Clubroot (*Plasmodiophora brassicae*) an imminent threat to the Australian canola industry

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Abstract

In the last 20 years canola (*Brassica napus*) has become an important broadacre crop and a valuable ($545 million) part of the Australian economy. This success follows similar rapid growth of this crop in other parts of the world. In many countries this rapid growth in production has been followed by yield decline due to clubroot disease caused by *Plasmodiophora brassicae*. This disease is already widespread in Australia where it causes significant problems in the vegetable brassicas. Internationally, clubroot causes losses of up to 1.6 t/ha (50% of yield) and is considered a serious disease of canola in France, Canada, Czechoslovakia, Sweden, Britain and Germany. Controls based on the use of fumigants, fungicides, nutrients (calcium and boron) and liming to increase soil pH have been developed for use in vegetable brassica crops, however, most of these are too expensive to be considered for use in broadacre crops. The screening and further development of clubroot resistant varieties however is well advanced, particularly in Europe. These developments are based on the transfer of effective genes from *B. campestris* using non-specific resistance in *B. campestris* and backcrossing with *B. napus* containing race specific genes. International experience suggests that clubroot may become a serious problem in canola in Australia. As clubroot is not seed-borne, contact with the vegetable Brassica industry and more importantly dormant inoculum in the soil from past fodder Brassica crops represent the greatest risk for incursion into canola. Opportunities for contingency planning are discussed.

Keywords resistance, control, contingency, disease cycle.

Introduction

In the last 20 years canola has grown from being a relatively minor crop, to become Australia’s third largest broadacre crop after wheat and barley (Australian Bureau of Statistics 2000–01). Currently valued at $545 million to the Australian economy (Australian Bureau of Statistics 2000–01), canola is part of the botanical family Brassicaceae which includes mustard, turnip, wild radish and the vegetable brassicas broccoli, cauliflower, cabbage and Chinese cabbage. Clubroot caused by *Plasmodiophora brassicae* Wor. has historically been considered the most economically important disease of these crops (Karling 1942). Clubroot is not a new disease. It is widespread throughout Europe, the United Kingdom, United States of America, Asia and more recently, Australia and New Zealand where it has become the most important disease of the vegetable brassicas. In spite of this, twenty years ago only 2.5% of canola crops from 18 countries were reported to be affected by clubroot (Crete 1981 cited in Rod and Havel 1992). Since then production of canola has expanded significantly. Consequently, clubroot is now a serious disease of canola in many countries including France (Rouxel and Regnault 1985), Canada (Vigier *et al.* 1989) Czechoslovakia (Rod and Havel 1992) Sweden (Wallenhammer 1996), Britain (Davies 1986) and Germany (Cristen *et al.* 1999).

Resting spores of *P. brassicae* are long lived in soil. Consequently once soils are infected costly control measures are required to maintain crop production. Economic losses due to clubroot can be substantial with losses of up to 1.6 t/ha (50% of total yield) being reported from heavily infested fields (Wallenhammer *et al.* 1999). This article provides a brief summary of current worldwide experience of clubroot in canola and evaluates the potential risk to Australian production of this crop.

The pathogen and disease cycle

Clubroot is caused by the Plasmodiophorid pathogen, *Plasmodiophora brassicae*, which is an obligate, intracellular plant parasite. Infection can occur at any stage of growth and is restricted to the roots. Primary stages of infection lead to the production of secondary zoospores in the root hairs and secondary, cortical stages lead to the production of resting spores and gall development. Primary (root hair) and secondary (cortical) stages follow sequentially and have been extensively documented (Ingram and Tommerup 1972).