Australasian Plant Pathology (1995) 24: 217-224

Twenty-five years of root disease research – could such a satisfying experience ever happen again?

G.C. MacNish

Department of Agriculture, Esperance, Western Australia 6450

Presidential address to the Australasian Plant Pathology Society, Lincoln 1995

Introduction

Being the first son of a farmer, it was assumed by my parents and me that I would take over the family farm. Thus, it was planned that I should attend Muresk Agricultural College to gain an academic and practical grounding in farming. This plan changed, when in Year 9 at high school, the principal suggested to my parents and me that I should go to University. With a 'farm-fix' in mind, I went to University and never gave thought to any course other than Agricultural Science.

Because of financial constraints while I was at university, I applied for and obtained a Cadetship in the Western Australian Department of Agriculture. This provided a living allowance of £4.10 ($9) while at university, jobs in the organisation during vacations and a requirement to join the Department on graduation. Graduate cadets were allocated where the need was most pressing and I was assigned to the Soil Conservation Branch. After two months in that branch surveying contour banks, I decided that there had to be a more satisfying job in some other part of the Department.

Fortunately, during my final year at university, I did a unit of plant pathology and was placed by the Department in the Plant Pathology Branch for both mid-term vacations. With this vacation experience to help me, I did very well in the plant pathology examination. Consequently, I approached the Chief of Biological Services and asked if there were any vacancies in plant pathology. He said it just so happened that a vacancy was about to be created and he would discuss my appointment with the Director of Agriculture. A few days later I was called to the Director's office and told that he was prepared to give me a trial period in plant pathology. I joined the Plant Pathology Branch in February 1961 and heard nothing more from the Director.

Thus, by default, I became a plant pathologist. I cannot help contrasting the ease with which I was appointed and the difficulties imposed on young people trying to qualify to join the profession these days. I will return to this theme later.

In 1968 I went to the Waite Institute and began my PhD under Professor Noel Flentje and Dr Bob Dodman and later, Dr Jack Warcup, after the untimely death of Professor Flentje. So began a 25 year association with the study of root diseases. Although I worked on take-all during my PhD, I was introduced to rhizoctonia bare patch disease during trips to Eyre Peninsular with Prof Flentje and Dr Dodman. An interest in rhizoctonia bare patch began then and has been with me ever since.

On my return to Western Australia, I became the cereal root disease specialist in the Plant Pathology Branch. Since then my most important work on take-all has included identifying the levels of take-all risk in various regions of Western Australia (MacNish 1980), work on the effectiveness of sources of nitrogen to reduce take-all (MacNish and Speijers 1982) and work on the development of grass-free cropping techniques to reduce take-all (MacNish and Nicholas 1987). Apart from positively identifying rhizoctonia bare patch for the first time in Western Australia in 1971 (MacNish 1983), my most important work on this disease has been the use of cultivation as a control measure (MacNish 1985) and an understanding of the aetiology and ecology of this disease. Although most of my work

on cereal root diseases over the past 25 years has been a very satisfying experience, I think my work on the patch dynamics of rhizoctonia bare patch disease has given me the most satisfaction.

Rhizoctonia bare patch disease is caused by *Rhizoctonia solani* Kühn [*Thanatephorus cucumeris* (Frank) Donk] AG-8 (Neate and Warcup 1985). This disease was first reported in Australia by Samuel in 1928 (Samuel 1928) and has subsequently been reported in England (Dillon-Weston and Garrett 1943), Canada (Benedict and Mountain 1956), Scotland (McKelvie 1978; Murray 1981) and the Pacific North West of the United States of America (Weller *et al.* 1986). In Australia the disease is reported from the cereal growing regions across all southern areas including Western Australia, South Australia and Victoria and the southern part of New South Wales (Murray and Brown 1987). The disease is a problem when conservation tillage systems are used with both zero tillage and reduced tillage exacerbating this disease (MacNish 1983; 1985; Neate 1984; Rovira and Venn 1985; Weller *et al.* 1986). Although this disease is greatly reduced by cultivation (MacNish 1985; Jarvis and Brennan 1986) it can be present in cultivated crops in a mild form or with the symptoms failing to be expressed. Bare patch disease has a wide host range affecting to different degrees, cereals, legumes, rapeseed, grasses and other pasture species.

Methods of studying bare patch disease

Three major techniques have been used in the study of bare patch disease. The removal of undisturbed soil cores (10 cm diameter x 10.5 cm deep) from within and outside patches has been a major technique (Dubé 1971; MacNish 1984). Isolating from the roots of wheat seedlings grown in undisturbed soil cores allows consistent recovery of the pathogen. Attempting to isolate from wheat seedlings grown in soil samples that have been mixed, drastically reduces recovery of *R. solani* AG-8. Isolation of the pathogen from mature field plants is also very difficult (Samuel and Garrett 1932; Hynes 1937; Kerr 1955; Murray 1981). Wheat seedlings are grown in the soil cores for 3 weeks at 15°C. The roots are washed clean of soil and gathered into a braid before cutting into 1 cm lengths and plating on water agar containing 25 ppm chlortetracycline HCl and 13 ppm of either metalaxyl or furalaxyl. The use of soil cores, the elimination of root surface sterilisation and the use of a semi-selective medium have allowed isolation of *R. solani* AG-8 throughout the year (MacNish and Sweetingham 1993*a*).

Another technique employed has been the use of pectic isozymes to produce zymogram patterns to identify isolates of *R. solani* obtained from wheat roots. This technology, developed by Sweetingham *et al.* (1986), has shown that there are at present only five zymogram groups (ZG1-1 to ZG1-5) within *R. solani* AG-8. These five groups appear to be very stable (MacNish and Sweetingham 1993*b*) and some have been isolated from all cereal growing states in Australia except Queensland. The use of these ZGs has allowed the tracking of isolates within patches and between patches.

Anastomosis techniques have also been used to study isolates of *R. solani* AG-8. There are four categories of anastomosis reaction between isolates of *R. solani* (Carling et *al.* 1988). Of the four categories, only Category 2 (C2) and Category 3 (C3) have been employed in this study of patch dynamics. It has been found that confrontation between isolates from different ZGs always gives a C2 anastomosis reaction (MacNish and Carling 1995). Similarly, if isolates from different ZGs are opposed on potato-dextrose agar amended with 0.4% charcoal (PDCA) Yang *et al.* (1992), they always give a 'tuft' reaction (MacNish and Carling 1995). The 'tuft' reaction between the opposing colonies is an area of distinct demarcation that is occupied by a band of hyphae raised above the general level of mycelium on the agar surface. In AG-8, the tuft is usually white but can occasionally be brown on one side (Yang *et al.* 1993). Confrontation of isolates from within the same ZG can either give a C2 and a tuft or a C3 anastomosis reaction and a 'merge' reaction on PDCA. The 'merge' reaction is where the two cultures come together with little or no evidence of demarcation.

The C2 anastomosis reaction is a vegetative incompatibility reaction while a C3 reaction is a vegetative compatibility reaction. Thus isolates from within a ZG that give a C3 reaction can be allocated to a Vegetatively Compatible Population (VCP) (MacNish and Carling 1995). It is thought that all isolates belonging to the

same VCP are closely related and, thus, the VCP can be used to show relationships between populations of the pathogen in patches. Patch studies using VCPs have been conducted in Western Australia. In one field at Kojaneerup, all patches were found to be caused by isolates of ZG1-2 and all the isolates were shown to belong to the same VCP (MacNish et *al.* 1993*a*). This suggests that all patches originated from the same source. The original isolate was probably introduced and spread by tillage leading to the establishment of all the patches.

Patch formation

There are two generally held explanations for patch formation. The first assumes that the pathogen is generally distributed in the soil and that patches are caused by localised environmental factors. The second explanation is that patches are caused by discrete colonies of *R. solani* AG-8.

Kerr (1955) proposed that patch strains of *R. solani* were widely distributed in soil and patches formed where there was a localised increase in mycelium. De Beer (1965) agreed when he wrote that 'patches do not result from the introduction of the fungus but from a local increase in the population of an already widely distributed pathogenic strain'. De Beer proposed that a population build-up in bare patches was the result of a reaction of the fungus to an unknown localised stimulus.

As an explanation for changes in patch configuration between seasons, MacNish (1985) proposed that 'within an infested field there is a balance of soil factors suppressive or conducive to rhizoctonia patch and that seasonal factors and local effects within a field can alter this balance'. He proposed that changes to these factors would interact with the non-random distribution of inoculum to produce a complex changing pattern of rhizoctonia patches. Under this hypothesis, when conditions are highly suppressive there would be no patches or if the conditions were less suppressive, only a few moderate patches would be observed. If conditions become more conducive, then there would be a cluster of patches with some being moderate and some severe. When conditions were very conducive there might be just one large severe patch incorporating all the patches in the cluster mentioned above plus all the area between these patches.

If, however, an environmental stimulus could cause many foci to combine into a single patch, it seems likely that within that patch there would be foci from a number of different ZGs. If it happened that all foci were from the same ZG, it seems likely that in at least some patches there would be foci from a number of different VCPs.

MacNish and Sweetingham (1993*a*) have tested the hypothesis that each patch is colonised by a single isolate of *R. solani* AG-8 and will not contain a mixture of ZGs. They examined a large number of patches over a period of five years. Most patches had multiple samplings with some being sampled 12 times and two 25 times. Samplings took place over periods of 2 to 104 weeks and the number of individual samples removed from each patch ranged from 14 to 814 with a mean of 109 samples per patch. With one exception, there was only one ZG isolated from each patch. The exception was in one patch where two samples out of 108 were from a different ZG and these may have been contaminants. It has also been shown that multiple isolates removed from different locations in the patch or removed over a period of as much as 10 months always gave the same VCP (MacNish et *al.* 1993*a*). These results support the hypothesis that each patch is colonised by a single isolate of *R. solani* AG-8 and that patches are not due to some factor causing many different foci to combine.

Changing patch configuration between seasons Kerr (1955) mapped 74 patches in one season and found that in the following season 22 patches had disappeared and 28 new patches had appeared. The remaining 52 were in the same position in both seasons but with increases in diameter of 0 to 50 cm. MacNish (1985) mapped an area (0.225 ha) of crop for rhizoctonia bare patch over four consecutive seasons. This study showed that less than 25% of patches were circular and that patches tend to be in clusters and elongated in the direction of sowing. More importantly, the study showed that there could be dramatic changes in shape, size and area of patch between seasons. The area of crop that was patch was 26.5% in 1979 and 27.9% in 1980, but in 1981 the area of patch dropped to 10.2% only to recover to 20.6% in 1982 (MacNish 1985).

Using overlays for a closer examination of the maps from 1980 and 1981, MacNish (unpublished data) demonstrated that there were some new patches and that some patches disappeared without a trace. Some patches enlarged while in others, some parts of the patch enlarged and other parts of the same patch disappeared. Some patches remained unchanged between seasons and for others only vestiges of the original patch remained. There are a number of contributing explanations that can be put forward to explain observed changes in configuration of patch over time. These include patch expansion, tillage effects, demise and decline of patches, coalescing of patches and soil suppressiveness.

Patch expansion It has been shown that patches expand at different rates (Kerr 1955; MacNish and Sweetingham 1993*a*). There are also differential rates of expansion around the circumference of individual patches. This will account for many patches not being circular (MacNish 1985). Ludbrook *et al.* (1953) proposed that patches expand between seasons, but the work of MacNish et *al.* (1993*b*) demonstrates that patch expansion takes place during the growing season and other data (MacNish, unpublished) show there is little or no expansion between seasons. The confusion about patch expansion probably comes about for the following reason. At the beginning of the season the young host is susceptible to root damage and stunting. Later in the growing season the soil around the patch circumference is colonised by the pathogen but without causing obvious stunting of the host. Thus the actual size of the patch (colonised soil) will not be evident until the following season when susceptible immature plants are affected by stunting. Thus quite large changes can occur without being noticed and could give the impression that a sudden change has occurred between seasons.

Tillage effects MacNish (unpublished data) demonstrated that patches can be established when soil colonised by *R. solani* AG-8 is introduced to areas free of the pathogen This suggests the pathogen can be moved in soil. There is considerable evidence that patches tend to be elongated in the direction of sowing (Ludbrook *et al.* 1953; MacNish 1985). This suggests that the pathogen is spread more rapidly by tillage (presumably by movement of soil) than by growth through the soil. There is also the possibility that the pathogen could spread along the drill row using the host as a bridge.

Demise and decline of patches It is well established that patches can disappear between seasons (Kerr 1955; MacNish 1985). MacNish et *al.* (1993*b*) have also shown that the pathogen in patches can decline during the growing season. This decline will not be obvious when based on the stunting of plants, but the isolation of the pathogen from the soil becomes increasingly difficult during the growing season. Within some patches affected plants can show some recovery towards the end of the growing season. In some cases this could be due to a pathogen decline but it could also be due to the mature plants being less susceptible to attack by the pathogen.

Coalescing of patches MacNish et *al.* (1993*b*) undertook a detailed study over 6 years of two patches that coalesced. Changes in the configuration of the patch over that period were shown to be partly due to differential growth rates. The most dramatic changes, however, were due to a decline of the pathogen in large parts of the patch and those parts becoming non-patch areas the next season.

Suppressiveness The above scenarios can explain many of the changes in patch configuration between seasons but do not appear to explain the complete picture. I proposed another explanation based on suppressiveness for some of the changes observed between seasons. This proposal has some elements that are similar to those proposed earlier by MacNish (1985) and discussed above. The difference is that the level of suppressiveness interacts with the level of pathogen virulence within the established individual patch and is not changing the environment to cause patches to form. Within any field, the individual patches nearly always show a wide range of disease severity (MacNish, unpublished data). Thus because patches vary in severity, in a suppressive season those patches of soil colonised by a less aggressive pathogen will be hidden and not expressed as patches. Only those patches of soil colonised with a very aggressive pathogen will be evident as patches and these may be expressed as mild patches.

However, in a very conducive season all the patches will be seen with a range from mild patches right through to severe patches. Thus in one season only a few mild patches will be observed, while in the next season there may be many patches with a range of severities. Further work is needed to test this hypothesis and to define possible causes for changes in suppressiveness.

It is now clear that patches are caused by *R. solani* AG-8 and within AG-8 there are five ZGs. Within these ZGs there are a number of VCPs. I believe that each patch is caused by a single isolate of *R. solani* AG-8 and is usually colonised from a single infection focus. However, some patches are the result of a coalescing of individual patches. Changes in patch configuration can be explained by differential growth rates, tillage effects, coalescing of patches, decline and demise of patches and possibly changes in suppressiveness.

Conclusions

Although I can express satisfaction with the results of 25 years of research in the field of root diseases, it may be time to look ahead and ask what is the future for the profession of plant pathology? If you read Phytopathology News you will have seen a whole stream of articles expressing concern about the sad state of affairs in plant pathology. There have been a number of letters and editorial comments about downsizing and demise of our profession. There have also been many comments about us being on the wrong track and being unable to attract funds because our work is no longer seen as relevant. There have even been statements that our research is so irrelevant to plant pathology that it might as well be incorporated in some other discipline like biology, physiology, biochemistry or genetics (Merrill 1994).

An examination of the Land Grant Universities in the United States of America may give a clue to their problems and the likely problem in Australasia. All the Land Grant Universities have a School of Agriculture and a faculty of Plant Pathology or Botany and Plant Pathology. These faculties had traditional plant pathologists like mycologists, virologists, nematologists, bacteriologists and extension plant pathologists. All the universities wanted to establish molecular biology units because research funding was being drawn to the latest biotechnology. This change in direction has been achieved at the expense of traditional plant pathology.

A similar trend appears to be taking place in our region. In the 25th anniversary issue of Australasian Plant Pathology (Volume 23 Number 4) there are eight excellent short papers by Society members on the future of plant pathology in the Australasian region. In one of these papers, O'Brien and Pegg (1995) point out that 'while it has much to offer, biotechnology will never replace the old premises on which the science of plant pathology was founded. It is merely one more pillar which adds further strength to our science. The challenge for the future will be to successfully marry good disease management with biotechnology. They must go forward together – not one at the expense of the other. Somehow, as the old guard pathologists fade into the sunset, provision must be made for pathologists who are skilled in the traditional science/art of plant pathology but can see application for the advances which will come through biotechnology'. The other authors all talk about the problems facing our profession, but they also all give a positive outlook for the future. These papers are all worth a second reading. Despite many problems facing the new age of science, I believe our profession has a bright future.

The future for our research is not so clear. I believe the swing to short term funding and the spreading curse of short term contract employment are leading to a reduction in long term research. As a consequence there will be a reduction in our knowledge base. I believe that the knowledge base and the experience base are an integral part of all science. Our experience base is being eroded because our senior plant pathologists are leaving rather than face the continued frustration of trying to obtain funding. Our younger plant pathologists are failing to build up an experience base because they are always moving to new projects.

I am not sure how we can reverse this trend. It is to be hoped that it is just part of a cycle that will come to an end when politicians realise that science should be run by scientists rather than accountants who know nothing about scientific principles. These people have the idea that science can be run like a local soft-drink factory.

In Western Australia, the Department of Agriculture is going through a complete reorganisation and one consequence of this is a move to market orientated activities. I have no objections to this change in emphasis. Unfortunately this change has been accompanied by a rejection of long-term research or 'blue skies' research as the reviewer called it. Our minders think that this type of research should be done by the CSIRO and universities. That attitude was probably correct in the 1950s and 60s, but my impression is that those institutions are also not in the position to undertake such research. The rural section of CSIRO is suffering funding cuts and is also constrained by short term funding sources that require 'quick fix' solutions with easily measured economic benefits. The universities with increased teaching responsibilities can no longer afford to have their academic staff spend years working in one limited area of research. Thus it seems to me the days when somebody like Professor Garret could spend a life-time working on take-all are gone for ever.

Having been fortunate enough to have worked in one area for 25 years, my title posed the question 'could such a satisfying experience ever happen again?'. I guess I have answered that question, but of course this does not mean that other types of satisfying experiences cannot be found in plant pathology. Plant pathologists will have to find this satisfaction in short term research. I am afraid it will be get in, get an answer and then on to something new.

These changes in attitude to science may require a re-examination of the training of future plant pathologists. I have explained how easy it was for me to become a plant pathologist and that I pity today's graduates and post doctorals trying to get a job. Despite this I believe that the system used today is superior because the chances of getting the best graduate are much better with active competition. Also I believe that those who have gone to the trouble and expense to obtain specialist training do deserve to be consider first. I am sure that the best person for the job did not necessarily get the job 25 years ago. The Department of Agriculture had to employ its cadets and had little interest in higher training. It believed that on-the-job training was best for its agricultural scientists. Thus we had plenty of practical experience but lacked an in-depth understanding of our field.

Nowadays I fear that we may have gone the other way with highly trained PhDs with lots of theoretical understanding but a lack of practical experience. As pointed out by O'Brien and Pegg (1995), 'research funds and young scientists have been drawn to the high profile areas of biotechnology'. Who wants to be a plant disease diagnostician or a mycology taxonomist when the wonders of molecular biology beckon. The question is how will this affect our profession in the future? To again quote from O'Brien and Pegg (1995), 'the challenge for the future will be to successfully marry good disease management with biotechnology'. To cope with the short term projects of the future, quick tests based on biotechnology will be essential for the plant pathologist.

Consequently, I would encourage young plant pathologists and students training to become plant pathologists to accept with open arms all the new technologies that are coming our way. However, I would also ask them not to forget the plants. The vocation of plant pathologists has always been to create plant health and the consequent benefits that flow to humanity from healthy plants.

References

Benedict, W.G. and Mountain, W.B. (1956) – Studies on the etiology of a root rot of winter wheat in south-western Ontario. *Canadian Journal of Botany* **34**: 159-74.

Carling, D.E. Kuninaga, S. and Leiner, R.H. (1988) – Relatedness within and among intraspecific groups of *Rhizoctonia solani*: A comparison of grouping by anastomosis and by DNA hybridization. *Phytoparasitica* **16**: 209-210.

De Beer, J.F. (1965) – Studies on the ecology of *Rhizoctonia solani*, Kühn. Ph.D. Thesis, University of Adelaide.

Dillon-Weston, W.A.R., and Garrett, S.D. (1943) – *Rhizoctonia solani* associated with a root rot of cereals in Norfolk. *Annals of Applied Biology* **30**: 79.

Dubé, A .J. (1971) – Studies on the growth and survival of *Rhizoctonia solani*. Ph.D. Thesis, University of Adelaide.

Hynes, H.J. (1933) – "Purple Patch" of wheat and oats. A disease caused by the fungus *Rhizoctonia solani*. *Agricultural Gazette of New South Wales* **44**: 879-883.

Jarvis, R.J. and Brennan, R.F. (1986) – Timing and intensity of surface cultivation and depth of cultivation affect rhizoctonia patch and wheat yield. *Australian Journal of Experimental Agriculture.* **26**: 703-708.

Kerr, A. (1955) – Studies on the parasitic and saprophytic activities of *Pellicularia filamentosea* (Pat.) Rogers and *Sclerotinia homeocarpa* Bennett. Ph.D. Thesis, University of Adelaide.

Ludbrook, W.V., Brockwell, J., and Riceman, D.S. (1953) – Bare-patch disease and associated problems in subterranean clover pastures in South Australia. *Australian Journal of Agricultural Research* **4**: 403-414.

MacNish, G.C. (1980) – Management of cereals for control of take-all. *Journal of Agriculture of Western Australia* **21** (4th series): 48-51.

MacNish, G.C. (1983) – Rhizoctonia patch in Western Australian grain belt. *Australasian Plant Pathology* **12**: 49-50.

MacNish, G.C. (1984) – The use of undisturbed soil cores to study methods of controlling rhizoctonia patch of cereals. *Plant Pathology* **33**: 355-359.

MacNish, G.C. (1985) – Methods of reducing rhizoctonia patch of cereals in Western Australia. *Plant Pathology* **34**: 175-181.

MacNish, G.C. and Carling, D.E. (1995) – Use of vegetatively compatible populations to determine field distribution of *Rhizoctonia solani* AG-8. Meeting of the Pacific Division of the American Phytopathological Society, Jackson, Wyoming, June 1995. *Phytopathology* **85** (In press).

MacNish, G.C., Carling, D.E. and Brainard, K.A. (1993*a*) – Characterisation of *Rhizoctonia solani* AG-8 from bare patches by zymogram and anastomosis techniques. *Phytopathology* **83**: 922-927.

MacNish, G.C., McLernon, C.K. and Wood, D.A. (1993*b*) – The use of zymogram and anastomosis techniques to follow the expansion and demise of two coalescing bare patches caused by *Rhizoctonia solani* AG-8. *Australian Journal of Agricultural Research* **44**: 1161-1173.

MacNish, G.C. and Nicholas, D.A. (1987) – Some effects of field history on the relationship between grass production in subterranean clover pasture, grain yield and take-all (*Gaeumannomyces graminis* var. *tritici*) in a subsequent crop of wheat at Bannister, Western Australia. *Australian Journal of Agricultural Research* **38**: 1011-1018.

MacNish, G.C. and Speijers, J. (1982) – The use of ammonium fertilisers to reduce the severity of take-all (*Gaeumannomyces graminis* var. *tritici*) on wheat in Western Australia. *Annals of applied Biology* **100**: 83-90.

MacNish, G.C. and Sweetingham, M.W. (1993*a*) – Evidence that each rhizoctonia bare patch is dominated by an individual zymogram group (ZG) of *Rhizoctonia solani* AG-8. *Australian Journal of Agricultural Research* **44**: 1175-1194

MacNish, G.C. and Sweetingham, M.W.(1993*b*) – Evidence of stability of pectic zymogram groups within *Rhizoctonia solani* AG-8. *Mycological Research* **97**: 1056-1058.

McKelvie, A. (1978) – Barley stunt disorder. *The North of Scotland College of Agriculture Digest* **1**: 45-50.

Merrill, W. (1994) – Teaching plant pathology: A myth. *Phyopathology News* **28**: 62-64.

Murray, D.I.L. (1981) – *Rhizoctonia solani* causing barley stunt disorder. *Transactions of the British Mycological Society* **76**: 383-395.

Murray, G.M. and Brown, J.F. (1987) – The incidence and relative importance of wheat diseases in Australia. *Australasian Plant Pathology* **16**: 34-37.

Neate, S.M. (1984) - Minimum cultivation and root diseases of wheat. Ph.D. Thesis, University of Adelaide.

Neate, S.M. and Warcup, J.H. (1985) – Anastomosis grouping of some isolates of Th*anatephorus cucumeris* from agricultural soils in South Australia. *Transactions of the British Mycological Society* **85**: 615-620.

O'Brien, R. G. and Pegg, K. G. (1994) – Plant pathology in Australasia: – a future perspective from the disease management point of view. *Australasian Plant Pathology* **23**: 213-215.

Rovira, A.D. and Venn, N.R. (1985) – Effect of rotation and tillage on take-all and rhizoctonia root rot in wheat. In *Ecology and Management of Soilborne Plant Pathogens*. (Eds C.A. Parker, A.D. Rovira, K.J. Moore and P.T.W. Wong), pp. 255-258. The American Phytopathological Society, St. Paul, USA.

Samuel, G. (1928) – Two "stunting" diseases of wheat and oats. *Journal of Agriculture, South Australia* **32**: 40-43.

Samuel, G., and Garrett, S.D. (1932) – *Rhizoctonia solani* on cereals in South Australia. *Phytopathology* **22**: 827-836.

Sweetingham, M.W., Cruickshank, R.H. and Wong, D.H. (1986) – Pectic zymograms and taxonomy and pathogenicity of the Ceratobasidiaceae. *Transactions of the British Mycological Society* **86**: 305-311.

Weller, D.M., Cook, R.J., MacNish, G.C., Bassett, E.N., Powelson, R.L and Paterson, R.R. (1986) – Rhizoctonia root rot of small grains favoured by reduced tillage in the Pacific Northwest. *Plant Disease* **70**: 70-73.

Yang, H.A., Sivasthamparam, K. and O'Brien, P.A.O. (1993) – Mycelial interactions and the potential use of tuft formation in characterising *Rhizoctonia solani* isolates infecting cereals. *Australian Journal of Botany* **41**: 253-262.

Yang, H.A., Tommerup, I.C., Sivasthamparam, K. and O'Brien, P.A.O. (1992) – Heterokaryon formation with homokaryons derived from protoplasts of *Rhizoctonia solani* anastomosis group eight. *Experimental Mycology* **16**: 268-278.