CSIRO PUBLISHING
Australasian Plant Pathology, 2010, **39**, 170–175 www.publish.csiro.au/journals/app

Phosphonate applied by trunk injection controls stem canker and decreases *Phytophthora* pod rot (black pod) incidence in cocoa in Sulawesi

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Abstract. Stem canker and *Phytophthora* pod rot (PPR) or black pod caused by *Phytophthora palmivora* are serious diseases of cocoa (*Theobroma cacao* L.) in Sulawesi, Indonesia, causing high yield losses for smallholders, possibly exceeded only by losses due to the cocoa pod borer (CPB), *Conopomorpha cramerella*. Potassium phosphonate (phosphite) applied by trunk injection has been demonstrated to effectively control canker and PPR in Papua New Guinea. The method was tested in a field trial in south-east Sulawesi. Fifty trees were injected with phosphonate, 50 with water and 50 were left untreated. Phosphonate was applied at a rate of ~16 g active ingredient per tree per year, depending on the size of each tree. Trees were evaluated each month for canker severity, for PPR incidence and for CPB incidence and severity. From 4 months after the initial injection, trees treated with phosphonate had negligible levels of canker. Over a 2.5-year period, phosphonate significantly decreased PPR incidence. Cycles of PPR infection occurred in the wet season with PPR incidence fluctuating from less than 30% to greater than 75%. These fluctuations might have been due to variations in rainfall causing natural cycles of sporulation and infection. CPB incidence did not differ significantly between treatments. Since trunk injection of phosphonate effectively controls stem canker and decreases PPR in the long term it provides a valuable option for the management of these diseases for cocoa smallholders.

Introduction

Two of the most serious diseases affecting cocoa (*Theobroma cacao* L.) in Sulawesi, Indonesia, are stem canker and *Phytophthora* pod rot (PPR or black pod) caused by the oömycete, *Phytophthora palmivora* (Guest 2007). Yield losses to PPR in the wet season are commonly as high as 80% (A. Purwantara, unpubl. data). Losses due to PPR are compounded by the coincidence of the peak in PPR incidence with the peak cocoa harvest late in the wet season (May/June).

Trunk injection of phosphorous acid, as its partially neutralised potassium salt (potassium phosphonate), has been tested for control of *Phytophthora* pathogens on several tree crops, initially avocado infected with *P. cinnamomi* causing root rot (Pegg *et al*. 1985). Phosphonate is most active against oömycetes (Drenth and Guest 2004) and appears to induce host resistance responses at relatively low concentrations (Dunstan *et al*. 1990; Grant *et al*. 1990; Guest *et al*. 1995; Daniel and Guest 2006). It has low mammalian toxicity and is non-persistent in the environment (Guest *et al*. 1995; Drenth and Guest 2004).

Phosphonate, applied by trunk injection, was first tested on cocoa in Papua New Guinea, proving efficacious for control of canker and PPR caused by *P. palmivora* (Anderson and Guest 1990; Holderness 1990, 1992; Guest *et al*. 1994). Later, the method was applied to cocoa in Ghana obtaining effective control of diseases caused either by *P. palmivora* or *P. megakarya* (Opoku *et al*. 1998, 2007). However, this method has not been applied on cocoa in Indonesia. Its practicability, using only simple equipment, and demonstrated long-term cost effectiveness could particularly suit Indonesian smallholders. To test the method in Sulawesi, a 4.5-year trunk injection trial was established on a working farm in south-east Sulawesi. Phosphonate was applied by trunk injection once each year, and the incidence of PPR and severity of stem canker monitored. For a portion of this time, the incidence and severity of cocoa pod borer (CPB) was also assessed. The trial provided a demonstration to local farmers of a potentially simple and cost-effective way to reduce stem canker and PPR.

Materials and methods

Establishment of the trunk injection trial

The trial was established on a cocoa smallholding in the province of south-east Sulawesi (Kolaka District, Ladongi subdistrict).

 10.1071/AP09078 0815-3191/10/020170

A plot of 100 trees, all planted from hybrid seed in 1992 but varying in size, were labelled and injected in alternate order with either water (as a control) or phosphonate, giving 50 trees for each treatment. The first injection was in June 2002, followed by repeated injections in December 2002, October 2003, December 2004, December 2005 and October 2006. Holes (25 mm deep) were drilled with a hand drill using either a 6.0 or 6.5-mm-diameter drill bit. The holes were positioned ~0.5 m above the ground and angled slightly downwards when drilled towards the centre of the trunk. Injectors (20 mL, spring-loaded, Chemjet, Bongaree, Qld, Australia) containing 20 mL water or phosphonate solution were screwed into the holes. Approximately 30 min was required to empty each injector and the injection of all trees was completed in 1 day. Each tree was injected with water or 20% potassium phosphonate solution prepared as described by Guest *et al*. (1994). Ten L of 40% phosphorous acid solution was added slowly to 10 L 40% potassium hydroxide solution and adjusted to a final pH of 6.0. Therefore, each 20-mL injection of phosphonate solution delivered 4 g of potassium phosphonate. Since the canopy diameter of most of the trees in the trial was ~2 m, over 90% of the trees were injected with 40 mL using two injectors, each with 20 mL water or phosphonate, inserted opposite each other on the trunk (i.e. ~8 g potassium phosphonate was injected per tree). Larger trees (~3-m canopy diameter) were given three injections (a total of 60 mL or 12 g potassium phosphonate per tree). Injection holes were plugged with engine grease. Six months later the trees were reinjected at the same rates. Therefore, the total amount of potassium phosphonate injected per tree in 2002 was 16 g for the majority of trees and 24 g for the few, larger, trees. Thereafter, trees were injected once per year with the same volumes as for the above treatments but using a commercially prepared 40% potassium phosphonate solution (Phosacid 400, Humibox (M) SDN.BHD, Selangkor Darul Ehsan, Malaysia). Hence, trees with a canopy diameter of ~2 m (over 90% of the trees) still received a total of 16 g potassium phosphonate annually but this was applied only once per year. Similarly, the larger trees (with a canopy diameter of ~3 m) were injected with 24 g once per year.

A further control treatment of 50 untreated (non-injected) trees was included in the trial in April 2004 to provide data on the background incidence of PPR, stem canker and CPB under a farm management regime typical of the area, and to assess the effect of the injection method itself on disease incidence and severity. These trees were in a block adjacent to the above block of 100 trees and were assessed monthly in the same way as for the trees injected with water or phosphonate.

Evaluation of stem canker, PPR and CPB

The severity of stem canker on each tree was assessed every month using the following scoring system: 0, no canker; 1, <10 cm^2 active canker; 2, 10–50 cm^2 active canker; 3, 50–100 cm^2 active canker; 4, >100 cm^2 active canker (or recent tree death associated with canker). The area of canker was assessed based on the canker visible as sunken tissue on the intact bark and the margins as shown by scraping off the surface of the bark with a knife. An average severity score for each month was obtained from the mean score of the trees assessed for each treatment.

Ripe pods and immature infected pods were harvested monthly and the PPR-infected pods counted. Harvested ripe pods were also assessed for CPB infestation. Ripe pods were split lengthways and CPB-infested pods, as shown by the discolouration and aggregation of beans and mucilage, were counted. Pods in which 50% or more of the beans had been affected by CPB were considered to be severely infested.

Statistical comparisons of PPR, CPB and canker incidences

For each treatment (water-injected, phosphonate-injected and untreated), cumulative PPR and CPB incidences (from April 2004 to December 2006) were calculated for each tree. Hence, the number of pods harvested and the numbers infected/infested with PPR or CPB from each tree were totalled for this period and the percent (cumulative) incidence calculated. To test the effect of the treatments on cumulative incidences, each tree was considered to be a separate replicate. Statistical tests were conducted using SPSS ver. 11 (SPSS Inc., Chicago, IL, US). For both PPR and CPB incidences, the Levine test gave no significant differences between variances indicating that the variances were homogeneous. The treatments were thus compared by one-way ANOVA followed by the Bonferroni *post hoc* test. Cumulative canker scores were obtained by summing the monthly scores for each tree for the period April 2004 to December 2006. As for the cumulative PPR and CPB incidences, one-way ANOVA was used to compare treatments, but since the Levine test indicated non-homogeneity among the variances the Games-Howell *post hoc* test was used.

Rainfall data

From 2003 onwards, rainfall data, indicating total rainfall (mm) each month, were recorded by Mars Inc. at a weather station situated nearby the trial site in Ladongi. Before 2003, rainfall data records, obtained from a neighbouring subdistrict, Mowewe, were supplied by the provincial government. Correlations between total rainfall per month and incidence of PPR were conducted on SPSS ver. 11 to give Pearson correlation coefficients (r).

Results

Effect of phosphonate treatment on stem canker

In the controls (water-injected and untreated trees), relatively high peaks of canker severity and incidence occurred in 2002, but from 2003 onwards incidence varied from 10 to 36%, and average severity scores from 0.2 to 1.0 (Figs 1–4). Fig. 3 indicates that canker incidence was similar in both of the control treatments varying from 10 to 36% after 2003. The average severity scores for water-injected and untreated trees (Fig. 4) indicate that the canker lesion area for most control trees remained under 10 cm^2.

Figure 1 shows the incidence of stem canker in the water- and phosphonate-injected trees from mid 2002 to the end of 2006. The average severity in water- and phosphonate-injected trees that were initially infected with stem canker is shown in Fig. 2 for the same period. A clear decrease in stem canker incidence and average severity resulting from the treatment with phosphonate was evident from about 4 months after the initial treatment. Incidence decreased to negligible levels in phosphonate-injected trees, indicating cankers were decreased substantially

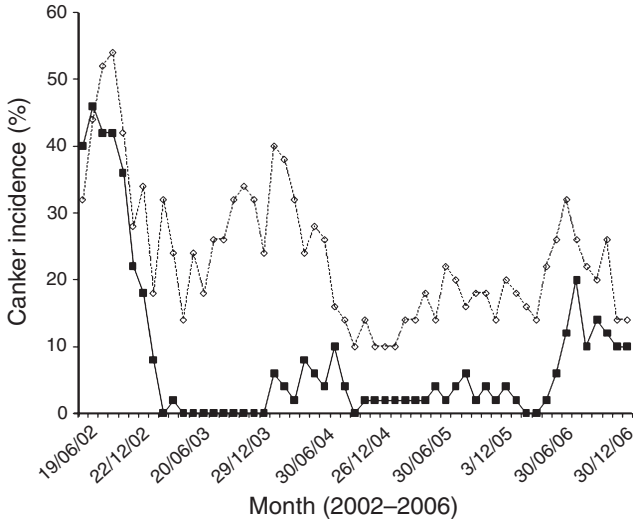

Fig. 1. Canker incidence (%) in phosphonate-injected (solid line and black squares) and water-injected (broken line, open diamonds) trees assessed from 2002 to 2006. Trees were injected in June 2002, December 2002 and then every year until the end of 2006.

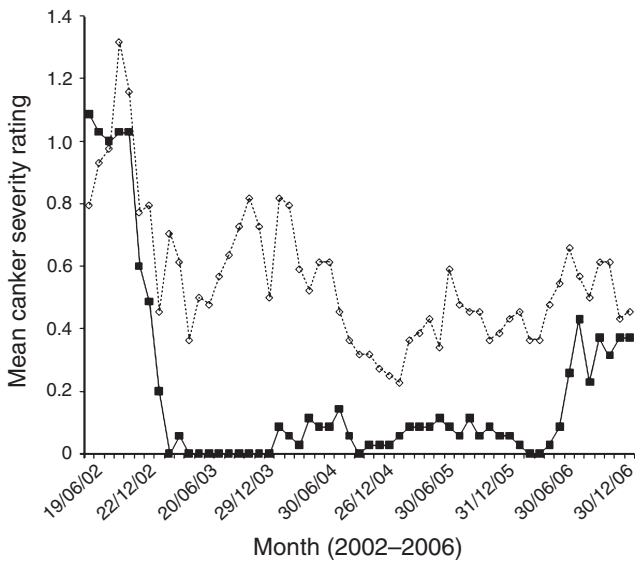

Fig. 2. Mean canker scores for phosphonate-injected (solid line and black squares) and water-injected (broken line, open diamonds) trees assessed from 2002 to 2006. Only trees that initially had canker infections are included. At the time of the first injection, there were 44 canker-affected trees in the water-injected treatment and 35 trees in the phosphonate-injected treatment. The scores refer to categories designating the area of canker lesions (see Materials and methods).

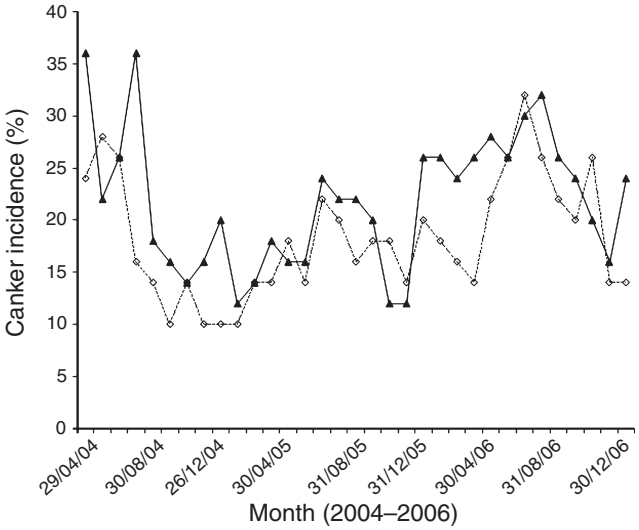

Fig. 3. Incidence of stem canker (%) in untreated or non-injected (solid line and black triangles) and water-injected (broken line, open diamonds) trees assessed from 2004 to 2006.

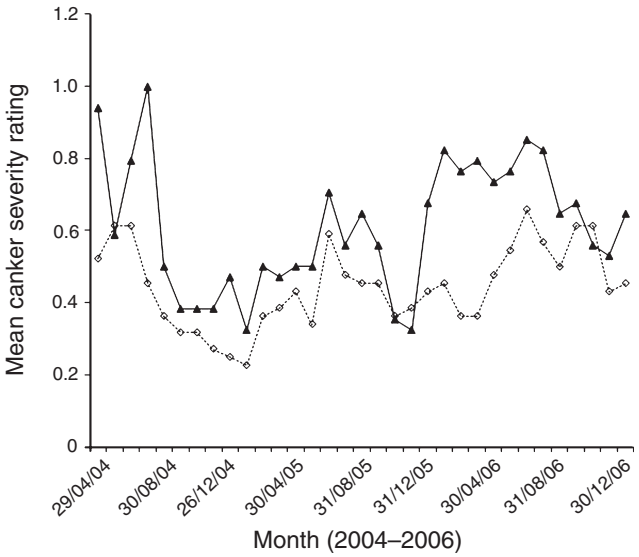

Fig. 4. Mean canker scores for untreated (solid lines and black triangles) and water-injected (broken line, open diamond symbols) trees assessed from 2004 to 2006. Only trees that initially had canker infections are included. At the time of the first injection, 44 trees in the water-injected treatment, and 34 untreated trees, were affected by canker. The scores refer to categories designating the area of canker lesions (see Materials and methods).

by the phosphonate treatment. The total cumulative canker scores (calculated by adding the scores for each month from April 2004 to December 2006) were significantly ($P < 0.05$) less in phosphonate-treated trees than in both sets of control trees, which were not significantly different (Table 1).

The similar levels of canker incidence and severity in the water-injected and the untreated trees indicate that the injection procedure itself had little or no effect on canker (Figs 3, 4). In both the control treatments (untreated and water-injected trees), a seasonal change in the incidence and average severity of canker was evident. From 2004 to 2006, in both control treatments, the highest average canker severities occurred in June/July, with scores of 0.6–1.0, and the lowest in November/December, with scores of 0.2–0.4. Canker incidence followed a similar seasonal fluctuation to severity (Fig. 3).

Table 1. Mean cumulative canker severity scores

For each tree, canker scores obtained each month from April 2004 to December 2006 were summed and the means (±s.e.) of the cumulative data obtained for each treatment. Means followed by the same letter are not significantly ($P < 0.05$) different

Treatment	Mean total canker score
Untreated	13.72 ± 3.15a
Water-injected	12.92 ± 3.82a
Phosphonate-injected	2.80 ± 1.17b

Effect of phosphonate on the incidence of PPR

In both water- and phosphonate-injected trees for the period of the trial from 2002 to 2006 a seasonal fluctuation was observed in PPR incidence with a lower incidence in the phosphonate-treated trees (Fig. 5). Considering the cumulative data from April 2004 to December 2006, the incidence of PPR was significantly ($P < 0.05$) lower in the phosphonate-injected than in the water-injected and untreated controls (Table 2). The incidence of PPR did not differ between the water-injected and untreated trees (Table 2, Fig. 6),

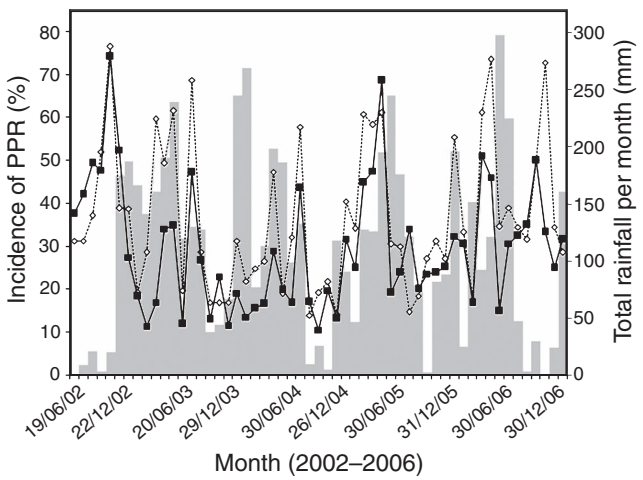

Fig. 5. Mean *Phytophthora* pod rot (black pod) incidence (%) in ripe pods for trees injected with phosphonate (solid line and black squares) and injected with water (broken line, open diamonds) assessed each month from 2002 until 2006 (left axis). Data shown are the mean incidence each month of the 50 trees in each treatment. Rainfall totals per month (mm), recorded at a local weather station (see Materials and methods), are indicated by the shaded bars (right axis).

Table 2. Mean cumulative incidences of *Phytophthora* pod rot (PPR), total cocoa pod borer (CPB) and severe CPB in ripe pods from April 2004 to December 2006

Mean incidences were calculated from the total number of ripe pods and infested/infected pods harvested from each tree in the 2.5 years. Each treatment had 50 trees (replicates). Means (±s.e.) within columns followed by the same letter are not significantly ($P < 0.05$) different

Treatment	Mean PPR (%)	Mean total CPB (%)	Mean severe CPB (%)
Untreated	43.4 ± 1.9a	69.8 ± 2.0a	25.7 ± 1.9a
Water-injected	40.2 ± 2.1a	69.0 ± 1.9a	23.8 ± 1.6a
Phosphonate-injected	26.3 ± 1.8b	64.0 ± 1.7a	19.9 ± 1.6a

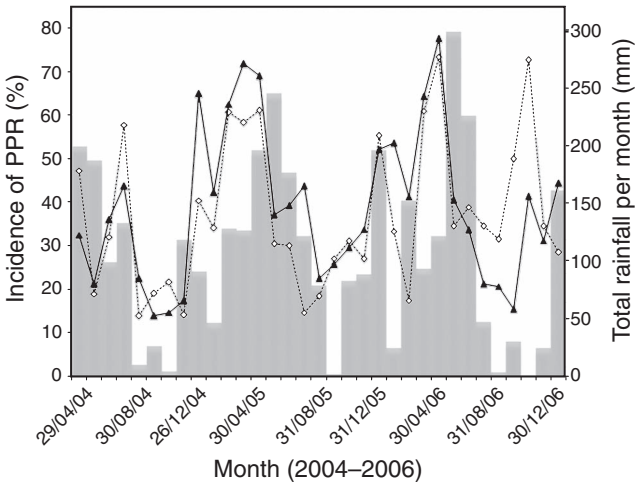

Fig. 6. Mean *Phytophthora* pod rot (black pod) incidence (%) in ripe pods for untreated (non-injected) trees (solid line and black triangles) and trees injected with water and (broken line and open diamonds) assessed each month from 2002 until 2006 (left axis). Data shown are the mean incidences for each month of the 50 trees in each treatment. Rainfall totals per month (mm), recorded at a local weather station (see Materials and methods), are indicated by the shaded bars (right axis).

indicating that the injection method itself did not influence the incidence of PPR.

Data for PPR incidence in the water-injected (control) trees indicate that during the dry season (around August/September) of each year from 2003 PPR incidence decreased to lows of 14–17% (Fig. 5). Generally low levels of PPR incidence of under 30% were maintained for about 4 months during the dry period of each year. The highest incidences of PPR occurred during the wet season with an early peak at the beginning of the rains (around December) and high levels also occurring later in the wet season, usually between March and July. Incidence of PPR in the untreated (control) trees, evaluated from April 2004 to December 2006, was significantly ($P < 0.05$) correlated with total monthly rainfall in the same period (Pearson correlation coefficient, $r = 0.325$*). However, no significant correlation occurred between total monthly rainfall and PPR incidence in either the water-injected control ($r = 0.265$) or in the phosphonate-treated trees ($r = –0.141$), in the 4.5 years of the experiment from June 2002.

Cycles of infection, apparently independent of the seasons, are evident in these data. Generally, increases in PPR incidence during the wet season were followed by sharp decreases. This is evident in both the water- and phosphonate-injected trees where high levels of PPR incidence (ranging from 55 to 78%) occurring in October 2002, March–May 2003, April 2004, April 2005, January 2006 and April 2006 were followed in each case by a sudden decrease in the following month to levels below 30% and even below 20% (Fig. 5). Similar decreases following peaks in PPR incidence were observed in the untreated trees (Fig. 6).

Effect of phosphonate on CPB infestation

Figure 7 shows the changes in CPB incidence in the 2.5-year period for each treatment and the total number of pods harvested from the 150 trees in the trial. CPB incidence decreased during the

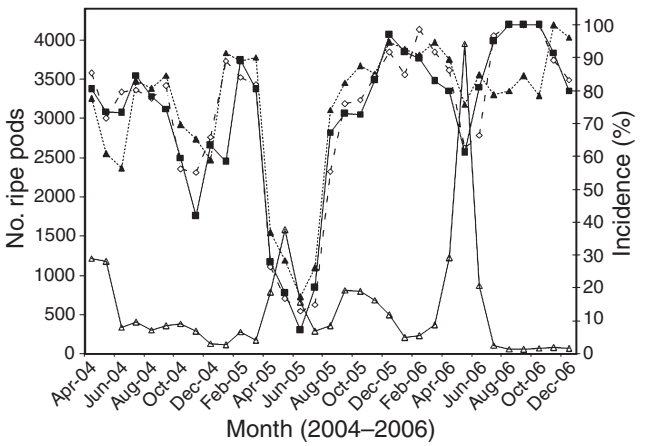

Fig. 7. The total number of ripe pods (left axis, solid line, open triangles) harvested each month from 2004 to 2006 from the 150 trees in the trunk injection trial and the mean % cocoa pod borer incidence (right axis) for each treatment in which groups of 50 trees were either untreated (broken line, black triangles), water-injected (dash line, open diamonds) or phosphonate-injected (solid line, black squares).

mid-year peak harvests, the decrease in mid 2005 being much greater than in mid 2006. A dilution effect caused by the increase in the number of pods could account for the decrease in the percent infestation by CPB during the main harvest period. Phosphonate had no significant effect on cumulative CPB incidence (Table 2).

Discussion

Canker incidence and average severity varied with the seasons as evident in the untreated and water-injected controls. From 2004 to 2006, both control treatments had peaks in canker severity scores (0.6–1.0) in June (at the end of the wet season in south-east Sulawesi), and the lowest scores (0.2–0.35) at the end of the year just before the rains began again. The changes in average severity (assessed as the mean canker score on the trees in each treatment) could be largely explained by fluctuations in incidence so that decreases in canker incidence in the drier periods (Figs 1, 3) caused a concomitant decrease in average severity (Figs 2, 4). Hence, during the dry season it appears that cankers are progressively lost until canker incidence reaches its lowest annual rate at the end of the dry season and that wetter conditions reverse this process with new cankers being formed. The apparent relation between canker development and rainfall is supported by the lower average incidence (and average severity) in 2005, which was also a year during the study period in which southern Sulawesi received a relatively low total amount of rainfall (http://www.weatheronline.co.uk accessed November 2008).

Phosphonate applied by trunk injection to canker-affected trees decreased or eliminated cankers, with long-term effects. Apart from a slight increase in canker incidence and severity at the end of the evaluation period, the phosphonate treatment maintained canker at zero or near zero levels. The reason for the increase observed is uncertain. Possibly, the application or uptake of phosphonate in the final injections (in the last year) was incomplete due to technical or other reasons. However, the data from the preceding years indicate clearly that the earlier injections were very effective in reducing cankers. The results provided a clear confirmation of the effectiveness of phosphonate trunk injection in reducing *Phytophthora* stem canker, as found previously in Papua New Guinea (Anderson and Guest 1990; Holderness 1992; Guest *et al*. 1994). Annual phosphonate injections providing 16 g active ingredient (a.i.) per tree resulted in decreases in canker incidence to negligible levels, consistent with the findings of Guest *et al*. (1994) who found injections providing an annual total of 15 g a.i. per tree gave optimum control of canker. Apart from some internal browning at the sites of injection, the phosphonate treatments in Sulawesi did not cause any visible phytotoxicity symptoms, consistent with Anderson and Guest (1990) that no phytotoxic reaction occurred to phosphonate even in trees where 16 g a.i. per tree was applied every 6 months.

As would be expected, PPR incidence decreased with the arrival of the dry season, with generally lower levels being maintained until the following wet season when it increased again sharply. Changes in PPR incidence also occurred during the wet season and were characterised by several peaks and troughs. The reason for the large decreases in PPR incidence during the wet season is uncertain. Possibly, they reflect the rainfall patterns within the wet period: drier times in the wet season might have caused the sharp declines in PPR incidence observed. The positive correlation found between PPR incidence in the untreated control trees and total monthly rainfall indicates a degree of dependence of PPR incidence on the amount of rainfall. However, the large decreases in PPR incidence observed within the wet season might also have been partly a result of a natural cycle of infection by the pathogen, with an initial infection event coinciding with the beginning of the rains followed by sporulation initiating a second round of infection. Therefore, the low levels of PPR incidence observed in the month following the first infection event of the new wet season might represent a lag period during which the pathogen undergoes sporulation and initiates a second round of infection. An alternative explanation for these fluctuations in the wet season is that they resulted from the experimental method, which involved regular complete removal of infected pods. A study conducted in Cameroon demonstrated that regular removal of pods infected with *P. megakarya* decreased infection rates (Ndoumbe-Nkeng *et al*. 2004). In the phosphonate injection trial at Ladongi, the regular harvesting of mature pods probably removed a large proportion of the *Phytophthora* inoculum source from the canopy, since the disease is most common and extensive on ripe pods.

Cumulative data taken over 2.5 years indicated that trunk injection of phosphonate significantly decreased PPR incidence over this period. This decrease might be accounted for (at least partly) by the eradication of stem canker by the phosphonate treatment and therefore removal of a source of inoculum for new PPR infections. Phosphonate probably also had a direct effect on the infection of pods by the pathogen (Anderson *et al*. 1989). Nevertheless, the very similar cycles of infection in the phosphonate-treated and control trees indicated that other sources (including pods on which the pathogen was sporulating) provide inoculum for new infections and that the eradication of canker by phosphonate injection did not greatly affect the cycle of pod infections occurring during the wet seasons. In Indonesia,

possible sources of inoculum, other than stem cankers, include infected pods, flower cushions and soil (A. Purwantara, unpubl. data). Insects may also transmit *Phytophthora*. In West Africa (Maddison 1981; Gregory *et al*. 1984) and Papua New Guinea (Konam 1999; Konam and Guest 2004), insects such as ants and beetles have been shown to transmit inoculum and initiate PPR infections. In Sulawesi, tent-building ant species are common on cocoa farms and holes bored by insects, probably coleopterans, commonly occur in pods infected with *P. palmivora* (D. I. Guest, pers. obs.).

Annual trunk injection of phosphonate would benefit smallholders by reducing the incidence of PPR and especially by controlling stem canker, improving the performance of cocoa trees and reducing tree death due to this disease. Although trunk injection is a low cost method of treating trees, particularly if a commercial supply is locally available, it requires a supply of special spring-loaded syringes and a drill (preferably a cordless electric drill). At present in Australia, potassium phosphonate products (in liquid form) cost ~A$7–10/L. Since commercial phosphonate products are not currently available in Indonesia they need to be imported, which increases the cost for farmers. In the future, potassium phosphonate products may become more readily available thus reducing the cost. Future studies could investigate the application of phosphonate as a bark paint or spray, using even cheaper and more readily available methods of application. Further studies are also needed to assess whether higher dosages of phosphonate could provide better control of PPR.

While phosphonate trunk injections on their own reduce losses due to *P. palmivora*, they are more effective when used as a component of an integrated disease management strategy. Such a strategy could include: (i) the selection of less susceptible genotypes; (ii) regular pruning of cocoa and shade trees and weeding to improve light penetration and reduce humidity in the cocoa canopy; (iii) sanitation involving regular harvesting of ripe pods and removal of all diseased pods and disposal of pod husks by burying or composting to reduce the inoculum load; (iv) measures to reduce the populations of insect vectors of *Phytophthora*; and (v) appropriate fertilisation to improve general tree health and growth (Konam *et al*. 2008).

Acknowledgements

This work was conducted as part of a project (CP/2000/105) funded by the Australian Centre for International Agricultural Research. The authors would like to thank Haji Sudirman who kindly provided a site for this experiment on his farm in Ladongi, south-east Sulawesi.

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Manuscript received 7 May 2009, accepted 27 October 2009