An updated pest risk assessment for spread of *Erwinia amylovora* and fire blight via commercial apple fruit

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Abstract

The phytosanitary risk associated with the movement of export-quality apple fruit to countries where fire blight does not occur is reassessed based upon additional data available since 1998 and clarification or correction of previously misinterpreted data present in the literature. The low epiphytic fitness of *Erwinia amylovora* (Ea) on apple fruit, the documented low incidence of viable Ea populations on mature apple fruit and the lack of a documented pathway by which susceptible host material could become infected from fruit-borne inoculum remain unchanged, and support the view that movement of Ea via commercial apple fruit is highly unlikely. With this new information, we updated a previously published model to re-estimate the likelihood of fire blight outbreaks in new areas because of commercial fruit shipment. This likelihood decreased in every scenario, and ranged from one outbreak in 5217 years to one in 753,144 years. By using the corrected and newly published data and by making assumptions based upon documented pathogen biology, the model gives more robust statistical support to the opinion that the risk of importing Ea on commercial apple fruit and the concomitant risk of establishing new outbreaks of fire blight is so small as to be insignificant.

Keywords: Trade restriction; Quarantine; Phytosanitary; Long-range spread

1. Introduction

Long-range dissemination of the fire blight pathogen, *Erwinia amylovora* (Burr.) Winslow et al., is an aspect of fire blight disease epidemiology that has received less attention from an experimental viewpoint than other, more easily studied aspects. Far from a purely academic concern, countries where fire blight disease has not been reported or thought to be endemic (e.g., Japan, Australia) have historically taken an aggressive regulatory posture against the importation of rosaceous plant host materials, including fruit, from countries where fire blight occurs. Whether or not it is appropriate to include commercially mature apple fruit amongst the prohibited host materials has been the subject of rigorous, sometimes rancorous, debate during the past two decades. Efforts to identify a scientific basis for such regulations date back to McLarty (1923), who sought to evaluate the likelihood that mature apples contribute to the spread of fire blight disease in response to Australia’s (then) recently enacted legislation prohibiting the importation of apples, pears and fruit from other hosts susceptible to fire blight disease.

More recently, Roberts et al. (1998) reviewed the extant pertinent scientific literature relative to the presence of the phytopathogenic bacterium *E. amylovora* (Ea) on mature, commercial apple fruit to develop a robust, quantitative estimate of the phytosanitary risk associated with movement of commercial apple fruit from countries with fire blight to those without. The risk of transmitting the bacterium and subsequent development of fire blight disease to areas where it is not believed to occur was estimated under three different scenarios (S1, S2, S3; Roberts et al., 1998), representing differing levels of fire blight present in the orchard and the presence or absence of various mitigative measures currently required by certain

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importing countries. These estimates were developed using a multiplicative model that assigned discrete probabilities to each of a sequence of events (nodes) that are required for the disease to be spread via commercial apple fruit. In all scenarios, the risk was found to be so small as to be “insignificant”.

For some nodes in the model there was scientific data to indicate they do not occur (e.g., parts of P1, P5). Reflecting the conservative approach taken with the model, positive values were assigned to these nodes (e.g., the node value for P5), even though there was no scientific evidence to support a positive value for them. This was in no way an inference or acknowledgement by the authors that a positive risk is associated with these nodes. Subsequent to the publication of Roberts et al. (1998), Taylor et al. (2002) demonstrated that the required event at P5 (transfer of *E. amylovora* from a contaminated apple onto a susceptible host and subsequent fire blight development) did not occur experimentally, even under fire blight-conducive conditions. Had Roberts et al. (1998) used the absence of any scientific evidence that a vector exists to transfer hypothetical bacteria from a discarded fruit to a susceptible host, the value for that step in the pathway would have been zero, and the model would have predicted the number of years before the first outbreak to be infinity. Thus, Roberts et al. (1998) used non-zero estimates for nodes, even those for which there was no evidence a step in the hypothetical pathway could be completed. Even using these inflated hypothetical values the theoretical probabilities were extraordinarily low.

Events since 1998, both scientific and political, provide rationales for a revision of the dataset used to estimate the values for the pest risk assessment (PRA). First, the accretion of data after 1998 from field experiments that are relevant to one or more nodes in the model provides a reason to recalculate the estimates provided in Roberts et al. (1998). Second, the US government sought relief from Japan’s very restrictive phytosanitary regulations in 2002 by requesting establishment of a Dispute Settlement Body Panel at the World Trade Organization (WTO), citing, among other reasons, a lack of scientific support for Japan’s regulations. The PRA of Roberts et al. (1998) was updated during the course of these proceedings and submitted to the Panel as an exhibit with one of the US submissions. The Panel’s decision, which was adopted by the WTO membership in 2005, supported the US claims, and found no valid scientific support for a buffer zone of any size, the need for absolute freedom from fire blight in export orchards, the need for any preharvest orchard inspections, or the need for chlorine treatment of fruit, harvest bins or packinghouse equipment. Accordingly, the Government of Japan modified its law regulating import of US apples so that the only phytosanitary requirement is that exported fruit be accompanied by a phytosanitary certificate documenting they are physiologically mature and free of fire blight disease symptoms. Third, in spite of the 2005 decision by the WTO, some countries such as Australia (Anonymous, 2006) maintain prohibitive regulations against the importation of pome fruit from countries where fire blight occurs. Fourth, comments on the validity of the methodology and conclusions in Roberts et al. (1998) by Yamamura et al. (2001) have been published and require a response.

Accordingly, this updated risk assessment includes additional relevant data published since 1998; data which either provide a quantitative basis for estimating probabilities that were initially based upon subjective evaluations with some degree of uncertainty (e.g., P2 and P5), or further substantiate probability estimates that were already strongly supported by published data (e.g., P1). Certain misinterpretations, misunderstandings, or misstatements regarding some of the previously published works that appeared in the original risk assessment paper, particularly as related to the calculation for P1, the probability that fruit is contaminated with *Ea*, are noted and corrected. Finally, we seek to make the model more robust by estimating the risk under several different levels of export that may be obtained should less restrictive phytosanitary measures be implemented by importing countries. Little information regarding the field biology of *Ea* and fire blight has been published beyond that already discussed in depth in the first risk assessment, and this will not be covered again here.

2. Materials and methods

2.1. Changes to node value estimates

F1: Number of fruit transported per year. The value used for F1 in Roberts et al. (1998) was 20,000,000 fruit. The revised model also estimates risk using three additional levels of fruit export. The 800,000,000 and 80,000,000 fruit values represent approximately 10% and 1% of an 80,000,000-box crop, respectively. Actual export statistics indicate that 6534 tonnes of apple fruit were exported to Japan in 1995. This is the highest volume year to date, and based upon 88 fruit per 19 kg box, is calculated to have been 30.1 million fruit, which is close to the value used in Roberts et al. (1998). Interestingly, the volume of exports has declined steadily since 1995—116 tonnes (about 561,000 fruit) in 2001. No apples have been exported to Japan from the US since 2001.

P1: Probability a fruit is contaminated with *E. amylovora*. Table 1 from Roberts et al. (1998) presented data on the detection of *Ea* from mature, symptomless apple fruit from various locations and harvested under differing levels of disease pressure. Several published studies have documented that populations of *Ea* associated with developing apple fruit decline during the growing season to zero or near-zero at harvest (Dueck and Morand, 1975; Hale et al., 1987), even when fire blight disease was present in the orchard. As immature apple fruit are not involved in commercial movement of apple fruit, only mature apple
fruit were considered appropriate for inclusion in this risk assessment. Upon further review it became apparent that some of the data reported in Roberts et al. (1998) from van der Zwet et al. (1990) were inappropriate for inclusion in Table 1 because either (1) the data resulted from assays of immature fruit, or (2) the data resulted from assays of stored fruit (van der Zwet et al., 1990). The data from the immature fruit assays were excluded from the revised model, so the value used for Washington State changed from 40 to 20 for the number of fruit harvested from lightly infected trees and trees without fire blight. The datum value of 0 detections from 105 fruit given in Table 1 of Roberts et al. (1998) for fruit harvested from trees near-moderately blighted pear trees or from trees without fire blight was removed from Table 1, because the fruit were assayed after storage, and the values may have reflected the natural decline of *Erwinia amylovora* populations observed during storage (Hale and Taylor, 1999). Also after additional review, the datum value for number of fruit assayed presented in the Roberts et al. (1998) for West Virginia (from van der Zwet et al., 1990), was changed from 80 to 40, because half of the fruit were determined to be immature at the time of assay based upon review of the harvest dates. This also changed the resulting number of fruit positive for *Ea* from 5 to 0 for fruit harvested from trees near moderately blighted pear trees and from 2 to 0 for fruit harvested from trees without fire blight. Additionally, another 175 apple fruit from West Virginia (van der Zwet et al., 1990) that were inadvertently excluded from Roberts et al. (1998) analysis were included in this revision, and resulted in the addition of 5 apples to the

<table>
<thead>
<tr>
<th>Location</th>
<th>FB present in orchard</th>
<th>No. fruit assayed</th>
<th>No. fruit positive</th>
<th>Used to calculate P1</th>
<th>Reference Notes</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Washington State, USA</td>
<td>Yes&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
<td>1455</td>
<td>0</td>
<td>S3</td>
<td>Roberts et al. (1989)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>100</td>
<td>0</td>
<td>S1, S2, S3</td>
<td>Roberts et al. (1989)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>900</td>
<td>0</td>
<td>S3</td>
<td>Roberts (2002)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes&lt;sup&gt;b&lt;/sup&gt;</td>
<td>20</td>
<td>0</td>
<td>S2, S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>20</td>
<td>0</td>
<td>S1, S2, S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td>West Virginia, USA</td>
<td>Yes&lt;sup&gt;c&lt;/sup&gt;</td>
<td>40</td>
<td>0</td>
<td>S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No&lt;sup&gt;e&lt;/sup&gt;</td>
<td>40</td>
<td>0</td>
<td>S2, S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No&lt;sup&gt;d&lt;/sup&gt;</td>
<td>175</td>
<td>5</td>
<td>S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td>Utah, USA</td>
<td>Yes&lt;sup&gt;b,d&lt;/sup&gt;</td>
<td>20</td>
<td>1</td>
<td>S3</td>
<td>van der Zwet et al. (1990)</td>
<td>Detected from calyx (external), S. Thompson, pers. commun.</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>20</td>
<td>0</td>
<td>S1, S2, S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td>New Zealand</td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>400</td>
<td>3</td>
<td>S3</td>
<td>Hale et al. (1987)</td>
<td>Calyx washings</td>
</tr>
<tr>
<td></td>
<td>Yes&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1400</td>
<td>0</td>
<td>S2, S3</td>
<td>Hale et al. (1987)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>300</td>
<td>0</td>
<td>S1, S2, S3</td>
<td>Hale et al. (1987)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes&lt;sup&gt;b&lt;/sup&gt;</td>
<td>173</td>
<td>0</td>
<td>S2, S3</td>
<td>Hale et al. (1996a)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes&lt;sup&gt;b&lt;/sup&gt;</td>
<td>150</td>
<td>0</td>
<td>S2, S3</td>
<td>Hale and Taylor (1999)</td>
<td></td>
</tr>
<tr>
<td>Ontario, Canada</td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>60</td>
<td>0</td>
<td>S3</td>
<td>Dueck (1974)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>40</td>
<td>0</td>
<td>S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>40</td>
<td>0</td>
<td>S1, S2, S3</td>
<td>van der Zwet et al. (1990)</td>
<td></td>
</tr>
<tr>
<td>British Columbia, Canada</td>
<td>Yes&lt;sup&gt;a&lt;/sup&gt;</td>
<td>54</td>
<td>54</td>
<td>S3</td>
<td>Scholberg et al. (1988)</td>
<td>Pooled samples, actual (+) number could be 18–54</td>
</tr>
</tbody>
</table>

<sup>a</sup>Fruit harvested from heavily infected trees.
<sup>b</sup>Fruit harvested from lightly infected orchards.
<sup>c</sup>Fruit harvested from trees located near moderately blighted pear trees.
<sup>d</sup>Fruit harvested from trees within 10 m of severe fire blight.
number of fruit positive for Ea. The data values reported for Utah were also revised because most of the fruit assayed were immature. Thus, the values for number of fruit assayed changed from 80 to 20 because none of the Rome apple fruit were mature at the times of assay, and only the last harvest of Red Delicious may have been mature (S. Thomson, personal communication). Accordingly, the value for the number of positive fruit changed from 27 to 1 for fruit harvested from lightly infected trees, and remained 0 for fruit harvested from trees without blight.

The design of several experiments from Scholberg et al. (1988) was misinterpreted by Roberts et al. (1998); the corrected data values are presented in Table 1. The value of 72 apples actually referred to a storage experiment, and was incorrectly reported in Roberts et al. (1998) as 72 positive isolations of Ea. The actual datum value after storage was 0. Furthermore, these data resulting from a storage experiment were inappropriate for inclusion in Table 1. For those fruit assayed by Scholberg directly after harvest, the assay technique used by the authors involved incubating bulked samples (3 bulked fruit per assay, with 3 replications and 6 treatments) in an enrichment medium after harvest, and plating this medium onto selective agar media. Therefore, the number of fruit assayed was changed from 72 to 54, and because the fruit were bulked into 3-fruit samples, the range of values for possible number of fruit positive for Ea is 18–54. The most conservative approach was taken by including the largest value (54) in the revised Table 1.

Additional data for Washington State were added to Table 1, based on the results of a joint US-Japan experiment that was conducted in Washington State in 2000 (Roberts, 2002; Mizuno et al., 2002). In this study, 900 mature fruit were harvested at varying distances (0–300 m) from point sources of fire blight inoculum, and assayed after harvest for the presence of Ea by both Japanese and US scientists. It is of interest and appropriate to note here that Mizuno et al. (2002) did not report data collected from apples harvested directly from the trees with fire blight disease that served as the point source of inoculum for the study. Even though Japan was afforded the opportunity to sample these fruit for the presence of internal Ea and development of fire blight during storage, they chose not to. *E. amylovora* was not detected from any of 900 fruit, and these data are reflected in revised Table 1 and were used to calculate the revised estimate for P1.

Results from a total of 5407 tested fruit are reported in Table 1. This should not be confused with or misrepresented as a random sample from all available fruit. Rather, it represents a highly biased sample given that most of the fruit were selected because of their close proximity to fire blight disease (e.g., harvested from a tree or branch with fire blight disease). Instead, the data can be associated with different levels of disease presence or sanitation practiced by growers in the region where apples are produced for export. To obtain a meaningful estimate of the overall rate of contamination of mature fruit by Ea, the experimental data must be sorted and weighted as if they were from a stratified random sample, the strata consisting of orchards meeting different phytosanitary standards (the scenarios S1, S2 and S3 in Roberts et al., 1998).

During the years between the publication of the original PRA and the current revision, a number of papers have proposed a viable but not culturable (VBNC) state for a range of environmental bacterial isolates. A VBNC state for Ea was described by Ordax et al. (2006) in response to continuous exposure to dilute aqueous copper solutions. Resuscitation of Ea from the VBNC state was claimed after removal of the copper and addition of nutrients. To date no information or data are available that would establish the existence of this state for Ea in nature; only laboratory induction of the VBNC state has been reported. Given that there are no data on the incidence of VBNC cells of Ea on mature apple fruit (or anywhere else in nature) or any evidence that such a state is epidemiologically significant with regards to natural populations of Ea or the initiation of fire blight disease, there is no path to inclusion of VBNC cells of Ea in the PRA other than speculation, which would be inappropriate and contrary to the stated goal of providing a quantitative assessment of risk. Given the total absence of such data, it is inappropriate to speculate how such a state, should it ever be demonstrated to occur naturally with Ea, might influence our understanding of fire blight epidemiology. Given that the extremely low incidence of culturable Ea cells reported in the scientific literature is entirely consistent with the historical fact of non-transmission of Ea and fire blight via commercial apple fruit and the lack of a demonstrated pathway by which such an event could occur, it would be a striking development indeed should data eventually be published proving a causal relationship between Ea in a VBNC state on fruit and the establishment of fire blight disease.

For P1, S1: P1 for S1 is estimated on the following evidence: 480 fruit from 5 trials were found free of Ea when harvested from orchards free of fire blight (300, 100, 40, 20 and 20). The 50% upper confidence limit on the value of IR, the probability that an individual mature fruit is contaminated with Ea, is calculated from the binomial distribution as

\[
\text{IR} = 1 - P(0)^{1/n} = 0.0014430,
\]

as follows:

\[
\text{IR} = 1 - 0.5^{1/480},
\]

\[
\text{IR} = 1 - 0.5^{0.0020833},
\]

\[
\text{IR} = 1 - 0.9985570,
\]

\[
\text{IR} = 0.0014430.
\]

The binomial distribution is used throughout for calculations of positive values for infestation rates in those instances where all the data indicate that no *E. amylovora* is found associated with a particular class of fruit. As discussed above, this should not be construed as an acknowledgement of risk where none has been indicated by scientific investigation, but as an acknowledgement by
the authors of the impossibility of proving that something does not exist or never occurs.

For P1, S2: Using the same logic offered in the original PRA, P1 for S2 is estimated as 0.0003998 on the following evidence. The estimated contamination rate for S2 orchards that would not meet S1 requirements is 0.0003887, and is calculated from Table 1 using data from lightly infected orchards or orchards near-moderately blighted pear trees, where 1783 fruit tested in 5 trials were found to be free of Ea (1400, 173, 150, 40 and 20).

Because in this revised model none of the fruit meeting S2 criteria were positive for Ea, the 50% upper confidence limit on the value of IR is estimated as for S1.

\[
IR = 1 - 0.5^{1/1783},
\]
\[
IR = 1 - 0.5^{0.0005608},
\]
\[
IR = 1 - 0.9996113,
\]
\[
IR = 0.0003887.
\]

An estimated 1% of apples originate from orchards that would meet requirements for S1, and 95% of apples are estimated to meet S1 or S2 requirements. Thus, 1/95 of the apples have an estimated contamination rate of 0.0014430, and 94/95 of the apples have an estimated contamination rate of 0.0003887, giving a weighted average value of 0.0003998.

\[
1/95 \times 0.0014430 = 0.0000152(S1),
\]
\[
94/95 \times 0.0003887 = 0.0003846(S2)
\]
\[
0.0003998.
\]

For P1, S3: P1 for S3 is estimated as 0.0013817. From nine trials marked S3 but not S2 or S1, 3144 mature fruit were tested and 63 were found to be externally contaminated with Ea, giving a contamination rate of 0.0200382 for orchards meeting only S3 requirements (1455, 900, 400, 175, 60, 54, 40, 40 and 20, Table 1). As 95% of apples met either S1 or S2 requirements, 5% of the apples met only S3 requirements. Thus, a weighted average is calculated as follows:

\[
5/100 \times 0.0200382 = 0.0010019(S3 \text{ orchards only})
\]
\[
95/100 \times 0.0003998 = 0.0003798(\text{orchards meeting S1 or S2 requirements})
\]
\[
0.0013817.
\]

P2: Probability that *E. amylovora* survives storage, transport and discard conditions: In the 1998 PRA, a subjective estimate of 0.1 was used for P2 because published data from which to make more supported calculations was lacking. The 160 fruit from Washington State that were incorrectly used in calculations to estimate P1 were inserted into the calculation for P2 (Table 2). Additionally, Hale and Taylor (1999) published data that directly addressed the question of survivability of Ea on stored fruit infested with various levels of Ea and then held for either 25 d in cool storage, or 25 d in cool storage followed by 14 d at room temperature. Ea populations on infested fruit calyces ranged from $10^0$ to $10^7$ cfu. Live Ea cells were detected on 59 of 570 contaminated fruit assayed after cool storage for 25 d only, all from fruit with initially high infestation levels. Live Ea cells were detected on 2 of 570 contaminated fruit assayed after cool storage for 25 d plus 14 d at room temperature, again from fruit with the highest initial infestation levels. As this node is intended to estimate the total effect of storage, transport and discard conditions on survival of Ea, we have used only the data from the 25 d in cold storage plus 14 d at room temperature, as it most closely reflects the cold storage requirement for US apples to Japan, which is 55 d at 2.2°C or less. Even so, the node value estimate used in this revised PRA overestimates survival of Ea on apple fruit, should it be present, because the additional population reductions that would be expected during the longer cold storage period, transport period and discard conditions are not considered. The node value estimate for P2 was calculated by dividing the total number of fruit positive for Ea after storage (2) by the total number of contaminated fruit placed into storage (570), yielding a value of 0.0003588. The value of this new, quantitative estimate was used for P2 in the revised model in place of the subjective estimate used previously.

P5: Probability that *E. amylovora* is transferred to a new host and infection occurs: As stated in Roberts et al. (1998), there is no documented vector that transfers bacterial cells from a hypothetically contaminated, discarded apple fruit to a susceptible host, which then (hypothetically) becomes infected. As a value of 0 for this node would reduce the probability of new outbreaks occurring from the entire model to 0, in the 1998 PRA we used an estimate of 0.0001 for P5, with a high degree of uncertainty because there was only one study available that provided pertinent data (Hale et al., 1996). Based upon additional studies we have revised the estimate for P5 to 0.0003786, with significantly less uncertainty than previously stated, because now there are data to support a quantitative estimate for this node value. As in Roberts et al. (1998), data from Hale et al. (1996) that evaluated lateral spread of Ea from 30 contaminated fruit placed into an orchard were included in this analysis. In a subsequent 2-year study in New Zealand, Taylor et al. (2002) placed 1800 apple fruit that were contaminated with a marked strain of Ea 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. As there was no lateral movement or fire blight development detected in any of these studies, the binomial formula was used, as before, to calculate a 50% upper confidence limit on the value for P5.

3. Results

Based upon the changes to certain node value estimates detailed above, revised values in the risk assessment,
expressed as years before first outbreak, were calculated for each of 4 levels of apple fruit export to Japan, and are presented in Table 2. For S1, the revised estimates for years before the first outbreak of fire blight ranged from 5217 years to 208,667 years. For S2, the revised estimates for years before the first outbreak of fire blight ranged from 18,829 to 753,144 years. For S3, the revised estimates for years before the first outbreak of fire blight ranged from 5448 to 217,925 years. In comparison, the estimates for years before the first outbreak for S1–S3 from the 1998 PRA (20 million fruit exported) vs. the estimates from this revised PRA were: 38,462 vs. 208,667 years (S1); 35,971 vs. 753,144 years (S2); and 11,364 vs. 217,925 years (S3), respectively.

4. Discussion

The removal of inappropriate data (from assays of immature or stored fruit) and the inclusion of new data published since 1998 (Roberts, 2002; Hale and Taylor, 1999; Taylor et al., 2002) profoundly affected the node value estimates for P1 and P2 and slightly modified the estimate for P5. The resulting estimates of the probability of an outbreak of fire blight due to trade in export-quality apple fruit were dramatically lower than those reported in the 1998 PRA, even at increased export levels (Table 2). These changes also greatly enhance our confidence in the current estimates over those in the 1998 PRA because several of the most subjective estimates have been replaced with estimates based upon published data, and demonstrate that the estimated probabilities of spreading Ea and fire blight on apple fruit in Roberts et al. (1998) were overestimated, even though extremely low. The new estimates strongly reinforce the conclusion drawn in the original 1998 PRA that international trade of commercial, export-quality apple fruit poses a negligible risk of introducing fire blight to importing countries.

Yamamura et al. (2001) sought to provide a “better statistical procedure” for estimating the probability of spread of fire blight disease via imported apple fruit using the data from Roberts et al. (1998), by considering the “variability of the proportion of infected fruits that may occur both temporally and spatially”. Thus, there are two cases to consider: models treating P1 as a constant estimated from actual published data (Roberts et al., 1998) and the model of Yamamura treating P1 as a random variable.

In the first case, Yamamura et al. (2001) estimated P1 by utilizing all of the data in Table 1 of Roberts et al. (1998). The sum of all infested fruit, from all studies, was divided by the sum of all fruit assayed, yielding the estimate $P1 = \frac{109}{4650} = 0.0234$. While this is the correct method of obtaining a ratio estimate of the proportion infested among all studies reported in Table 1 (Roberts et al., 1998), it is inappropriate for the purpose at hand. This is because the data in Table 1 do not represent a random sample of fruit shipped from any production area, as assumed by Yamamura et al. (2001). Rather, the data in Table 1 represent the results of several studies of the pathogen Ea conducted under various circumstances for various

<table>
<thead>
<tr>
<th>No. fruit/ year transport</th>
<th>F1</th>
<th>P1 Probability that a fruit is contaminated with Ea</th>
<th>P2 Probability pathogen survives storage, transport and discard</th>
<th>P3 Probability fruit is discarded near host</th>
<th>P4 Probability host is at receptive stage</th>
<th>P5 Probability of Ea transfer to new host and infection occurs</th>
<th>F2 Years until first outbreak</th>
</tr>
</thead>
<tbody>
<tr>
<td>Revised estimation for S1, from revised PRA (Table 1)</td>
<td>800,000,000</td>
<td>0.0014430</td>
<td>0.0035088</td>
<td>0.0025</td>
<td>0.05</td>
<td>0.0003786</td>
<td>0.0001916926914240</td>
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<td>97,000,000</td>
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<td>0.05</td>
<td>0.0003786</td>
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<td>Revised estimation for S2, from revised PRA (Table 1)</td>
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<tr>
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<tr>
<td>Revised estimation for S3, from revised PRA (Table 1)</td>
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<td>0.0013817</td>
<td>0.0035088</td>
<td>0.0025</td>
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</table>

800,000,000 fruit approximates 10% of an 80 million box crop.
97,000,000 fruit approximates 485 sea containers, and is the approximate amount of fruit shipped to Japan in 1995.
the model’s parameters, variation in a proportion. The authors correctly estimated the role of variation in the parameter’s value and the issue. In particular, it is certain to be too high, over-estimate for the production areas of concern in this trade export to any country. Rather, it is an estimate that pertains only to the biased data set reported in Table 1. And, as Yamamura et al. (2001) noted, the $\beta$-distribution did not appear to fit this unrepresentative data set particularly well. Because Yamamura’s two models (one treating $P_1$ as a constant, the other treating it as a random variable) employed very different estimates of the mean infestation rate (0.0234 and 0.1200, respectively), the resulting predicted frequency of outbreaks and time between infestations cannot be compared. The differences found by Yamamura et al. (2001) in the predictions of these two models were due entirely to the different mean values of $P_1$, not to variation in the parameter $P_1$ per se. Thus, the primary point and the key conclusion of Yamamura et al. (2001) are both negated.

Yamamura et al. (2001) additionally stated, “Our results are not conclusive...”. Results based on sampling, statistics and modelling are never certain and, in this sense, are never “conclusive.” However, at any given level of knowledge, conclusions can be drawn. If appropriate quantitative tools are applied properly to data collected in a scientifically acceptable manner, and if the results are correctly interpreted and explained, then these results may be convincing and even difficult to dispute. The likelihood that quantitative results are within any desired distance of the true value can be estimated. In the case of Yamamura et al. (2001), the above statement continued, “...since the data used in the estimation, i.e. Table 1 of Roberts et al. (1998) were not obtained from a random sampling.” Moreover, the authors admitted “the validity of the beta distribution itself might be suspect.” As both the use of the data from Roberts et al. (1998) as if it represented a random sample and the use of the $\beta$-distribution to represent variation in the proportion of infected fruit were vital to the analysis of Yamamura et al. (2001), it may be concluded that the authors themselves considered their results to be invalid, rather than inconclusive.

Acknowledgements

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References


