STUDIES ON BACTERIAL SPECK OF TOMATOES

CAUSED BY *PSEUDOMONAS SYRINGAE* PV *TOMATO*

A thesis presented in fulfillment of a
Masterate of Science by thesis only
at Massey University

by

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1980

ADDENDUM

Pg 1, Para 3: Agrobacterium tumefaciens - 'e' not 'a'

Pg 6, Para 1: feasibility - 'i' not 'a'

Pg 10, para 1: 1st sentence reads:
...oxidase reagant using the method of Kovacs (Lelliott et al 1966)

Pg 16, para 2: 'sera' not 'serum'

Pg 16, para 3: 'antiserum' not 'antisera'

Pg 21, para 2: distinguished - 'g' not 'q'

Pg 24, para 1: Klement (1963)

Pg 24, para 1: 24, 28 or 72 '48' not '28'

Acknowledgements

I am sincerely grateful to Professor K.S. Milne for his advice and encouragement throughout the course of this study and in the preparation of this thesis.

I would also like to thank:

Dr M.J. Hill for helpful discussion and for use of the facilities in the Seed Technology Centre, Massey University.

Mr H.F. Neilson for technical assistance and discussion.

The Central Photographic Unit, Massey University for photography.

Mrs J. Hay for typing this thesis.

Odering's Nurseries, Palmerston North and Persson's Nurseries and Garden Centre, Palmerston North for the use of facilities and for tomato plants and tomato seed.

Mr B. MacKintosh (Wattie Industries Ltd) for tomato plant material and tomato seed.

Arthur Yates and Company for making funds available for this project.

Special thanks to Shirley.

Table of Contents

List of Plates

List of Figures

List of Tables

Page

Page

Abstract

The taxonomy of the causal agent of bacterial speck of tomatoes is discussed and the trinomial *Pseudomonas syringae* pathovar *tomato* (Okabe) Young, Dye and Wilkie is adopted.

A vacuum infiltration method of artificially inoculating seed was used and *P.syringae* pv *tomato* was detected in both artificially and naturally infested seed using sensitive enrichment culture techniques. The pathogen can remain viable between seed harvest and sowing in association with seed but seed-plant transfer was only occasionally demonstrated.

The acid seed extraction method and other germicidal seed treatments were evaluated for their effect on the seedborne pathogen. Streptomycin sulphate as a slurry treatment (2.5g a.i./Kg of seed) just prior to seed sowing was the only totally effective seed treatment tested.

The potential for survival in infected crop debris, soil and on alternative hosts was shown. However, the pathogen was not isolated from weeds in infected tomato crops and no conclusive evidence of systemic infection was found.

1. INTRODUCTION

The tomato, *Lycopersicum esculentum* Mill., is an important fresh-market and processing crop in New Zealand. The North Island is responsible for 83% of the total production (Anon, 1977). A limited export market for fresh-market tomatoes, mainly to the Pacific Islands, also exists (Anon, 1978).

The area cultivated to tomatoes has been static for the last few years with approximately 800 hectares producing at an average of 68 tonnes per hectare (Anon, 1979). Processing tomatoes, one of the two major processing crops in New Zealand, occupy approximately two-thirds of the total hectarage in tomatoes but only produce about half the total annual yield of approximately 54,500 tonnes. Fresh-market tomatoes occupy the remaining area and produce the other half of the total yield.

This yield does not reflect the genetic potential of the crop due to the influence of a variety of adverse environmental factors, of which diseases frequently are an important part. A number of bacterial, fungal and viral pathogens infect tomatoes and often cause economic loss. One of the bacterial diseases, 'bacterial speck', caused by *Pseudomonas tomato* (Okabe) Alstatt, is regularly of economic importance causing significant yield losses (Tate and van der Mespel, 1976), usually in outdoor tomatoes but occasionally under glass. Other bacterial pathogens may also cause disease in tomatoes in New Zealand including:- *Agrobacterium tumefacians* Smith and Townsend causing crown gall; *Corynebacterium michiganense* (Smith) Jensen causing tomato canker; *Pseudomonas cichorii* (Swingle) Stapp causing stem bacteriosis; *Pseudomonas solanacearum* Smith causing bacterial wilt; *Pseudomonas syringae* van Hall causing stem necrosis; *Pseudomonas viridiflava* (Burkholder) Dowson causing internal stem rot and *Xanthomonas vesicatoria* Doidge causing bacterial spot.

Bacterial speck of tomatoes was first recorded in 1933 (Okabe 1933; Bryan 1933) and was first found in New Zealand, at Hastings, in 1944 (Reid, 1948) where it was causing serious stunting of a dwarf variety in the field and extensive leaf infection of seedlings in boxes. The disease is now widespread in New Zealand causing economic loss by way of reduced yields and delayed fruit maturity.

Yields are reduced when the tomato foliage is diseased (Schneider, Hall and Grogan, 1975; Grogan, Kimble, Schneider and Ioannou, 1974). The percentage yield loss varies with the plant growth stage at the time of infection; slight yield losses are to be expected even in diseased tomato seedlings which may recover when only the lower branches are diseased. The yield reduction in plants inoculated at the following stages:- (i) third true leaf; (ii) third true leaf and 50% flowering; (iii) third true leaf 50% flowering and first green fruit as compared to a healthy control, were 12, 13 and 13% respectively (Schneider, Hall and Grogan, 1975).

Grogan *et al* (1974) found significant yield reductions in all plants inoculated with the pathogen at seven weeks. Total yields in this trial were reduced by an average of 15% and total ripe fruit yield was reduced by 59%. The disease had no affect on the number of fruit set or on the average weight of either green or red fruit. However, there was a total yield loss due to the delayed maturity because ripe fruit weigh more than green fruit.

If plant roots are diseased the total or red fruit yield, as compared to a healthy control, is not significantly reduced. However, in diseased plants there is a delay of approximately six days to the 50% red stage (Grogan *et al* 1974). Top inoculation of 14 day old plants reduced total yield by 16% and red fruit yield by 20% while top and root inoculation of 14 day old plants reduced total yield and red fruit yield by only 11 and 13% respectively. The bacterium also had a synergistic effect on

maturity, delaying it by as much as 10 days. Root inoculation of 60 day old plants under glass reduced both root and stem weights by 38% and 26% respectively, although the general plant appearance was unaffected (Grogan *et al* 1974).

Although the disease may occur at any stage during the growing season, Schneider and Grogan (1977a) reported that infections occurring in the early spring caused the greatest economic loss. Pohronezny, Volin and Stall (1979) estimated a 50% cull rate of fruit harvested from diseased plots due to the small raised speck lesions which make the fruit unmarketable.

The disease is readily recognized by the characteristic symptoms on both fruit and foliage. The fruit lesions appear as small, dark, raised pustules on the fruit surface while foliar lesions are dark brown to black necrotic areas surrounded by yellow halos (Bryan 1933).

The species causing bacterial speck in tomatoes, *Pseudomonas tomato*, was erected by Okabe in 1933 and named *Phytomonas tomato* Okabe. In the same year Bryan (1933) described a bacterial pathogen of tomatoes and named it *Bacterium punctulans* Bryan. In Bergey's Manual of Determinative Bacteria (1939) *Bacterium punctulans* is listed as a probable synonym of *Phytomonas tomato*. Dowson in 1943, referred to the organism as *Pseudomonas punctulans* (Bryan) Dowson (Reid 1944). The name *Pseudomonas tomato* was proposed in 1944 (Alstatt 1944) and is now most commonly used in the nomenclature of the pathogen. However, recently Young, Dye, Panagopoulos, Bradbury and Robbs (1978) proposed a nomenclature and classification system for plant pathogenic bacteria in which *Pseudomonas tomato* is named *Pseudomonas syringae* pathovar *tomato* (Okabe) Young, Dye and Wilkie. Although the merits of this taxonomic system will be discussed subsequently, all pathogens shall, hereafter in this study, be referred to by the nomenclature of Young *et al* (1978).

Study Objectives

Although bacterial speck of tomatoes has the potential to cause considerable economic loss, the possible sources of primary inoculum have not been positively identified in New Zealand. This study aimed to identify and determine the importance of the possible sources of primary inoculum and investigate the effectiveness of different control measures. As conflicting opinions exist as to the importance of any seedborne spread of *Pseudomonas syringae* pv *tomato*, most of this dissertation deals with the possible importance of tomato seed as a source of primary inoculum. The importance of any other sources of inoculum was also investigated.