Attachment 1: Excerpts from "Australia – Measures Affecting the Importation of Apples from New Zealand (WT/DS367): Third Party Submission of the United States of America"

2. Mature, Symptomless Apples are Not a Pathway for Fire Blight Disease

29. Mature, symptomless apples do not transmit fire blight because they are not a pathway for the disease, and Australia has provided no evidence that proves the contrary. As the United States explained in *Japan – Apples*, the scientific evidence indicates that: (1) *Erwinia amylovora* are not associated internally with mature, symptomless apple fruit; (2) *Erwinia amylovora* are rarely associated externally with mature, symptomless apple fruit, even when harvested from blighted trees and orchards; (3) even if a mature, symptomless apple were externally contaminated with *Erwinia amylovora*, such bacteria are unlikely to survive normal commercial handling, storage, and transport of fruit; and (4) even if the imported commodity were externally contaminated with *Erwinia amylovora*, there is no dispersal mechanism or vector to allow movement of such bacteria from the fruit to a suitable host.[49] Imported apples are not a means of transmission of fire blight bacteria because the chain of transmission – from association of bacteria with fruit to bacterial survival of handling, storage, and transport to vectoring of bacteria to a suitable host – is never completed.[50] Accordingly, the United States considers that Australia lacks a scientific basis to restrict imports of mature, symptomless apple fruit because they are not a pathway for the transmission of the disease.

30. The scientific evidence indicates that mature symptomless apples do not harbor fire blight bacteria internally and that external bacteria on mature, symptomless apples are rarely found. In a 1989 study, Roberts *et al.* found no internal or external bacteria either in or on the surface of 1,555 mature, symptomless apples harvested from blighted orchards in the State of Washington.[51] The Roberts (2002) study cited by New Zealand was a major investigation that sampled 30,900 apple fruit and also found no internal disease symptoms.[52] As part of that study, nine hundred fruit were sampled at harvest from trees that actually had fire blight disease, but no *Erwinia amylovora* were found when scientists from the Japanese and U.S. governments tested them simultaneously. Moreover, the study evaluated an additional 30,000 apples harvested at various distances from these infected trees for the incidence of fire blight disease development during commercial storage, but not a single apple developed the disease.

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[49] *Japan – Apples (Panel)*, para. 4.82.

[50] *Japan – Apples (Panel)*, para. 4.83.

[51] R.G. Roberts *et al.*, *Evaluation of mature apple fruit from Washington State for the presence of Erwinia amylovora*, Plant Disease 73: 917-921 (1989) (Exhibit NZ-97).

[52] NZ FWS, para. 4.11 (citing R.G. Roberts, *Evaluation of buffer zone size on the incidence of* Erwinia amylovora *in mature apple fruit and associated phytosanitary risk*, Acta Horticulturae 590: 47-53 (2002) (Exhibit NZ-20)).

31. Even in the rare event that mature, symptomless apples were externally contaminated with *Erwinia amylovora*, the bacteria would be unlikely to survive normal commercial handling, storage, and transport conditions. This is evidenced by the Hale and Taylor (1999) study cited by New Zealand, which examined the survival of *Erwinia amylovora* on apple fruit subject to normal commercial cooling and storing by surface-inoculating fruit with varying numbers of bacteria and measuring surviving bacteria after storage.[53] The study found that under both "commercial conditions" and "laboratory conditions," of 570 inoculated fruit, bacteria were eliminated on all but two fruit after storage for 25 days at cool temperatures and 14 days at room temperature. Bacteria were only isolated from some of the fruit that had been inoculated with extremely large numbers of bacteria, levels far higher than those that have been found on harvested mature, symptomless fruit.[54]

32. The scientific evidence further demonstrates that there is no documented vector or dispersal mechanism to transfer external fire blight bacteria from mature, symptomless apple fruit to a susceptible host. As the Roberts *et al.* 1998 literature review explained, "[t]here are no specific pathways recorded that document movement of *E. amylovora* fruit, either imported or domestic in origin, to susceptible host tissues in an orchard or nursery."[55] This is true despite studies that attempted to vector the bacteria to susceptible hosts. For instance, New Zealand points to a study by Hale *et al.* (1996). In that study, heavily inoculated apple fruit were suspended in the canopy of apple trees "as close as possible to blossom clusters containing open flowers," but there "was no spread of *E. amylovora*" to "any of the immature or mature fruit [in such trees] sampled," and "[n]o symptoms were seen in any blossom clusters" in the immediate vicinity of the inoculated fruit.[56] In a 2003 study, Taylor and Hale placed 1,800 apple fruit that had been contaminated with a marked strain of fire blight bacteria into an orchard. Even under conditions conducive for fire blight development, the discard of contaminated fruit in an orchard led neither to lateral spread of the bacterium to new host material nor to the development of fire blight disease in surrounding trees that could be attributed to the marked strain.[57] Taken together, this scientific evidence indicates that mature, symptomless apples are not a pathway for fire blight disease.

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[53] NZ FWS, para. 4.18 (citing C.N. Hale & R.K. Taylor, *Effect of cool storage on survival of Erwinia amylovora in apple calyxes*, Acta Horticulturae 489: 139-43, (1999) (Exhibit NZ-24)).

[54] Hale, C.N. and R.K. Taylor, *Effect of cool storage on survival of Erwinia amylovora in apple calyxes*, Acta Horticulturae 489: 139-43, 141 (1999) (Exhibit NZ-24).

[55] R.G. Roberts *et al.*, *The potential for spread of Erwinia amylovora and fire blight*, Crop Protection 17: 19-28, 23 (1998) (Exhibit NZ-22).

[56] C.N. Hale *et al.*, *Ecology and epidemiology of fire blight in New Zealand*, Acta Horticulturae 411: 79-85, 83 (1996) (Exhibit NZ-27).

[57] Taylor, R.K., Hale, C.N, Gunson, F.A., and Marshall, J.W., *Survival of the fire blight pathogen, Erwinia amylovora, in calyxes of apple fruit discarded in an orchard*, Crop Protection 22 (4): 603-608 (2003) (Exhibit US-1).

4. Australia's Measures for Apples from New Zealand

36. The United States considers particularly problematic some of the measures imposed by Australia that are the same or similar to those that the Dispute Settlement Body ("DSB") in *Japan – Apples (Article 21.5)* found were being maintained without sufficient scientific evidence. For instance, Australia requires apples to be sourced from areas free of fire blight symptoms, orchard inspections, and the suspension of an orchard/block if visual symptoms of fire blight are detected.[68] But the *Japan – Apples (Article 21.5)* panel found that requirements that an "orchard be free of apple trees or other plant infected with fire blight, that the orchard...be inspected once per year at the early fruitlet stage, and that detection of a blighted tree in this area by inspection will disqualify the orchard as a whole cannot be considered to be supported by sufficient scientific evidence."[69]

37. Australia further requires disinfection of apples at the packing house and cleaning and disinfecting of packing house equipment before each Australian packing run.[70] These requirements, however, are contrary to the conclusions of the *Japan – Apples (Article 21.5)* panel that "surface disinfection is not justified by scientific evidence" and that "the scientific evidence does not justify chlorine disinfection of packing facilities in order to prevent contamination of mature, symptomless apples by *E. amylovora*."[71] Australia also requires that packing houses registered for export source apple fruit only from registered orchards, which essentially imposes a separation requirement on apples exported to Australia.[72] But in *Japan –Apples (Article 21.5),* the panel concluded that "separation of fruit destined for Japan is not supported by sufficient scientific evidence."[73] In light of the findings of the *Japan – Apples (Article 21.5)* with respect to the aforementioned measures, the United States is of the view that the similar measures imposed by Australia are also maintained without sufficient scientific evidence, in violation of Article 2.2 of the SPS Agreement.

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[68] Final Import Risk Analysis Report for Apples from New Zealand ("IRA"), Part B, Biosecurity Australia (November 2006) pp. 106, 316, 318 (Exhibit NZ-1).

[69] *Japan – Apples (Article 21.5)*, para. 8.89.

[70] IRA, p. 318 (Exhibit NZ-1).

[71] *Japan – Apples (Article 21.5)*, paras. 8.97 and 8.102.

[72] IRA, p. 317 (Exhibit NZ-1).

[73] *Japan – Apples (Article 21.5)*, para 8.107.

C. The Scientific Evidence on European Canker

38. New Zealand and Australia have set forth competing interpretations of the scientific evidence regarding whether mature, symptomless apples are a pathway for transmitting European canker. The United States does not address all of the scientific evidence in this debate, but instead offers its views below on three key factors necessary for the infection of apple fruit with European canker, in part based on its own experience. These three factors are: 1) conducive climatic conditions; 2) the presence of a susceptible host; and 3) a sufficient concentration of inoculum. Favorable occurrence of all three of these factors is necessary for infection of apple fruit to occur. In light of these three factors, and the U.S. knowledge of the disease, the United States does not consider that Australia has adduced sufficient scientific evidence to establish that apples will be latently infected with European canker and can transfer the disease to susceptible hosts.

39. Preliminary, the United States notes that it is important to distinguish between the infection of trees and the infection of fruit with European canker. Although trees may be infected with European canker, this does not necessarily mean that fruit will likewise become infected. For instance, during a 1956 outbreak of European canker in Sonoma County, California, wood canker was the only phase of the disease that was of concern, and no infection of fruit occurred during the outbreak.[74]

40. Conducive climatic conditions is the first factor that is needed for the infection of apple fruit with European canker. European canker has not been reported as present in the major apple producing regions of central Washington State. The United States believes that the absence of European Canker in these areas is because the climate is not suitable to the development of the disease. A range of factors is necessary for the climatic conditions to be conducive to the infection of apple fruit, including favorable temperatures and the timing, duration, and quantities of rainfall. During a 1965 outbreak of European canker in Sonoma County, California in which fruit were infected, rainfall above 100 centimeters per year, foggy weather, and moderate temperatures seemed to be the unifying factors that resulted in the appearance of the causal organism in the orchards. This outbreak was also the result of favorable epidemiological and biological conditions, such as leaf fall at the appropriate time and conidial production.[75]

41. In terms of suitable climatic conditions, a 1975 study by Dubin and English found that several consecutive days of wetness, without a dry period, are necessary to achieve a high level of European canker infection. Conidia – the asexual fungal spores of *Nectria galligena* – are dispersed by water in liquid form and easily dry out, even at high levels of relative humidity. Dubin and English (1975) found that over 90 percent of conidia germinated in water in liquid form, but the ability of conidia to germinate dropped significantly in lower humidity. For instance, spore germination was reduced

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[74] Nichols, C.W. and Wilson, E.E., *An outbreak of European canker in California*, Plant Disease Reporter 40: 952-953 (1956) (Exhibit US-2).

[75] Dubin, H.J. and English, H., *Epidemiology of European Apple Canker in California*, Phytopathology: 65: 542-550 (1975) (Exhibit US-3).

by half when conidia were subjected to high relative humidity of 100 percent, but with no free water, and temperatures of 19 degrees Celsius for 12 hours.[76] This study indicates that inoculum potential will be lower in periods without rain and when relative humidity falls below saturation.

42. The second factor that is necessary for the infection of apple fruit is the presence of a susceptible host. Although the infection of apple fruit with European canker in the United States is rare, the presence of a susceptible host has been studied in other countries, particularly in relation to the timing of fruit infection. Swinburne (1971) found that fruit in storage were more likely to develop rots if they had been infected on the tree late in the summer.[77] Fruit infected early in the season contained a natural resistance to European canker in the form of benzoic acid, which is toxic to the pathogen.[78]

43. The third factor necessary for infection of apple fruit is a high concentration of spores to serve as an inoculum. Dubin and English (1974) found that five conidia per leaf scar wound were not sufficient to cause infection, 50 conidia per leaf scar wound caused only 20 percent of the leaf scar wounds to be infected, and 500 conidia resulted in infection of 80 percent of the leaf scar wounds.[79] Furthermore, the susceptibility of leaf scar wounds to infection by *Nectria galligena* declines with time. Another study found that only 6 percent of the leaf scar wounds were infected after 28 days, as compared with a 20-percent rate of infection for fresh scar wounds.[80]

44. As for whether European canker infection could be transmitted to a host orchard, apple fruit has never been reported to be an important source of inoculum for the spread of European canker. Individual apple fruits that have been discarded on the ground will most likely either decompose or be consumed by animals before any latent infection that might exist would have a chance to cause decay, and the fungus can sporulate. In the unlikely event of an apple fruit producing spores, these spores will be unlikely to cause an infection of European canker in trees because lengthy wet periods, as well as high levels of inoculum, are needed.

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[76] Dubin, H.J. and English, H., *Effects of Temperature, Relative Humidity, and Dessication on Germination of Nectria Galligena Conidia*, Mycologia: 67: 83-88 (1975) (Exhibit NZ-12).

[77] Swinburne, T.R., *The Seasonal Release of Spores of Nectria Galligena from Apple Cankers in Northern Ireland*, Annuals of Applied Biology. 69: 97-104 (1971) (Exhibit Aus-76).

[78] Swinburne, T.R., *European canker of Apple (Nectria galligena)*, Review of Plant Pathology. 54: 787-799 (1975) (Exhibit NZ-9).

[79] Dubin, H. J. and English, H., *Factors affecting apple scar infection by Nectria galligena conidia*, Phytopathology 64: 1201-1203 (1974) (Exhibit Aus-67).

[80] Wilson, E. E., *Development of European canker in a California apple district*, Plant Disease Reporter. 50:182-186 (1966) (Exhibit NZ-64).

45. Furthermore, in the unlikely event that a sporulating apple is discarded on the ground, it would be a poor source of inoculum for trees in an apple orchard because conidia are dependent on splashing rain drops for dissemination, and the concentration of spores a few meters from the sporulating fruit will likely be well below the threshold required for infection. And spores that are dispersed by air will be subject to even greater dilution than spores dispersed by rain. Australia also posits that birds and insects may be a possible means for European canker to be transmitted from a sporulating apple on the orchard floor to a host tree.[81] But there is no scientific evidence that supports this proposition.

46. In closing, the United States notes that Australia's risk assessment acknowledges that fruit are unlikely to spread European canker. The risk assessment states that "[n]o studies exist in the literature to demonstrate long-distance disease spread from fruit infections...."[82] Later, the risk assessment recognizes that, "[t]here is no evidence in the literature that indicates that long distance spread of the disease is due to movement of fruit." Rather, the risk assessment explains that, "[l]ong-distance movement of European canker is primarily the result of movement of infected nursery stock."[83]

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[81] Aus. FWS, para. 615.

[82] IRA, p. 142 (Exhibit NZ-1).

[83] IRA, p. 142 (Exhibit NZ-1).