Importation of Fresh Bananas from the Philippines

Revised Draft IRA February 2004

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Introduction

This submission is limited in scope to aspects of Moko disease of dessert and cooking bananas caused by *Ralstonia solanacearum* race 2 relevant to this IRA.. The objective has been to clarify understanding of this disease and its consequences, where there have been apparently conflicting statements in the scientific literature.

The submission is made in my capacity as a member since April 2001 of the Technical Working Group (Pathogens) responsible for giving advice to the Import Risk Analysis Team.

Summary of Main Conclusions

1.The internationally recommended name for the wilt of dessert bananas and plantains (cooking bananas) is Moko bacterial wilt. The internationally recommended name for the fruit rot of cooking bananas in the Philippines is Bugtok (=bacterial hard pulp). Both diseases are caused by *Ralstonia solanacearum* race 2. DNA-based methods and pathogenicity tests show conclusively that both diseases in the Philippines are caused by an identical agent.

2.Although there is little published evidence it is likely that Moko bacterial wilt of plantains in Central and South America and Bugtok of the cooking bananas Saba and Cardaba in the Philippines have been spread on infected bunches to new locations and then disseminated by insects to healthy plants. Moko bacterial wilt of dessert bananas is in most instances known to have been introduced to new areas on infected planting material (rhizomes). There is no published information showing that dessert banana fruit has served to introduce Moko bacterial wilt to a new location.

3.Moko bacterial wilt of dessert bananas does not result in devastating losses but must be held in check through the implementation of control measures involving eradication of infected plants. There are no instances where Moko bacterial wilt has been successfully eradicated once the disease has become established.

4. Insect transmission of Moko bacterial wilt is rampant only where certain cooking banana genotypes are grown on a large scale (e.g. Bluggoe in Central and South America and Saba and Cardaba in the Philippines). These or similar cooking bananas are not grown in Australia. The B strain of *R.solanacearum* present in the Philippines is not associated with a high incidence of insect transmission on dessert bananas in other countries.

5.Moko bacterial wilt is a disease of the tropics; there are no confirmed records of the disease in any subtropical location. There is nothing in the literature to suggest that *Ralstonia solanacearum* can readily adapt to cool climate conditions.

6. The Draft IRA has set stringent Area of Low Pest Prevalence (ALPP) requirements for areas from which bananas can be exported to Australia; a standard which is rarely achieved in the Philippines at present. At this stage it is not known whether the ALPP is attainable in the Philippines.

7. The Draft IRA has made a conservative estimation of the proportion of banana plants infected with the Moko bacterium which develop (symptomless) infected bunches. A high proportion of infected fingers are found only on cooking bananas as a result of insect transmission which is not known to occur on dessert bananas affected by the B strain of *Ralstonia solanacearum* in the Philippines.

8. The probability of distribution of Moko bacterial wilt from discarded banana waste has been estimated very conservatively in the Draft IRA. There is no evidence to support the contention that insects could transmit the disease from discarded banana waste to healthy banana plants.

9. If the second risk management measure were adopted, involving restriction of the distribution of Philippine bananas in Australia, it would be a criminal offence to discard Philippine banana waste in any part of the area of exclusion (maximum penalty 10 years imprisonment).

The following subjects are covered:

(i) Terminology: common names of banana diseases and their causal agents

(ii) Implications of the changed understanding of the relationships of Moko bacterial wilt disease of dessert bananas and Bugtok disease of cooking bananas in the Philippines in the past decade

(iii) Evidence that fruit of cooking bananas has served as a means of distribution of Moko bacterial wilt and Bugtok

(iv) Economic importance of Moko bacterial wilt on dessert banana and of Bugtok on cooking bananas

(v) Effect of environmental factors on disease expression of Moko bacterial of banana and plantain and implications for geographical distribution of the disease

(vi) Areas of low pest prevalence

(vii) Estimation of the proportion of banana plants infected with the Moko bacterium which develop (symptomless) infected bunches

(viii) Potential for the distribution of Moko bacterial wilt from discarded banana waste

(ix) Bibliography

(i) Terminology: common names of banana diseases and their causal agents.

The International Society for Plant Pathology has set up a *Committee on Common Names of Plant Diseases* under the Chairmanship of Dr. David Teakle. A subcommittee consisting of Dr. David Jones, Dr. Chris Hayward and Dr. John Thomas has recently released the results of its deliberations on banana diseases: Common Names of banana diseases and their causal agents (www.isppweb.org/names_banana_common.asp).

The recommended common name for the wilt of dessert bananas and plantains (cooking bananas) caused by *Ralstonia solanacearum* race 2 is Moko bacterial wilt.

The recommended common name for the fruit rot of the cooking bananas Saba and Cardaba (ABB/BBB genotype) in the Philippines caused by *Ralstonia solanacearum* race 2 is Bugtok (=bacterial hard pulp). The recommendation to retain "Bugtok" as a common name is potentially a source of confusion and requires justification.

In Central and South America Moko bacterial wilt occurs on plantains of the Bluggoe subgroup (ABB genotype: Bluggoe, Moko, Cachaco and Chato) as well as other subgroups. Transmission of Moko bacterial wilt in these plantains occurs principally through insects such as bees, wasps and flies which visit the flowers. Once the flower of any susceptible variety has been infected, the bacteria move through the vascular system of the peduncle and pseudostem to the rhizome and other organs, which can lead to mechanical transmission by machete during agricultural activity. When 'seeds' (rhizomes) from diseased fields are used to establish new plantations, the disease is readily transmitted. (French and Sequeira, 1968). Moko bacterial wilt of Bluggoe and other plantains in Central and South America shows symptoms of both fruit rot and wilting of the foliage.

There is considerable variation among cooking bananas of the AAB and ABB genotypes in their susceptibility to insect transmission of Moko bacterial wilt. According to Stover (1972): "Those varieties with persistent bracts are less liable to infection by insect-transmission through the male flower bud than are varieties with dehiscent bracts".

The disease on the cooking bananas Saba and Cardaba in the Philippines is different in several respects from Moko bacterial wilt on plantains in Central and South America and is given the common name "Bugtok (=bacterial hard pulp)". The differences are listed below.

Moko bacterial wilt on Bluggoe Plantain: the disease becomes systemic and there are wilt symptoms; rhizomes become infected and serve as a vehicle for disease transmission when planted elsewhere; there are obvious symptoms in diseased fruit which may show premature ripening (yellowing) of fingers among green uninfected fingers. If the fruit is cut some of the infected fruit will show a rotting that goes from brown and viscous to grey and dry (French and Sequeira, 1968)

Bugtok disease of the cooking bananas Saba and Cardaba in the Philippines: following insect transmission to the inflorescence infection progresses through the peduncle and pedicels to the developing fruit and downwards through the pseudostem. The disease rarely becomes fully systemic and there are no symptoms of wilt. By contrast with Moko bacterial wilt on bluggoe plantain, suckers from infected plants do not serve to transmit the disease when planted in pristine areas (Soguilon *et al.*, 1994). Infected fingers are outwardly normal and do not show premature ripening; when cut they show a black or reddish discolouration. The fruit are firm uncooked and the pulp hardens after cooking. (N.B. the terms "Bugtok", "Tibaglon" and "Tapurok" which have been used to describe this disease in different parts of the Philippines all mean "hardening" of the fruit pulp). Bugtok disease is an insect transmitted fruit rot and wilt symptoms are absent.

(ii) Implications of the changed understanding of the relationships of Moko bacterial wilt of dessert bananas and Bugtok disease of cooking bananas in the Philippines in the past decade

On visits to Southern Mindanao in 1996 (with Dr. Luis Sequeira) and again in 1998 I gained the impression that plant pathologists and agronomists working in the banana industry did not consider that Bugtok disease of cooking bananas growing in backyards near to plantations of dessert bananas was of any consequence in the epidemiology of Moko bacterial wilt on dessert bananas.
In August, 2001, the Chairs of the Technical Working Groups appointed by Biosecurity Australia to evaluate importation of fresh banana fruit from the Philippines visited banana plantations in the Philippines. In their report released in January, 2002, the following statement shows the understanding current at that time.

"Moko and bugtok

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There appear to be two schools of thought on the causal agent of Moko and bugtok.

- The first school of thought believes that Moko and bugtok are caused by different strains of the bacterium *Ralstonia solanacearum* because Moko is found on Cavendish bananas and is not insect-transmitted while bugtok is found on local cultivars and is insect-transmitted. The TWG chairs were advised that there is no evidence of insect-transmission of bugtok from backyard plants of local cultivars to commercial plantations of Cavendish bananas.

- The second school of thought is saying that Moko and bugtok are caused by the one and the same strain of *R.solanacearum* because the isolates of bugtok and Moko are almost identical based on DNA analysis. Limited cross inoculation experiments have indicated that bugtok isolates infect young tissue cultured plantlets of Cavendish bananas. This issue needs clarification and the Philippines have agreed to provide further information."

In the past three years more evidence has been gathered showing conclusively that Moko bacterial wilt of dessert bananas in the Philippines and bugtok disease of cooking bananas are caused by identical agents (Fegan, 2002; Draft IRA Report, p.291); the first school of thought no longer has any credence. The evidence was incomplete until cross infection tests with Moko and bugtok isolates on Cavendish bananas were carried out in the field in the Philippines during 2002-2003. This work done at the instigation of Biosecurity Australia, showed that the isolates of either source were equally pathogenic on Cavendish banana (Soguilon, 2003).

The implication for the Draft IRA is that any control measure involving Area Freedom should take into account distribution of infected cooking bananas as well as of dessert bananas. If the Philippine authorities expect to meet the stringent Area of Low Pest Prevalence requirements specified in the Draft IRA area surveys should include both diseases. To quote the Executive Summary: "The low pest prevalence (LPP)level for Moko in an approved ALPP would not exceed 0.005 cases (infected mats) per hectare per year - i.e. no more than one in 6,800 infected plants per year." Infected cooking bananas could be a source of infection in plantations of dessert banana, probably mainly through mechanical means and use of contaminated implements rather than through movement of soil. Whether insect transmission to Cavendish bananas is also involved requires further investigation.

(iii). Evidence that fruit of cooking bananas has served as a means of distribution of Moko bacterial wilt and Bugtok disease in Central and South America and the Philippines

There is no published record stating that dessert bananas have served to introduce Moko bacterial wilt into a locality previously free of the disease. The disease has been spread in Central and South America and the Caribbean on rhizomes for planting and locally by mechanical means, movement of soil and in some instances by insects (Buddenhagen, 1961). The bulk of dessert bananas are transported from the tropics and subtropics to regions such as Western Europe and countries such as Japan where bananas are not grown commercially. If Moko bacterial wilt has been detected in such export fruit the information is not public. By contrast it is probable that Moko bacterial wilt of plantains in Central and South America and Bugtok of cooking bananas in the Philippines have been spread on infected fruit from one locality to another. French and

Sequeira (1968) made an extensive survey of Moko bacterial wilt in the tributaries of the Amazon extending into Peru. They indicate that river traffic could serve to spread the disease, and comment that, "All plantains and bananas that are aboard the boats (even though they may only be for consumption by the crew) should be inspected, as it is possible that affected racemes will sprout in the water or on the bank of the river, where they can serve as a source of infection for transmission of the bacteria by insects."
Bugtok disease of cooking banana occurs widely in the southern, central and parts of the northern Philippines (Draft IRA p. 384). There is no published information on the mode of dissemination among the islands of the Philippines. It is unlikely that infected planting material (rhizomes or suckers) were involved in view of the evidence of Soguilon *et al.* (1994) that suckers from diseased plants did not transmit the disease when planted in pristine land. Although insect transmission of Bugtok occurs within plantings of cooking bananas there is little information on the ability of insects to carry the disease over long distance overland (Buddenhagen and Elsasser, 1962) and none at all of dissemination over water. It seems unlikely that this would occur given the sensitivity of *Ralstonia solanacearum* to UV radiation and desiccation. It is likely that Bugtok could have been spread between islands on infected bunches. The fingers show no external signs of infection and being less perishable than dessert bananas are capable of being transported for marketing between islands. The cut peduncles of infected bunches are a source of bacterial ooze which could be carried by insects to the inflorescences of healthy plants, in a manner similar to that postulated by French and Sequeira (1968) to have occurred along the tributaries of the Amazon river.

(iv) Economic importance of Moko bacterial wilt on dessert bananas and of Bugtok on cooking bananas

The losses caused by Moko bacterial wilt on bananas and plantains in Central and South America and Bugtok on cooking bananas in the Philippines are well covered on pp.158 and 159 of the Draft IRA. The literature is confusing where it is not made clear that the term banana is used to include both dessert banana and plantain. For example Phelps (1987) and Coelho Netto and Nutter (2002), among other authors, do not always distinguish between losses caused by Moko bacterial wilt on dessert and cooking bananas. The disease is most devastating on the plantains (cooking bananas) such as Bluggoe and Saba and Cardaba where there is a high incidence of insect transmission. According to Stover (1972) plant losses attributable to Moko disease were as low as 1% annually but this is only achieved as result of the application of expensive control measures without which losses could increase to 5% or more. On this evidence Moko bacterial wilt on dessert bananas is not a devastating disease comparable to several diseases caused by fungi, viruses and nematodes. According to Phelps (1987) on large estates in Panama, Costa Rica, Honduras, Nicaragua and Guatemala, "Moko disease is rated second to Sigatoka, on the basis of its effects on banana as well as on the cost of control measures". In contrast Moko bacterial wilt on Bluggoe plantain or Bugtok on Saba and Cardaba in the Philippines can be devastating unless bagging of inflorescences or debudding are practised (Molina,1996).

In the Philippines the B strain of *R. solanacearum* is present (Draft IRA p. 389; Fegan, 2002) on both dessert and cooking bananas; in the latter insect transmission is highly significant and often results in a high incidence of fruit rot. There is no evidence that insect transmission occurs in dessert bananas but more investigation is required. Where the SFR strain is present there is potential for insect transmission on both dessert and cooking bananas. According to Phelps (1987): "If the SFR strain is introduced into an area where the susceptible bluggoe is in mixed planting with or near to commercial banana, the spread of Moko will be rapid in both species."

The paragraphs headed 'The direct impact of Moko" on pages 158 and 159 of the Draft IRA should probably be redrafted to make clear where disease losses attributable to Moko bacterial wilt are on cooking bananas rather than dessert bananas.

Moko bacterial wilt of dessert bananas occurs in all of the major banana producing countries (Ecuador, Costa Rica, Columbia, the Philippines etc, Draft IRA p.33) but is held in check through application of well understood control measures, including destruction of infected mats. Australia is probably unique in having a higher degree of mechanisation and substantially higher labour costs than the other countries affected by the disease. Control through destruction of infected mats would be a much more expensive undertaking in North Queensland if the disease had become established.

The spread of Moko bacterial wilt from island to island in the Caribbean and from South America to Central America has been the result of planting of infected seed pieces (rhizomes) (Buddenhagen, 1961). There appear to be no published records of successful eradication of Moko bacterial wilt from any country once the disease has become established. Eradication of infected mats limited the spread of the disease in Grenada (Hunt, 1987).

(v) Effect of environmental factors on disease expression of Moko bacterial wilt of banana and plantain and implications for geographical distribution of the disease

There have been no studies under controlled environment conditions of the effects of environmental factors on expression of Moko bacterial wilt on banana. The environmental factors include diurnal temperature regime, soil and air temperature, photoperiod, soil moisture and humidity. Inspite of the paucity of information on banana some useful information has been obtained for *Ralstonia solanacearum* on other hosts , such as tomato, potato and peanut. The integration of this information enables prediction of the likely impact of the pathogen when distributed to new environments.

The distribution of bacterial wilt worldwide in relation to climate is well understood. The following conclusions are based on the exhaustive and authoritative work of Kelman (1953). "On the basis of the records of geographic distribution certain generalizations can be made. The land areas of the world in which bacterial wilt occurs are bounded by the latitudes 45 degrees N and 45 degrees S. Within these widely-separated boundaries, the disease is most common in the regions characterized as follows: average rainfall above 40 inches per year; average length of growing season not less than six months; in the northern hemisphere mean temperature in January and July not lower than 50 degrees and 70 degrees F, respectively; in the southern hemisphere mean temperatures in January and July not lower than 70 degrees and 50 degrees F, respectively; and an annual temperature range not exceeding 40 degrees F."

Since Kelman completed his study evidence has been gathered to show that at the altitudinal and latitudinal limits of the distribution of bacterial wilt all of the records are of bacterial wilt (brown rot) on potato. The locations include southern Argentina and parts of western Europe (the Netherlands, France, Belgium) and northern Europe (Sweden) where the disease was introduced on potatoes for processing (Olsson, 1976). There are indications that the disease has become established in parts of western Europe but not in Sweden where the disease has probably been eradicated. A specific strain of *Ralstonia solanacearum* adapted to lower temperature environments referred to as race 3 (pathotype), biovar 2 (phenotype) and RFLP 26A (genotype) is associated with all of these disease outbreaks. The tuber-bearing *Solanum* species evolved in the high Andes of Bolivia and Peru and the low temperature strain of the pathogen is believed to have co-evolved with the potato, probably over a period of time measured in millennia. Moko bacterial wilt of banana is a tropical disease (Draft IRA p. 384). There are no confirmed reports in any sub-tropical location. There is no evidence of the evolution of strains of the Moko bacterial wilt pathogen adapted to low temperatures in sub-tropical locations, or reason to believe that such an evolution and adaptation analogous to what has happened on potato would readily occur.

In the United States where southern-grown tomato seedlings are shipped to wilt-free midwestern and northeastern states, there have been numerous reports over decades of bacterial wilt in fields planted with seedlings grown in Florida or Georgia (Vaughan, 1944; Kelman, 1953). There has been a similar experience in Canada where tomato transplants raised in Georgia served to introduce bacterial wilt in southwestern Ontario (Layne and McKeen,1967). Although there is some evidence of overwintering in the short term there is no published evidence of adaptation to or establishment of the pathogen in these cold environments.

Bacterial wilt is more severe at high temperature regimes and the resistance to the disease in tomato tends to breakdown with increase in daily maximum temperature. Krausz and Thurston (1975) found that elevated temperature (32 C) in controlled environment chambers significantly increased severity of bacterial wilt in two tomato lines bred for resistance to bacterial wilt. Similar results were obtained by Mew and Ho (1977). Resistant cultivars differed in response to high soil temperatures; some maintained a moderate resistance at 26, 30 and 32 C, and others were resistant at 26 C, and either moderately susceptible or susceptible (more than 60 % of plants wilted) at 32 C. The rate of onset of wilt was more rapid as temperatures increased in some resistant cultivars. Vaughan (1944) showed that from 70 F to as high as 110 F soil temperatures the rate of development of the disease increased with increase in temperature. In growth chamber experiments with potato French and De Lindo (1982) showed that resistant and susceptible potato cultivars showed increased susceptibility at warm (28/16 C day/night) compared with a cool temperature regime (20/8 C day/night).

Soil temperatures have a greater influence than air temperatures on the onset of bacterial wilt of tomato as shown by Gallegly and Walker (1949) in greenhouse experiments. When air temperatures were maintained at 22 C, disease development after inoculation was most rapid in plants growing in soil maintained at 30 and 36 C. When the soil temperature was maintained at 28 C and the plants were grown at air temperatures of 16, 20, 24 and 28 C, maximum disease indices were recorded in the series with the highest post-inculation air temperature.

Diurnal temperature regime has a strong influence on the development of bacterial wilt of peanut (Subandiyah and Hayward, 1990). Mean symptom scores were high at day/night regimes of 30/25 and 35/30 C and low at the regimes of 25/20 and 20/15 C. Disease progress curves showed that symptoms were generally less severe in winter than in summer suggesting an effect of day length.

The growing conditions in six Australian banana production areas and two in the Philippines are compared on pages 38 and 39 of the Draft IRA. Daily temperature maxima and minima show little variation throughout the year in both Lowland Davao and Highland Bukidnon, the Philippines. The climate in the Philippines enables uniform production of bananas throughout the year and also uniform conditions conducive for expression of Moko bacterial wilt.. By comparison conditions in North Queensland show greater variation; for about six months of the year conditions would be considered highly favourable for expression of Moko bacterial wilt because of the high daily maxima and minima temperatures, as well as conditions highly favourable for disease dissemination because of the very marked wet season. From the experience gained with other host/bacterial wilt interactions it can be concluded that plants will be more susceptible at higher temperature regimes; there will be more disease and more inoculum released to the soil from diseased root systems to infect neighbouring plants. The greater the seasonal variation in temperature maxima and minima (cf. Fig. 2, graph 3, for Northern New South Wales p. 38, Draft IRA) the greater the difference in conditions optimal for expression of Moko bacterial wilt. The disease is more likely to persist in soils where environmental conditions are uniformly favourable for disease expression and susceptible plants are always available.

(vi) Areas of low pest prevalence

According to the Draft IRA, p. 148, "Data provided by BPI (Philippines Dept. Agriculture, 2002a) indicate that the number of cases (infected mats) of Moko detected in routine control operations during 1998-2001 varied between 0.024 per hectare per 4 week period to 0.134 cases per hectare per 4 week period. The average incidence was 0.055 cases per hectare per 4 week period, and only 5% of the incidence values were greater than 0.1 cases per hectare per 4 week period. As these data were actually derived from weekly inspection data, it was assumed that the incidence of Moko in any single week is 0.1 cases per hectare divided by 4 weeks, or 0.025 cases per hectare per week."

Two feasible management measures have been proposed for Moko bacterial wilt: sourcing of fruit from areas of low pest prevalence (ALPP), and restricting the distribution of Philippine bananas in Australia (Draft IRA Executive Summary). The low pest prevalence (LPP) level for Moko in an approved ALPP would not exceed 0.005 cases (infected mats) per hectare per week, which is about 1 case per 4 hectares per year - i.e, no more than one in 6,800 infected plants per year. This is a conservative estimate given that the estimated number of cases per hectare per week is fivefold higher. No detailed information is available on the distribution of Moko bacterial wilt in southern Mindanao; accordingly it is not known whether the ALPP prescribed in the Draft IRA is attainable.

(vii). Estimation of the proportion of banana plants infected with the Moko bacterium which develop (symptomless) infected bunches.

In the Draft IRA p. 149 the data used for calculation of the likelihood that a tonne of harvested fruit will contain at least one symptomless infected fruit is from the book by Stover (1972) on diseases of banana and plantain. Stover does not provide a supporting reference for his data, suggesting that he was drawing on unpublished information. He gives a figure of 15% for the insect-transmitted SFR strain in Central American Cavendish bananas. The Draft IRA p. 149, second paragraph correctly states that this figure is likely to be lower for the B strains occurring in the Philippines which has a lesser potential for insect transmission. On this basis the Draft IRA makes a conservative estimation of the number of symptomless infected fruit. The authors of the Draft IRA have used the best data available to them in making their calculations.

In the work of Soguilon (2003) Cavendish banana plants were inoculated by spraying the cut surface of the peduncle at all hands out stage with Moko and Bugtok isolates. The plants remained externally symptomless 11 weeks after inoculation but vascular discolouration was observed on all of the inoculated plants at the end of the observation period. All of the inoculated plants were internally symptomatic. Soguilon reisolated the bacterial pathogen from discoloured vascular tissue in a relatively low proportion of the sampled fruit (two fingers per hand). This may be because of the dilution effect in her isolation procedure, but the low rate of isolation may also reflect the fact that vascular discolouration is a defence reaction by the plant perhaps analogous to the hypersensitive response; as a result viable bacteria may no longer be recoverable from some of the discoloured vascular tissue.`More work is required to investigate this possibility.

The Cavendish banana plants 11 weeks post-inculation were all without external symptoms but all were internally symptomatic. The cut peduncle tissue showed vascular tissue on inspection. However targeted inspection for internal symptoms of Moko infection was not an effective risk management measure. When the restricted likelihood was taken into account the restricted risk for Moko was found to be low, the same as for the unrestricted likelihood (Draft IRA p. 277) which exceeds Australia's ALOP.

(xiii) Potential for the distribution of Moko bacterial wilt from discarded banana waste

The Probability of Distribution of Moko bacterial wilt from imported bananas is considered on pp. 151-155 of the Draft IRA. A step-wise, highly conservative assessment has been made of the likelihood of survival and spread of the disease from banana waste. The Draft IRA rightly stresses that the most probable means of dispersal would be by rain-splash or movement of contaminated soil by flood water. The involvement of insects is highly improbable. The insects involved in transmission of infection within a cooking banana plantation forage around the inflorescence where they are probably attracted by the sugar content of floral nectaries. There is no evidence of which I am aware that insects are attracted to bacterial ooze, neither is there any evidence of the transmission of infection from banana waste. Stover (1972) states: "Undoubtedly, considerable insect transmission occurs to freshly cut sucker surfaces following pruning" but this is in the context of a plantation where there is Moko bacterial wilt and high numbers of the insects involved in disease transmission. For transmission to occur sufficient numbers of the pathogen must come into contact with the exposed vascular tissue of a susceptible host. The Draft IRA has concluded (p. 154) that the likelihood of movement of contaminated soil or rain-splash , given the distribution of symptomless infected bananas to an area where bananas are farmed commercially, and the discarding of the organism in banana waste, is low. This estimation must be considered highly conservative. Banana waste has a high sugar content, capable of rapid fermentation by saprophytes in the environment. The numbers of the pathogen will be low in symptomless fruit and it is very unlikely that *R. solanacearum* could compete in such an environment.

Australia may adopt the second risk management option in which Philippine bananas are restricted in distribution to those parts of Australia where commercial banana production does not occur. Any person disposing of a banana fruit originating in the Philippines in any part of the area of exclusion such as the Tully valley or anywhere else would be guilty of a criminal offence and liable to up to 10 years imprisonment (Draft IRA p.)

(ix) Bibliography

Buddenhagen, I.W. 1961. Bacterial wilt of bananas: history and known distribution. Tropical Agriculture (Trinidad) 38: 107-121.

Buddenhagen,I.W. and Elsasser, T.A. 1962 An insect-spread wilt epiphytotic of bluggoe banana. Nature, London 194: 164-165.

Coelho Netto, R.A. and Nutter, F.W. 2002 Use of GPS and GIS technologies to map the prevalence of Moko disease of bananas in the Amazonas region of Brazil. In, Proceedings of the Third International Bacterial Wilt Symposium, 4-8 February, 2002, White River, Republic of South Africa. 60 pp.

Fegan, M. 2002 Study of Moko disease of banana caused by Ralstonia (Pseudomonas) solanacearum race 2: a draft report to the Australian Quarantine and Inspection Service. The University of Queensland, St. Lucia, Australia. 31 pp.

French, E.R. and De Lindo, L. 1982 Resistance to Pseudomonas solanacearum in potato: specificity and temperature sensitivity. Phytopathology 72: 1408-1412

French, E.R. and Sequeira, L. 1968 Marchitez bacterial o Moko del platano en el Peru. Fitopatologia 3: 27-38 (English translation provided by AFFA)

Gallegly, M.E. and Walker, J.C. 1949 Relation of environmental factors to bacterial wilt of tomatoes. Phytopathology 39: 936-946

Hunt, P. 1987 Current strategies for Moko control in Grenada: technical and logistical constraints. In, Proceedings of the Conference on Improving Citrus and Banana Production in the Caribbean through Phytosanitation, 2-5 December 1986, St. Lucia WI. CTA/CARDI (pubs.). Wageningen, The Netherlands. pp 121-129

Kelman, A. 1953 The bacterial wilt caused by Pseudomonas solanacearum. North Carolina Agricultural Experiment Station Technical Bulletin 99. pp.194

Krausz, J.P. and Thurston, H.D. 1975 Breakdown of resistance to Pseudomonas solanacearum in tomato. Phytopathology 65: 1272-1274

Layne, R.E.C. and McKeen, C.D. 1967 Southern bacterial wilt of field tomatoes in southwestern Ontario. Canadian Plant Disease Survey 47: 94-98

Mew, T.W. and Ho, W.C. 1977 Effect of soil temperature on resistance of tomato cultivars to bacterial wilt. Phytopathology 67: 909-911

Molina, G.C. 1996 Integrated management of 'Tibaglon', a bacterial fruit rot disease of cooking bananas under farmer's field. Philippine Phytopathology 32: 83-91

Olsson, K. 1976 Experience of brown rot caused by Pseudomonas solanacearum (Smith) Smith in Sweden. EPPO Bulletin/Bulletin OEPP 6: 199-207

Phelps, R.H. 1987 The status of Moko disease of banana in the Caribbean. In, Proceedings of the Conference on Improving Citrus and Banana Production in the Caribbean through Phytosanitation, 2-5 December 1986 St. Lucia WI, CTA/CARDI (pubs). Wageningen, The Netherlands, pp. 100-107

Soguilon, C.E. 2003 Asymptomatic or sub-clinical infection of Cavendish banana fruit. Unpublished report, Philippines Bureau of Plant Industry, DNCRDC, Bago Oshiro, Davao City, The Philippines.

Soguilon, C.E., Magnaye, L.V. and Natural, M.P. 1994 Bugtok disease of cooking banana in the Philippines. ACIAR Bacterial Wilt Newsletter 10: 5-7

Stover, R.H. 1972 In, Banana, Plantain and Abaca Diseases. Commonwealth Agricultural Bureau (CAB), Commonwealth Mycological Institute, Kew, Surrey, UK, pp. 189-203

Subandiyah, S. and Hayward, A.C. 1990 Virulence screening and the influence of temperature regime on the interaction of some isolates of Pseudomonas solanacearum E.F.Smith with peanut (Arachis hypogaea L.). In, Proceedings 3rd International Conference on Plant Protection in the Tropics, March 20-23, 1990, Genting Highlands, Pahang, Malaysia pp.96-101

Vaughan, E. 1944 Bacterial wilt of tomato caused by Phytomonas solanacearum. Phytopathology 34: 443-458

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March 24, 2004