelihood value of 1 presented in Australia's IRA regarding the probability that N. Galligena would survive palletisation, quality inspection, containerisation and transportation (importation step 6). Is this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, p. 127; paras. 4.293-4.295 of New Zealand's FWS; and paras. 595-598 of Australia's FWS)

Dr Deckers:

474. The same remark as on question 80 should be made here.

Dr Latorre:

475. There is no scientific literature to support the assumption that *N. Galligena* can survive palletisation, quality inspection, containerisation and transportation (Step 6). Comments: (i) It appears to be reasonable to assume that post-harvest processing does not affect survival of latently infected fruits, and then a value of 1 could be acceptable only for the survival likelihood of the internal inocula. (ii) However, these post-harvest processes can affect survival of the external inoculum, epiphytically contaminating the fruit surface, which may be negligible. Then a value of 1 would be unacceptable. (iii) The Biggs (1995) article is not relevant; it does not deal with *N. Galligena*.

Dr Sgrillo:

476. IRA states that "Some infected fruit not detected during sorting may be identified at quality inspection."[59]

477. Therefore the parameters of the distribution should reflect the probability of some infected fruits being detected in quality inspection. The choice of the value 1 for the probability of survival of *N. Galligena* means that infected fruits will never be detected in the quality inspection. This implies to accept that efficacy of sorting to detect infected fruits is 100% and the efficacy of quality inspection is 0%.

Question 83
Please comment on the likelihood values and distribution presented in importation step 7 of Australia's IRA regarding clean fruit being contaminated by N. Galligena during palletisation, quality inspection, containerisation and transportation (uniform distribution between 0 and 1 x 10E-6). Is

[59] (*footnote original*) IRA p. 127.

this evaluation objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this evaluation fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, p. 128; para. 4.296 of New Zealand's FWS; and paras. 599-601 of Australia's FWS)

Dr Deckers:

478. This risk is extremely low when the fruits are prepared after a longer period of storage, because most of the fruits that have internal infections of NG will be developed into rotten fruits and can be removed during preparation.

Dr Latorre:

479. There is no experimental evidence allowing us to assume that the likelihood that packed clean fruit is contaminated with *N. Galligena* would be different from zero, at Step 7. Comments: (i) At this stage it is unlikely that latently infected fruits develop symptoms and less likely that *N. Galligena* sporulates on the surface of latently infected fruits. (ii) Even if the inoculum (conidia) is present, post-harvest dissemination by fruit contact would be extremely unlikely because fruit injuries are needed for infection. (iii).This evaluation (Step 7) falls within a range that could not be considered legitimate. I suggest discounting this step from the risk analysis.

Dr Sgrillo:

480. See responses 133 – 134.

Dr Swinburne (Response to Questions 78, 80, 81, 82 and 83):

481. The assignment of necessarily subjective probabilities within a risk assessment model is not within the normal ambit of researchers whose stock in trade is data derived from experiments. Additionally, the importation steps described in the model do not fit comfortably with the steps in commercial marketing, as practised in, say, the UK. Consequently it is inappropriate to comment here on the veracity of the conclusions of each step individually, but the overall model does need comment:

482. It is usual for dessert apples to be harvested into bulk bins that are transported to on-site CA (controlled atmosphere) stores, where they are cooled, and sealed in chambers with adjusted CO2 and O2 concentrations. At intervals dictated by marketing strategies they are removed from these stores and dispatched to specialist pack houses, where the operations of washing, grading and packed for retail. The process may extend over many months, which has not been factored into the IRA model. Any infections present may develop into rots during this time, and this will be strongly influenced by both the store temperature and environment (Berrie et al 2007, appendix1). At grading these would be removed, so the numbers of infected fruit will diminish with time, consequently the statement that "none of the pack house measures would reduce infection" is incorrect, as it must also embrace the CA store period.

483. At several of the Imp steps the IRA emphasises the risk of superficial contamination but omits reference to any opportunities for these to convert to infections. Curiously it also overlooked the possibility that an infected fruit could develop into a rot with spores in store. These could be redistributed in the wash water to healthy fruit if they were not removed beforehand. This has not been found to be a problem in the UK, probably because of disinfectants in the wash water and the fact that apples are allowed to dry rapidly before packing.

484. The IRA model does show a reduction in the number of infected/contaminated fruit between harvest and dispatch, which in general would be expected, but how that was derived in the light of the observations above is not clear.

Question 84
Please comment on the conclusion in Australia's IRA regarding the probability of importation of N. Galligena. In particular, is the conclusion regarding a mean estimate for infection/infestation rate (0.0068% of the total proposed number of apples imported from New Zealand) objective and credible on the basis of the available scientific evidence? Is it based on respected and qualified scientific sources? Does this conclusion fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, p. 128; para. 4.299 of New Zealand's FWS; and paras. 602-603 of Australia's FWS)

Dr Deckers:

485. The final infection rate of the fruits will strongly depend on the presence of the NG infections in the orchards in New Zealand. Maybe it should be good the take the differences in susceptibility of the different apple varieties for NG into account when making the final estimation.

Dr Latorre:

486. Considering that mature apple fruits are from areas where climate conditions are not particularly conducive for fruit infection, a mean infection/infestation rate of 0.0068% falls out of the range that could be considered legitimate on the basis of general knowledge regarding the European canker. This value may not explain the real infection/infestation rate; therefore, it needs to be validated before acceptance.

Dr Sgrillo:

487. The proportion of 0.0068% of fruits infected was generated by Monte Carlo simulations executed by the IRA Team. Considering the importation volume expected by Australia[60] this proportion would result in 3,400 to 27,200 infected fruits imported by year.

488. The steps that compose the model are hypothesis about the real system. If each of these hypotheses is evaluated and validated, the model would also be validated and it result could be accepted. However the data presented in the IRA were not considered sufficient to validate each of the hypotheses proposed because most of the values of the distributions were established by guesses and not by sampling of the real world.

489. The result of a mathematical model is as good as the value assigned to its variables. Many of the parameters used in the simulation were considered overestimated because they didn't reflect the meaning of the qualitative category in the population. As consequence, the final result could also be overestimated.

490. As IRA does not present real data to validate the final result of the simulation is supposed that these data do not exist.

Question 85
Taking into account the quantitative data from the literature cited by Australia's IRA regarding the inoculum dose necessary for an N. Galligena infection to occur, please comment on the conclusions contained in the IRA on the ultimate assignment of probability values for exposure. Are those

[60] (*footnote original*) IRA p. 19.

conclusions objective and credible on the basis of the available scientific evidence? Are they based on respected and qualified scientific sources? Do these conclusions fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, pp. 134-139; para. 4.314 of New Zealand's FWS)

Dr Deckers:

491. The success of an infection of NG is not only inoculum dependent, but will largely depend on the conditions under which the infection has to be realised and on the presence of an host plant in a susceptible stage at the moment the inoculum is high enough to realise an infection. Question here is if there will be a host plant in a susceptible stage when a rotten fruits releases spores in the environment.

Dr Latorre:

492. The factors considered by Australia's IRA are acceptable; however, it is difficult to judge the likelihood assigned to each parameter. Although it is described in AUS-2BA (p. 136), it is not clear how Australia's IRA relates the inoculum dose necessary for infection and the probability of exposure to susceptible host plants.

Dr Sgrillo:

493. The quantitative data regarding the inoculum dose necessary for an *N. Galligena* infection to occur, cited by IRA, were not used by the IRA Team, at least in a direct way.

494. The minimum and maximum parameters elected for the Exposure are not directly derived from the source data. The IRA states: "These values [exposure] are based on the IRA team's view taking into account all the factors discussed above."[61] However the IRA Team does not explain how the available data were used.

495. The considerations of response 36 also apply to *N.galligena.*

496. The estimated likelihood of the exposure of *N.galligena* to susceptible host plant varies from "negligible" to "extremely low".[62] However when the values assigned to these categories are analyzed together with the total population (150.000.000 of fruits) the numbers that come out, in the lower categories, are much above than the qualitative description of the categories. See also responses 133-134.

Dr Swinburne (Response to Questions 84 and 85):

497. The end point of this analysis, predicting that 0.0068% of apples imported will "carry" the disease is predicated on a starting point presumption of a probability that each picked fruit is infected between 10x E-6 & 10xE-3, which at best is far too large to be credible. (see Q75). Given all the uncertainty in the calculations for the intermediate steps (see Q78-83) this outcome does not inspire confidence.

498. In theory one spore can initiate infection; in practice the probability of successful infection with one spore is very small. The literature cited in the IRA (p136) all indicate that the probability of securing infection increases with dose, and that at approximately 1000 spores per fresh leaf scar almost 100% infection will occur. More recent data (Lolas, 1999, Ph.D. thesis University of London)

[61] (*footnote original*) IRA p. 138.
[62] (*footnote original*) IRA p. 139.

also demonstrates that the number of conidia needed to achieve 50% infection varies with cultivar; another factor.

499. The "exposure value" quoted, assuming it is credible to deduce such a factor, seems to make assumptions regarding the year-round availability of infection sites, and that all discarded apples discharge spores all year, which are not correct. Moreover by stating "*a significant exposure factor for* N. galligena *is the fact that the fungus has a specific mechanism for spore dispersal*" in the conclusion on p 138, suggests that the outcome was heavily reliant on the erroneous presumption that rotted fruit would release ascospores. Unless the meaning of these values has been misunderstood, and based on the import of 2X10E8 apples of which 0.0068% is infected, the worst case scenario is that c14 infection events/year are anticipated, or at the lower probability, it would take nearly 100 years to get one such event.

Question 86
Please comment on the consequences assessment of "E" contained in Australia's IRA regarding the direct impact of European canker on plant life or health. Is that assessment objective and credible on the basis of the available scientific evidence? (IRA, Part B, pp. 145-147; paras. 4.326-4.327 of New Zealand's FWS; paras. 676-686 of Australia's FWS)

Dr Deckers:

500. The impact of an infection on plant life or health can indeed have substantial consequences for some regions in Australia. A rainfall of 1000 mm alone is not enough to estimate the risk of one region; more important are the rainfall conditions during specific periods of the growing season like during the leaf fall period or during harvest period when there are a lot of natural infection wounds available on the trees.

Dr Latorre:

501. According to the definition provided by Australia's IRA (AUS-2 p. 38-39, Table 10), the impact score, "E", contained in Australia's IRA implies measurable (permanent or reversible) effects on the economy at national, regional, district and local levels. Specifically, it implies highly significant impact at district ("A geographically or geopolitically associated collection of aggregates–generally a recognized section of a state…") and local levels ("An aggregate of households or enterprises–e.g., a rural community, a town or a local government area"). A "highly significant" impact would threaten economic viability through a large increase in mortality/morbidity, or a large decrease in production.

502. Based on the knowledge of European canker, and according to the general experience observed in other apple exporting countries where European canker is present, considering the consequences impact as "E" is not credible particularly because:

(a) Its economical effects (increased costs of winter pruning, fungicide treatments and the removal of stem lesions and infected branches; fruit yield reduction) can be absorbed by farmers with no major consequences to the farmer or to the local apple industry.

(b) The economical effects can be reversible rather than permanent at local levels.

(c) Disease severity can be high at tree level but disease prevalence and disease incidence are commonly far less than 100%.

(d) Even under highly-prone environmental conditions, disease progress rate would be low rather than high. Therefore, it would be very unlikely that *N. Galligena* could attack a high proportion of the apple population in a single growing season at local levels.

(e) Although European canker is often classified as one of the most economically damaging diseases of apple, this is a relative concept, indicating that European canker is a major disease of apple. In many apple-producing areas, this means that it requires adequate control measures annually. However, it cannot be interpreted as being a devastating disease limiting apple production at districts or local levels.

(f) "Fruit rot generally develops in the field or before harvest, although storage losses of 10–60% of the stored fruit crop have been reported in various parts of the world (Swinburne, 1964; Swinburne, 1975)." This may be true in some years and only under highly favourable environmental conditions at harvest. On the basis of the climate analysis presented, weather in Australia is favourable but not highly favourable for disease development (fruit infection phase) in summer months.

(g) There are no published records indicating that losses due to European canker affect apple production to the extent that they can threaten the economic viability of the apple industry locally or nationwide.

503. Therefore, the "E" score is unreal because it is unlikely that losses can be severe at the district or local level.

Question 87
Please comment on the consequences assessment contained in Australia's IRA regarding the control or eradication of European canker. Is that assessment objective and credible on the basis of the available scientific evidence? What is your view on the relevance of New Zealand's assertion in this regard that, in case of an outbreak of European canker, routine orchard control of the disease is possible as part of routine controls of other apple diseases already present in Australia, such as apple scab? (IRA, Part B, p.148; para. 4.331 of New Zealand's FWS; paras. 696-702 of Australia's FWS)

Dr Deckers:

504. When NG infections would occur in Australia, it would be necessary to consider a combination of specific fungicide treatments in combination with eradication measures at orchard level . It will be much more than the routine controls made for other diseases like apple scab. Also the timing of some specific treatments during leaf fall period will be necessary.

Dr Latorre:

505. A rating of "D" was assigned to control or eradicate European canker, which implies that European canker is unlikely to be discernible at a national level and would be of minor significance at the regional level, but would be significant at the district level and highly significant locally. This rating appears to be too high considering: (i). Control of European canker would be unlikely to be too high, physically and economically (ii). Control strategies for other apple diseases (e.g., apple scab, powdery mildew) would help to control European canker. (iii). The rate of disease progress is commonly low, which implies that eventual outbreaks of European canker must be localized, facilitating control and eradication. (iv) The presence of European canker has no effect on apple trade internationally, except with Australia. Thus, eradication of eventual European canker outbreaks would not affect the international trade.

Question 88
Please comment on the findings contained in Australia's IRA that birds and insects are suspected to act as dispersal mechanisms of N. Galligena because birds may be able to transfer spores of infected fruit to a host by feeding on fruit discarded as waste and then flying on to branches of trees. Are those findings objective and credible on the basis of the available scientific evidence? Are they based on respected and qualified scientific sources? Do these conclusions fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, pp. 135-136; para. 615 of Australia's FWS; para. 45 of the United States' Third Party submission)

Dr Deckers:

506. This way of infection has not been proven scientifically. Whenever birds or insects would be a vector for a NG spores, there would be also a need for a wound or a fresh leaf scar on the branch before the NG infection can take place.

Dr Latorre:

507. There is no scientific evidence demonstrating that birds and insects can disperse *N. Galligena*. These considerations are not acceptable and would not be legitimate according to the standards of the scientific community.

Dr Swinburne:

508. There is no scientific evidence that birds or other vectors are involved in the dissemination of infective agents of *N. Galligena*.

Question 89
Please comment on whether the consideration in Australia's IRA of the risk associated with the practice of packing houses leaving orchard wholesaler waste uncovered and exposed to the elements on the premises or in landfills is objective and credible, taking into account the likelihood of this situation occurring in packing houses in Australia. (IRA, Part B, p. 130 (European canker); paras. 4.130 and 4.419-4.421 of New Zealand's FWS; paras. 784-785 and 898-900 of Australia's FWS; and R 100 by Australia)

Dr Deckers:

509. Leaving high amounts of fruit waste in the neighbourhood of orchards should always be considered as a risk and should be avoided anyway. The treatment of the fruit waste should be included in the standard operation procedures of each packing house.

Dr Latorre:

510. This possibility would be extremely unlikely considering that: (i) Removal of fruit waste is essential to preventing infestation with other pests. (ii) Removal of fruit wastes is needed to comply with good agricultural practices. (iii) Dropping infected fruits or leaving wastes uncovered on the ground runs against the standards of packing houses and against the cultural attitude of Australian people. This possibility should be disregarded from the risk analysis.

Dr Swinburne:

511. There are several factors that mitigate against the possibility that dumped infected fruit pose a hazard to nearby orchards. Firstly, such a dump would have to be extremely close to potential host

trees, as the conidia are unlikely to be splashed dispersed more than a few meters, vertically or horizontally (seeQ73). Secondly, this has to occur not only during rain, but when the host is receptive to infection, which in commercial practice would be in spring at bud burst; consequently all the fruit discarded during the winter would have to remain *in situ* until then, which is unlikely in normal commercial practice.

512. In the UK fruit is picked and then stored in CA facilities on farm, but then sent to specialised pack houses with grading facilities. These are generally located in industrial parks, often close to other wholesale distribution centres. These sites are usually devoid of any amenity planting, and remote from orchards. If comparable practices are followed in Australia, it seems unlikely that fruit discarded at pack houses poses any great risk.

Question 90
Please comment on the reasoning contained in Australia's IRA in relation to the Tasmanian outbreak. Is that reasoning objective and coherent? In particular, please comment on the relevance of the fungus having no asci or ascospores, and whether the latter are better suited to long distance dispersal than conidia. Please also comment whether it is possible that the Tasmanian outbreak may have been due to a unique strain of N. Galligena that required another mating type for reproduction and whether this could explain why there was a limited spread of the disease during the Tasmanian outbreak. Please comment on whether climate is a relevant factor and whether the local climatic conditions during the relevant time period in Spreyton, Tasmania, may have been unfavourable to that particular strain of the disease. Please also comment on whether the chemicals used to control apple scab could have had an impact on the limited spread of N. Galligena during the Tasmanian outbreak. (IRA, Part B, pp. 117-118, 141-149; paras. 4.86, 4.92-4.93, 4.415-4.416, 4.323-4.324 of New Zealand's FWS and pp. 224-225 of Annex 3 to New Zealand's FWS; paras. 632, 654-669, 691 and 890-892 of Australia's FWS; and R 73 by New Zealand, para. 160)

Dr Deckers:

513. Different factors are probably involved in the limited spread of the NG infection in Spreyton in Tasmania: the combination of strict eradication measures and of specific fungicide treatments will have limited the further spread to other regions. Probably the climatological conditions were not optimal for a strong disease development. The hypothesis of the presence of a unique strain seems unlikely to me. The introduction could have been here trough infected plant material. The lack of ascospores can be the consequence of the fact that there was no formation of the perithecia in which the ascospores can be formed.

Dr Latorre:

514. The reasoning contained in Australia's IRA in relation to the Tasmanian outbreak seems reasonable. Comments:

(a) One would expect slow movement if environmental conditions for establishment and spread of European canker are rather unfavourable.

(b) The absence of ascospores is irrelevant, considering that in most parts of the apple-producing areas, European canker is successfully disseminated by conidia.

(c) It is true that ascospores can be moved by wind currents facilitating dispersal. Nevertheless, I doubt that the absence of ascospores limits the spread of European canker under Tasmanian conditions. It is possible that the environmental conditions in Tasmania were unfavourable for ascospore production.

(d) Unfavourable climatic conditions and the use of chemicals to control apple scab, or even specific fungicide treatments to control *N. Galligena*, may have limited disease spread in Tasmania.

(e) Ascospores and conidia can be dispersed locally (meters from the source of the inoculum). Long-distance dissemination (kilometres) is explained by the transportation of infected plants.

Dr Swinburne:

515. The outbreak of canker at Spreyton, Tasmania is used by both sides to support opposing views of the likely hood of the disease establishing on mainland Australia.

516. There seems to be acceptance that this outbreak originated in nursery infection (seeQ53) prior to the trees being planted, which is consistent with the report that it was confined to a few blocks or orchards on some farms. Failure to spread beyond this limited area is attributed by the parties to conflicting reasons:

517. The Australian position is that spread did not occur due to a combination of the eradication programme, fungicide regimes used to control scab and the possibility that this outbreak was caused by an aberrant strain of the pathogen that lacked the ability to produce the ascospores necessary for long distance dispersal. The removal of all infected trees would reduce and finally eliminate the opportunity to spread but does not explain the failure to do so in the earlier years. There is no doubt that fungicide regimes for scab do reduce the severity of canker incidence (seeQ53) but would not explain the failure of existing cankers to form perithecia. There is no evidence in the literature that there are distinct strains of the pathogen responsible for European canker in the apple, including the data of Flack & Swinburne (1975). The alternative explanation put forward for the lack of ascospores is that the pathogen is heterothallic, and one of the mating types was missing. There are conflicting reports in the literature concerning sexual reproduction in *Nectria* (El-Gholl, Barnard & Schroeder 1986; Kruger, 1974), but the former (homothallic) seems the most convincing. To assume that failure to form mature perithecia was due to the presence of only one mating type also requires the assumption that the entire epidemic originated, somewhere, with one spore, which is most unlikely.

518. The NZ position is that climatic conditions in Tasmania are marginal for the development of canker, and this accounts for the failure of the disease to spread. The impact of climate generally is discussed in Q56. In California ascospores fail to develop in most seasons, (Wilson, 1966). That this is due simply to climate and not to problems involving heterothallism or unusual strains of the pathogen is revealed by observing that in some years they do develop in California.

519. Australia additionally invokes the Tasmanian experience as evidence that the disease can establish elsewhere on the mainland, and cites the difficulties of eradication there as a guide to the economic impact that would result if it did so. The weakness of any predictive model based solely on annual rainfall has already been discussed (Q66). The key factor has to be related to the days of rain-fall, (leaf-wetness duration), associated with spore formation, dissemination and finally infection. The Beresford and Kim model (Annex 3 NZ FWS), which crucially includes rain days, predicts that Tasmanian districts have a climate which is only marginally congenial for canker, thus explaining the duration of the outbreak.

Question 91

Does Australia's IRA provide an objective and coherent assessment of the likelihood and implications of New Zealand apples being repacked at rural packing houses in close proximity to orchards, when assessing the risks related to fire blight, European canker and ALCM? Was such assessment made with proper methodological rigour? (Para. 4.418 of New Zealand's FWS; R 99 by Australia)

Dr Deckers:

520. I don't see the necessity to repack fruits that are exported from New Zealand to Australia.

Dr Latorre:

521. The assessment and implication of the repacked apples at rural packing houses in close proximity to the orchards is a possibility that cannot be denied. However, the impact of repacked fruits can be minimized if fruit is exported in retail-ready packs, as New Zealand has suggested.

Dr Sgrillo:

522. See question 46.

Dr Swinburne:

523. Fruit shipped from New Zealand in bulk bins to pack houses in Australia for final grading could have had time to develop rots, including, if present, time for quiescent infections by Nectria to become progressive. Fruit graded in NZ into retail ready packs need only to go to distribution centres, and, as any rots will have already been removed, have only a relatively short time to develop any more. (Q53). The removal of any rots during grading in NZ would in any event reduce the number arriving in Australia; this does not seem to have been allowed for in Step 6 (IRA p127) (see Q82).

524. It is not possible to comment on the marketing strategies that might be adopted. But R99 by Australia makes it clear that the IRA indicates that fruit discarded by consumers constitutes the greatest threat for the disease to enter the country. Fruit out of store would be held at considerably less than 100%RH. To become infectious units, discarded apples would require a period of "leaf wetness" to develop spores. It is most unlikely that in the prevailing climate all rotted apples so discarded would become infectious units.

Question 92
Is the requirement identified in Australia's IRA that a packing house provide details of the layout of the premises, sufficiently justified by the scientific evidence relied upon? (IRA, Part B, pp. 317; para. 4.149 of, and pp. 242 and 247 of Annex 4 to, New Zealand's FWS; and para. 963 of Australia's FWS)

Dr Deckers:

525. It is not clear which risk Australia wants to reduce with this measure.

Dr Latorre:

526. The general rules that registered packing houses must comply with normal procedures in accordance with good agricultural practices is acceptable. However, there is no risk of fruit contamination at packing houses unless infected fruits with visible sporulation are processed along with healthy fruits, but this possibility is negligible and in practice it should be regarded as non-existent. At present, there are no scientific reports demonstrating the spread of European canker at packing houses.

Dr Swinburne:

527. The requirement to provide details of pack house layout seems out of all proportion to the minute risk posed by any threat of cross contamination of apples supposedly coming from infected orchards. The inability of superficial conidia to survive on the surface of fruit has already been discussed. (Q49 etc). The same applies to any that might be transferred on boxes, grading machines or any other piece of equipment.

Question 93
Please comment on whether, from a technical perspective and as described in Australia's IRA, the 17 specific measures that have been challenged by New Zealand can be distinguished as either measures active in risk reduction, or measures designed to implement or support active measures. (R 14-26 by the Parties)

Dr Deckers:

528. All measures have the intention to reduce the infection risk actively, directly or indirectly.

Dr Latorre:

Measures challenged by New Zealand		Active in risk reduction	To implement or support active measures
Nº	Description		
Fire blight			
1	The requirement that apples be sourced from areas free from fire blight disease symptoms.	Yes	
2	The requirement that orchards/blocks be inspected for fire blight disease symptoms…		Yes
3	The requirement that an orchard/block inspection methodology be developed and approved that addresses issues such as visibility of symptoms in the tops of trees ,…		Yes
4	The requirement that an orchard/block be suspended for the season on the basis that any evidence of pruning …		Yes
5	The requirement that an orchard/block be suspended for the season on the basis of detection of any visual symptoms of fire blight.		Yes
6	The requirement that apples be subject to disinfection treatment in the packing house.	Yes	
7	The requirement that all grading and packing equipment that comes in direct contact with apples be cleaned and disinfected (using an approved disinfectant) immediately before each Australian packing run.	Yes	
8	The requirement that packing houses registered for export of apples process only fruit sourced from registered orchards.		Yes
European canker			
9	The requirement that apples be sourced from export orchards/blocks free of European canker (pest-free places of production).	Yes	
10	The requirement that all trees in export orchards/blocks be inspected for symptoms of European canker,…		Yes

11	The requirement that all new planting stock be intensively examined and treated for European canker	Yes*	
12	The requirement that an orchard/block be suspended for the season on the basis that any evidence of pruning or other activities carried out before the inspection could constitute an attempt to remove or hide symptoms of European canker.		Yes
13	The requirement that exports from an orchard/block be suspended for the coming season on the basis of detection of European canker and …		Yes
Apple leafcurling midge, ALCM			
14	The requirements of inspection and treatment for apple leafcurling midge,…	Yes	
General			
15	The requirement that Australian Quarantine and Inspection Service officers be involved in orchard inspections for European canker and fire blight, in direct verification of packing house procedures, and in fruit inspection and treatment.		Yes
16	The requirement that New Zealand ensure that all orchards registered for export to Australia operate under standard commercial practices.		Yes
17	The requirement that packing houses provide details of the layout of premises.		Yes**

*To my understanding, this measure would prevent further dispersal of European canker; however, it does not directly reduce the risk of fruit contamination. I suggest eliminating this measure.
**To my understanding, this measure does not apply, considering that there is no scientific evidence supporting the possibility that European canker can be disseminated at the packing houses.

Dr Swinburne:

529. The following measures would be deemed to be in support of active measures, and not active measures in risk reduction: planting healthy stock as replacement trees, disqualification of an orchard if pruning began before inspection, requirement of pack house layout, and indeed the direct involvement of AQIS officers in the inspection processes.

IV. ALCM

Question 94
Please comment on whether the evaluation in Australia's IRA of the following issues was objective and credible, relying on respected and qualified scientific sources, and based on sufficient support in the available scientific evidence: (i) the conditions for adult emergence of ALCM; (ii) the number of ALCM generations per year; (iii) the flight range of ALCM; (iv) the adult life span of ALCM; (v) the climatic factors in respect of ALCM spread; (vi) the host range and conditions required for ALCM egg laying; (vii) the absence for the need for a vector for ALCM to be able to spread from its initial location; and (viii) the life stage in which ALCM could enter Australia on export apples imported from New Zealand. (Paras. 85, 743-745, 796-797 and 802-815 of Australia's FWS; R 81 and 85 by New Zealand)

Dr Cross:

(i) the conditions for adult emergence of ALCM

530. There is a disagreement between the parties as to the time that would be needed for ALCM adults to emerge after being removed from cold storage. NZ claims that 13-18 days would be required but supply no evidence to verify this. For such a narrow time window of emergence, all individuals would have to be at the same stage of development (pre-diapause mature 3rd instar larvae) at harvest, be forced into diapause by a possibly short period of cold exposure, be held in a long enough period of cold exposure to break diapause in all individuals and then be raised to a constant high temperature (> 25 °C) until emergence occurred. Even if this happened it is unlikely that emergence would occur over a period of 5 days. New Zealand's claim must be based on a supposition that all pupae and individuals at other stages of development are killed by cold storage, even if the cold exposure were for short periods. This is probably not the case and evidence needs to be provided by New Zealand before the 13-18 days emergence delay for all individuals should be considered true.

531. Australia is probably correct to assert that (some) adults could emerge as soon as the appropriate triggers are encountered by the pupa. It is likely that individuals would emerge over quite an extended period ranging from <1 day to > 60 days and possibly to > 1 year depending on conditions. In many midge species, a proportion of individuals need two or more "winter" periods of cold to break diapause. However, no studies appear to have been done to resolve the issue definitively for ALCM.

532. It is likely that ALCM in cocoons on apple fruits emerging from cold storage would be at a wide range of stages of development including:

1. Third instar larvae that have just cocooned and need to complete diapause and post diapause development. Note the word "pre-pupa" is sometimes used to refer to the mature third instar larva in a cocoon before it pupates. Whilst this is perhaps a convenient label it is meaningless in the case of ALCM. There is not a fourth instar that might be recognised as a pre-pupal stadium, as in some other insects. Darvas, Skuhrava & Andersen (2000), in "Agricultural Dipteran Pests of the Palaearctic Region" record that prepupae of the third generation overwinter in cocoons and pupate in the spring and give Alford (1984) as one of their references for that statement. But Alford (1984) does not mention prepupae. Gagne, in his major works on North American and Neotropical gall midges (Gagne 1989, 1994) makes no mention of prepupae.

   In any case, these individuals would require a period of exposure to cold to break diapause followed by a period of post diapause development to adult emergence which would vary considerably according to temperature. The time requirements for development and how they vary with temperature for these individuals have not been determined for ALCM. Note that the emergence of first generation ALCM males in spring can occur over a period of several weeks (Tomkins et al., 2006 (Aus exhibit 92)), a considerably greater time span than the 5 day range between 13 and 18 days indicated by NZ. In any case, the time requirements for these individuals could be greater than the requirements for individuals in 2 below.

2. Mature third instar larvae that have just cocooned that do not require diapause but have to complete pupal development. Periods of cold may force all these individuals into diapause but this has not been proven and the conditions (exposure times/cold severities) to force pre-pupae into diapause have not been studied and are thus unknown. It is quite possible that some mature third instar larvae in cocoons in cold

store would not go into diapause if cold exposure time were short. Barnes (1948) reports that pupal development takes 13-18 days but this was an approximate time range for individuals held at ambient conditions in a laboratory. The rate of insect development is usually proportional to temperature above a threshold temperature. Unfortunately, the threshold temperature and the day degree requirement for ALCM have not been determined, though some information on which to calculate them is provided by Shaw et al. (2005) (NZ exhibit 16). Using development times to 50% emergence of 61, 35, 23, 19 and 14 days at 11, 15, 19 23 and 27 °C, respectively (taken from figure 2 on page 309) and performing liner regression between the rate of development against temperature, an excellent linear fit is obtained (R2=0.991) giving a threshold development temperature of 6.44°C and a requirement of 295 degree-days above the threshold temperature for 50% emergence. If temperatures are only just above the developmental threshold of 6.44 °C when the ALCM on apples were removed from cold store, pupal development could take a long time, perhaps many weeks If temperatures were high, development would proceed rapidly and emergence could perhaps occur in less than 13 days, though it is probable that prolonged exposure to high temperatures would result in high mortality and development times much shorter than 13 days may be impossible. Note that Shaw et al. (2005) (NZ Exhibit 16) only explore temperatures in the range 11-12 °C and do not provide the time distributions of emergence.

3. Pupae at various stages of development including some at late stages of development that were nearly ready to emerge as adults at the moment of putting into cold storage. These individuals would be ready to emerge more or less as soon as suitable conditions for emergence were restored.

NZ assertions seem to be based on an assumption that cold storage of apples would either kill individuals that were pupating or perhaps force them into diapause, though no evidence to support this has been supplied. Indeed, if pupae were killed by exposure to cold, then ALCM's existence would be threatened by post diapause cold periods in spring. No evidence has been produced that AA or CA storage with very low oxygen conditions (< 1%) lead to rapid mortality of ALCM pre-pupae or pupae. Respiration rates of these life stages would be very low and it's likely that individuals could survive long periods in CA.
Gagne in his major works on North American and Neotropical gall midges Gagne (1989, 1994) says that pupal diapause, though known in the Cecidomyiidae, is rare, having been recorded in *Contarinia negundinis* in North America, in *Resseliella lavandulae* in France and in *Hasegawia sasacola* in Japan. It is most unlikely to occur in *Dasineura mali*.

What is lacking is good quality studies investigating 1) the effect of temperature on the rate of development (duration) of the different life stages of ALCM 2) requirements for diapause induction and breaking in ALCM 3) the effect of exposure to varying degrees of cold and CA storage on survival of the different ALCM life stages 4) The requirements for temperature, humidity and windspeed for emergence of adults after pupation. Until such studies are done this question cannot be resolved with certainty.

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 to mate and lay eggs to start an infestation.

(ii) the number of ALCM generations per year

533. Para 3.75 of NZ FWS that there are usually four generations in NZ per annum though 5 have been reported ignores the report of up to 7 generations par annum in NZ by Tomkins (1998)(Aus exhibit 89). However, the occurrence of 7 generations seems unlikely and would require a long season of continuously warm conditions with no interruptions by dry periods and continuous availability of growing shoots for galling. Tomkins (1998) may have based his estimate of 7 generations on a simple calculation of the length of the season and the absolute minimum generation time of ALCM, rather than observing successive life cycles of the pest through the season, which would be very difficult. The occurrence of 4 or exceptionally 5 generations is more likely. Note that in Cross, Hall, Shaw and Anfora, Crop Protection 28(2009), 128-133, data, including from New Zealand, shows generation times of 52, 46 and 39 days for the first 3 generation of ALCM between 41 and 51 ° latitude. However, the precise number of generations has only limited bearing on the risk assessment.

(iii) the flight range of ALCM

534. There don't appear to have been any studies into the flight range of ALCM females where females are forced to find a host. The distance in Aus IRA of 200 m is perhaps plausible but not supported by evidence. The 30 m distance (over 3 generations) given in Suckling et al (2007) (NZ exhibit 15) is based on the decline in shoot infestation rates in new apple trees adjacent to an old established planting in a sex pheromone mating disruption trial, a situation which is not directly analogous to the situation required for the risk assessment of a mated female flying to a distant host. It does not indicate that females are only able to fly a maximum of 30 m. The value of 200 m suggested by Australia appears to be based on the background rate of infestation up to 200 m shown in Figure 4 of Suckling et al. (2007) but this data does not show that these infestations were caused by longer range movement of females from the adjacent block. Suckling et al. (2007) suggest that low levels of infestation were present on the new trees when they were planted. Cross (pers. comm. 2005) (now published in Table 5 of Cross & Hall, Crop Protection 28 (2009), 139-144) found that male ALCM could be attracted over a distance of 50 m (the greatest distance investigated) away from the host plant by a sex pheromone lure. It is possibly that the flight range of females is shorter than males because females are carrying an egg burden. However, females tend to have slightly larger, perhaps stronger bodies and wings which might compensate for this. The flight range of females relative to males has not been investigated. It is possible that gusts of wind could increase the range flight range of females and this would seem logical though there appears to be no evidence to support this. ALCM avoids flying in windy conditions. However, the example of the lettuce aphid invasion of Tasmania from NZ (2600 km) is not relevant as aphids are known to have long range dispersal mechanisms whereas leaf midges do not. However, the assertion (Aus WS para 808) of a flight range of 30-50 m would be ample in many cases between an orchard and pack house co-located within an apple orchard is reasonable.

535. Australia's IRA with respect to this issue was objective and plausible and relied on what little real evidence there was, but the available evidence was insufficient for a scientifically sound assessment.

(iv) the adult life span of ALCM

536. The life span of females in the field will depend very much on microclimatic conditions at the time. The life span of 2-6 days appears to be based on cursory laboratory observations and not on a properly conducted study of the distributions of longevity of males and females and how they are affected by conditions. The meaning of the terminology "life span of 2-6 days" is vague. It is unclear whether it means that the average life span is somewhere between 2 and 6 days or whether it means

all or at least most individuals live between 2 and 6 days. The lower limit of 2 days does not include the probability that many individuals in some circumstances will die on the day of their emergence. The life span of males and females is likely to be short in warm, dry, windy conditions. Hall & Cross (2006) in the process of rearing many hundreds of virgin male and female ALCM adults to identify the midges sex pheromone, found that females held in glass entrainment vessels at 18-20 °C and low (<500 ml/min) airflow conditions mostly survived < 1 day with a small number of individuals surviving for 2 days and none for longer. Males held in the same conditions survived < 24 hours. Females held in small tubes with moisture provided in fridge conditions ~ 4°C survived 4-5 days, rarely 6. Again, quantitative field studies are lacking but a life span of 1-2 days in the field is probable. Males probably have a shorter life span than females. However, there appears to be no substantive disagreement between the parties on this issue even though in my view it has very substantial bearing on the risks of establishment and spread.

537. The Aus FWS was objective and credible and used the available scientific evidence.

(v) the climatic factors in respect of ALCM spread

538. In its assessment of the likelihood of spread of ALCM in Australia, Australia's IRA considered the spread and distribution of ALCM throughout NZ since 1950. Victoria, Tasmania and New South Wales were considered to have a suitable cool climate for the spread of ALCM but the assessment was broadly qualitative, not quantitative. ALCM can tolerate quite a wide range of climates and it is inevitable that conditions conducive to ALCM establishment and spread are present in several, perhaps many areas of Australia, especially in areas where the climate is favourable to apple growing. 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.

539. In the northern hemisphere, in the continent of Europe, where ALCM has long been present and has reached it's distributional equilibrium, ALCM does not appear to occur at latitudes less than approximately 38°. It is absent from southern Spain and does not occur in Israel where there are extensive apple orchards. This minimum latitude value needs to be confirmed by more detailed investigation. In the USA, ALCM is absent from the southern states of Carolina and Georgia although it is present in New York State and has spread westward from there. The most northerly part of New Zealand is about 35°.

540. Based on this evidence, it seems unlikely that ALCM would pose a significant threat in areas north of ~38° latitude in Australia. So the threat of ALCM invasion appears to be confined largely to SE Australia and Tasmania.

541. ALCM needs sufficiently long cool (< 5 C°) periods in winter for diapause breaking and reasonably regular summer rainfall to survive. The winter chill intensity and duration requirements for diapause breaking have not been established nor have the summer rainfall requirements. However, the current distribution provides evidence to establish limits. For rainfall for instance, ALCM has established in areas of Washington State, USA, west of the Rockies, but is as yet absent to the east of the Rockies where there is little summer rainfall and apples are grown using irrigation water. Climatic conditions in SE Australia, which have been exceptionally hot and dry, have been quite unsuitable for ALCM survival.

542. The current distribution of ALCM could have been used to establish climatic conditions that are especially favourable to ALCM and climatic boundary conditions for its existence. A climatic analysis would also have given a better assessment of the likely impact of ALCM in different areas of

Australia. However, the IRA did not assume that ALCM would spread to all areas of Australia where apples are grown commercially or domestically and the overall assessment is correct.

543. In conclusion, Australia's IRA was objective and credible, but did not draw sufficiently on available information and did not conduct a sufficiently detailed analysis.

(vi) the host range and conditions required for ALCM egg laying

544. It is well known and both parties agree that apple & related *Malus* sp are the only hosts of ALCM and that eggs are laid in tender leaves in growing shoots. In New Zealand, oviposition and larval attack has been observed in flowers resulting in damage to young developing fruitlets. This appears to be a very rare occurrence which has only been reported from New Zealand. It evidently occurs when there is a heavy early emergence in spring. Minimal temperatures and maximum wind speeds for female flight and oviposition have not been quantified.

(vii) the absence for the need for a vector for ALCM to be able to spread from its initial location

545. In biology, the term "vector" is normally used for an organism that transmits infection. ALCM does not have a vector in this sense. In this context, the term vector is referring to an agent, biological or physical, that can passively carry ALCM. ALCM females can fly short distances and thus it is self evident that a vector for short range spread is not needed. In successive generations it could spread long distances provided there was a chain host plants each separated by no more than the flight range of females. However, ALCM would require a "vector", i.e. a commodity or conveyance with which it could move, for long range spread between host plants separated by greater distances than the maximum flight range of females. It is considered that normally ALCM spreads to new areas on nursery material. The question of flight range has been dealt with under (iii) above.

(viii) the life stage in which ALCM could enter Australia on export apples imported from New Zealand

546. The answer is essentially the same as given in (i) above. Unless New Zealand can provide evidence to the contrary, ALCM could enter Australia as mature 3rd instar larvae (in the full range of stages of diapause) or as pupae (in the full range of stages of post diapause pupal development) in cocoons round the stalk or eye of apple fruits or possibly attached to bulk bins used in the orchard for picking apple fruits.

547. Australia's IRA relating to this issue was objective and credible and relied on what little scientific evidence there was but the available scientific evidence is insufficient for proper resolution.

Dr Deckers:

548. The adult emergence of ALCM will not be immediately after the cold storage; it will take some time depending on internal processes of development of the insect in the pupae that will depend on the environmental conditions.

549. In Europe we have only 3-4 generations per year, depending on the presence of active growing shoots on the apple trees where the adults can put their eggs on the young leaves. The presence of young leaves on active growing shoots is necessary.

550. The flight range in an orchard is normally limited, but introduction in a newly infected orchard indicate a general spread over the whole orchard in a very short time. This could indicate that wind distribution can play a role. There is no need for a vector.

551. In Europe the adult life span of ALCM is about 4 weeks for the first generation and 6 weeks for the later generations (Frankenhuizen, 1992, Wageningen, pp 207-209: schadelijke en nuttige insecten en mijten in fruitgewassen ; uitgave nederlandse fruittelers organisatie, NFO, Nederland ISBN 90 9002 363 1).

552. The life stage in which ALCM could enter Australia is the larvae in the cocoon externally present on the apple fruit. But the import by contaminated fruit trees is considered to be much more important for the introduction of ALCM than the import on infected apple fruits.

Question 95
Please comment on whether the evaluation in Australia's IRA of the potential entry and establishment of ALCM in Australia was objective and credible, relying on respected and qualified scientific sources with respect to the life span of ALCM and its flight range? Are the conclusions in Australia's IRA as to the relevant probability values in this respect chosen by the Australia's IRA sufficiently supported by the available scientific evidence? (IRA, Part B, pp. 175-183; paras. 4.350-4.366 of New Zealand's FWS; paras. 792- 824 of Australia's FWS; R 83-85 by New Zealand)

Dr Cross:

553. With respect to the narrow issue of the life span and flight range of ALCM, the evaluation in Australia's IRA was objective and credible (see answers to Question 94, parts (iii) & (iv) above).

554. With respect to the conditions for adult emergence of ALCM Australia's IRA was objective and credible and relied on what little scientific evidence was available. (see answer to Question 94, part (i) above).

Dr Deckers:

555. The chances for a New Zealand apple to bear a ALCM cocoon is already low and will decrease during grading when the fruits are washed and brushed. Crucial point in the further risk assessment is the survival rate of the larvae in the cocoons entering in diapause when they come into the storage room and when they come out the storage room some months later.

Question 96
Please comment on whether the evaluation in Australia's IRA of the potential biological and economic consequences of ALCM incursion in Australia was objective and credible? In assessing consequences of ALCM introduction, should current or historical data be considered to be more relevant? (IRA, Part B, pp. 184-187; paras. 4.367-4.377 of New Zealand's FWS; paras. 825-850 of Australia's FWS; R 86 by New Zealand)

Dr Cross:

556. Australia's methodology for assessing impacts (IRA p 35-40) is qualitative and some of the terms used are relativistic and are not clearly defined. In Table 10, much rides on the differences between "minor", "significant" and "highly significant" but the differences between these impacts is not defined in terms of economic loss, the need to apply insecticides or social consequences. Because of this, Australia's assignment of an impact score of D (national level-unlikely to be discernable; regional level – minor, district level – significant; local level – highly significant) of the direct impacts on plant health is credible though a C rating (district level – minor; local level - significant) in my view would be more appropriate. ALCM is a moderately damaging pest in nurseries, in young orchards during the early years of establishment and is perhaps most damaging to newly grafted trees. In these situations it reduces shoot growth and could reduce the rate at which newly planted orchards reach their full cropping potential. In many countries where ALCM is ubiquitous and the climate very

favourable for it (e.g. the UK), growers generally pay limited attention to it and live with it as a minor irritation and do not apply insecticides. In recent years it has become apparent that trying to control it with broad-spectrum insecticides does more harm than good because such insecticides are harmful to the pest's natural enemies and short term gains result in longer term resurgences. Several of the important natural enemy groups occur in Australia but the parasitoid *Platygaster demades*, an important natural limiting factor, appears to be absent, so it is likely that ALCM would be on average more damaging than it were if *Platygaster demades* were present. If the ALCM established in Australia, its impact could be mitigated by introduction of *Platygaster demades*. ALCM damage could be reduced by applications of selective insecticides if such materials become available in future but currently there are evidently no suitable insecticides for control of ALCM in Australia. In my view, ALCM is significant in particular orchards at a local level and this would deserve a C score.

557. Reliance on the survey of Nelson growers in the mid 1990s by Smith & Chapman (1995) coupled with the absence of *Platygaster demades* has perhaps led to the D categorisation. However, NZ is right to point out that the pest status in NZ has reduced since the introduction of integrated fruit production programmes and a reduction in the use of broad-spectrum pesticides which disrupted natural enemies of ALCM. This mirrors the European experience where most growers live with ALCM without apparently suffering serious losses and seldom make treatments in newly planted orchards to control it.

558. The impact scores of "A" for direct impacts on human life and other aspects of environmental effects in the IRA are objective and credible.

559. The D categorisation is also somewhat severe in the case of the indirect impact on the requirement for control measures and a C rating would be more objective and credible. The control or eradication alternatives are very different and should not be lumped together. Eradication of a first initial outbreak would certainly be difficult and costly and would justify a D score. It would require the grubbing of the infested trees or orchard (s) and of others in the vicinity. The consequences of doing this would very much depend on the local circumstances and how extensive the initial outbreak had become before it was first detected. If an attempt was made to eradicate the pest from a whole district, an E score would be appropriate. However, as stated above, the consequences of possibly having to apply control measures in nurseries on newly planted orchards at a local level only justifies a C score.

560. The categorisations in the IRA of the impact of ALCM on domestic trade or industry, international trade and environment and community are credible and objective. However, effects of ALCM infestation on skin finish or fruit quality are rare. The type of damage reported from New Zealand has not been reported from elsewhere and is extraordinary. The international trade rating would very much depend on the scale and importance of exports from Australia.

561. However, the re-categorisation of the direct impacts on plant health and the need for control treatments would not result in a change in the rating of the overall consequences as "low". In this respect, the conclusion of Australia's analysis was objective and credible.

Dr Deckers:

562. Why not decide on the need for a fumigation only when there is a problem with ALCM found on an apple in the orchard or during grading and packing; this would allow to reduce the risk of a potential introduction into Australia substantially.

563. The problem of an ALCM infection in an apple orchard is not considered in Europe as a major problem, but more as a secondary parasite that makes some damage on the leaves without interfering too much with the productivity of the fruit trees.

Question 97

Please comment on whether the consideration in Australia's IRA of the mortality and occupancy rates of ALCM cocoons on New Zealand apples was objective and credible, relying on respected and qualified scientific sources? Is such consideration sufficiently supported by the available scientific evidence? Did the consideration in Australia's IRA in this regard take into account relevant viability data regarding ALCM insects or cocoons present on New Zealand apples, from respected and qualified scientific sources? (Paras. 67 and 103 of New Zealand's opening statement and Exhibit NZ-102; paras. 731 and 734 of Australia's FWS; and R 87 and R 88 by Australia)

Dr Cross:

564. The sources of data on occupancy and viability of ALCM in cocoons on New Zealand apples are very sparse. In my view the data on viability rates is very critical and the data is inadequate for an objective and credible assessment.

565. Two estimates are available for occupancy rates: In one survey of 30 blocks of Braeburn in the Waikato region and one in the Bay of Plenty in April – May 1994, 63% of cocoons were found to be empty (Tomkins et al, 1994; Exhibit NZ 43). The second study of Rogers et al. (2006) (Exhibit NZ 17) gives estimates of 36.6%-42.2 % empty cocoons on samples from 3 different varieties from the Nelson region in 2005.

566. Good data provided by NZ from endpoint inspection of 4,556,564 fruit over the years 2001-2004 from which a total of 7297 occupied cocoons were found is presented in Table 40 of IRA and used to set appropriate values for the probability of importation. The data in the table refers to occupied cocoons (unoccupied cocoons are evidently not reported). It is regrettable that the viability and parasitism rates of this large sample were not determined. Thus, the only data on the viability of ALCM in occupied cocoons is that of Rogers et al. (2006) (Exhibit NZ 17) from samples from 3 different varieties from the Nelson region in 2005. In this study estimates were made of the % of occupied cocoons that contained viable pupae. Larvae or pupae that were shrivelled were considered to be non-viable. Australia is also right to question the use of the prodding test to establish whether or not non-shrivelled individuals are alive or dead as this would not be a very accurate test. Some individuals that did not move in response to prodding could be viable. Rearing to adult is necessary for establishing mortality. However, if this were done it could well be found that mortality is considerably higher than established by examination and prodding.

567. The original paper was ambiguous in the way the data on mortality was reported as it was unclear whether the % mortalities were expressed as a percentage of the whole sample (including occupied and non-occupied cocoons) or just of the occupied cocoons. This was clarified in the letter of the author of the paper to Biosecurity NZ of 18 August 2008 (Exhibit NZ 102). A value of 75.1% mortality of occupied cocoons is given in this letter. Values for % mortality ranged from 45.2 to 93.9% in the 24 samples of 25 Braeburn fruits reported. Unfortunately, there are several discrepancies between the values presented for the % mortality based on all cocoons in original paper and in the letter which brings the soundness of the data itself into question.

568. The outcome of this is that there is only one bulk estimate of the mortality % of occupied cocoons of 75% from the Rogers et al (2006) data. This data is not sufficient for a reliable estimate of this parameter or of its variability. Given the crucial importance of viability in calculating risks and determining appropriate sample sizes it does seem important that a more rigorous study is conducted over several seasons. Australia's IRA does need to take viability into account. Until good data is produced, it would be entitled to conservative estimate of 50% viability (= 50% mortality) given the lack of data and likely variable nature of this parameter.

Dr Deckers:

569. The mortality of the ALCM cocoons is an important discussion point between both countries, but what I miss in the publication of Rogers et al. 2006 is the real level of survival of the cocoons hatching to new adult insects after the whole traject of exportation and transportation.

Question 98
Please comment on whether the proportion of apples shipped from New Zealand to Australia in packages ready for retail use (i.e., retail-ready), compared with the proportion shipped from New Zealand to Australia in bulk, subject to packaging prior to retail sale, is relevant for the assessment of the likelihood of entry, establishment or spread of ALCM? If yes, how? (IRA, Part B, p. 9; R 8-10 by the Parties)

Dr Cross:

570. With respect to ALCM, the proportion of apples shipped retail ready from NZ to Australia is crucial. If all fruit were shipped as retail ready and held in a cool chain conditions until sold to consumers, the risk of importation, establishment and spread would be greatly reduced, perhaps to negligible levels. The arguments presented in paras 4.361 – 4.363 of NZ FWS with respect to this appear valid.

571. The effect of the proportion of apples shipped retail ready on the risk is also evident in Australia's IRA. The probabilities presented in Tables 44 and 45 of Australia IRA part B 175-183 with respect to commercial fruit crops arriving at the 7 orchard wholesalers are very much higher than the risks associated with the other utility points and are thus particularly critical to the overall risk assessment.

572. The quantities of fruit and the way they are is held and handled, if at all, at these 7 orchard wholesalers, which evidently are in close proximity to apple orchards (Aus exhibit 66 shows a helpful aerial photo of one orchard wholesaler and the orchards in the vicinity), appears to be critical. Two very different scenarios would give very different risks of entry and establishment: 1) Higher value, retail ready fruit in packs or cartons ready for sale held in cold stores and redistributed to markets with minimal breaks in the cold chain and minimal losses resulting in disposal of fruits in the vicinity of orchards. The potential risks in this scenario are very low: There would be virtually no opportunity for ALCM adults to emerge, mate, exit the pack house and locate a susceptible apple tree. 2) Fruit arriving in bulk bins being graded and packed, or perhaps fruit being repacked with larger volumes of discarded fruit being held temporarily at ambient temperatures outside before being disposed of perhaps nearby in the vicinity of an apple orchard. The potential risks for this scenario are much higher. It appears to be because of this analysis that the overall IRA for ALCM is comparatively high.

573. Tables 42 and 43 of the IRA present a very wide range of values for the weekly indicative estimates of numbers of apples arriving at the 7 orchard wholesaler utility points as well as ay the other utility points. Data in Table 42 should be discarded as it relies on old inadequate published data. The estimates in Table 43 should be used because they are based on recent, good quality data taken from large numbers of samples over 4 years. Australia does not appear to have challenged the quality or provenance of the August 2005 data presented in IRA Table 40 but continues to give the old estimates (based on much more limited data) equal weight. In response to recent questions, NZ confirmed that the fruit was not subject to mitigation procedures or handled in any way to reduce the incidence of ALCM cocoons. They have also confirmed that the efficacy of detection of cocoons is very high, close to 1.0.

574. Within the cells of Table 43 two estimates of the numbers of apples arriving at utility points are given based on two extreme scenarios, 0.1%-5% of imported apples being distributed to orchard packing houses and the remainder (95%-99.9 being distributed to one urban wholesaler). The other scenario was based on 70-100% of imported apples being distributed to the orchard wholesalers 1) in an upper row of values based on P1 = Uniform (10^{-3}, 5 x 10^{-2}) and 2) the lower row of values based on P1= Uniform (0.7,1). The values for urban wholesaler are inverted because of the assumption that if fruit didn't go to the orchard wholesalers then it would go to the urban wholesaler. The median and mean value estimates based on these widely varying P1 values vary by a factor of 33. But if no fruit were handled in this way at the 7 orchard wholesalers or the urban wholesaler, the high risks should be excluded from the IRA.

575. Furthermore, packed and graded ready-for-sale fruit is unlikely to have green leaf material attached and the packaging itself would be much less likely to be infested with ALCM than say bulk bins into which the fruit was picked in the orchard, further reducing any risks.

576. In conclusion, requiring fruit to be packed and graded in NZ ready for sale would be an effective SPS measure to minimise the risk of ALCM entry and establishment in Australia. SPS measures would need to deal with the critical issue of disposal of waste fruit well away from apple trees. No detailed study has been presented by NZ to quantify these risks in similar pack houses in NZ.

Dr Deckers:

577. The risk for importation of the ALCM into Australia is greater for the apples exported in bulk than in ready-retail. By the preparation read-retail part of the cocoons can be washed of.

Question 99
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 level of infestation of viable ALCM cocoons on New Zealand apples? Is such consideration sufficiently supported by the available scientific evidence? From the information provided in the IRA, is it possible to determine the impact considered by the IRA of factors such as cocoon viability rates and the biology of ALCM on the likelihood of entry, establishment and spread of ALCM? (IRA, Part B, pp. 161, 163, 166 and 171; paras. 4.106-4.133 and 4.334-4.366 of New Zealand's FWS; paras. 721-824 of Australia's FWS)

Dr Cross:

578. The IRA estimation of the probability of importation using an assessment of 8 importation steps lead to a much higher estimate of the mean infestation rate of imported fruits of 4.1 x 10^{-2} (mean), 2.1 x 10^{-2} (5th percentile) and 6.5 x 10^{-2} (95th percentile) (IRA Results importation, p165) than the findings from the end point inspections presented in Table 40 which led to a most likely value of 1.3 x 10^{-3} being estimated (minimum 10^{-3}, maximum 3.8 x 10^{-3}). The first estimate of the mean infestation rate is 31.5 times higher than the second. In my view the second estimate is the more trustworthy because it is based on actual observations of large samples taken over 4 years. The first estimate should be discarded as the risk estimates at critical importation steps are subject to large uncertainties because they are based on inadequate old published data.

579. The data in Table 40 gives the frequency of occurrence of occupied cocoons. The actual infestation rate of viable cocoons would be substantively lower as a significant proportion of occupied cocoons are not viable (see answer to Question 97 above). Data on this aspect is comparatively scanty so a conservative estimate of 50% cocoons occupied by viable larvae or pupae might be appropriate given the likely variable nature of these parameters.

Dr Deckers:

580. In the whole discussion of the introduction of the ALCM trough mature apple fruit from New Zealand I don't see scientific data indicating that the occupied cocoons after storage are able to hatch and to form viable adult ALCM. This is an important information you need when you want to evaluate the risk of introduction.

Question 100
Please comment on the relevance, if any, of seasonal population development (i.e. whether by harvest, some adults may have emerged leaving behind their empty cocoons) and of parasitism by Platygaster demades, or both, on the evaluation in Australia's IRA regarding the viability of ALCM cocoons on New Zealand apples. (IRA, Part B, pp. 159-160; paras. 4.108 of New Zealand's FWS; and paras. 845-846 of Australia's FWS)

Dr Cross:

581. Seasonal population development is highly relevant. By harvest 3 generations of ALCM or more would have occurred and a substantial proportion are likely to have emerged, leaving behind their empty cocoons. In each generation a proportion of larvae may go into diapause and not emerge till the following year or the year after that. The proportion doing this varies considerably between generations and years. Barnes 1948 gives an excellent overview of extensive earlier studies by Whitcomb in Massachusetts in the late 1930s and early 1940s.

582. As stated in the answers given to Question 97 above, the survey of Tomkins et al (1994) found 63% of cocoons were found to be empty and the second study of Rogers et al (2006) (Exhibit NZ 17) gives estimates of 36.6%-42.2 % empty cocoons. These are two snap shots of what is likely to be a very variable parameter, due to variations in the availability of shoot growth and temperature and rainfall patterns as well as geographical and topographical variations. Better data is required.

583. However, it is important to remember that the August 2005 data gives rates of infestation by occupied cocoons (so the numbers of empty cocoons is not included).

584. Parasitism by *Platygaster demades* would also reduce the proportions of viable cocoons. Todd (1959) reports parasitism rates of 61.7, 78.7 and 47.3 in the 3rd generation larvae in the 1955-56, 1956-57 and 1957-58 seasons respectively. Parasitism in the second generation was much lower due to asynchrony of host and parasitoid populations. However, parasitism in the 3rd or possibly 4th generation are likely to be more relevant as apple fruits are most likely to be infested with cocoons from the generations which occur close to harvest. This is clearly a significant though highly variable factor. A conservative estimate might consider 30% of cocoons to be parasitised on average.

Dr Deckers:

585. The number of generations of ALCM can be different from one year to another and the population development will depend also on the presence of shoots in an active growing stage in the orchard. When there is a severe drought period, the vegetative growth of the trees can be stopped and this can have a decisive influence on the further development of the populations of ALCM.

586. The parasitism of ALCM by *Platygaster demades* can influence the final survival of the ALCM. Therefore it is important to know the number of the hatching adults coming out of the cocoons at the end of the whole export process.

Question 101
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. Is such consideration sufficiently supported by the available scientific evidence? Did the consideration in Australia's IRA in this regard take into account relevant viability data regarding the possibility of damage or infestation on leafy stipules? (IRA, Part B, pp. 161; paras. 4.113 of New Zealand's FWS; and paras. 739-740 of Australia's FWS)

Dr Cross:

587. At picking, depending on the firmness of attachment of the fruit, the way the fruit is picked and the degree of care taken to remove leaf material from picked fruits, a proportion of fruit will have mature leaves and leafy stipules attached. However, at harvest these leaves and leafy stipules are old and unlikely to be infested with ALCM. During picking, some mature larvae (and perhaps the odd ALCM gall) may fall from infested shoots above into the picking bucket and be transferred to the bulk bins where they may further contaminate fruits or the bins themselves. If bins were under the trees or the trees were tall, some larvae, leaf material possibly including occupied galls. The degree of leaf contamination in bulk bins is likely to be very variable, but could be high (Aus exhibit 64 illustrates that high levels of leaf contamination in bins can occur).

588. The leaf material does therefore pose some limited additional risk of increasing the frequency of fruit contamination by ALCM compared to the situation just prior to picking. However, the August 2005 endpoint inspection data will have already taken that into account with respect to the fruits.

589. The values given in the IRA Importation step 3 (page 161) for the likelihood that clean fruit is contaminated by apple leaf curling midge during picking and transport to the packing house is given as Uniform(10^{-3}, 5×10^{-2}). The basis for these estimates is unclear.

590. If fruit imported is into Australia prior to grading and packing in these bulk bins which contain leafy material, the leafy material and the bins would pose an additional risk to the fruit alone because there is a significant chance both leafy material and bins would be contaminated with ALCM cocoons.

591. However, there is a requirement for the apples to be free of trash. Furthermore, as stated above, if the fruit is retail ready and already packed and graded in NZ, the risks would be negligible Uniform (0, 10^{-6}). Furthermore, if the use of NZ Aug 2005 end point inspection data is accepted, no additional risk should be attributed as it is already taken into account.

Dr Deckers:

592. This way of infection of the clean fruit during picking or trough leaf material is not considered to be a real infection possibility and is not sufficiently covered by scientific science references. The leafy stipules are not the type of leaves that are infected by the ALCM ; the leaves in the top of the shoots are the ones that are infected.

Question 102
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 effects that cool storage of apples has on attached ALCM cocoons, specifically relating to the delayed developmental stage of diapause? Is such consideration sufficiently supported by the available scientific evidence? Did the consideration in Australia's IRA in this regard take into account relevant data regarding the life stages at which ALCM can enter diapause, the conditions required for adult emergence of ALCM from diapause and the time that it takes for adult emergence to occur following completion of diapause? (Paras. 4.118-4.120, 4.131 and 4.355 of New Zealand's FWS; paras. 765-766 of Australia's FWS)

Dr Cross:

593. This question has already been answered in detail in the response to Question 94 part (i) the conditions for adult emergence of ALCM.

594. The overall effect of the range of ALCM development stages and conditions to which they would be exposed after transport to Australia is a prolonged period of emergence of viable individuals. This substantially decreases the chances of a male and female emerging within the time frame of a few days which is required for successful mating. The risk of establishment is thus substantially reduced and this important factor has not been taken into account in Australia's IRA or in the subsequent intercourse between the parties. Some important further analyses is provided in the answer to question 104.

Dr Deckers:

595. It is not clear in the scientific literature when the ALCM will produce adult insects hatching from the cocoons after the diapause imposed by the cold storage period under normal atmosphere condition or under controlled atmosphere condition, with low oxygen and CO2 concentrations. The presence of young leaves on the shoot tips is also a necessary condition for a successful introduction of the ALCM in a new area.

Question 103
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 flight distance of male and female ALCM? Is such consideration sufficiently supported by the available scientific evidence ? Is the reasoning articulated by the IRA on the basis of such scientific evidence, including the methodologies applied, objective and coherent? Do the results of the IRA's assessment in this regard sufficiently warrant the challenged requirements relating to ALCM? (IRA, Part B, pp. 168-171; para. 4.123 of New Zealand's FWS; and paras. 802-804, 806 and 923 of Australia's FWS)

Dr Cross:

596. This question has already been answered in my response to Question 94 part (iii) the flight range of ALCM.

597. Australia's IRA with respect to this issue was objective and plausible and relied on what little real evidence there was, but the available evidence was insufficient for a scientifically sound assessment.

Dr Deckers:

598. The fact that the flight distance of the ALCM is limited in distance, reduces the real risk of contamination starting from apples carrying the pupae of the insect.

Question 104
Please comment on whether standard statistical techniques support the view that a 600 fruit sample would provide 95% confidence that no more than 0.5% (1 in 200) fruit have cocoons?

Dr Sgrillo:

599. The threshold infestation (minimum infestation that could be detected) of an inspection system, for large lots, sufficiently mixed, may be calculated rearranging formula 4 of the Appendix 3 of the ISPM 31.[63]

$$TI = \frac{1-(1-CL)^{\frac{1}{n}}}{e}$$

Where *TI* is the threshold infestation, *CL* is the confidence level, *n* is the number of units in the sample and *e* is the efficacy of the detection method.

600. Making the confidence level equal to 0.95 (95%), the sample size equal to 600 fruits and the efficacy of the method equal to 1 (100%), then the threshold infection would be 0.005, what means that the sample would contain, at least, one fruit with cocoons, if 0.5%, or more, of the fruit in the lot are infested. This is true if the efficacy of the inspection is 100% (every infested fruit in the sample is detected).

Please comment on whether an infestation level of 0.5% of apple fruit with ALCM cocoons would be enough to initiate an ALCM population? What are the factors that would be relevant in the consideration of the risk of establishment of ALCM? (IRA, Part B, Table 40, p. 166; paras. 4.127 and 4.137 of New Zealand's FWS; and paras. 734-736 of Australia's FWS)

Dr Sgrillo:

601. To evaluate if 0.5% of fruit with ALCM cocoons would be enough to initiate an ALCM population it should be considered that founder populations are typically small and consequently are at great risk of extinction. Generally, the smaller the founder population, the less likely is establishment. Though many scientists have referred to a "minimum viable population" there is rarely a distinct threshold. Instead it is more realistic to consider the probability of establishment as being a continuous function of the initial population size. This function reflects many characteristics of the species, such as its intrinsic rate of reproduction, mate location abilities, and genetic diversity.[64]

602. It is important to consider also that the dispersal pattern and the probability of finding a mate are critical for pest establishment. Insects that mate before dispersal have a higher probability of establishment. In the destination area, the initial population numbers are extremely low. So if insects disperse first, then they will probably never find a mate. This is true even for insects with very

[63] (*footnote original*) Exhibit AUS-30: International Plant Protection Convention, International Standard for Phytosanitary Measures No. 31: Methodologies for sampling of consignments, 2008, from Report of the Third Session of the Commission on Phytosanitary Measures, Rome, 7-11 April 2008.

[64] (*footnote original*) Reference 01 Liebhold, A.M., W.L. Macdonald, D. Bergdahl, and V.C. Mastro. 1995. Invasion by Exotic Forest Pests: A Threat to Forest Ecosystems. *Forest Science Monographs* 30.49 pp.

sensitive pheromone communication mechanisms[65], as ALCM. IRA describes the reproductive strategy of ALCM, informing that the males emerge 1-2.5 hour earlier than females. There is no information whether the males disperse just after emergence or not.[66]

603. A rough evaluation can be done, considering that theoretically a couple of adults with the right age and conditions, in the right time and at the right place could initiate a population.

604. Considering a sex ratio of 0.6[67] (1.5 females per male) it would be necessary 3 viable pupae to obtain a couple of adults. To estimate the number of fruits that carries 3 pupae it is necessary first to estimate the proportion of fruits that have alive pre-pupae. This is calculated multiplying the proportion of cocoons that have a pre-pupa (at the harvest) by the proportion of pre-pupae that is alive. The available data to estimate this proportion is variable. Tomkins et al. (1994)[68] estimates that 37% of the infected fruits have a pre-pupa while Rogers et al. (2006)[69] estimates that 60% of the infected fruits have a pre-pupae. This author estimated also that 41.1% of the pre-pupae are alive. These numbers generates proportion of 0.15 and 0.25, respectively, of infected fruits with viable pupae. The number of fruits necessary to obtain 3 viable cocoons if estimated dividing 3 by the proportions (0.15 and 0.25) what generates 20 and 12 fruits respectively. Finally if 0.5% of the population is infested then it will be necessary 4,000 and 2,400[70] fruits disposed in the same place and almost in the same time, respectively, to generate 1 pair of adults that could mate.

605. In the real world would be necessary much more than a pair of adults to start a new population because many low probability events have to take place successively for a population to establish.

606. Also the data available is not fully reliable. Rogers et al. (2005) states: "Naturally infested apples were selected from Nelson orchards with uncharacteristically high levels of ALCM during 2005". Therefore the data is not representative of the average New Zealand conditions. The experiment of Tomkins et al. (1994) was developed in Waikato and Bay of Plenty that contains only 3%[71] of the pip fruit production in New Zealand. See also response 108.

Dr Cross (Response to whole question):

607. Standard statistical techniques do support the view that a 600 fruit sample would provide 95% confidence of no more than 0.5% (1 in 200 fruit have cocoons) providing that the efficacy of detection is 100%, i.e. all the cocoons on the sample would be detected on examination.

608. The sampling is destructive (i.e. each apple sampled is not returned to the whole lot where it would be equally likely to be re-sampled), it is assumed that the midge cocoons are randomly distributed so the underlying distribution is the hypergeometric. The methods and calculations are set

[65] (*footnote original*) Dr Alexei Sharov (Virginia Polytechnic Institute and State University - personal communication).

[66] (*footnote original*) IRA p. 176.

[67] (*footnote original*) Reference 06: Heath, J et al. Flight Activity and Further Evidence for a Female-Produced Sex Pheromone of the Apple Leaf Midge, Dasineura mali, in Nova Scotia. Northeastern Naturalist 12(1): 93-102. 2005.

[68] (*footnote original*) Exhibit NZ-43: Tomkins AR, Wilson DJ, Hutchings SO and June S (1994) "A survey of Apple Leafcurling Midge (Dasyneura mali) management in Waikato Orchards", Proceedings of the 47th New Zealand Plant Protection Conference, 346-349.

[69] (*footnote original*) Exhibit NZ-17: Rogers DJ, Walker JTS and Cole LM (2006) "Apple Leafcurling Midge Cocoons on Apple: Pupal Occupancy and Mortality", HortResearch, Havelock North, New Zealand.

[70] (*footnote original*) 12 fruits/0.005 (infestation)=2400 fruits; 20 fruits/0.005=4000 fruits.

[71] (*footnote original*) Exhibit NZ-3: Pipfruit New Zealand Incorporated (2006 and 2008) Pipfruit Industry Statistical Annuals, January 2006 and March 2008, New Zealand pp. 18 and 20.

out in ISPM No. 31 Methodologies for sampling of consignments (Aus Exhibit 30). Table 1 gives the numbers of fruit that would need to be sampled from for lots of increasing size at the 95% and 99% confidence levels. For a lot of >200,000 fruit at the 95% confidence level, 597 fruit would need to be sampled for a 0.5% level of detection, close to the 600 fruit sample size specified, taking New Zealand's assurances that the efficacy of detection is close to 1.0.

609. However, assuming that the sampling proportion is small (i.e. the number of fruit sampled is small compared to the size of the whole lot), the binomial approximation (Table 3) is a reasonable one for the hypergeometric (this is equivalent to assuming sampling with replacement). This is much easier to calculate:

prob(≥ 1 infected fruit) = 1 – prob(zero infected fruit) $= 1-(1-p)^n$

For the figures given, with n = 600 & p = 0.005, prob = 0.0494 (close to 0.05, i.e. 95% confidence)

610. Alternatively, to evaluate n for a given underlying p and required confidence level 95%, the calculation is

$$n = \frac{\log_e(0.05)}{\log_e(1-p)}$$

611. Thus, with p = 0.005, n ≥ 598, i.e. a sample size n of approximately 600 as suggested.

612. Values for 95% and 95% confidence levels for different levels of detection and different % efficacies are set out in Table 3.

613. An infestation level of 0.5% of apple fruit with ALCM cocoons would be enough to initiate an ALCM population providing a sufficiently large number of fruits were disposed of within the female flying distance of an apple tree.

614. The main factors that are relevant in the consideration of the risk of establishment of ALCM are:

(a) The % fruits infested with occupied ALCM cocoons

(b) The proportion of these occupied cocoons that contain viable ALCM

(c) The proportion of ALCM that are parasitized by *Platygaster demades*

(d) The sex ratio of the ALCM cocoons (it is assumed that the sex ratio is 1:1)

(e) The period over which emergence occurs and it's statistical distribution

(f) The numbers of fruits placed or disposed of in close proximity to each other

(g) The distance of the site of disposal to the nearest apple trees

(h) The time of season, weather and the presence or otherwise of growing shoots on the apple trees

615. Below the probabilities of at least 1 male and 1 female emerging from different numbers of fruit placed or discarded within the flight range of ALCM females of a susceptible host with initial

infestation rates of 0.5%, 0.1% and 0.05% are calculated. Note that this does not take into account the fact that actual infestation rates would be reduced by mortality and parasitism nor the greatly reduced probability of two individuals emerging within a life span time frame of each other over a protracted period of adult emergence, which would further reduce the effective infestation rate substantially.

616. Using the same notation as above (a sample of n from a large population N (N>>n), with an underlying probability of infestation of p), we want to calculate the probability of at least 1 male and 1 female emerging from a sample. Assuming the binomial distribution can be used as an approximation to the hypergeometric distribution and also assuming that males and females are equally likely and that there is never more than one infection per fruit:

617. Let k = number of infected fruit, p_s = probability of all one sex (i.e. not at least one male of one female), prob(k) = prob of k infected fruit from binomial distribution.

618. If in the sample there are no infected fruit or only 1 infected fruit then there is no chance of having at least 1 male and 1 female.

619. If in the sample there are 2 or more infested fruit $p_s = \text{prob}(k)/2^{k-1}$ as for $k \geq 2$ there are 2^k different (ordered) combinations of males and female (e.g. for k = 3 can have MMM, MMF, MFM, FMM, MFF, FMF, FFM, FFF) of which 2 (MMM, FFF) will all be the same sex, so probability of k infested fruit with all same sex = $\text{prob}(k) \times 2/2^k$.

620. Thus Prob(at least one male and 1 female) = p_{mf} = 1 – prob(0, 1 infested fruit) – prob(all same sex)

$$= 1 - \sum_{k=2}^{n} \frac{1}{2^{k-1}} \binom{n}{k} p^k (1-p)^{2-k} - (1-p)^n - np(1-p)^{n-1}$$

621. The final two terms are the probability of 0 and 1 infested fruit respectively.

622. It is not possible to re-arrange the above formula to calculate n for a given probability of at least 1 male and 1 female, but it is possible to calculate that probability for various values of n. Using the GenStat statistical package, it is possible to do this using the above formula for $n \leq 150$, but the factorial parts of the formula then become too large; for $150 < n \leq 1000$ the program uses the probability function for the Binomial distribution. After n = 1000, this part of the program also fails to work because of large numbers within the calculation. It is likely that there are other numerical methods to do this calculation for n > 1000 if one cared to investigate.

623. The Table overleaf gives estimates of the probability of at least 1 male and 1 female emerging from different numbers of discarded fruit (n) where the initial infestation rate of occupied cocoons is 0.5%, 0.1% or 0.05%.

Table of probabilities of at least 1 male & 1 female for infestation rates of 0.5%, 0.1% and 0.01% with viable cocoons for given n							
n	p_{mf}			n	p_{mf}		
	0.5%	0.1%	0.05%		0.5%	0.1%	0.05%
25	0.0035	0.000148	0.000037	525	0.5346	0.05325	0.01511
50	0.0136	0.000598	0.000151	550	0.5587	0.05775	0.01648
75	0.0290	0.001338	0.000341	575	0.5818	0.06238	0.01791
100	0.0487	0.002357	0.000604	600	0.6040	0.06712	0.01938
125	0.0718	0.003645	0.000940	625	0.6252	0.07198	0.0209
150	0.0976	0.005191	0.001346	650	0.6454	0.07694	0.02247
175	0.1254	0.006986	0.001823	675	0.6647	0.08200	0.02409
200	0.1547	0.009019	0.002368	700	0.6831	0.08716	0.02575
225	0.1850	0.01128	0.00298	725	0.7007	0.09241	0.02746
250	0.2159	0.01376	0.003658	750	0.7173	0.09774	0.02921
275	0.2472	0.01646	0.004400	775	0.7331	0.1032	0.031
300	0.2784	0.01936	0.005206	800	0.7481	0.1086	0.03283
325	0.3095	0.02245	0.006074	825	0.7624	0.1142	0.03471
350	0.3402	0.02572	0.007003	850	0.7759	0.1198	0.03662
375	0.3703	0.02918	0.007991	875	0.7887	0.1255	0.03858
400	0.3998	0.03281	0.009038	900	0.8008	0.1313	0.04057
425	0.4285	0.0366	0.01014	925	0.8122	0.1371	0.0426
450	0.4564	0.04054	0.0113	950	0.8231	0.1429	0.04466
475	0.4834	0.04464	0.01252	975	0.8333	0.1488	0.04677
500	0.5095	0.04887	0.01379	1000	0.8430	0.1548	0.0489

624. The 600 fruit sample would provide 95% confidence that no more than 0.5% (1 in 200 fruit) have occupied cocoons, but the actual infestation rate would be reduced by a factor 0.5 x 0.7 for reduced viability and parasitism and probably by a further factor of 0.1 – 0.5 for the protracted emergence relative to the short life span, actual effective infestation rates of 0.1% or even 0.05% would be more realistic.

625. Note also that the average rate of infestation of NZ apples by ALCM as indicated by the August 2005 data is 0.16%, 3 x lower than the 0.5% 95% confidence value that would be detected by a 600 fruit sample.

626. Thus approximately 100, 500 or >1000 fruits would have to be discarded for infestation rates of 0.5, 0.1, and 0.05% in one place for a 5% chance of at least one male and one female emerging to start an infestation.

627. So to answer the latter part of the question, a 0.5% infestation rate of occupied cocoons would have a 5% chance of initiating an infestation if perhaps 500-1000 fruits were discarded in one place within the female flight range of a susceptible host.

Dr Deckers (Response to whole question):

628. The estimation of 1 fruit per 200 fruits carrying a cocoon seems to be rather high and indicate a relatively high infection pressure in the orchard. There will be surely circumstances that there will be no fresh vegetative growth at the end of the season and this factor will reduce the overall infection risk seriously.

Question 105
Please comment on whether the consideration in Australia's IRA regarding the inadequacy of an inspection and treatment system based on a 600 fruit sample to manage the risk for ALCM was objective and credible, relying on respected and qualified scientific sources. Is such consideration sufficiently supported by the available scientific evidence? (IRA, Part B, p. 188-190; paras. 1094-1096 of Australia's FWS; paras. 4.517-4.522 of New Zealand's FWS)

Dr Cross:

629. The adequacy of the 600 fruit sample size will very much depend on the way fruit is handled in Australia in particular the number of fruit that are likely to be placed or disposed of in the vicinity of an apple tree or trees. As set out in my answer to question 98, two very different fruit handling scenarios would give very different risks of entry and establishment and quite different sampling sizes would be appropriate.

630. If higher value, fruit is retail ready in packs or cartons ready for sale held in cold stores and redistributed to markets with minimal breaks in the cold chain and there were minimal losses resulting in disposal of fruits in the vicinity of orchards, the potential risks in this scenario are very low: There would be virtually no opportunity for leaf midge adults to emerge, mate, exit the pack house and locate a susceptible apple tree. A 600 fruit sample size would be very adequate to give a very minimal risk from disposal of small numbers of fruits by consumers etc in gardens or near orchards etc.

631. If fruit arrived in bulk bins for grading and packing with larger numbers of discarded fruit being held temporarily at ambient temperatures outside before being disposed of possibly nearby in the vicinity of an apple orchard, the potential risks for this scenario are much higher.

Dr Deckers:

632. The necessity to treat all the lots when a fruit sample of only 600 apples is inspected is surprising: why not only treat the lots of apples when they come from an infected orchard or when some ALCM have been found.

633. I don't understand why the fumigation treatment applied on the infected apple plots would not be able to reduce the ALCM populations sufficiently. Is there a clear scientific evidence for this view.

Question 106
Is the requirement identified in Australia's IRA that a packing house provide details of the layout of the premises, sufficiently justified by the scientific evidence relied upon? (IRA, Part B, pp. 317; para. 4.149 of, and pp. 242 and 247 of Annex 4 to, New Zealand's FWS; and para. 963 of Australia's FWS)

Dr Cross:

634. It is unclear how a detailed knowledge of pack house premises in NZ could be used to identify areas of risk with respect to ALCM. The locations where end point inspections for ALCM and freedom from trash take place clearly do need to be identified.

Dr Deckers:

635. There is no clear scientific background for this requirement.

Question 107
Please comment on what factors, other than the volume of trade in apples between New Zealand and Chinese Taipei, would need to be taken into account to support a contention that New Zealand's experience in exporting apples to Chinese Taipei may be used to draw conclusions on the potential for ALCM to enter, establish or spread in Australia as a result of imports of apples from New Zealand? (Para. 4.133 of New Zealand's FWS; para. 819 of Australia's FWS; R6 by Chinese Taipei)

Dr Cross:

636. The geographic location, the climatic conditions and the availability and locations of suitable hosts would need to be taken into account.

637. ALCM does not appear to occur at latitudes < 38°. Chinese Taipei is at ~25° latitude, much too far south to have a climate suitable for ALCM. Australia FWS para 821, states that Chinese Taipei has a subtropical, oceanic climate. There are no records of ALCM in tropical or sub-tropical areas (note also that apple trees are not normally grown in such areas because there is insufficient winter dormancy). If ALCM did temporarily establish there, populations would probably be of short duration and may go unnoticed.

638. Because of the unsuitable climate of Chinese Taipei, New Zealand's experience in exporting apples to there should not be used to draw conclusions about the risks of importation into Australia.

Dr Deckers:

639. The climatological situation in both countries as well as the list of possible host plants presenting both Australia and Chinese Taipei should be compared when one wants to compare the risk for ALCM introduction in both countries.

Question 108
Are the conclusions in Australia's IRA as to the probability range and distribution patterns (triangular distribution with a minimum value of 1.5 x 10E-2, a maximum value of 0.115, and a most likely value of 5 x 10E-2) for importation step 2 sufficiently supported by the available scientific evidence?

Dr Sgrillo:

640. The numerical values of the parameters of the distribution were based in a single survey of 30 orchard blocks, in 1993-1994 seasons, conducted by Tomkins et al. (1994)[72] as explained by IRA:

> "A survey of 30 orchard blocks in the Waikato region and one in the Bay of Plenty during the 1993–94 season recorded up to 11.5% of apples as being contaminated with apple leafcurling midge cocoons in the Waikato region, and around 1 to 2% in the Bay of Plenty."[73]

641. The IRA explains also how the distribution and the respective parameters were chosen:

> "The IRA Team decided to represent Imp2 as a triangular distribution with a minimum of 1.5 x 10–2, a maximum of 0.115 and a most likely value of 5 x 10–2. This was based on the evidence that contamination rates for pupae or larvae of apple leafcurling midge range from 1–2% to 11.5% of apples in the Bay of Plenty and the Waikato region respectively, and taking into account that these rates are not indicative of apple leafcurling midge abundance which is affected by rainfall leading to higher levels in wet districts or lower levels in dry districts".[74]

642. In assessing the scientific evidence have to be considered that there is no indication, whether these 31 orchards are representative of the population of orchards in New Zealand neither if the season 1993/94 may be considered typical from the climatic aspect. For these data be used with confidence would be necessary, also, to evaluate whether the management of orchards in 1993/94 is representative of the management of all orchards in New Zealand currently.

Are the conclusions in Australia's IRA on this issue scientifically justified and reasonable? Do these conclusions fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA?

Dr Sgrillo:

643. The conclusions were drawn from a survey conducted 15 years ago in 31 orchards, in a region where only 3% of the pip fruits are grown, where the climate is not representative and probably, where the orchard management were different.

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[72] (*footnote original*) Exhibit NZ-43: Tomkins AR, Wilson DJ, Hutchings SO and June S (1994) "A survey of Apple Leafcurling Midge (Dasyneura mali) management in Waikato Orchards", Proceedings of the 47th New Zealand Plant Protection Conference, 346-349.

[73] (*footnote original*) IRA p. 159.

[74] (*footnote original*) IRA p. 160.

644. The conclusions derived from this data should not be applied to the whole country without further consideration.

Is there any difference in this regard between the different areas in New Zealand where apples are produced for export? (IRA, Part B, pp. 159-160; para. 4.336-4.338 of New Zealand's FWS; paras. 726-738 of Australia's FWS)

Dr Sgrillo:

645. Hawkes Bay contains 61% of the pipfruit area for exportation while Waikato contains 3%[75]. According to New Zealand Waikato have warm wet climates, more conducive to ALCM[76].

Dr Cross (Response to whole question):

646. Australia IRA should use the August 2005 end point inspection data provided in table 40 of its IRA and discard steps 2 & 3 of its 8 step importation analysis. Step 2 relies on old and inadequate published data and the August 2005 data appears to be of much better quality being recent and based on large sample sizes over 4 years. New Zealand has given assurances that the August 2005 data was not from fruit subject to any risk or infestation mitigation procedures and that the efficacy of detection is close to 1. The risk values in step 3 of the IRA appear to be guesses. Australia does not appear to have challenged the quality of the August 2005 data but continues to give the old estimates based on much poorer quality data to which it has given equal weight. The most likely value of 5×10^{-2} for importation step 2 results in a 38.5 fold higher estimation of the most likely risk value for the August 2005 data (most likely 1.3×10^{-3}).

647. Figures X and Y in Australia's exhibit 51 also present good quality data on the incidence of ALCM cocoons on fruit and include additional information on the small percentages of lines that have > 2% ALCM as well as the maximum percentages of ALCM infestation.

648. There will be local differences in the degree of infestation but these may vary from year to year.

Dr Deckers (Response to whole question):

649. It is not because the likelihood that picked apple fruits are infested with ALCM with a value of 5x10-E2, that this is the value of the apples that care the ALCM after storage and after retail-ready preparation of the fruits when they enter the Australian market.

Question 109
In the context of its analysis regarding importation step 2, does Australia's IRA appropriately take into account the "viability" of cocoons? (IRA, Part B, pp. 159-160; para. 4.337 of New Zealand's FWS; and para. 729 of Australia's FWS)

Dr Cross:

650. The work of Rogers et al (2006) on cocoon occupancy and viability is cited in Australia's IRA importation step 2 analysis, but then it doesn't appear to have been taken into account when fixing the probability values in the summary analysis of importation step 2. If only 25% of cocoons contain viable ALCM then the values should be 4 times smaller. In view of the need for caution, 50% might

[75] (*footnote original*) Exhibit NZ-3: Pipfruit New Zealand Incorporated (2006 and 2008) Pipfruit Industry Statistical Annuals, January 2006 and March 2008, New Zealand pp. 18 and 20.
[76] (*footnote original*) New Zealand FWS para. 4.336.

be a suitably conservative estimate of viability, in view of the paucity of the available data. However, in my view the analysis in step 2 relies on old and inadequate data and the August 2005 data should have been used (see above). The analysis of risks does need to take into account parasitism by *Platygaster demades* and, more importantly, the protracted emergence of ALCM adults relative to their short life span.

Dr Deckers:

651. There is no clear scientific evidence of the survival of the insects in the pupae stadia of ALCM during storage and preparation of the fruits in the packing houses in New Zealand and Australia.

Question 110
Please comment on whether the finding in Australia's IRA that Rogers et al (2006) may have underestimated the number of cocoons containing viable ALCM, because the authors assumed that all occupants of the cocoons tested were pre-pupae, is objective and credible, relying on respected and qualified scientific sources. Is such finding sufficiently supported by the available scientific evidence? (IRA, Part B, p. 163; paras. 4.112 and 4.126 of New Zealand's FWS; and para. 733 of Australia's FWS)

Dr Cross:

652. Above the header to table 2 in Rogers et al (2006) it is stated that "Many of the dissected ALCM cocoons contained pre-pupae that were shrivelled and obviously dead". This presumably means that prodding was used as a final test on those that were not obviously dead and shrivelled with death characterised as failure to move when prodded. There is some uncertainty here as to the proportion of the individuals that were pupae and it is possible that some live individuals were scored as dead as they failed to move when prodded. The prodding test is not particularly good because some viable individuals may not move when prodded. But equally, a substantive proportion of those scored as live because they did move when prodded may have died subsequently and failed to emerge as adults. Australia is right to assert that a better test would have been to see what proportion of individuals actually emerged as adults but such a test may well have shown even lower viability rates.

Dr Deckers:

653. The definition of the pupae to be death was that they don't react when touched by a needle. It would have been better to look to the real number of hatching insects from the cocoons at the end of the storage period.

Question 111
In the context of importation step 3, are the conclusions in Australia's IRA in respect of the probability range and distribution pattern for fruit contamination by ALCM during picking and transport to the packing house (uniform distribution between 1 x 10E-3 and 5 x 10E-2) sufficiently supported by the available scientific evidence?

Dr Sgrillo:

654. The only scientific data provided by IRA is a publication by Tomkins (1998) who found out that typically a leafroll contains 20–30 larvae, but numbers up to 500 have been observed.[77]

[77] (*footnote original*) IRA p. 161.

655. There is also another source, identified as "industry sources" estimating a typical figure of up to 200 leaves per bin.[78] However further information would be needed to qualify and establish the credibility of these sources.

656. The quantitative data available do not guarantee that the parameters established describe the true population.

(ii) Are the conclusions in Australia's IRA on this issue scientifically justified and reasonable?

Dr Sgrillo:

657. The conclusions in IRA are a hypothesis about the contamination of the fruits. This hypothesis could be validated when scientific results are available.

658. However it is not clear which is the main mechanism of contamination of the fruits.

659. First the IRA Team explains how the contamination may occur:

> "Contamination may occur when infested leaves are picked during harvest along with the fruit".[79]

660. Australia subsequently informed that their main concern is not what is explained in the IRA, but the leaves that could be picked:

> "However, the main area of concern for the IRA Team was not the chance of contamination by ALCM cocoons and leaves directly adjacent to the fruit harvested."[80]

> "It is quite probable that pickers will brush against leaves or branches of other parts of trees which may sometimes harbour ALCM leaf rolls, occasionally causing ALCM larvae to fall from elsewhere on the tree into a picking bag or bins on the ground."[81]

661. The IRA Team does not explain which mechanism was considered to choose the values of the parameters of the distribution. The conclusions of the IRA need further justification.

Do these conclusions fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, pp. 161; paras. 4.339-4.343 of New Zealand's FWS; and paras. 741-746 of Australia's FWS)

Dr Cross (Response to whole question):

662. The values given in the IRA Importation step 3 (page 161) for the likelihood that clean fruit is contaminated by apple leaf curling midge during picking and transport to the packing house is given as Uniform (10^{-3}, 5 x 10^{-2}). The basis for the choice of values in the estimates is unclear. The basis is given is that the contamination only occurs when infested leaves are picked and the number of leaves picked but there is no information on which to quantify the risk. Uniform(10^{-3}, 5 x 10^{-2}) is a vague and wide range of values but Uniform (10^{-6}, 10^{-3}) may have been an equally valid guess!

[78] Ibidem.
[79] (*footnote original*) IRA p. 161.
[80] (*footnote original*) Australia FWS para. 742.
[81] Ibidem.

Dr Deckers (Response to whole question):

663. There is not sufficient scientific evidence for this step in the IRA importation step 3. If there are leaves that are harvested, it will be for the majority leaves coming from the bourse structures where the fruits are hanging on. In addition these leaves are not the leaves where the ALCM midges put their eggs on because they prefer the young leaves in the growing shoot tips.

Question 112
In the context of its analysis regarding importation step 3, does Australia's IRA appropriately take 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? (IRA, Part B, p. 161; paras. 4.114 and 4.342 of New Zealand's FWS; and paras. 743-744 of Australia's FWS)

Dr Cross:

664. In some years in some orchards, there could well be flushes of new growth in the weeks running up to and including harvest. This generally occurs if the fruit load is light and rainfall occurs or irrigation is applied. However, heavy fruit loads can greatly reduce or shut down shoot growth completely in the weeks running up to harvest. In the work in NZ, Italy and UK to establish a sex pheromone trap threshold for ALCM recently reported in Cross, Hall, Shaw and Anfora, Crop Protection 28(2009), 128-133, it was found that the third and fourth generation emergences of adult ALCM often occurred when few viable shoots for galling were present and this made it difficult to establish a valid relationship between trap catches and the numbers of galls that developed for the 3rd and 4th generations.

Dr Deckers:

665. Under standard orchard management condition, the majority of the vegetative shoot growth will be stopped some weeks before harvest time. Exceptionally it is possible to have late regrowth reactions at the end of the season but phytotechnically, these reactions have to be avoided because these late regrowth reactions mean a loss of terminal flower buds on the shoots.

Question 113
Please comment on the data for the period of 2001-2004 referred to in para. 4.344 of New Zealand's FWS, in terms of the possibility that a certain level of viable ALCM could survive packing house, quality inspection and export processes, and could arrive in Australia. In the context of importation step 8, are the conclusions in Australia's IRA in respect of the probability range and distribution pattern for ALCM surviving and remaining with the apple fruit after on-arrival minimum border procedures (uniform distribution between 0.7 and 0.9) sufficiently supported by the available scientific evidence? Are the conclusions in Australia's IRA on this issue scientifically justified and reasonable? Do these conclusions fall within a range that could be considered legitimate according to the standards of the scientific community and the methodology applied in the IRA? (IRA, Part B, p. 165; paras. 4.344-4.349 New Zealand's FWS; and para. 749 of Australia's FWS)

Dr Cross:

666. A 600 fruit sample taken in NZ prior to export would ensure with 95% confidence that the maximum infestation rate of 0.5% occupied cocoons was not exceeded. If there is a second mandatory independent border inspection of 600 fruits were done then this is essentially the same as a total inspection of 1200 apples. Substituting n = 1200 and using a 95% confidence value in the binomial formula gives the proportion of fruit as 0.0025, i.e. 0.25% or 1 in 400. So a second inspection would reduce the likelihood by a factor of 2, giving a probability factor for this stage of 0.5

not including any other mortality factors. This is not far from the value of 0.46 given in para 3.346 of NZ FWS (calculated on an assumption of an infestation level of 0.13%) and appreciably lower than the mid point value of 0.9 given in importation step 8 of Australia's IRA.

667. There appears to be a fundamental disagreement between the parties as to whether a second inspection would be carried out, Australia asserting that such an inspection would either not occur or would be ineffective. This needs to be resolved. The effects depend on whether or not a second inspection in carries out, the sample size and method and the efficacy of detection. The 0.25% infestation level that would be detected with 95% confidence from two 600 fruit samples is above the 0.13% average infestation level found in the August 2005 data.

668. Australia's evaluation of importation step 8 seems to suggest there would only be minimum border procedures and that there would be no second inspection, the values of 0.9 being given to account for possible natural mortality.

Dr Deckers:

669. The number of viable insects after the whole process of fruit preparation, storage and exportation is the decisive parameter that should be taken into account. The number of cocoons on the fruits can be substantially different from the number of hatching insects.

Question 114
Please comment on the decision in Australia's IRA not to limit itself to the August 2005 data indicating the level of ALCM infestation of export quality New Zealand apples destined for the United States, on the grounds that such data did not reflect unrestricted risk but a level of importation where risk mitigation measures were already applied? (IRA, Part B, pp. 166, 172-174, 178-183, 190-192; para. 755 of Australia's FWS)

Dr Cross:

670. As stated above, Australia should have discarded its original figures based on a small number of old spot estimates in the literature and used the good quality August 2005 data. New Zealand have given assurances that the fruit on which the August 2005 data was based was not subject to any risk mitigation procedures or other procedures that would reduce the infestation rate and have stated that the efficacy of detection of inspections is close to 1.0.

Dr Deckers:

671. It is not clear which risk mitigation measures were undertaken by New Zealand that should have influenced the 2005 data. Anyway these data indicate an infection percentage that is about 10 times lower than the infection percentage proposed by the IRA.

Question 115
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 whether adult emergence of ALCM from diapause may take place in seasons other than spring (for example, if the cool chain is broken and day length and temperature replicate spring, such as in a controlled environment like a supermarket or packing house, or in specific areas of Australia). Would this be relevant to the establishment of ALCM, taking into account host specificity and specific environmental requirements for establishment? Is the consideration in Australia's IRA on this issue sufficiently supported by the available scientific evidence? (IRA, Part B, p. 161; para. 4.118 of New Zealand's FWS; and para. 766 of Australia's FWS)

Dr Cross:

672. Adult emergence of ALCM (which occurs after pupation and not immediately after diapause is completed by 3rd instar larvae in cocoons) may take place in seasons other than spring if suitable environmental conditions occur either outdoors in summer or autumn or at any time in a controlled environment in a supermarket or pack house. On average, this would decrease the risk of establishment because some adults would emerge in winter when they are unable to find a suitable host outdoors. Adults emerging from fruit sold in late autumn, winter or early spring (when conditions outdoors are not suitable for ALCM establishment) would pose no risk. If the fruit were discarded in the vicinity of an apple tree, some of the adults would have already emerged reducing the numbers that could emerge later when environment and host plant conditions were suitable for survival.

Dr Deckers:

673. It can be important to consider the conditions when the adults emerge after a period of diapause. To be successful the hatching process should be under conditions comparable to the situation of the cocoons in the soil after the dormancy period, where temperature and moisture will be important factors. When the hatching occurs under other circumstances like in a supermarket or in a packing house, the chances of normal development and survival could be different from the normal situation.

674. Also from the side of the host plant there should be a coincidence in the development stages necessary for the midges to have young leaves on the host plants at the moment of hatching. Both factors will strongly reduce the real infection risk.

Question 116
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 whether some ALCM larvae that have entered diapause may have progressed beyond the pre-pupal stage to the pupal stage and will be ready to emerge as adults as soon as, or shortly after, the appropriate environmental triggers are encountered by the pupa, or whether they will necessarily take the 13-18 days (as indicated by Barnes (1948)) to emerge once they have encountered appropriate environmental triggers? In other words, is it possible for diapause in ALCM to end due to climatic conditions and then for ALCM development to be completed in less time than 13-18 days? (IRA, Part B, pp. 171 and 180; para. 4.361 of New Zealand's FWS; and paras. 772-773; 780-784; 795-797 of Australia's FWS)

Dr Cross:

675. This has already been answered in detail under Question 94 (i), the conditions for adult emergence of ALCM. ALCM could emerge in less than 13-18 days because the cocoons may contain mature pupae close to emergence. No data has been presented to show that AA or CA storage will have killed such pupae.

Dr Deckers:

676. I think the situation is more complex than it is described here; the situation for development of 13 to 18 days can vary following the conditions of temperature and moisture. What is the length of the period of diapause and what are the determining factors to bring this period to an end ? Question is if the same factors will be available when the cocoons hatch after they have been in storage for some months and when the fruits are exported.

Question 117
Please comment on whether the consideration in Australia's IRA of the likelihood of spread of ALCM in Australia was objective and credible, relying on respected and qualified scientific sources with respect to the climatic conditions suitable for the spread of ALCM. Is the consideration in Australia's IRA on this issue sufficiently supported by the available scientific evidence? (IRA, Part B, pp. 177-180; paras. 4.364-4.365 of New Zealand's FWS; paras. 811-813 and 821 of Australia's FWS; and R 81 from New Zealand)

Dr Cross:

677. Australia's IRA on this point seems objective and credible but, as stated in the answer to question 94, a weakness in the IRA was that Australia failed to quantify (or at least delimit) the geographic range and range of conditions which are necessary for ALCM establishment and spread, both in terms of temperature and rainfall and their seasonal occurrence. The geographic and climatic limits were not established. The distribution of ALCM in the continent of Europe, where it has long been present and it's range and distribution have more or less reached equilibrium, used in conjunction with evidence from different regions of the American continents, could have been used to establish climatic conditions that are especially favourable to ALCM and geographic and climatic boundary conditions for its existence. There are extensive areas where apples are cultivated that are unsuitable for ALCM. It is considered to be absent from hot, dry areas of southern Europe and Israel for instance and in the USA it evidently does not occur in the southern apple growing regions of Carolina or Georgia though it has spread westward from New York State. In the northern hemisphere, ALCM does not appear to occur at latitudes less than approximately 38°. It also appears to be absent from areas with greater latitudes where summer rainfall is very low or absent. It is most troublesome in areas with high rainfall. A climatic analysis would also have given a better assessment of the likely impact of ALCM in different areas of Australia. Recent hot droughty climatic conditions in SE Australia have been unsuitable for ALCM, though the climate of Tasmania does appear favourable. However, the IRA did not assume that ALCM would spread to all areas of Australia where apples are grown commercially or domestically and the overall assessment is correct.

Dr Deckers:

678. The spread of the ALCM will be limited to the regions with cool temperate zone climatology and this aspect was maybe not sufficiently taken into account in Australia's IRA and this could have over estimated the likelihood of spread. Nevertheless there are regions in Australia with this type of cool temperate climatology where ALCM could spread easily.

Question 118
Please comment on the relative potential impact of factors such as the suitability of local climatic conditions and the volume of ALCM infesting apples imported from New Zealand for ALCM establishment in the territory of a Member such as Chinese Taipei. (Paras. 4.364-4.365 of New Zealand's FWS; para. 821 of Australia's FWS)

Dr Cross:

679. This question has already been partially answered in answers to Questions 107 and 117. The climatic limits under which ALCM can survive need to be quantified and the climatic conditions in other member states such as Chinese Taipei need to be evaluated in comparison. Chinese Taipei is at a latitude of 25°, well south of the known range of ALCM in the northern hemisphere. Australia's FWS para 821, states that Chinese Taipei has a subtropical, oceanic climate. There are no records of ALCM in tropical or sub-tropical areas. ALCM is unlikely to establish in Chinese Taipei because the climate is unsuitable.

Dr Deckers:

680. The suitability of the local climatic conditions is decisive for the potential introduction of the ALCM into a new area and this is more important than the volume of infested apples imported.

Question 119
In the context of the assessment of consequences of an incursion of ALCM in Australia, please comment on whether the reasoning and conclusions in Australia's IRA were objective and credible with respect to the potential for an increased use of insecticides, disruption of existing pest management programs, increases in control measures and increased costs to producers. Is the consideration in Australia's IRA on this issue sufficiently supported by the available evidence? (IRA; Part B, p. 185; para. 4.371 of New Zealand's FWS; and paras. 839-840 of Australia's FWS)

Dr Cross:

681. Australia's IRA part B page 185 does not indicate that ALCM is only likely to be a significant pest problem in nurseries and young trees in orchards that are establishing and was deficient in this respect as pointed out by NZ in para 4.371 of its FWS. However, Australia is right to point out in paras 840 of their FWS that invasive species do not necessarily behave in the same way when they are introduced to different parts of the world. If ALCM were to establish in Australia with out its parasitoid *Platygaster demades*, in regions with a suitable climate it could be more numerous and damaging than it currently is in similar regions in NZ. However, where invasive outbreaks of ALCM have occurred in other countries, the grower response has not been to treat established orchards with insecticides. In general, growers have learnt that controlling ALCM in established orchards with broad spectrum pesticides is counter productive because of destruction of natural enemies and is not necessary because it does not lead to increases in yield or quality.

682. The establishment of ALCM in Australia could lead to increased use of insecticides if suitable insecticides were available in Australia but this would probably be confined to nurseries and possibly young trees. This would lead to marginally increased costs to producers.

Dr Deckers:

683. Diazinon is also in Europe a standard compound in the control of the ALCM. Problem will be here that in the near future there will be a serious reduction in the number of insecticides available within the IPM production method for the control of diseases like ALCM.. In Belgium the Diazinon is not longer available for the control of ALCM on apple. The fact that ALCM would need an insecticide treatment does not mean that there will be a disruption of the existing pest management program.

Question 120
Please comment on whether the conclusion in Australia's IRA, that an inspection of a 600 fruit sample from each import lot would not achieve Australia's ALOP for ALCM, was objective and credible, in light of the IRA's view that the unrestricted risk for ALCM is "low". How does this alternative measure, proposed by New Zealand, compare to the relevant measures imposed by Australia, namely (i) inspection of a 3000 fruit sample from each lot with a find resulting in mandatory treatment or rejection for export or (ii) treatment of all lots with a suitable treatment to kill ALCM? Under what circumstances, if at all, could the alternative measure proposed by New Zealand achieve Australia's ALOP? (IRA, Part B, pp. 4-5, 165-166 and 187-192; paras. 4.138, 4.513-4.523 of New Zealand's FWS; paras. 734-735, 824, 954-957 1089-1105 of Australia's FWS; and R 140-141 by the Parties)

Dr Cross:

684. The unrestricted risk estimation presented in Table 49 of Australia's IRA part B p 187 needs to be recalculated for two different importation scenarios:

(a) Mature apple fruit free of trash, either packed or sorted and graded bulk from New Zealand

(b) Retail ready fruit which would not be handled at sensitive utility points

685. The August 2005 infestation rate data should be used and viability, parasitism and the time span of adult emergence relative to adult longevity need to be taken into account in the recalculation. The inclusion or exclusion of different utility points for the two importation scenarios is crucial. Consideration needs to be given to the numbers of fruit that are likely to be placed or discarded within the flight range of a susceptible host at the relevant utility points in formulating the risk estimates. It might be found that the unrestricted risk estimates for one or both of these scenarios then falls below Australia's ALOP.

686. If not, then the sample sizes required to meet Australia's ALOP should then be recalculated for fruit subject to fumigation and not subject to fumigation for each of the two importation scenarios. Note that 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.

687. Until this is done, then it is inappropriate to comment on the sample sizes and the need or otherwise for fumigation treatment required to meet Australia's ALOP.

688. The requirements for a 3000 fruit inspection or for fruit fumigation are clearly restrictive and alternative measures coupled with a 600 fruit inspection would be preferable provided they met Australia's ALOP.

Dr Deckers:

689. The imposed measure for a chemical control of the problem of ALCM in the New Zealand orchard should concentrate on the last generations of the ALCM. Together with the proposed inspection of a 600 fruit sample after harvest, this could help to achieve Australia's ALOP for ALCM.

Question 121
Please comment on whether the consideration in Australia's IRA of the risk associated with the practice of packing houses leaving orchard wholesaler waste uncovered and exposed to the elements on the premises or in landfills is objective and credible, taking into account the likelihood of this situation occurring in packing houses in Australia. (IRA, Part B, p. 170 (ALCM); paras. 4.130 and 4.419-4.421 of New Zealand's FWS; paras. 784-785 and 898-900 of Australia's FWS; and R 100 by Australia)

Dr Cross:

690. The quantities and way that waste fruit is handled at the 7 orchard wholesalers is of crucial importance. If large numbers (>>100) of fruit are held in proximity to or discarded at orchard wholesalers in the vicinity of susceptible apple trees then the risk of establishment is clearly high. The risk would be considerably reduced, perhaps eliminated, if the fruit were enclosed so the midge adults could not escape.

Dr Deckers:

691. When fruit waste should remain uncovered and exposed for a short time, the ALCM will not have got the opportunity to hatch from the cocoon because this step will need more time (days) after coming out of the storage room. A professional fruit packing station will not leave fruit waste uncovered for a long period.

Question 122
Does Australia's IRA provide an objective and coherent assessment of the likelihood and implications of New Zealand apples being repacked at rural packing houses in close proximity to orchards, when assessing the risks related to fire blight, European canker and ALCM? Was such assessment made with proper methodological rigour? (Para. 4.418 of New Zealand's FWS; and R 99 by Australia)

Dr Cross:

692. Australia's IRA did provide an objective and coherent assessment with respect to "apples free from trash either packed or sorted and graded bulk from New Zealand" but appears it did not consider the case of retail ready fruit. It took into account two scenarios of different amounts of fruit being handled by the orchard pack house, 70-100% versus 0.1-5%. This lead to very large (33 fold) differences in the estimates of the numbers of infested apples being handles at the orchard wholesaler utility points (Tables 42 and 43) which resulted in the estimates of the partial probabilities of entry, establishment and spread which are high for the orchard wholesalers (Tables 44 and 45). However, if fruit were supplied from New Zealand "retail ready" or "just in time", then it seems most unlikely that any fruit would be returned to the orchard wholesalers for repacking. The IRA needs to be recalculated with respect to this scenario.

Dr Deckers:

693. I don't see the necessity for repacking the fruit when New Zealand exports the apples retail-ready. The waste will thus be for the majority in the New Zealand packing house. Australia describes the risk in detail in the two different scenario's with only a few apples be repacked or with many apples repacked. But this risk described in the IRA doesn't look to correspond with the reality.

Dr Sgrillo:

694. The Australia's IRA considers two extremes scenarios: a) from 70% to 100% of apples being repacked at rural packing houses and b) from 0.1% to 5% of apples being repacked at rural packing houses.[82]

695. The scenarios have a large effect in the predicted number of infected fruit imported weekly, as is shown in IRA's Tables 42 and 43 (the data are in the first and second lines, respectively, of the two first rows of the tables).[83]

696. This data were used by experts, together with other pieces of information, to estimate the partial probability of entry, establishment and spread as stated by IRA:

> These estimates are based on expert opinion taking into account the sequence of events for successful transfer of the pest to a susceptible host, the estimated numbers of infested apples at each utility point, the availability and susceptibility of hosts at

[82] (*footnote original*) IRA p. 172.
[83] (*footnote original*) IRA pp. 173-174.

each utility point, information relevant to establishment and spread of the pest and all relevant information provided by stakeholders.[84]

697. However, IRA is not clear in explaining how the estimated numbers of infested apples were used to estimate the partial probability of entry, establishment and spread. There are no specific estimates for each of the two scenarios, as can be seen in IRA's Table 44 and 45[85]. It seems that the differences in the estimated numbers of infested apples had not generated any difference in the partial probability of entry.

Question 123
Please comment on whether, from a technical perspective and as described in Australia's IRA, the 17 specific measures that have been challenged by New Zealand can be distinguished as either measures active in risk reduction, or measures designed to implement or support active measures. (R 14-26 by the Parties)

Dr Deckers:

698. I don't think it make sense to split up the specific measures in measures active in risk reduction and measures designed to implement or support active measures. All measures aim to reduce the risk for the different problems concerned.

V. RISK ASSESSMENT

Question 124
Please comment on whether Australia's IRA takes into account internationally accepted standards or guidelines from the IPPC in applying a semi-quantitative methodology. (Paras. 4.161-4.167 of New Zealand's FWS; and paras. 288-294 of Australia's FWS)

Dr Latorre:

699. Australia's IRA has taken into account internationally accepted standards and guidelines from the IPPC in applying the semiquantitative methodology. However, the likelihood values and midpoint values used for the semiquantitative analysis (Table 12, AUS-2BA, p. 43) should be validated before acceptance.

Dr Schrader:

700. Pest risk can be carried out with quantitative or qualitative data, a combination of both is also possible. The approach depends on the situation, different approaches could be appropriate under different circumstances – this depends also on the quality and quantity of available data. Up to now, there are no standard definitions of quantitative or qualitative pest risk assessment provided by the IPPC. So far, these terms are used differently in different contexts by different Regional and National Plant Protection Organisations.

701. ISPM No. 11 states under 2.2.4 (Conclusion on the probability of introduction and spread): "The overall probability of introduction should be expressed in terms most suitable for the data, the methods used for analysis, and the intended audience. This may be quantitative or qualitative, since either output is in any case the result of a combination of both quantitative and qualitative information. The probability of introduction may be expressed as a comparison with that obtained from PRAs on other pests." Under 2.3 (Assessment of potential economic consequences), it is stated

[84] (*footnote original*) IRA p. 178.
[85] (*footnote original*) IRA p. 179.

that: "Requirements described in this step indicate what information relative to the pest and its potential host plants should be assembled, and suggest levels of economic analysis that may be carried out using that information in order to assess all the effects of the pest, i.e. the potential economic consequences. Wherever appropriate, quantitative data that will provide monetary values should be obtained. Qualitative data may also be used. Further, under 2.3.2.3 (Analytical techniques): "The use of analytical techniques is often limited by lack of data, by uncertainties in the data, and by the fact that for certain effects only qualitative information can be provided." and 2.3.2.4 (Non-commercial and environmental consequences): "If quantitative measurement of such consequences is not feasible, qualitative information about the consequences may be provided. An explanation of how this information has been incorporated into decisions should also be provided." Also 2.3.3 (Conclusion of the assessment of economic consequences): "Wherever appropriate, the output of the assessment of economic consequences described in this step should be in terms of a monetary value. The economic consequences can also be expressed qualitatively or using quantitative measures without monetary terms. Sources of information, assumptions and methods of analysis should be clearly specified." as well as 2.5 (Conclusion of the pest risk assessment stage): "[…] A quantitative or qualitative estimate of the probability of introduction of a pest or pests, and a corresponding quantitative or qualitative estimate of economic consequences (including environmental consequences), have been obtained and documented or an overall rating could have been assigned" refer to both qualitative and quantitative estimates. A pest risk assessment/analysis is in most cases based on qualitative approaches and expert judgment, but it is essential that this is explained and made transparent. The use of modelling is also possible. The important point is, that the model used is appropriate and applied in an adequate and correct way.

Dr Sgrillo:

702. The International Standards of Phytosanitary Measures (ISPM) of the IPPC do not make reference to "semi-quantitative" methodology.

703. However the semi-quantitative approach is described in the *IPPC pest risk analysis training course-Participant Manual* as follows: "A semi-quantitative pest risk assessment combines elements of both quantitative and qualitative assessments, adding precision using quantitative methods where these are applicable, and incorporating qualitative methods for those parts of the assessment where data is not available or the same degree of precision is not required."[86]

704. There are no international guidelines for development of semi-quantitative models in Plant Protection. A number of different methodologies could be applied, under the denomination of "semi-quantitative". The Australia's IRA is based in the development of a quantitative stochastic model that relies on repeated random sampling from statistical distributions (Monte Carlo method). The values of the parameters of the distributions were chosen from qualitative likelihoods. The numeric results of the simulations were then translated to qualitative terms.

705. In quantitative modeling the statistical distributions and the corresponding parameters are derived from sampling of the real world. The semi-quantitative methodology used by IRA could introduce bias in the model because the parameters and the shapes of the distributions are mostly based in guesses and not derived from sampling. Assigning numbers to subjective estimation does not result, necessarily, in a more objective assessment.

706. It is not possible to evaluate the potential bias that each step could introduce in the model because the IRA does not present the sensitivity analysis.

[86] (*footnote original*) Reference 02_FAO/IPPC- 2007. IPPC Pest risk analysis training course-Participant Manual

Question 125
In the light of internationally recognised and accepted practice and standards for risk assessment in horticultural products that are traded internationally, does a proper risk assessment need only evaluate measures that reduce or otherwise manage risk? Can or should risk assessments also evaluate or consider measures that do not, by themselves, have the objective of reducing risks? (Paras. 855-859 of Australia's FWS; para. 53 of the United States' Third Party submission; and R 23 by the Australia)

Dr Latorre:

707. To my understanding, risk assessment analysis should only consider measures that are directly related to reducing the risk of entrance, establishment and spread of a new pest or disease in a new geographical area. However, the method of pest risk assessment can vary according to the circumstances, with other measures used when appropriate.

Dr Schrader:

708. The SPS-Agreement defines risk assessment in Annex A as "the evaluation of the likelihood of entry, establishment or spread of a pest or disease within the territory of an importing Member according to the sanitary or phytosanitary measures which might be applied, and of the associated potential biological and economic consequences […]". Sanitary or phytosanitary measures are defined in Annex A as "Any measure applied: (a) to protect animal or plant life or health within the territory of the Member from risks arising from the entry, establishment or spread of pests, diseases, disease-carrying organisms or disease-causing organisms; […]Sanitary or phytosanitary measures include all relevant laws, decrees, regulations, requirements and procedures including, inter alia, end product criteria; processes and production methods; testing, inspection, certification and approval procedures; quarantine treatments including relevant requirements associated with the transport of animals or plants, or with the materials necessary for their survival during transport; provisions on relevant statistical methods, sampling procedures and methods of risk assessment; and packaging and labelling requirements directly related to food safety."

709. The IPPC (1997) and ISPM No. 5 separate management measures from risk assessment. According to the IPPC definitions, pest risk assessment is the "evaluation of the probability of the introduction and spread of a pest and the magnitude of the associated potential economic consequences (see Glossary Supplement No. 2) [FAO, 1995; revised ISPM No. 11, 2001; ISPM No. 2, 2007]", whereas pest risk management is the "evaluation and selection of options to reduce the risk of introduction and spread of a pest [FAO, 1995; revised ISPM No. 11, 2001]"

710. Both are subsumed under the definition of pest risk analysis, which is defined as "the process of evaluating biological or other scientific and economic evidence to determine whether an organism is a pest, whether it should be regulated, and the strength of any phytosanitary measures to be taken against it [FAO, 1995; revised IPPC, 1997; ISPM No. 2, 2007]".

711. In ISPM No. 11, stage 3, Risk Management, it is outlined that "the conclusions from pest risk assessment are used to decide whether risk management is required and the strength of measures to be used. Since zero-risk is not a reasonable option, the guiding principle for risk management should be to manage risk to achieve the required degree of safety that can be justified and is feasible within the limits of available options and resources. Pest risk management (in the analytical sense) is the process of identifying ways to react to a perceived risk, evaluating the efficacy of these actions, and identifying the most appropriate options."

712. ISPM No. 14 (systems approach see below, answer to question 140) requires the evaluation of the efficacy of measures, which includes both independent and dependent measures.

713. According to my understanding, all potential measures that are proposed for managing the risk, either principal or designed to implement the principal measures, should be evaluated in a proper pest risk analysis. This is also necessary for technical justification ("justified on the basis of conclusions reached by using an appropriate pest risk analysis or, where applicable, another comparable examination and evaluation of available scientific information", IPPC 1997).

Dr Sgrillo:

714. According to the IPPC it should be recognized first that only measures that are necessary to prevent the introduction and spread of quarantine pests are allowed to be required by the importing country, as the Principle "Necessity" of the IPPC states:

> "Contracting parties may apply phytosanitary measures only where such measures are necessary to prevent the introduction and/or spread of quarantine pests, or to limit the economic impact of regulated non-quarantine pests. In this regard, the IPPC provides that: *"Contracting parties shall not, under their phytosanitary legislation, take any of the measures specified in ... unless such measures are made necessary by phytosanitary considerations ...*" (Article VII.2a). Article VI.1b states that "*Contracting parties may require phytosanitary measures for quarantine pests and regulated non-quarantine pests, provided that such measures are …limited to what is necessary to protect plant health…*"."[87]

715. Secondly all the measures required shall be assessed and technically justified, as the Principle "Technical justification" of the IPPC, provides:

> "Contracting parties shall technically justify phytosanitary measures "...on the basis of conclusions reached by using an appropriate pest risk analysis or, where applicable, another comparable examination and evaluation of available scientific information." (Article II.1). In this regard, the IPPC provides that "Contracting parties shall not, under their phytosanitary legislation, take any of the measures specified in paragraph 1 of this Article (VII) unless such measures ... are technically justified." (Article VII.2a)."[88]

716. The IPPC recognizes also "Operational principles" that are related to the establishment, implementation and monitoring of phytosanitary measures, and to the administration of official phytosanitary systems. The recognized Operational Principles are: "pest risk analysis, pest listing, recognition of pest free areas and areas of low pest prevalence, official control for regulated pests, systems approach, surveillance, pest reporting, phytosanitary certification, phytosanitary integrity and security of consignments, prompt action, emergency measures, provision of a National Plant Protection Organization, dispute settlement, avoidance of undue delays, notification of non-compliance, information exchange and technical assistance."[89]

717. The conclusion is that only measures necessary to prevent the introduction/spread of quarantine pests may be established and that each of these measures should be assessed and justified.

[87] (*footnote original*) Reference 10: IPPC, ISPM No. 1: Phytosanitary principles for the protection of plants and the application of phytosanitary measures in international trade, 2006.
[88] Ibidem.
[89] Ibidem.

Question 126
Do internationally recognised and accepted practice and standards for risk assessment, such as the ISPMs, provide any guidance as to who may participate in a risk assessment panel? (Para. 4.468 of New Zealand's FWS; and R 89-91 by Australia)

Dr Latorre:

718. I am not aware of any document providing guidance as to who may participate in a risk assessment panel. It is possible that this should be in accordance with the Commission on Phytosanitary Measures (IPPC, Art. XI). In my opinion, specialists should be included in the panel to analyse the information and make final conclusions. However, this does not prevent requesting the opinions of other people, including farmers, horticulturists, mathematicians, biometricians or politicians, when appropriate.

Dr Schrader:

719. According to the International Plant Protection Convention, National Plant Protection Organisations are responsible for the conduct of pest risk analyses (Article IV 2 f). ISPM No. 2 refers to this article on page 7: "Article IV.2f states that the responsibilities of the National Plant Protection Organization (NPPO) include "*the conduct of pest risk analyses*". No further guidance is given here. ISPM 11 states under 2.3.2.3 (Analytical techniques) that there are analytical techniques which can be used in consultation with experts in economics to make a more detailed analysis of the potential economic effects of a quarantine pest. In addition, ISPM No. 11 states under 1.1.1: "A list of pests likely to be associated with the pathway (e.g. carried by the commodity) may be generated by any combination of official sources, databases, scientific and other literature, or expert consultation". Both standards refer to "expert judgment".

720. Common practice is that *ad hoc* experts are included in risk assessment panels which may not be employed by an NPPO. This is e.g. the case in the Expert Working Groups conducting PRAs that are organised and lead by the European Plant Protection Organisation (EPPO). The European Food Safety Authority (EFSA), which reviews and also conducts PRAs, has established a plant health panel that is doing this work, and most of the members are not employed by an NPPO, the same is true for *ad hoc* experts involved in specific cases. However, EFSA requests from any expert involved in the work a declaration of interest. If an expert has to declare an interest, he/she may be excluded from the work or parts of it.

Dr Sgrillo:

721. There is international no guidance on who may participate in a risk assessment panel.

Question 127
Please comment on whether it would be a legitimate methodological approach for a risk assessor to assess the unrestricted risk in the first instance, and then assess the extent to which potential risk management measures could mitigate that risk. Likewise, would it be a legitimate methodological approach for a risk assessor to only assess the unrestricted risk, without considering the extent to which potential risk management measures could mitigate that risk? (IRA, Part B, pp. 23, 40-41, 44; and paras. 824 and 902 of Australia's FWS)

Dr Latorre:

722. There is not a single accepted methodology to assess the risk of entrance, establishment and spread of a pest or disease. It is legitimate to assess firstly the unrestricted risk and then following the

same or similar methodology to assess measures to mitigate the risk. It would also be acceptable to assess only the unrestricted risk, if appropriate.

Dr Schrader:

723. Pest risk analysis, as is outlined in ISPM 2 and 11, comprises risk assessment and risk management. The definition of pest risk analysis as provided in ISPM No. 5 (Glossary of Phytosanitary Terms) is as follows: "The process of evaluating biological or other scientific and economic evidence to determine whether an organism is a pest, whether it should be regulated, and the strength of any phytosanitary measures to be taken against it [FAO, 1995; revised IPPC, 1997; ISPM No. 2, 2007]". To assess the unrestricted risk in the first instance, and then assess the extent to which potential risk management measures could mitigate that risk is therefore a legitimate methodological approach. It is also legitimate to only assess the unrestricted risk in accordance with the definition for pest risk assessment (see also answer to question 125), however this would not make any sense with regard to the purpose of a pest risk analysis/import risk analysis when an unacceptable risk is identified in this assessment. In case, the assessment reveals that the (unrestricted) risk with regard to introduction and spread of the pest is acceptable however, the consideration of potential risk management measures is not necessary.

Dr Sgrillo:

724. ISPM 11[90] describes the methodology that shall be used in the PRA process:

> "Stage 1 (initiation) - The aim of the initiation stage is to identify the pest(s) and pathways which are of quarantine concern and should be considered for risk analysis in relation to the identified PRA area.
>
> Stage 2 (Pest risk assessment) - The process for pest risk assessment can be broadly divided into three interrelated steps:
>
> (a) Pest categorization
>
> (b) Assessment of the probability of introduction and spread
>
> (c) Assessment of potential economic consequences (including environmental impacts).
>
> Stage 3: Pest Risk Management - The conclusions from pest risk assessment are used to decide whether risk management is required and the strength of measures to be used. Since zero-risk is not a reasonable option, the guiding principle for risk management should be to manage risk to achieve the required degree of safety that can be justified and is feasible within the limits of available options and resources. Pest risk management (in the analytical sense) is the process of identifying ways to react to a perceived risk, evaluating the efficacy of these actions, and identifying the most appropriate options. The uncertainty noted in the assessments of economic consequences and probability of introduction should also be considered and included in the selection of a pest management option".

725. Therefore it is a legitimate methodological approach for a risk assessor to assess the unrestricted risk in the first instance, and then assess the extent to which potential risk management measures could mitigate that risk.

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[90] (*footnote original*) Exhibit AUS-6: IPPC, ISPM No. 11: Pest risk analysis for quarantine pests including analysis of environmental risks and living modified organisms, 2004.

726. It would not be a legitimate methodological approach to only assess the unrestricted risk, without considering the extent to which potential risk management measures could mitigate that risk (to assess the necessary strength of measures).

Question 128
What factors might be relevant in deciding whether to adopt a qualitative, a semi-quantitative, or a quantitative methodology for a particular risk assessment?

Dr Sgrillo:

727. See also question 124.

728. When reliable specific numeric data are available and more precision is required then quantitative model shall be used. The main advantage of this approach is that it generates objective and reproducible conclusions to satisfy the technical justification for phytosanitary measures.

729. When reliable numeric data are not available then qualitative methods will be used.

730. According to the OIE the semi-quantitative method would not be recommended because:

> "However, a number of significant problems may arise from adopting a semi-quantitative approach in an import risk analysis. It is some times employed as a means of combining various qualitative estimates, by assigning numbers to them, to produce a summary measure or to priorize risks. The numbers may be in a form of probabilities ranges or scores, which may be weighted before combined by addition, multiplication, etc. The numbers, ranges, weights and methods of combination chosen are usually quite arbitrary and need careful justification to ensure transparency. It should be recognised that numbers assigned to categories cannot be manipulated mathematically and statistically. It is impossible to assign precise numbers unless a quantitative assessment has already been carried out. Semi-quantitative assessments often give a misleading impression of objectivity and precision and may not adequately reflect relativities, which can lead to inconsistent outcomes. Assigning numbers to subjective estimates does not result in a more objective assessment, particularly when the numbers chosen and their method of combination are arbitrary. Semi-quantitative methods will rarely offer any advantage over a well researched, transparent, peer reviewed qualitative assessment".[91]

Does Australia's IRA provide an objective and coherent explanation for the fact that it uses a semi-quantitative methodology in this case for only the second time for a plant product?

Dr Sgrillo:

731. There is no such an explanation for the use of a semi-quantitative approach. Australia has noted, however, that SPS Agreement does not prescribe a particular risk assessment methodology.[92]

What is the relevance, if any, of the Senate Inquiry referred to by New Zealand in its opening statement? (Para. 110 of Australia's FWS; para. 73 of New Zealand's opening statement; and R 92-94 by the Parties)

[91] (*footnote original*) Exhibit NZ-47: OIE (2004a), Handbook on Import Risk Analysis for Animals and Animal Products Volume 1: Introduction and Qualitative Risk Assessment, World Organization for Animal Health. pp. 27-28.

[92] (*footnote original*) R 93 by Australia.

Dr Latorre (Response to whole question):

732. Qualitative, semi-quantitative, or quantitative methodologies can be appropriate. The decision will depend on the pests or diseases under study and on the quantitative data available in the literature. However, to my understanding, Australia's IRA does not provide a technical explanation for the use of a semi-quantitative approach, except to say that this procedure apparently facilitates the interpretation by stakeholders and reinforces objectivity and transparency (AUS-2BA p. 11). The decision to use a semi-quantitative methodology to assess the risks must be taken by experts. Therefore any intervention of the Senate in this regards, if ever happens, would be improper and should be clarified.

Dr Schrader (Response to whole question):

733. In accordance with ISPM 11, PRAs can be qualitative or quantitative (or include both methodologies). This is mostly depending on available data and information. In most cases, available information is in such a way, that a (fully) quantitative risk assessment is not possible (see also answer to question 124).

Question 129
Does Australia's IRA provide an objective and coherent explanation for the fact that it applies a semi-quantitative methodology to establish the likelihood of the entry, establishment and spread of certain pests, including the three at issue in this dispute, while applying a qualitative methodology to some other pests? (Para. 110 of Australia's FWS; para. 56 of the United States' Third Party submission; para. 73 of New Zealand's opening statement; and R 92-94 by the Parties)

Dr Latorre:

734. To my understanding, Australia's IRA does not provide an explanation for the fact that it uses a semi-quantitative approach to establish the likelihood of the entry, establishment and spread of certain pests, while it uses a qualitative methodology in other cases.

Dr Sgrillo:

735. Australia informs that the choice of methodology "was based on the available information about the various pests, the nature of the pests themselves, the scientific literature including other relevant risk analyses, the need for transparency and consistency, and the complexity of the pathway".[93]

736. However, in most cases the data and specific information needed were not available. So the IRA team has chosen the distributions and their parameters through guesses. The guesses represent hypotheses about the system, and these hypotheses were not validated because the necessary actual data were not available.

737. Australia has not explained, also, how the nature of the pests could favor the choice of the semi-quantitative method.

Question 130
Does Australia's IRA provide an objective and coherent assessment of the likelihood and implications of New Zealand apples being repacked at rural packing houses in close proximity to orchards, when

[93] (*footnote original*) R 94 by Australia.

assessing the risks related to fire blight, European canker and ALCM? Was such assessment made with proper methodological rigour? (Para. 4.418 of New Zealand's FWS; and R 99 by Australia)

Dr Latorre:

738. To my understanding, this was an estimation based on general knowledge of the biology of *N. Galligena*, considering two scenarios with low (0.1-5%) and high (70-100%) proportions, respectively, of the fruit being repacked in orchard packing houses. Probability values were estimated. However, the rationale involved in these estimations is not clear. For instance, probability from 0.7 to 1.0 was assigned in the highest scenario. Probability of one would mean that if a single apple fruit had visible sporulating lesions of *N. Galligena* and was repacked (or discharged?) at the packing houses, *N. Galligena* would invariably spread to the nearest apple trees. If so, this is hard to believe. If apples were repacked at rural packing houses in close proximity to the orchards, I would expect that chances of dispersal and establishment of *N. Galligena* to increase slightly. Knowing the biology of *N. Galligena*, there are many factors (primarily environmental), that would have to concur in order to successfully disseminate *N. Galligena* under these circumstances. Mitigation measurements can be proposed at this step.

Dr Sgrillo:

739. For the fire blight and European canker see Response 46. For ALCM see Response 122.

Question 131
Please comment on the applicability of OIE guidelines when referring to plant related risks. (R 102-103 by the Parties)

Dr Latorre:

740. In biology, there are general principles that can be applied universally, independent of the nature of the organisms. Therefore, the use of OIE guidelines (Handbook on Import Risk Analysis for Animals and Animal Products) seems appropriate in the context to which New Zealand applied it.

Dr Schrader:

741. For plant health issues, clearly the International Plant Protection Organisation and its standards are the competent and responsible framework. OIE guidelines deal with animal diseases, where situations may be quite different. IPPC standards are concretely adopted for plant health issues. This is also specified in the SPS agreement in Annex A, which states under (c) that with regard to plant health, "the international standards, guidelines and recommendations developed under the auspices of the Secretariat of the International Plant Protection Convention in cooperation with regional organizations operating within the framework of the International Plant Protection Convention" are relevant.

Dr Sgrillo:

742. The *Codex Alimentarius Commission*, the *International Office of Epizootics (OIE)* and the *International Plant Protection Convention (IPPC)* are recognised by the SPS agreement as the international organization responsible for developing standards, guidelines and recommendations for food safety, animal health and zoonoses and for plant health, respectively.[94] These organizations share the same scientific principles and concepts. The general concepts, procedures and methods

[94] (*footnote original*) SPS Agreement, Annex A, pg 77.

based in scientific principles, including risk analysis, developed by these Organizations have mutual applicability.

Question 132
Does Australia's IRA provide an objective and coherent explanation regarding the period of time for completion of the IRA?

Dr Sgrillo:

743. Australia's IRA provides background information on the development of the IRA including on the previous risk analysis starting in 1996.[95] A timeline for the current risk analysis is also presented In Annex 1 of the Australia's First Written Submission.[96]

744. The development of the current IRA began in February 1999 and the final IRA was released in November 2006, after almost eight years. Three public consultation periods were open (11/2000, 02/2004 and 12/2005).[97]

745. However the previous risk analysis, that began in 1996 and was released in 1998, required only two years to be developed. To develop the previous risk analysis IRA states:

> "AQIS reviewed the available scientific literature, sought opinion from stakeholders, considered all the material provided during the consultation process and followed ISPM No. 2: Part 1 – Import regulations: Guidelines for pest risk analysis (FAO, 1996a)".[98]

746. Consequently much of the data and information necessary for the development of the current IRA had already been revised and was available when the development of the current IRA began.

747. There are no explanation regarding the difference on the necessary time to develop the previous IRA (two years) and the time spent in the development of the current IRA (eight years).

Please also provide information on comparable plant protection IRAs that were concluded in a significantly shorter or longer period of time than this IRA, and the factors that might have determined the time necessary for completing such IRAs.

Dr Sgrillo:

748. Australia provide the information that the previous IRA was developed in two years.[99]

Please comment on the experience of the United States referenced in its Third Party submission. (Para. 92 of the United States' Third Party submission; and R 142 and 145 by Australia)

Dr Sgrillo:

749. Australia inform that during this period (1999-2008) it has completed five import risk analyses (namely, table grapes, bulk maize, sweet corn, finfish and pig meat) according to the priorities identified by the United States and it has also completed eight import policy reviews that

[95] (*footnote original*) IRA pp. 7-8.
[96] (*footnote original*) Australia FWS, Annex1, pp. 332-336.
[97] Ibidem.
[98] (*footnote original*) IRA p. 8.
[99] (*footnote original*) IRA p. 7-8.

improved existing access for United States' products.[100] These facts makes the delay (for the development of the IRA for USA's apples) appears less severe.

Dr Latorre (Response to whole question):

750. The period of time allowed for completion of the import risk analyses (IRA) was too long (1999 to 2007). An acceptable explanation regarding the time taken for completion of this analysis was not presented. I have no information regarding the time taken for other IRA and I am not aware of the factors that have determined the time necessary for completion of other IRAs.

751. The assertions made by the United States, although external to the present dispute, are another example of the unjustified slowness observed by the Australian IRA team in undertaking and completing the IRA. This is suggesting that the overall procedures should be reviewed in the future.

Dr Schrader (Response to whole question):

752. I can not adequately answer this question. However, I would like to refer to the IPPC and to ISPMs Nos. 1, 2, and 24 that deal with the avoidance of undue delay:

> Article VII.2h IPPC:
>
> Contracting parties shall, as conditions change, and as new facts become available, ensure that phytosanitary measures are promptly modified or removed if found to be unnecessary.
>
> ISPM 1: 2.14 (Avoidance of undue delays): When a contracting party requests another contracting party to establish, modify or remove phytosanitary measures, when conditions have changed or new facts have become available, this request should be considered without undue delay. Associated procedures, which include, but are not limited to, pest risk analysis, recognition of pest free areas or recognition of equivalence, should also be performed promptly.
>
> ISPM No. 2: 3.6 (Avoidance of undue delay): Where other contracting parties are directly affected, the NPPO should, on request, supply information about the completion of individual analyses, and if possible the anticipated time frame, taking into account avoidance of undue delay (section 2.14 of ISPM No. 1: Phytosanitary principles for the protection of plants and the application of phytosanitary measures in international trade, 2006).
>
> ISPM No. 24: 2.7 (Timeliness): Contracting parties should endeavour to determine the equivalence of phytosanitary measures and to resolve any differences without undue delays. And Annex 1, step 7: If equivalence is recognized by the importing contracting party, implementation should be achieved by the prompt amendment of the import regulations and any associated procedures of the importing contracting party. The amendments should be communicated in accordance with Article VII.2b of the IPPC (1997).

Question 133
Does Australia's IRA provide objective and coherent explanations for the probability intervals assigned under the IRA to the qualitative descriptions of events, which are based on ranges of pre-

[100] (*footnote original*) R 142 by Australia.

determined probability values set out in Table 12 at p. 43 of Part B of the IRA, when applied on a per apple basis?

Dr Sgrillo:

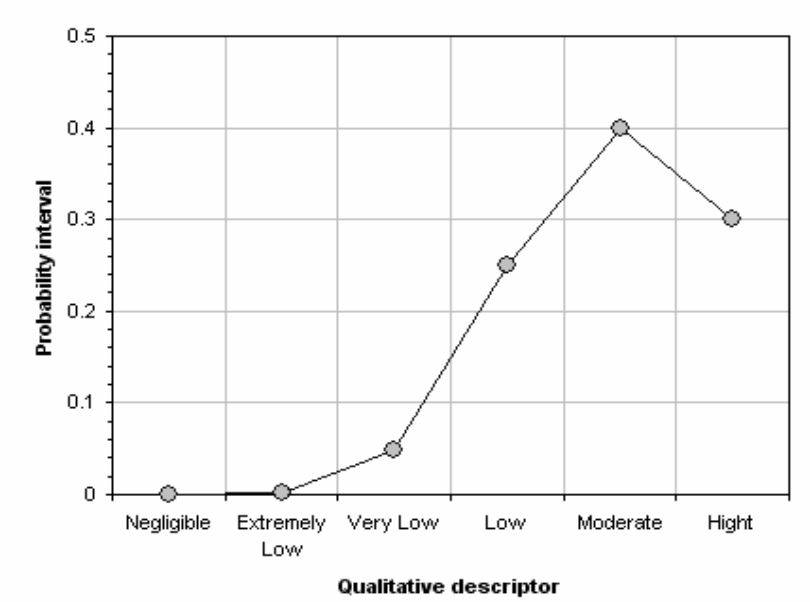
753. The methodology is described in the Australia's *Guidelines for Import Risk Analyses.*[101]

> "... to divide explicitly the 0–1 interval into a small number of mutually exclusive categories, or 'probability intervals'. These categories may subsequently be correlated with an equal number of descriptors..."

754. The descriptors are those presented in table 15 of the *Guidelines for Import Risk Analyses* (high, moderate, low, very low, extremely low and negligible).[102]

755. The probability interval seems to have been arbitrarily chosen to represent the qualitative descriptors. There are no perceived criteria for assigning probabilities intervals to the qualitative scale. No mathematical relationship between the categories was found, as presented in Figure 2.

> Figure 2. Relationship between the probability intervals (ranges) and the qualitative descriptors.



Are those probability intervals based on a legitimate methodological approach and supported by sufficient scientific evidence?

Dr Sgrillo:

756. The IRA uses six categories, but could have been five, seven or eight categories. The values used for the ranges of probabilities seem to have been arbitrarily settled.

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[101] (*footnote original*) Reference 07: Biosecurity Australia. Guidelines for import risk analysis. Draft September 2001. p. 86.

[102] (*footnote original*) Reference 07: Biosecurity Australia. Guidelines for import risk analysis. Draft September 2001. p. 84.

(iii) Are they in line with internationally accepted risk assessment standards and methodology?

Dr Sgrillo:

757. The semi-quantitative methodology is not mentioned in the international standards of the IPPC and is not supported by the OIE: "Semi-quantitative methods will rarely offer any advantage over a well researched, transparent, peer reviewed qualitative assessment"[103] (see Question 128).

Does the IRA provide objective and coherent explanations for assigning a range of 0 to 1 x 10E-6, regarding the probability of a "negligible" event when applied on a per apple basis?

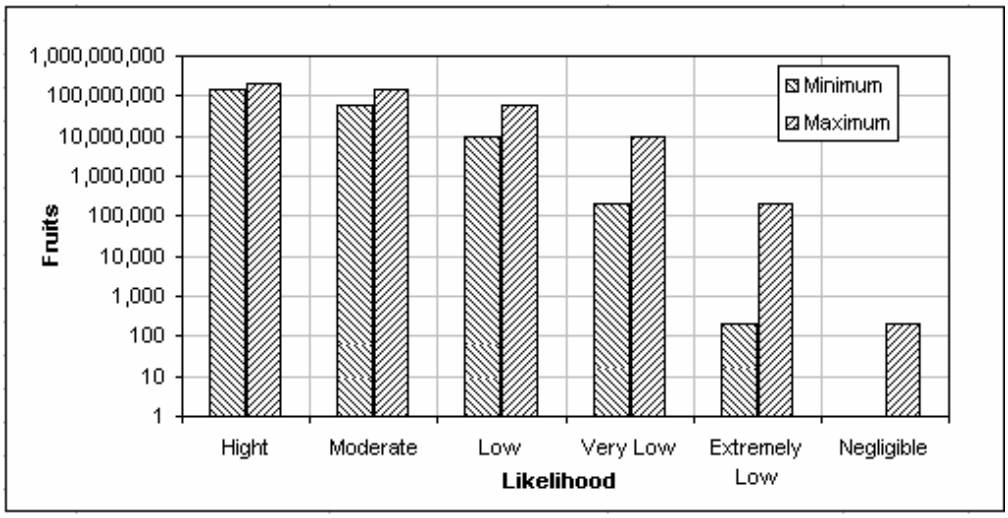
Dr Sgrillo:

758. IRA defines the "negligible" category as "*The event would almost certainly not occur*".[104] The range of probabilities assigned to this event is 0 to 1E-6. This choice is arbitrary because other ranges, as 0 to 1E-9 or 2E-6, could also be used, after adjustment of the remaining ranges, without violating any scientific principle.

759. The likelihood of 1E-6 corresponds to an occurrence in each 1.000.000 units, what seems to be negligible. However in stochastic pest risk models (see question 137), the number of expected occurrences is found multiplying the probability of occurrence by the number of units in the population.

760. Figure 3 shows, in logarithmic scale, the number of fruits that corresponds to each likelihood category, considering a population of 200.000.000 fruits.

Figure 3. Number of fruits in each likelihood category, from a total of 200.000.000 fruits.



[103] (*footnote original*) Exhibit NZ-47: OIE (2004a), Handbook on Import Risk Analysis for Animals and Animal Products Volume 1: Introduction and Qualitative Risk Assessment, World Organization for Animal Health. p. 28.

[104] (*footnote original*) Reference 07: Biosecurity Australia. Guidelines for import risk analysis. Draft September 2001. p. 86.

761. Note that the "very low" category corresponds to "*The event would be very unlikely to occur*"; the "extremely low" category is described as "*The event would be extremely unlikely to occur*" and the "negligible" category is "*The event would almost certainly not occur*".[105]

762. There is a difference between *likelihood per unit* and *number of expected occurrences* in the population. 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.

763. It can be noted that, in the lower part of the categories, some distortion become evident. The "very low" category contains up to 10,000,000 fruits, the "extremely low" category contains up to 200,000 fruit and the "negligible" category 200 fruits.

764. In the phytosanitary context "negligible" should represent, in populational terms, one event in several years and not 200 events in one year.

Is such an approach based on a legitimate methodological approach and sufficiently supported by scientific evidence? (IRA, Part B, pp. 42-45; paras. 4.174-4.186 of New Zealand's FWS; paras. 269-271 and 295 of Australia's FWS; para. 73 of New Zealand's opening statement; and R 107 by New Zealand)

Dr Sgrillo:

765. This approach seems to be based in an arbitrary choice and not in scientific principles.

Dr Latorre (Response to whole question):

766. Australia's IRA provides little insight as to how the probability values were assigned to each of the six qualitative descriptors (high, moderate, low, very low, extremely low and negligible). To my understanding, these probability intervals were arbitrarily assigned based on the general knowledge of the pests and diseases, considering the opinions of experts and stakeholders, to ensure that these probability ranges contained the actual values. However, and at least in connection to European canker, these probability ranges are difficult to believe. One of the main weaknesses is the range used to numerically explain the negligible descriptor. The term "negligible" denotes something of so little consequence as to warrant no attention. In other words, the probability of a negligible biological event would be almost certainly zero but different from zero. Therefore, by no means can a negligible event range from 0 to a maximum of $1x10^{-6}$ with a midpoint of $5x10^{-7}$. In doing so, the likelihood of a particular biological event is overestimated. As stated before, $5x10^{-7}$ is a relatively high probability value, even considering the lowest possible total volume of apples (50,000,000, AUS-2 p.19) that can ultimately be imported from New Zealand. To my knowledge, little or no experimental information has been published with regard to the likelihood of most of the biological events concerning the probability of entrance, establishment and spread of European canker. Therefore, unless the IRA team has additional information (e.g., experimental data) not presented in this IRA, assigning these probability values can lead to weak conclusions. I would strongly suggest reviewing the probability values given in Table 12 (AUS-2 p. 43), accepting that the maximum probability to be assigned to a negligible event should be such that one can be almost certain that this event will not occur in a given population, and that the minimum value should be different from zero. Then probability values for other descriptors can be assigned, considering that if an event has a probability of one, there is certainty that the event will occur.

[105] (*footnote original*) Reference 07: Biosecurity Australia. Guidelines for import risk analysis. Draft September 2001. p. 86.

Question 134
Does Australia's IRA provide objective and coherent explanations for defining "negligible" as an interval between 0 and 10^{-6}?

Dr Sgrillo:

767. Australia doe not explain why the maximum value for the "negligible" range is 1E-6. There is no scientific base to support the exclusive choice of 1E-6 because other ranges, as 0 to 1E-9 or to 2E-6, could also be used, after adjustment of the remaining ranges, without violating any scientific principle.

Is that definition based on a legitimate methodological approach? Please comment on whether the intervals assigned by Australia's IRA correspond to an event that would almost certainly not occur. (Para. 4.160 of New Zealand's FWS; para. 274 of Australia's FWS; para. 73 of New Zealand's opening statement; and R 96-98 by the Parties)

Dr Sgrillo:

768. It is recognized that the numeric probabilities representing the qualitative descriptors in the IRA are to be interpreted in a *per unit* basis. However they should reflect the category concepts also in populational terms but this is not occurring in the present case.

769. Considering the importation of 200.000.000 fruit per year the category negligible could represents 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 Latorre (Response to whole question):

770. Please refer to the answer to Question 133.

IV. Question 135

When performing a biological risk assessment, how does the uniform distribution compare to the triangular distribution and to the beta-PERT distribution, in particular when modelling events that have a low or even "negligible" likelihood of occurring?

Dr Sgrillo:

771. Firko and Podleckis (2000)[106] describe the characteristics of the uniform distribution in pest risk assessment:

> "Uniform distributions are the simplest PDF (Probability Distribution Function); only a minimum and maximum value is needed to specify the distribution. Every value between the minimum and the maximum has an equal probability of being selected by the sampling algorithm. Uniform distribution may be appropriate when there is little justification for assuming the some values are more likely than others or when data do not suggest a central tendency. Uniform distribution used for probability values that range over an order of magnitude should be used with caution. If the analysts were thinking on a log scale, results could be overly conservative. For

[106] (*footnote original*) Reference 04: Firko, M.J. and Podleckis, E.V. Likelihood of Introducing Nonindigenous Organisms with Agricultural Commodities: Probabilistic Estimation. In: Ferson S, Burgman M., editors. Quantitative methods for conservation biology. Springer; New York: 2000. pp. 77–95.

> example, consider a uniform distribution covering two orders of magnitude (minimum=0.0002, maximum=0.02). Values between the minimum and the geometric mean (0.002) will be chosen only about 9% of the time, and values between the geometric mean and the maximum will be chosen about 91% of the time".

772. For the same authors[107] the limitations of the triangular distribution refers to the minimum and maximum values:

> Triangular distributions are specified by three values: minimum, most likely, and maximum. The relative frequency of the various values in the distribution is indicated by the shape of the curve, which is determined by the relative positions of the three parameters. Values at or near the most likely value are selected for calculations more often than values at or near the minimum and maximum. Triangular distributions can be used when it is fair to assume that some values are more likely than other, but have the disadvantage of not selecting values below the minimum and above the maximum. Choice of the minimum and maximum values can be considered more important than choice of the most likely value because the minimum and maximum imply a high degree of assurance about the limit of the underlying distribution. We use triangular distribution occasionally but avoid them because biological systems are seldom well represented by distributions with strict limits. The arbitrary shape of the triangular distribution and its strict limits are indications that the precise nature of the distribution is not known.

773. Also, by using a strict triangular shape about the mode, the triangular distribution may place too much emphasis on the most likely value, at the expense of the values to either side.

774. The Pert distribution also uses the most likely value, but it is designed to generate a distribution that more closely resembles realistic probability distribution as normal or lognormal distributions.

775. Realistic stochastic models, however, are only developed when available numeric data are sufficient to estimate, directly from real populations, the shape of the distributions and the values of the respective parameters.

(ii) Can you explain the advantages and disadvantages of one with respect to the other?

Dr Sgrillo:

776. The uniform distribution requires only the maximum and minimum values but it generates less realistic samples.

777. The triangular distribution is based on knowledge of the minimum and maximum and an inspired guess as to what the modal value might be[108], however it emphasizes on the modal value, at the expense of the values to either side.

778. From the three options the Pert distribution is expected to be the more realistic because it reduces the emphasis on the modal value.

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[107] Ibidem.

[108] (*footnote original*) http://rule-of-thumb.net/2008/09/06/random-numbers-out-of-triangular-distribution/. Retrieved 23/February/2009.

Please comment on the implications of a methodological approach for a risk assessment that defines the minimum and maximum of a distribution, but not the central tendency of the distribution. Could this type of risk assessment result in a distorted view of the actual likelihood of an event? (IRA, Part B, p. 42; para. 4.187 of, and pp. 242 and 247 of Annex 4 to, New Zealand's FWS; para. 312 of Australia's FWS; para. 73 of New Zealand's opening statement; and R 109 by New Zealand)

Dr Sgrillo:

779. The results of the simulations from a uniform distribution will converge to its arithmetic mean ([minimum+maximum]/2). However, if the actual probability distribution of the process is skewed then the risk assessment will result in a distorted view of the reality.

Dr Latorre (Response to whole question):

780. I am not in a position to answer this question.

Dr Schrader (Response to whole question):

781. A Uniform distribution represents a distribution for which each value in the continuous range of values between minimum and maximum limits occurs with the same probability. It is the simplest and least realistic method of the three methods mentioned here and is useful in situations, where a minimum and maximum value are available, but no sufficient information to determine the most likely value. This method implies a high degree of uncertainty.

782. The triangular distribution can be applied if a "most likely" estimate in addition to the minimum and maximum estimates is available. This probability distribution favors the most likely value and should provide a better estimate of the probabilities of reaching other values. The distribution does not need to be symmetrical about the mean; by this it can model a variety of different circumstances. The disadvantage is that it may emphasize too much the most likely value, at the expense of the values to the two other sides towards the minimum or maximum. This distribution is easy to calculate and generate, but still limited with regard to modelling real-world estimates.

783. Though the beta-PERT distribution also uses and emphasizes the most likely value, it generates a smooth distribution curve that resembles more closely a realistic probability distribution and places progressively more emphasis on values close to the most likely value. The advantage of the PERT distribution is that its shape is similar to the normal curve, without knowing the precise parameters of the related normal curve. This relates to the assumption that realistic events or situations are normally distributed.

Question 136
Please explain what weight a uniform distribution range, as used by Australia's IRA, attributes to the maximum value, as compared to the minimum value. Is there any indication that the maximum value assigned to the uniform distribution range in the model used by the IRA gives appropriate weight to the maximum probability of a "negligible" event occurring?

Dr Sgrillo:

784. See Table 1, below.

Does using a risk analysis software package which performs simulations that randomly select numbers from within the uniform distribution, effectively average the higher and lower ends of the probability range?

Dr Sgrillo:

785. Yes, even general software, as MS Excel, presents an acceptable performance. For example 15 simulations of 1.000 random uniform values between 0 and 1E-6 generates an average of 4.9961E-7, ranging from 4.84E-7 to 5.17E-7 what corresponds to an error of ± 3.5 % when compared with the expected average 5.00E-7.

Please comment on whether the IRA's use of the "negligible" range gives more weight to the upper bound of the uniform distribution range than the minimum bound.

Dr Sgrillo:

786. The data of Table 1 represents the percentage of sampling, in each range, from a uniform distribution with minimum value = 0 and maximum value = 1E-6. The table was prepared dividing the amplitude of each interval by the total amplitude (1E-6).

Table 1. Percentage of sampling from a uniform distribution with minimum value = 0 and maximum value = 1E-6

Range	Sampling (%)
0<>1E-12	0.0001
1E-12<->1E-11	0.0009
1E-11<->1E-10	0.009
1E-10<->1E-09	0.09
1E-09<->1E-08	0.9
1E-08<->1E-07	9
1E-07<->1E-06	90
Total	**100**

787. Table 1 shows that 90% of the random numbers generated in a simulation will fall in the range 1E-7 to 1E-6 and only 0.0001% of the random numbers will fall in the range 0 to 1E-12.

If so, does the IRA give undue weight to the maximum probability of a "negligible" event occurring by assigning the upper bound of their uniform distribution to be 1 x 10E-6 bearing in mind the per apple methodology and the estimated annual volume of apples imported?

Dr Sgrillo:

788. In a logarithmic scale, where the weight is equally distributed among the various magnitude orders, the probability of sampling a number very close to zero is the same of sampling a number close to the upper bond of the distribution.

789. However, as shown in table 1, in a decimal scale, there is much more chance of sampling a number close to the upper bond than close to zero.

790. It is recognized that 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 category concepts also in populational terms.

791. Considering 150.000.000 fruits imported per year the category negligible could represents 150 fruits per year. However, an event that "would almost certainly not occur" should be expected to occur only once in each several years.

Does each step in the model have to be estimated separately and then the average value taken forward as the input into the next step? Or at each step of the model, are the full distribution of output values effectively taken forward to the next step – not just the average value? (IRA, Part B, pp. 17-19 and 42; paras. 4.190-4.192 and 4.194-4.195 of New Zealand's FWS; and paras. 314-316 of Australia's FWS)

Dr Sgrillo:

792. The model runs according the following steps:

(a) Generate a set of random inputs, one for each probability function;

(b) Evaluate the model;

(c) Store the result.

793. The steps 1, 2 and 3 are repeated 1000-2000 times[109] and then the output distribution is computed from the stored values.

794. The fifth, 50th (or median) and 95th percentiles of the output distribution are then compared with the probability intervals in IRA's Table 12.[110]

Dr Latorre (Response to whole question):

795. I am not in a position to answer this question.

Question 137
Please comment on the relevance of the conflicting assumptions made by the Parties as to the varieties of apples and the magnitude of traded volumes that New Zealand could successfully export into Australia. What role does volume of trade play in the risk assessment used by Australia? If volume of trade was overestimated, what significance would this have on the overall probability of entry? (IRA, Part B, pp. 19 and 24; paras. 4.194-4.203 of New Zealand's FWS; paras. 113-121 and 325-343 of Australia's FWS; and R 110 by New Zealand)

Dr Latorre:

796. First, the risk of entrance would be related to the size of the population. In this case, it will depend on the total volume of mature apples annually imported by Australia. In general, as the volume increases, so does the probability that a given biological event may occur, increasing the chances that *N. Galligena* will gain entrance into Australia. Therefore, it is very important for an accurate IRA to define objectively the eventual volume of mature apple that Australia would be importing from New Zealand annually.

Dr Sgrillo:

797. The general pest risk model can be expressed as follows[111]:

[109] (*footnote original*) Reference 07: Biosecurity Australia. Guidelines for import risk analysis. Draft September 2001. p. 87

[110] (*footnote original*) IRA p. 43.

[111] (*footnote original*) Reference 05: Bigsby, H. and J. Crequer. 1996. Conceptual model for the management of pest risk. Statistics in Ecology and Environmental Monitoring, Dunedin, June 1996, pp. 7-16.

$$Risk = Prob\_Intro \times Conseq \quad ...1$$

798. Where *Risk* is the probability of introduction (*Prob_ Introd*) multiplied by the potential economic impact (*Conseq*).

799. The probability of introduction can be estimated by[112]:

$$Prob\_Intro = 1 - (1 - Prev \times Prob\_Estab)^{Entered} \quad ...2$$

800. Where *Prob_Intro* is the probability of introduction, *Prev* is the prevalence (proportion of units in the consignment that is infected/infested), *Prob_Estab* is the probability of establishment and *Entered* is the number of infected/infested unities that has entered into the importer country.

801. The number of entered units can be estimate by:

$$Entered = Volum\_Imported \times Prob\_Ingress \quad ...3$$

802. Where *Volum_Imported* is the volume (units) imported and *Prob_Ingress* is the probability of ingress into the importer country.

803. Making the necessary substitutions the general equation for pest risk can be derived as:

$$Risk = \left(1 - (1 - Prev \times Prob\_Estab)^{Volum\_Imported \times Prob\_Ingress}\right) \times Conseq \quad ...4$$

804. This is the demonstration that the risk is directly proportional to the volume imported.

805. Figure 4 below presents a chart generated with equation 4, applying the following theoretical values: consequences = 1; prevalence = 0.001 (0.1%); probability of establishment = 0.005 and Probability of ingress = 0.001.

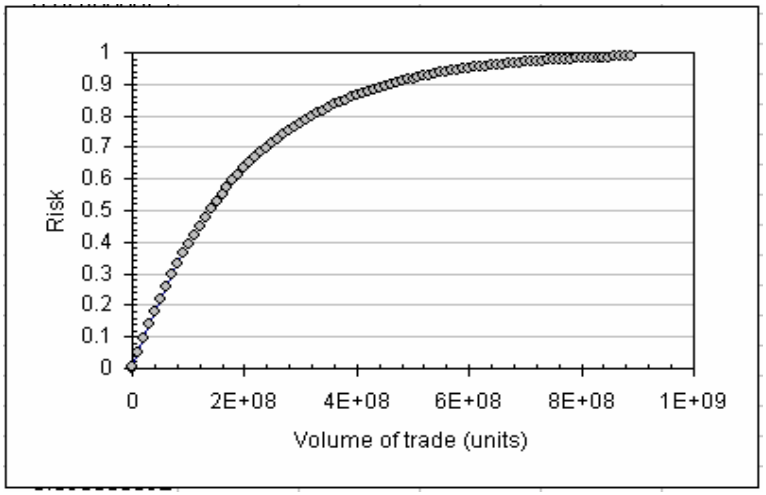

Figure 4. Effect of the volume of trade on the phytosanitary risk

[112] (*footnote original*) Reference 08: Baker, R.T., J.M. Cowley and D.S. Harte. 1993. Pest risk assessment. Lynfield Plant Protection Centre Publications, New Zealand, N°. 1, 12 pp.

806. Figure 4 shows that the final risk is a continuous function of the volume of trade. In many cases the probability of establishment is a function of the size of the population that has entered. Therefore the introduction process become non-linear and more complicated model are necessary. However always the risk will be proportional to the volume of trade.

807. Limiting the volume to be imported can be used as valid phytosanitary measure to reduce the risk to an acceptable level.

Question 138
In defining risk, please comment on whether is it appropriate to suggest that where Australia's IRA assigns "negligible" likelihoods to steps in a given pathway, such events should be treated as breaking the chain of causation for disease transmission, and that therefore, the assessment of risk should cease? Would it be a legitimate methodological approach for risk assessors to consider disease transmission pathways with steps that approach a probability of zero? Please comment on whether it is a legitimate methodological approach to continue a risk analysis if the likelihood of a step in a continuum of events is considered negligible or the step is considered almost certain not to occur? (Para. 4.160 of New Zealand's FWS; para. 274 of Australia's FWS; para. 59 of the United States' Third Party submission; para. 73 of New Zealand's opening statement; and R 96 by New Zealand)

Dr Latorre:

808. There are multiple importation steps (eight steps) to explain the possible flow of *N. Galligena* during the process necessary for importing mature apple fruits from New Zealand. Australian's IRA assigned a probability value to each step. However, some of these steps (e.g., Steps 3, 5 and 7) are indeed mere possibilities (hypothesis rather than true facts) that need to be confirmed. In such cases, a probability equal to zero should be assigned or even better, disregard the steps considered almost certain not to occur.

Dr Schrader:

809. If one risk element is rated "negligible", it has to be put into question, whether it makes sense to proceed further with the risk assessment. As an example, the Canadian Risk Assessment Scheme (Plant Health Risk Assessment, Commodity Risk Assessment, Canadian Food Inspection Authority, Plant Health Risk Assessment Unit, Science Advice Division) uses a risk estimation matrix, where ratings for likelihood of introduction are combined with ratings for consequences of introduction. The scheme uses three different scores for ratings: negligible, low, medium and high. "Negligible" in combination with any other rating (including high) results in "negligible". In comparison to this, the Australian IRA guidelines assign "negligible" to a high likelihood of entry, establishment and spread combined with a negligible impact, but assigns "very low" (which is still meeting Australia's ALOP) to a negligible likelihood of entry, establishment and spread combined with an extreme impact.

Dr Sgrillo:

810. It depends on what is the meaning of "almost certain not to occur".

811. If "almost certain not to occur" refers to the probability of occurrences in a per fruit bases and is described by the probability range 0 to 1E-6 then the risk assessment should proceed because the consequences have to be considered and the combination of a negligible probability of introduction with huge consequences could generate an unacceptable risk.

812. If "almost certain not to occur" refers to the likelihood of occurrences in the population as one occurrence in each several years, for example, then the probability range could be many times lower.

In this case the path could be assessed to be removed from the model to increase the clarity and simplicity; and the causal chain could be broken.

813. If "almost certain not to occur" mean that the possibility to occur is only a theoretical supposition and there are no records that the event has ever occurred then the path can be removed from the model and the causal chain would be broken.

Question 139
Please comment on whether, from a technical perspective and as described in Australia's IRA, the 17 specific measures that have been challenged by New Zealand can be distinguished as either principal or ancillary? (R 14-26 by the Parties)

Dr Latorre:

814. As stated in the answer to Question 93, phytosanitary measures which act directly to lower the risk of entrance, establishment and spread of *N. Galligena* should be considered as principal measures, while those measures implemented to support any active measure (principal measures) should be regarded as ancillary measures. The latter measures make sense only if principal measures exist.

Dr Schrader:

815. The IPPC Standards do not use the terms "principal" and "ancillary". However, it can be interpreted that Australia attempts a systems approach as described in ISPM No. 14. In this standard, independent and dependent measures are differentiated. A systems approach needs at least two independent measures. See also answer to question 140.

Dr Sgrillo:

816. The distinction between "principal" and "ancillary" measure is not recognized by IPPC and, therefore, there is no international guidance to distinguish the measures in plant protection context.

817. However the measures could be technically distinguished supposing that the "principal" measures are the ones having the direct purpose to prevent the introduction and/or spread of quarantine pests and that the "ancillary" measures support, verify and operationalise the principal measures.

Question 140
Please comment on the extent to which IPPC standards and other relevant documentation recognize any distinction between principal and ancillary measures, that is, between measures active in risk reduction and those which are designed to implement or support such measures. (R 24 by Australia)

Dr Latorre:

818. To my understanding, principal and ancillary are terms not used officially to distinguish between main and auxiliary (secondary) phytosanitary measures. However, the meaning of these two terms is understandable.

Dr Schrader:

819. As outlined in question 139, the 17 specific measures having been challenged by New Zealand, can be interpreted to be an attempt of a systems approach according to ISPM No. 14: "The use of integrated measures in a systems approach for pest risk management". According to this standard, systems approaches integrate measures for pest risk management in a defined manner and "could provide an alternative to single measures to meet the appropriate level of phytosanitary protection of an importing country." A systems approach requires integrating different measures, with a cumulative effect." It includes at least two measures that are independent of each other, and may include any number of measures that are dependent on each other.

820. The standard further outlines, that the importing country is making the decision regarding the acceptability of a systems approach, "subject to consideration of technical justification, minimal impact, transparency, non-discrimination, equivalence, and operational feasibility. A systems approach is usually designed as an option that is equivalent to but less restrictive than other measures."

821. Especially where the alternative is prohibition, it is "important to consider systems approaches among risk management options because the integration of measures may be less trade restrictive than other risk management options." An advantage of the systems approach is the ability to address variability and uncertainty by modifying the number and strength of measures to meet the appropriate level of phytosanitary protection and confidence.

822. A systems approach may include:

- measures applied in the place of production, during the post harvest period, at the packinghouse, or during shipment and distribution of the commodity
- cultural practices
- field treatment,
- post harvest disinfestation,
- inspection and other procedures
- risk management measures designed to prevent contamination or re-infestation
- pest surveillance, trapping and sampling
- measures that do not kill pests or reduce their prevalence but reduce their potential for entry or establishment (safeguards)

823. According to ISPM No 14, "systems approaches may be considered when one or more of the following circumstances apply:

- a particular measure is:
  - not adequate to meet the appropriate level of phytosanitary protection of the importing country
  - not available (or likely to become unavailable)
  - detrimental (to commodity, human health, environment)
  - not cost effective
  - overly trade restrictive
  - not feasible".

824. An important requirement of the systems approach is that "importing countries, in consultation with the exporting country where appropriate should select least trade restrictive measures where there are options."

Dr Sgrillo:

825. The terms "principal measures" and "ancillary measures" are not defined in the IPPC.

826. There are methods and operations designed to implement and support phytosanitary measures, as defined in the ISPM 5 (Glossary of phytosanitary terms-2008).[113]

> "Phytosanitary action: An official operation, such as inspection, testing, surveillance or treatment, undertaken to implement phytosanitary measures;"
>
> "Phytosanitary procedure**:** Any official method for implementing phytosanitary measures including theperformance of inspections, tests, surveillance or treatments in connection with regulated pests;"
>
> "Phytosanitary measure**:** Any legislation, regulation or official procedure having the purpose to prevent the introduction and/or spread of quarantine pests, or to limit the economic impact of regulated non-quarantine pests;"

Question 141
Please comment on whether, in general, "ancillary" and "principal" measures can share a common scientific basis. Does the scientific justification of an ancillary measure necessarily depend on whether its related principal measure is found to be scientifically justified? (R 25 by the Parties)

Dr Deckers:

827. No, the ancillary measures don't share always a common scientific base with the principal measures. So is the requirement of the inspection of the new planted trees on European canker and the necessity to treat them for NG totally different from the NG contamination of apples on adult trees in an orchard. The same is true for the pruning of fire blight infections: technically this should be done to reduce the impact of the disease, but of course this pruning could hide the presence EA infection in an orchard.

Dr Latorre:

828. Yes, ancillary measures and principal measures can share a common scientific basis, but the justification of the ancillary measures depends on the existence of scientifically justified principal measures.

Dr Schrader:

829. Article 2 (2) of the SPS-Agreement states that: "Members shall ensure that any sanitary or phytosanitary measure is applied only to the extent necessary to protect human, animal or plant life or health, is based on scientific principles and is not maintained without sufficient scientific evidence, except as provided for in paragraph 7 of Article 5.

830. Article 3 (3) of the SPS-Agreement states that: "Members may introduce or maintain sanitary or phytosanitary measures which result in a higher level of sanitary or phytosanitary protection than would be achieved by measures based on the relevant international standards, guidelines or recommendations, if there is a scientific justification, […]"

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[113] (*footnote original*) References 03: International Plant Protection Convention, International Standard for Phytosanitary Measures No. 11: Glossary of Phytosanitary Terms, 2008.

831. According to the IPPC, phytosanitary measures have to be technically justified ("Contracting parties shall not, under their phytosanitary legislation, take any of the measures specified in paragraph 1 of this Article unless such measures are made necessary by phytosanitary considerations and are technically justified." Article VII 2a, IPPC 1997). The definition given in the IPPC for "technically justified" is as follows: "justified on the basis of conclusions reached by using an appropriate pest risk analysis or, where applicable, another comparable examination and evaluation of available scientific information." The IPPC does not use the term "scientifically justified".

832. There is no direct indication (neither in the IPPC nor in the SPS Agreement), that the scientific justification of a dependent (ancillary) measure depends on the scientific justification of an independent measure.

Dr Sgrillo:

833. Some times the "principal" and "ancillary" measures can share the same technical justification. Measures that confirm or inspect other measures could share the same scientific justification.

834. However this is not always true. It does not necessarily follow that where a "principal" measure is found to be technically justified related measures are also automatically consistent with those justifications.

Question 142
Please comment on whether the alternative measure proposed by New Zealand relating to inspection by AQIS officials, verification of standard commercial practice and provision of packing house details imposed by Australia on the importation of apples would achieve Australia's ALOP.

Dr Sgrillo:

835. It is not possible to evaluate if the measures required by Australia and the alternative measures proposed by New Zealand have the same effect because Australia does not describes the expected effect of the required measures in achieving Australia's ALOP. It would be necessary to know how much of the estimated risk would be reduced by each of the measures required by Australia.

How does this measure compare to the relevant requirements identified by Australia's IRA in terms of risk mitigation? Under what circumstances, if at all, could this alternative measure proposed by New Zealand achieve Australia's ALOP? (IRA, Part B, pp. 4-5 and 314-315; paras. 4.524-4.539 of New Zealand's FWS; para. 1106-1113 of Australia's FWS; and para. 73 of New Zealand's opening statement)

Dr Sgrillo:

836. See to the first part of this response.

Dr Deckers (Response to whole question):

837. It seems to be logic that the existing quality assurance procedures in New Zealand apple production would be audited by AQIS officials during inspection and that not all the work should be done in double. Orchard inspections for fire blight and for NG will not be included by the standard procedures of apple production and will be a supplementary task. The very intensive control row per row, proposed by AQIS for European canker for each orchard will not be evident to realise at orchard level. Why not concentrating the observations on the most susceptible apple cultivars? The same

consideration can be made for fire blight inspections at orchard level: why not concentrate on the most susceptible varieties? This will increase the chances to find infections in the orchards.

Dr Latorre (Response to whole question):

838. It is reasonable to audit rather than inspect, considering that both Australia and New Zealand have a highly professional and very reliable Quarantine Service. Moreover, both countries have previously agreed to establish a system of auditing (e.g., stone fruits and tomatoes) rather than *in situ* inspection by officials from the Australian Quarantine and Inspection Service (AQIS). This auditing system is technically feasible, economically less restrictive, and should satisfy the appropriate level of protection (ALOP) established by Australia. Certainly, it can facilitate trade activities between both countries.

839. I see no reason why auditing would interfere with mitigation measures. Mitigation measures should be applied in accordance with a final protocol accepted by both countries, while auditing should verify only the correct application of the approved mitigation measures. On the other hand, if *in situ* inspection is performed, there are no substantiated reasons to allow thinking that this procedure *per se* would lower the risk of entrance, establishment and spread of *N. Galligena* in Australia.