

Sclerotinia rot losses in processing tomatoes grown under centre pivot irrigation in central Brazil

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Sclerotinia rot caused by *Sclerotinia sclerotiorum* is one of the most important diseases of processing tomatoes in Central Brazil. Yield losses in tomato cultivars (cv.) IPA-5 were assessed in mature plants from 1995 to 1997, and related to different disease intensities, in a naturally infested area irrigated by centre pivot. Over the 3 years, there were no differences (Tukey at 5%) in fruit numbers between plants without symptoms (NS) and with intermediate symptoms (IS), which yielded higher numbers than plants with severe symptoms (SS). The greatest reduction in fruit number was 56.8% in 1997. Significant differences were observed in fruit weight and size among NS, IS and SS plants in 1995 and 1997. In 1996, NS and IS plants were similar, but different from SS, which yielded significantly less. Weight and size reductions in SS plants reached 84.3% and 62.0%, respectively, in 1997. In 1996 and 1997, yield losses related to time of symptom appearance and physiological age were also assessed. Significant correlations were found ($P < 0.01$), with nearly total losses observed when plants were infected from early to mid bloom, as opposed to plants infected near harvest, which had lower disease incidence and produced economically acceptable yields. Quadratic and exponential models best fitted the relationship between yield and time of symptom appearance, and yield vs physiological age could be explained by logistic and Gompertz functions.

Keywords: disease intensity, epidemic development, *Sclerotinia sclerotiorum*, tomatoes, yield loss

Introduction

Processing tomatoes are an important agricultural commodity in Brazil, which is now one of the leading tomato producers in the world (FAO, 1996; Sullivan, 1996). Sclerotinia rot, caused by *Sclerotinia sclerotiorum*, is one of the most important diseases affecting the tomato crop in central Brazil, where most fields are cultivated under centre pivot irrigation (Lopes & Santos, 1994). Negligible in the early 1980s (Nasser & Hall, 1997), sclerotinia rot can now be found in more than 50% of the irrigated fields in this region (Mitsueda & Charchar, 1992). During the winter, the average temperature is around 20°C (Ferraz, 1987; Nasser *et al.*, 1990; Morais, 1994) and beans, soybeans, peas and tomatoes are the main crops.

Plant infection occurs either by myceliogenic germination of sclerotia or by ascospores produced in apothecia from a sclerotium (Purdy, 1979). In central Brazil, myceliogenic germination of sclerotia provides the main source of infection on processing tomato crops,

resulting in rotting of aerial parts of the plant in contact with the soil (Lopes & Santos, 1994).

Successive cropping of susceptible species, particularly tomato monocultures, has resulted in direct and indirect economic losses, caused by the yearly increase of sclerotinia rot. Despite their obvious importance, losses from this rot have not yet been quantified for tomatoes in Brazil.

Losses to diseases caused by *S. sclerotiorum* have been quantified for various crops in different countries (Kerr *et al.*, 1978; Morral *et al.*, 1984; Holley & Nelson, 1986; Tu, 1989; Conn & Tewari, 1990). The objective of this work was to quantify losses of processing tomatoes grown under centre pivot irrigation in central Brazil, taking into account disease intensity on the mature plant and the developmental stage of the plant, based on chronological and physiological age. The term disease intensity is used here as synonymous with disease severity (Zadoks & Schein, 1979; Nutter *et al.*, 1993).

Materials and methods

Field trials and crop management

Experiments were conducted from 1995 to 1997 at the

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Accepted 28 June 1999.

National Vegetable Crop Research Center, EMPRAPA Hortaliças, Brasília, DF. Processing tomato cultivar (cv.) IPA-5, the most popular in Brazil, was direct seeded yearly in April, under centre pivot irrigation, in an area naturally infested with *S. sclerotiorum*. The tomato area was part of a crop rotation study started in 1991 (Silva *et al.*, 1997). Processing IPA-5 tomatoes have an indeterminate growth, and require 120–125 days from sowing to harvest. The crop was managed according to local recommendations (Silva *et al.*, 1994). Airborne pathogens were successfully controlled by fungicide sprays, and no other soilborne pathogen was recorded in the experimental field during the experiment.

Relationship of yield losses with disease intensity near harvest

In each year, tomato plants were marked 10 days before harvesting according to three levels of disease intensity: apparently healthy (NS), where no symptoms were observed; intermediate (IS), with distinct symptoms, but plants still alive and with good fruit set; and severe (SS), where most of the stems were destroyed by the disease and fruit set was poor. For each intensity level, five plants were collected randomly, from each of six replications in a complete randomized block design, where blocks represented tomato plots under different crop rotations.

Relationship of yield losses and time of symptom appearance, based on chronological age of tomato plants

To establish the relationship between yield losses and time of symptom appearance (TSA), plants were marked at different developmental stages in which disease appeared in each of six replications in 1996 and 1997. At biweekly intervals, from appearance of the first diseased plant, five plants with initial symptoms were randomly marked with a wire stake and a coloured strip, in order to obtain a gradient of disease intensities at harvest. All other plants with initial symptoms were also marked at each evaluation time, to ensure that only initial symptoms occurring during the next interval would be counted. Care was taken to mark only plants with symptoms typical of infections from myceliogenic germination. The blocks were the same as for the preceding experiment. In both years the first diseased plants were observed in the first week of July, approximately 60 days after sowing (DAS), when flowering had just begun and before adjacent rows became close. Therefore five evaluations were conducted at 60, 75, 90, 105 and 120 DAS, corresponding to TSA 1, 2, 3, 4 and 5.

Relationship of yield losses and time of symptom appearance, based on physiological age of tomato plants

Fruit number, weight and size, assessed at different chronological ages, were transformed into percentage

values, as the proportion of those characters for plants with no disease symptoms. Tomato physiological ages were calculated according to minimum temperatures for different crop stages, given by Geisenberg & Stewart (1986), using daily temperatures obtained at Embrapa Hortaliças weather station, located nearby.

The number, total weight and size (g per fruit) of fruits on plants at the three disease intensities were compared by analysis of variance and the means were compared by the Tukey test ($P < 0.05$). The relationship between these fruit measurements and TSA in chronological age was examined by Pearson's correlation and regression analysis (Snedecor & Cochran, 1981) and according to mathematical models proposed by Zadoks & Schein (1979). Models were fitted according to higher coefficient of determination, lower sum of squares of error, and a random residuals distribution in a scatterplot. All statistical analyses were performed with SAS software (SAS Institute, 1988), and carried out with *glm*, *corr* and *reg* procedures. As statistically indicated, the variance in ANOVA was stabilized when the data were transformed to square root ($x + 0.5$) for analysis.

Results

Relationship of yield losses and disease intensity near harvest

In each of the 3 years, there were no differences in fruit numbers between plants without symptoms (NS) and with intermediate symptoms (IS), both of which yielded more fruits than plants with severe symptoms (SS) (Fig. 1a). The reductions of fruit numbers from SS plants as compared with NS plants were 23.6%, 47.9% and 56.8%, respectively, for 1995, 1996 and 1997.

When total fruit weights were compared, significant differences were observed among NS, IS and SS plants in 1995 and 1997. In 1996, NS and IS plants were similar, but different from SS, which yielded significantly less. Weight reductions in SS plants varied from 69.5% in 1996 up to 84.3% in 1997. Results from the 3 years combined showed significant differences between healthy plants, and plants with intermediate and severe symptoms (Fig. 1b).

Fruit size reduction in 1995 was estimated at 21.3% and 53.1% in IS and SS plants, and in 1996, fruit size in SS plants was 41.5% less than in NS plants. In the final year, fruit size was reduced by 28.4% in IS and 62.0% in SS plants. Differences were found among NS, IS and SS plants in 1995 and 1997, but not in 1996, when SS and IS plants were similar. Combined data from the 3 years (Fig. 1c) showed significant differences in fruit size among NS, IS and SS plants, as for total fruit weight.

Relationship of yield losses and time of symptom appearance, based on chronological age of tomato plants

All yield components were correlated ($P < 0.01$,

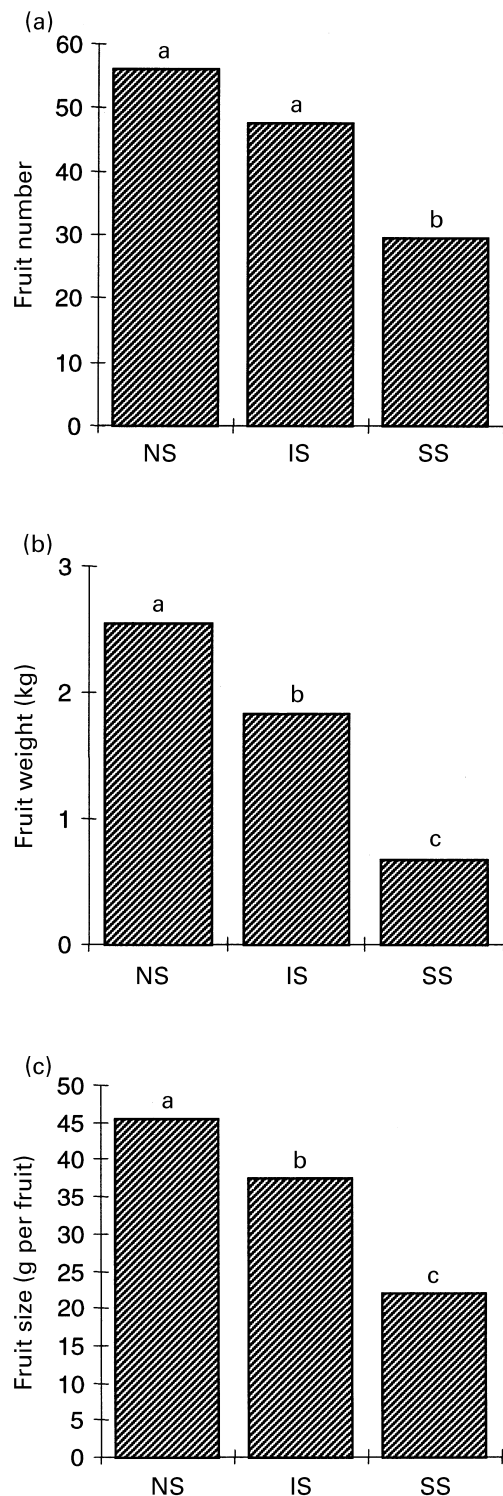


Figure 1 Tomato cultivar IPA-5 fruit numbers for five plants (A), total fruit weight (B) and size (C) produced in plants with no symptoms (NS), with intermediate (IS) and with severe symptoms (SS) of sclerotinia rot, assessed 10 days before harvest, from 1995 to 1997. Means followed by different letters were statistically different ($P=0.05$), according to the Tukey test.

$r=0.73-0.82$ in 1996, and $P<0.01$, $r=0.84-0.90$ in 1997) with TSA in chronological age. Regressions of yield components with TSA were best fitted by exponential models in 1996 and quadratic models in 1997, which fitted a nonlinear relationship observed between yield and TSA (Fig. 2a–c). A drastic yield reduction was detected in plants attacked early in the cycle, usually resulting in plant death before complete fruit set. Near-total losses were found for TSA from 1 to 4 (from 60 to 105 DAS), demonstrating that infections from early to mid bloom promoted severe losses, and only plants infected near harvest had a reasonable yield. Predicted values by regression analysis produced nearly parallel curves, showing a stable relationship between times of symptom appearance for all yield components.

Relationship of yield losses and time of symptom appearance, based on physiological age of tomato plants

When fruit set was plotted against physiological age of tomato plants, sigmoid curves were found (Fig. 2d–f), similar to results of Ho & De Hewitt (1986) for fruit growth on healthy plants. Sclerotinia rot at early development stages prevented the plant from proceeding to the next physiological stage, therefore resulting in higher losses. High correlations between TSA and fruit number, weight and size were found between yield and TSA in physiological age ($P<0.01$, $r=0.79-0.84$ in 1996, and $P<0.01$, $r=0.86-0.90$ in 1997). The logistic model was the most suitable for fitting the relationship between yield components and physiological age in both years, although the Gompertz model could also explain these relationships.

Discussion

The grouping of diseased plants according to intermediate or severe symptoms 10 days before harvest allowed the identification of different levels of loss caused by sclerotinia rot. Plants which were NS, IS and SS could be distinguished easily by their symptoms, and marked differences in fruit weight and size were found. This has practical importance for tomato growers and may help in evaluating yield loss. Statistical analysis showed no interactions between disease intensities and years of disease assessment ($P<0.2605$), suggesting that grouping plants on intermediate or severe symptoms offered an efficient discrimination of yield between apparently healthy plants and those with different disease intensities, in particular for fruit weight and size.

In 1996 yield was higher than in 1995 and 1997, probably because of higher temperatures and rainfall, after irrigations were over. Rains are unusual in Brasilia from May until September and the similar performance of NS and IS plants could be explained by the unusually high amounts of water in the soil and by higher temperatures. Increased temperatures accelerate plant growth and fruit ripening (Geisensberg & Stewart, 1986). The lower yield

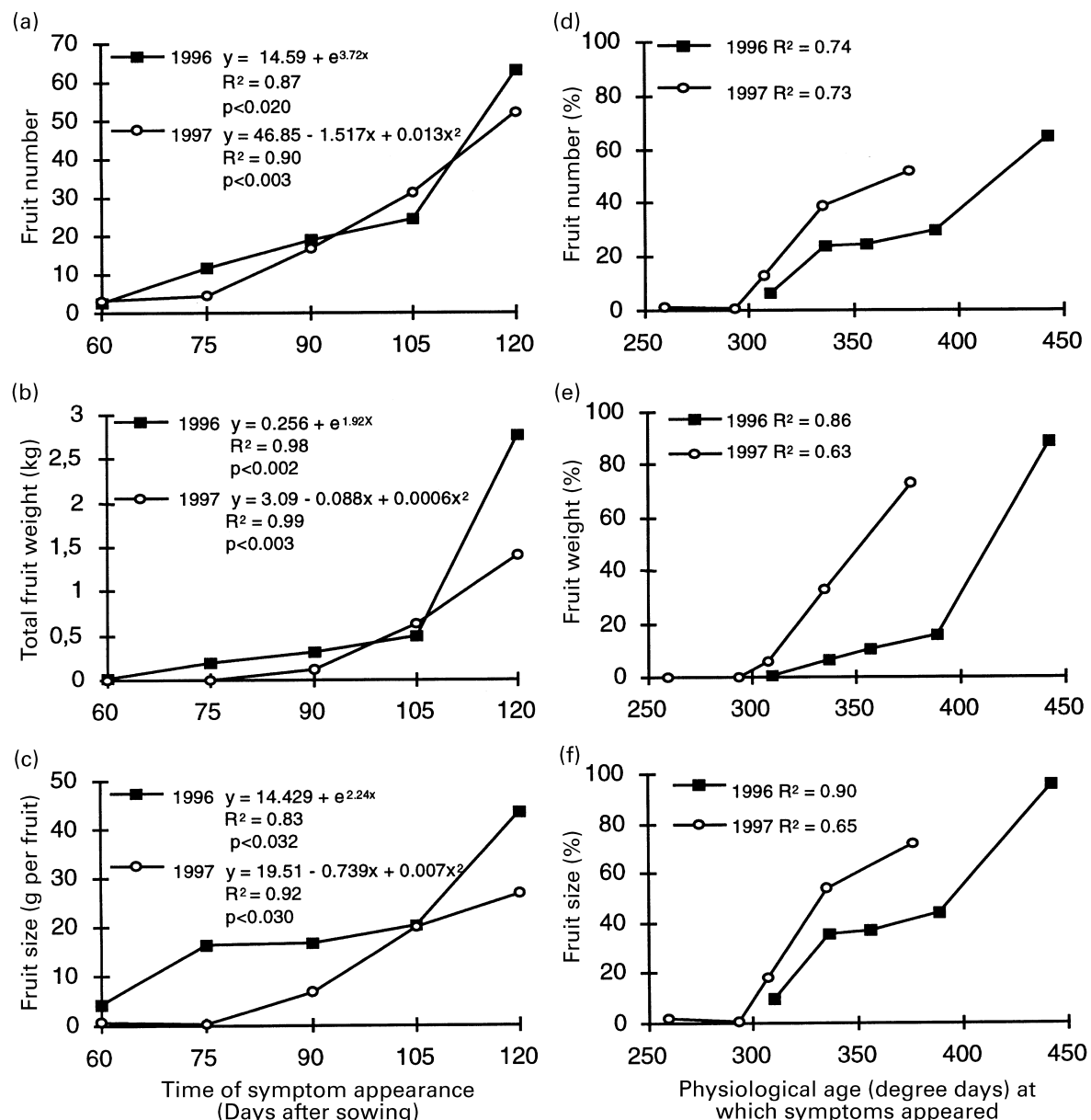


Figure 2 Relationship between fruit number, weight and size of IPA-5 tomatoes and time of sclerotinia rot symptom appearance, according to chronological (A, B, C) and physiological age (D, E, F), assessed in 1996 and 1997. Plants with initial sclerotinia rot symptoms were marked at 60, 75, 90, 105 and 120 days after planting, and assessed 10 days before harvest.

in 1997 was probably related to a higher inoculum density rather than to an earlier disease onset, because epidemics started around 60 DAS in all years.

The effect of disease intensity on yield components differed when results for the 3 years were grouped (Fig. 1), or compared for each individual year. Fruit number was already set in IS plants when symptoms appeared, and therefore would not differ from that in healthy plants. The closeness of fruit weight and size in 1996 for SS and IS plants was not observed in 1997, and, despite similarity in numbers, these fruits could not increase their weight and size as did NS plants. Lower scores for all yield

components remained in SS plants, and were a consequence of earlier infections that were visually obvious.

The recording of disease intensity and yield relationships is usually conducted during different growth stages (Sah & Mackenzie, 1987; Nutter, 1990; Hartman & Wang, 1992; Agrios, 1997), as yield can be highly affected by time of infection with plant pathogens (Madden & Nutter, 1995). This is particularly important in crops such as tomatoes, when a long period is necessary for fruit development (Francel *et al.*, 1997), as disease onset in different periods of fruit development may account for different yield losses. As for tomato

spotted wilt tospovirus (Moriones *et al.*, 1998), sclerotinia rot intensity had a close relationship to TSA, and this may be useful for future studies on yield loss prediction. Different weights could be attributed to differences in TSA, with a higher damage potential for earlier TSA.

At harvest, plants with early disease onset (TSA 1 and 2) were highly affected, with only one or two stems, and in some cases, with only a few olive-sized fruits that wilted after plant death. Plants with TSA 3 and 4 were infected at an intermediate stage of flowering and when adjacent rows were closing, and their yields were also low, with a few small fruits, hardly marketable. As most infections took place at stem base, fruit filling and weight gain was almost halted, also restricting the production of new fruits. Only plants with TSA 5, infected in the 2 weeks before harvest, produced a satisfactory yield. Visually, the disease intensity was similar to that of intermediate symptoms. Whenever the disease appeared during fruit ripening, direct losses were insignificant, and this might lead to an underestimation of the importance of the disease if most infections occur near harvest.

Sclerotinia rot quickly prevents water and nutrient uptake, mainly because of lesion depth rather than length, which is typical of stem rot diseases (Gaunt, 1995). Plant development almost ceases in the phenological stage after infection, and severe incidence of rot in the early and middle stages of the crop may be very damaging, particularly when the weather is conducive for disease development. Under these conditions, losses of 100% were observed in a commercial tomato field near Brasilia. Tomato crops have a relatively long period from the onset of fruit development until ripening, about 60 days in cv. IPA-5.

Yield of processing tomatoes depends mainly on the concentrated production of flowers early in the flower development stage, allowing only one mechanical harvest. For cv. IPA-5, most flowers are produced in the first 20 days of flowering, and only those produced in the first third of the flowering stage are able to develop into marketable fruits. The lower fruit size and weight in earlier TSA suggested that plants could not provide the carbohydrates needed for fruit development (Gaunt, 1987), with a negative effect on yield, even if most flowers were already developing fruits.

A similar situation was found by Ristaino *et al.* (1989), who described a severe impact of phytophthora root rot on vegetative and reproductive stages of processing tomatoes. The disease caused a water stress at these critical stages, with reductions in stand, fruit dry weight, and number of flowers and leaves. The same effect may occur with other diseases that strongly reduce water uptake and cause irreversible wilt.

When a plant is affected in its early stage of development, there is a compensatory effect on the yield of adjacent plants, which have more space and nutrients for growth. Therefore, losses resulting from death or damage to plants from 60 to 120 DAS may be partially compensated (Stofella & Sonoda, 1984) by healthy neighbouring plants, which must be taken into

consideration in yield loss assessment. However, for diseases such as sclerotinia rot, early infected plants die and function simply as missing plants when they are surrounded by other diseased plants, resulting in minimal compensation.

The mathematical models presented for yield vs TSA or physiological age are the result of statistical fit to the data observed. No mechanistic (biological) model is inferred or proposed based on the statistical fit, which was produced by an empirical process to obtain curves that best agreed with the actual data obtained.

Tomato growers should avoid early incidence of disease, focusing control measures from the earliest crop stages until 100 DAS, when the highest yield losses are produced. It is expected that if the disease begins early but with a low incidence, or if disease is controlled successfully from the beginning, or even if higher disease incidence occurs, but only near harvest, losses will not be extensive. However, an increase in the frequency of sclerotia in soil may account for higher disease severity and losses in subsequent crops. Only plants that showed symptoms in the last 2 weeks of the crop cycle could support disease incidence without damage. After the critical stage (100 DAS), fruits were physiologically developed and at the ripening stage. Although stopping irrigation did not prevent disease progress, the environment became drier and unfavourable to the pathogen.

Acknowledgements

First and second authors are CNPq fellows. All authors would like to thank Dr A. C. Café Filho from Brasilia University, Dr L. A. Maffia from Viçosa Federal University and Dr R. N. G. Miller from the National Center for Horticultural Research, for reviewing this manuscript.

References

- Agrios GN, 1997. *Plant Pathology* 4th edn. San Diego, USA: Academic Press.
- Conn KL, Tewari JP, 1990. Survey of alternaria blackspot and sclerotinia stem rot in Central Alberta in 1989. *Inventaire Des Maladies Des Plantes Au Canada* 70, 66–7.
- FAO, 1996. *Agriculture Yearbook*. Rome, Italy: FAO v.50.
- Ferraz EC, 1987. A ecofisiologia vegetal e a produção de alimentos no cerrado. In: Castro PRC, Ferreira SO, Yamada T, eds. *Ecofisiologia Da Produção Agrícola*. Piracicaba, SP, Brazil, Potafos, 101–11.
- Franc L, Neher DA, Campbell CL, 1997. Multiple-point regressions of yield loss. In: Franc L, Neher DA, eds. *Exercises in Plant Disease Epidemiology*. St Paul, USA: APS Press, 147–51.
- Gaunt RE, 1987. A mechanistic approach to yield loss assessment based on crop physiology. In: Teng PS, ed. *Crop Loss Assessment and Pest Management*. St Paul, USA: APS Press, 150–9.
- Gaunt RE, 1995. The relationship between plant disease severity and yield. *Annual Review of Phytopathology* 33, 119–44.
- Geisensberg C, Stewart C, 1986. Field crop management. In: Atherton JG, Rudih J, eds. *The Tomato Crop – A Scientific*

- Basis for Crop Improvement*. London, UK: Chapman & Hall, 511–57.
- Hartman GL, Wang TC, 1992. Black leaf mold development and its effect on tomato yield. *Plant Disease* **76**, 462–5.
- Ho LC, De Hewitt JD, 1986. Fruit development. In: Atherton JG, Rudih J, eds. *The Tomato Crop – a Scientific Basis for Crop Improvement*. London, UK: Chapman & Hall, 201–39.
- Holley RC, Nelson BD, 1986. Effect of plant population and inoculum density on incidence of sclerotinia wilt of sunflower. *Phytopathology* **76**, 71–4.
- Kerr ED, Steadman JR, Nelson LA, 1978. Estimation of white mold disease reduction of yield and yield components of dry edible beans. *Crop Science* **18**, 275–9.
- Lopes CA, Santos JRM, 1994. *Doenças do Tomateiro*. Brasília, Brazil: EMBRAPA/CNPB.
- Madden LV, Nutter FW, 1995. Modeling crop losses at the field scale. *Canadian Journal of Plant Pathology* **17**, 124–37.
- Mitsueda T, Charchar MJD, 1992. Mofo branco do feijoeiro (*Sclerotinia sclerotiorum*) nos Cerrados: estudos recentes e métodos de controle. In: *Seminário Sobre Os Progressos Da Pesquisa Agrônômica Na Região Dos Cerrados. Cuiabá, 23 a 24 de Outubro de 1991*. Brasília, Brazil: CPAC/EMBRAPA and JICA, 75–80.
- Morais MVR, 1994. Dinâmica do meio ambiente no Distrito Federal. In: Pinto MN, ed. *Cerrado*. Brasília, Brazil: UnB, 543–66.
- Moriones E, Aramburu J, Riudavets J, Arnó J, Lavinã A, 1998. Effect of plant age at time of infection by tomato spotted wilt tospovirus on the yield of field-grown tomatoes. *European Journal of Plant Pathology* **104**, 295–300.
- Morral RAA, Dueck T, Verma PR, 1984. Yield losses due to sclerotinia stem rot in western Canadian rapeseed. *Canadian Journal of Plant Pathology* **6**, 265 (Abstract).
- Nasser LCB, Hall R, 1997. Practice and precept in cultural management of bean diseases. *Canadian Journal of Plant Pathology* **18**, 176–85.
- Nasser LCB, Resck PV, Charchar MJD, 1990. Soil management, crop sequences and plant diseases in the Cerrado of Brazil. In: *Proceedings of the International Workshop on Conservation Tillage Systems*. Passo Fundo, RS, Brazil: CNPT/EMBRAPA, 190–203.
- Nutter FW, 1990. Generating plant disease epidemics in yield loss experiments. In: *International Rice Research Institute. Crop Loss Assessment in Rice*, 139–50, Manila, Philippines.
- Nutter FW, Teng PS, Royer MH, 1993. Terms and concepts for yield, crop loss, and disease thresholds. *Plant Disease* **77**, 211–5.
- Purdy LH, 1979. *Sclerotinia sclerotiorum*: History, diseases and symptomatology, host range, geographic distribution, and impact. *Phytopathology* **69**, 875–80.
- Ristaino JB, Duniway JM, Marois JJ, 1989. Phytophthora root rot and irrigation schedule influence growth and phenology on processing tomatoes. *Journal of the American Horticultural Society* **114**, 556–61.
- Sah DN, MacKenzie DR, 1987. Methods of generating different levels of disease epidemics on loss experiments. In: Teng PS, ed. *Crop Loss Assessment and Pest Management*. St Paul, USA: APS Press, 90–6.
- SAS Institute Inc., 1988. *SAS's User Guide: Statistics*, Release 6.03. Cary, SAS Institute Inc.
- Silva JBC, Giordano LB, Boiteux LS, 1994. *Cultivo do Tomate (Lycopersicon esculentum Mill.) para industrialização*. Brasília, Brazil: EMBRAPA. Instruções técnicas do CNPH no. 12.
- Silva WLC, Lopes CA, Pereira W, Fontes RR, 1997. Crop rotation systems for irrigated processing tomatoes in Central Brazil. In: *Proceedings of the 1st International Conference on the Processing Tomato. Recife, PE, Brazil, 21–2 November, 1996*. ASHS Press, 80.
- Snedecor GW, Cochran WG, 1981. *Statistical Methods* 7th edn. Ames, USA: The Iowa University Press.
- Stofella TJ, Sonoda RM, 1984. Influence of number and arrangement of missing plants on tomato yields. *Tropical Agriculture* **61**, 317–9.
- Sullivan GH, 1996. Economic performance, trade and competitive position in the north American tomato processing industry. *California Tomato Grower* **39**, 4–8.
- Tu JC, 1989. Management of white mold of beans in Ontario. *Plant Disease* **73**, 281–5.
- Zadoks JC, Schein RD, 1979. *Epidemiology and Plant Disease Management*. New York, USA: Oxford University Press.