A P P S NEWSLETTER

AUSTRALIAN PLANT PATHOLOGY SOCIETY NEWSLETTER

VOLUME 4 NUMBER 1 MARCH 1975

Presidential Address

The need for greater public awareness of the importance of plant diseases

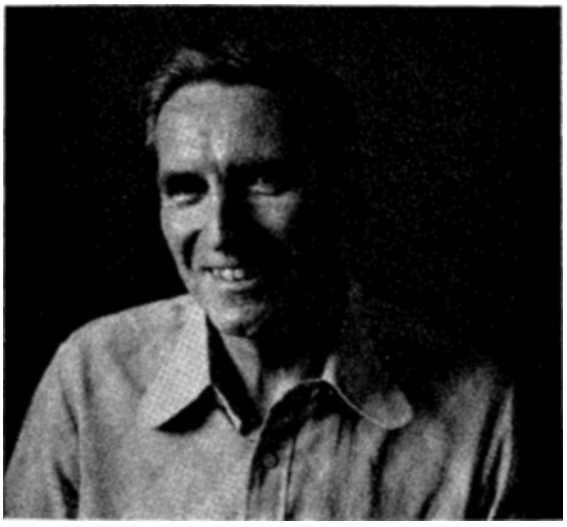
I have chosen this subject for my address because for the whole of my working life in plant pathology I have been irritated and frustrated by the lack of public awareness of the importance of plant diseases to mankind.

During the past six years, working in a university environment where plant pathologists are relatively rare species, I have found much interest and curiosity among colleagues and acquaintances in other disciplines, but generally abysmal ignorance of plant pathology. It is not surprising, therefore, that the 'man-in-the-street' is more ignorant.

In a society continuously bombarded with propaganda about developments in medical science — the great majority of which are concerned with fighting disease — we should have a ready-made and receptive audience, particularly if we can relate our findings to the welfare of mankind.

We should also by mind-association with human and animal diseases, have a public-relations advantage over our colleagues in other agricultural disciplines, but I see no evidence that we have attempted to exploit it.

The difference in value ratings between veterinary pathology and plant pathology was sheeted home to me recently during a discussion with a Dean of a Faculty of Agriculture in another Australian university, who is a veterinary science graduate. We were comparing our respective agricultural science courses when he remarked that he could never understand why agricultural graduates attached



Lionel L. Stubbs

A.P.P.S. President 1974

much less importance to plant diseases than veterinary graduates to animal diseases. This comment touched a raw edge, as it followed an unresolved argument with a number of my academic colleagues at Melbourne who oppose the teaching of plant pathology, but not entomology, to all undergraduate students. Illogically these same colleagues did not oppose a compulsory course in plant pathology for Forestry students.

To identify reasons for ignorance of our discipline and, hopefully, prescribe some remedies, it is necessary to look critically at both past and present performance of plant pathologists and university teaching in relevant courses.

Few would deny that, in Australia, we had an impressive beginning. Daniel McAlpine, Nathan Cobb and Henry Tryon firmly established the discipline in Victoria, New South Wales and Queensland in the 1890's and early 1900's, and all States except South Australia developed plant pathology laboratories in their respective Departments of Agriculture. South Australia was unique in sponsoring the development of a university department at the Waite Agricultural Research Institute.

During the past thirty years there has been continuous improvement in both staffing and facilities, including some late-developing forest pathology laboratories, and the output of research publications has generally been better than that of other agricultural disciplines in state departments.

Unfortunately, the reverse has occurred in the C.S.I.R.O. where, after an equally impressive beginning, plant pathology has been relegated to a minor role. Perhaps this is one reason why my colleagues in other agricultural disciplines are often relatively ignorant about the importance of plant diseases. Obviously, they are not sufficiently important to receive equal ranking with insect pests otherwise the Organization would have continued to be involved at its initially high level!

There are few, however, who know the complex series of events – personal problems, accident, tragedy and (unwise?) political decisions – that led to the change in emphasis in the Organization's Division of Plant Industry. I never fail to be amazed, however, that it proved possible in a country where disease limits, and in some cases inhibits, the production of practically every species of plant we cultivate.

The present standing of plant pathology in the universities provides no grounds for complacency. In all except one State teaching and research in plant pathology is vested in the agricultural faculties (in the case of Melbourne now Agriculture and Forestry), but only two universities have separate plant pathology departments. In Adelaide, this has occurred at the expense of the development of the discipline in the South Australian Department of Agriculture, and in Sydney it is a recent development combined with entomology.

It is understandable, therefore, that the universities give greater emphasis to teaching and research in agronomy, soil science and animal production disciplines. The relatively recent development of the animal disciplines, which followed the wool boom and gained further impetus when high export meat earnings appeared to be a permanent feature of our economy, has had the effect of reducing the amount of time allotted to the plant oriented disciplines.

The limit of increasing the workload of agricultural courses had been reached, and the alternative, now adopted by a number of universities, was to give students a choice of subjects. Thus, it is now possible for students to graduate with either an imperfect understanding of the importance of plant diseases to agriculture or, as in the case of Melbourne, with no understanding whatsoever.

The State Departments of Agriculture are necessarily industry-oriented, and from the beginning those concerned mainly with methods of production (and often farthest from the laboratory), have had the closest contact with farmers, farmer organizations and politicians. As a result the illusory belief has been created that the crop production expert is a generalist and the plant pathologist a specialist.

This is a belief that I make strenuous efforts to combat, because I believe that any plant pathologist worth his salt can only be successful in research involving an applied problem if he is also knowledgable about all the factors that influence plant growth, including pathogens. I doubt that such a claim could be made for most research workers in other agricultural disciplines.

These are some of the events and influences that I believe have contributed to the lack of public awareness of plant diseases and of the most successful research that has been responsible for keeping so many of them under control.

It would be false, however, to imply that plant pathologists are entirely blameless for this state of ignorance. Although we can claim credit for being among the first to recognize the need for and to obtain laboratories housing sophisticated equipment, and glasshouses and other controlled environment facilities to supplement our investigations in the field, some of us have become dominated by the tools with which we work. Increasingly we have become cloistered in our laboratories to the neglect of our public relations activities. Some of us grudgingly pay lip-service to extension activities involving our clients, which once were an important and mandatory segment of our duties, and occasionally we give talks to amateur horticultural societies and similar bodies. How many of us have acquired communication skills, other than learning by rote how to write papers for technical journals that are read only by plant pathologists? How many of us, after elucidating a plant disease problem or making some important contribution new to science, have taken the initiative to prepare or have prepared a popular report for wider publication?

Once again I use to illustrate my point the example of the medical scientist, where every research advance of significance, even some new link in a remote chain of evidence which may some day lead to the control of a disease at present uncontrollable is widely publicized, and the headlines invariably state that the control of X-disease is now just around the corner. This type of publicity obviously helps to keep public and private purse-strings open to support medical research, and it keeps the medical profession high in public esteem.

How many of us take the opportunity, when we have the right kind of audience, to tell the story of some of the great disasters inflicted on mankind by past plant disease epidemics, and of their enduring effects? I have had a number of opportunities in recent years, and I have varied the fare to suit the audience. The response has invariably been one of great interest and incredulous amazement, to learn of catastrophes not reported in the history books.

I admit that I have gained a lot of my knowledge of historical disease events from Carefoot and Sprott's book *Famine on the Wind,* which is the most fascinating story ever written about the profound influence of plant diseases on the history of Man.

This book, which is now available as a low-priced paperback, provides ready-made propaganda for our discipline ranging from fascinating speculation to well-documented facts, all presented in a most readable form. It should be required reading for all students of plant pathology if not biology, and certainly for all practicing plant pathologists. If it fails to educate the latter it should at least be good for their ego!

A public relations activity would be more effective if there were a number of plant pathologists in Australia with postgraduate training in agricultural extension. Such training is now available in three Australian universities and several colleges of advanced education. The Melbourne course, with which I am most familiar, has been in existence

since 1966. Since that time it has produced 73 diploma and three master's graduates. Only one of these was a plant pathologist and he was soon appointed to a district office only marginally concerned with plant diseases.

It would seem to me that the time has arrived for plant pathology administrators in each State to aim for one or two members of their staff to be trained as extension plant pathologists. The type of person most likely to benefit from such training would be a plant pathologist who has a genuine liking for public involvement. Preferably he should have demonstrated ability in research, and certainly not the reverse, as a trained extension plant pathologist would be an ineffective communicator of research results if his attitude to research lacked sympathy and enthusiasm.

Finally, I would like to point out that present day agricultural science students have a far greater interest in the social science disciplines than many of us had when we were students. For example in the 1975 final year B.Agr.Sc. course at Melbourne University, 28 students have elected to take the subject Rural Sociology and Extension as one of four subjects they have a right to choose, as compared with 15 for Plant Pathology. Four are quite adamant that they wish to take both, and these may be the kind of people that we should be looking for as the extension plant pathologists of the future.

It is a custom of professional societies to give a retiring president the opportunity to air his personal views on the eve of his retirement from office. I have taken full advantage of this privilege on this occasion.

I realize that there will be some who will say that our field of endeavour should be adequately publicized by our scientific achievements. Unfortunately, in present day society that is not the order of things. If we don't push our personal barrow no-one will ever push it for us, and I think that the time has arrived that we should start to push it harder than we have done in the past. We have taken the initial step of organizing ourselves into a Society which, after a slow beginning, is now becoming increasingly active. Let us keep it this way, and also recognize that the adoption by our Society of a more active public relations role would benefit not only plant pathology but also our members, most of whom are constrained in publicity matters by the inflexibly rigid procedures of the public service.

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RESEARCH NOTES

Late blackleg infections in rape are important

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Blackleg disease, (fungus *Leptosphaeria maculans*), caused great losses in Western Australian rapeseed (*Brassica napus* and *B. campestris*) crops in 1972 and 1973. In 1972 49,200 hectares of rape produced only 7,500 tonnes of seed and in 1973 1,000 tonnes were produced from 3,200 hectares, with an average yield of 159 kg per hectare and 318 kg per hectare respectively, compared to an expected average yield of at least 1,000 kg per hectare. Blackleg disease was considered to be the most important factor contributing to these losses.

Ascospores, liberated from infected rapeseed stubbles by rainfall, are considered to be the major source of crop infection (1,4). Crown (basal stem) infections constitute the most devastating phase of the resulting disease syndrome, involving leaf, crown, stem and pods. They result in death of seedlings and lodging of maturing plants. Pycnidiospores produced in lesions on the plants are responsible for secondary disease spread within the crop.

In France, Brunin and Lacoste (2) considered that, in nearly every case, severe crown cankers result from infections which occur during the first three to four weeks of growth. That this pattern of infection may not be typical of all situations is indicated by the following data.

Field observations were made in 1974 on a five hectare area of 60 m x 2.1 m plots of *B. campestris* cv. Span at Lancelin, 150 km north of Perth. Ascospore fallout from a neighbouring two year old blackleg infected stubble half a kilometre away was monitored with a Burkard Volumetric spore trap. It was commenced at the date of sowing, May 30, seven days prior to emergence, and discontinued from 14 weeks after emergence. There was no rainfall for 24 days after sowing and the first ascospore release was recorded 27 days after sowing and continued for 14 consecutive days. Further ascospore showers were not detected subsequently until the plants were 8½ weeks old. (See Table 1).

Cotyledons began to senesce three weeks after germination and most had disappeared by 4½ weeks. Cotyledonary infection, measured weekly, at no stage affected more than 0.01% of plants.

Five weeks after emergence 0.5% of plants had leaf lesions randomly located over the upper surface and this increased to 51% a week later. Masses of pycnidiospores in pycnidia were produced in these lesions by week 7. In subsequent weeks there was a gradual increase in the level of leaf infection, reaching a peak of 92% infected plants at 13 weeks after germination. The level of leaf infection then declined during the flowering and maturation period as leaves senesced.

Crown cankers were first detected seven weeks after germination on 0.1% of plants. They increased to 5% at 8 weeks, 7% at 9 weeks, and then to 35% at 10 weeks. Subsequently, the levels of crown canker gradually increased to 80% of plants by mid-flowering at 14 weeks after germination. Half of these cankers were rated as severe, according to the method of McGee (3).

In up to 40% of cases, crown cankers commenced at the node at the base of the petiole of leaves showing primary lesions. In the majority of the remaining cases the point of commencement of crown canker development was directly below the node. In many instances secondary infections were observed extending from leaf lesions down the entire length of the petiole to the node, and into the stem.

These observations suggest that direct ascospore infection was not the cause of most of the crown cankers observed. Further evidence to support this premise comes from observations that cankers become clearly visible in up to 50% of infected plants by four weeks after inoculation of plants growing over the widely differing temperature regimes of 12/7°C, 18/11°C, and 24/15°C (M.J. Barbetti, unpublished data). Hence, if the crown cankers had mainly been a result of direct infection from the initial ascospore fallout, then high levels of cankers would have been expected to have appeared by the time the plants were seven weeks old. The fact that high numbers of crown cankers were not observed until the plants were some 10 weeks old, taken in conjunction with the visual evidence above, strongly indicates that the majority of cankers were not the result of direct ascospore infection. The fact that cotyledonary infection at no stage affected more than 0.01% of plants also supports the hypothesis that the high levels of subsequent crown canker were not the result of cotyledon infection. It is considered that crown cankers resulted from extension of mycelium, from leaf lesions, down the petiole to the stem and/or from pycnidiospores produced in these lesions and washed down to the nodes, when the plants were 6 or more weeks old.