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Opening Remarks

by

Sir Emrys Jones

Two years ago, at the 7th Conference, we were expressing our anxieties about the world food situation. If anything the situation has deteriorated since we last met in this room. Nearly two thirds of the world's population live in regions where the food supply is in jeopardy, or insufficient to provide their basic needs throughout the year. It is generally accepted that around 500 million, mainly in Asia and Africa, are suffering from serious malnutrition and in danger of starvation and early death.

These are world problems; few of them can be overcome by countries acting unilaterally. Out of the chaos of the economic upheaval of the last two years or so there has at least emerged a movement to examine the world economic structures and their interactions. An attempt is now being made to seek logical international adjustments which might lead to an equilibrium with which all nations can live. There is a growing consensus that adjustments have to be made on an international level which will require the greatest possible degree of co-operation among nations. The World Food Conference, held in Rome at the end of last year, was an example of this movement, where an attempt was made to initiate international arrangements for improving the production of food and its world distribution. Many countries were disappointed with the outcome of this Conference but at least it was a solid beginning to the process of seeking international solutions to the problems associated with the future production and distribution of food in the world as a whole.

Out of it emerged the World Food Council, which has the responsibility for organising international action to overcome rural poverty, malnutrition and lack of food-producing capabilities. The first Meeting of the W.F.C., held last June, was somewhat disappointing in that a great deal of time was wasted in arguments about procedural matters, but there was a clear decision taken to aim for the following objective:-

"that within a decade no child will go to bed hungry, that no family will fear for its next day's bread and that no human being's future will be stunted by malnutrition."

No-one in this Conference Hall, I am sure, will deny that this is a laudable objective. We all fervently hope that it will be achieved. The great question is - HOW?

Will the countries of the Third World be able to feed their growing populations from their own land resources or will they become increasingly dependent on imported food, which they will be able to finance only at the expense of their overall economic growth? This is the agonising dilemma which confronts the world's politicians today.

Mr. McNamara has stated that there are 100 million farms in the developing countries of less than 5 hectares which are operating at a very low level of productivity. This level must be improved and there are five ways this can be achieved:-

1. Accelerate land reform
2. Better agricultural credit facilities
3. Provision of water for irrigation
4. Improved methods of rural distribution
5. Provision of adequate supplies of fertilisers and pesticides

The last of these is the main interest of this meeting but it is not just a question of providing sufficient fertiliser, pesticides are of major importance. Public funds must flow if anything like a 5% growth per annum of food production is to be achieved. The immediate answer lies in raising crop yields from the existing acreage of crop land. The technology to do this lies in this room at this conference where no less than 38 countries are represented which virtually spans the world, and provides a unique opportunity to create an effective dialogue and trust. The politicians can take a lesson from us in this. Professor Fletcher's book 'The Pest War' ought to be compulsory reading for all politicians. The pressure on land to produce food, if we are to achieve our objective, will be tremendous. What emerges from this book is the tragic waste of food which is taking place, waste and losses during germination, growth and losses after harvest. If all these losses could be overcome, the world's food supply would double.

Although we are very concerned at this conference with recent developments and new thinking in crop protection, we ought not to miss the opportunity of discussing the political background in which we are operating. We cannot any longer ignore what is happening in food production as a whole. Although there are only one or two sessions dealing with International Affairs, nevertheless it is my hope that the problem will be discussed informally during the course of this conference. A 5% increase of food production per annum seems an easy target and we know how it could be achieved by crop protection techniques. Its realisation lies in the hands of the governments of the world.

With these few thoughts, I have pleasure in declaring the conference open.

THE BAWDEN MEMORIAL LECTURE

THE ROLE OF PLANT PATHOLOGY

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As you must all be wondering now, so I wondered what prompted the British Crop Protection Council to ask me to succeed men of such accomplishment as Sir Henry Plumb and Dr. Pereira. I learnt that they aimed at a gentle transition from Sir Frederick Bawden's equals, through a junior disciple, towards future lecturers most of whom will inevitably have known him little, if at all. Also they knew there were few senior statesmen of agriculture to maintain the recent standard and so decided to divert the theme temporarily from policy towards science. Nothing would have pleased Bawden more than that they should begin with a plant pathologist. Nevertheless their request gives me an awesome task, yet one that I did not want to refuse to attempt.

I chose a general title that would interfere little with more detailed topics in subsequent lectures or in papers later during this meeting, but one that shows plant pathology as wholly and proudly a service science, which needs to use every specialism to help improve crop health. I may risk some displeasure from this audience by not talking mostly about chemical control. I make no apology because I know this represents but one of the methods to lessen diseases and that the agro-chemical industry depends on knowing the pathogens, their sources and spread, how they damage plants and our economy, as well as the ways in which they may be controlled. There is no time to attempt, and you are too expert an audience to need, a comprehensive introduction to pathology, so I shall give the view of an epidemiologist of foliage fungi on parts of plant pathology, especially some present weaknesses. I shall define 'plant pathology' within the confines traditional in Britain; banishing nematodes within nematology and dismissing as 'disorders' rather than diseases, the effects of minor element deficiencies, of nutrient imbalance and of atmospheric pollutants.

The identity of the pathogen.

Laymen often think of 'disease' as one entity, but specialists recognise many types of pathogen and many plant hosts, so that the total of possible interactions between them - that is of diseases - is prodigious.

Identifying each pathogen is traditionally the task of the taxonomists. When they change the familiar Ophiobolus into Gaeumannomyces or stick a Pseudo- prefix in front of a long respected Cercosporiella, agriculturists may be tempted to think them a very mixed blessing, but in reality taxonomic skill is both necessary and scarce. Sadly, taxonomists acquired the stigma of the worst of biological teaching based on 'types'. Not surprisingly, progress passed them by and they never learnt to make those puzzling differentiations between physiologic races, or isolates for which no diagnostic genetic characters are known, such as the forms

of *Puccinia striiformis* that have recently attacked some winter wheats or forms of other fungi that now prove insensitive to fungicides. Whoever in future identifies and classifies such isolates will need to be very different from today's taxonomists, with access to much new equipment and technique.

#### Quantitative ecology of hosts and pathogens.

Identifying pathogens is but the first step towards understanding their ecology quantitatively. It is easier to count weeds, insects and nematodes than to identify and count individual hyphal fungi, and it is often profitless to count bacterial cells or virus particles because they multiply and disappear so quickly. Pathologists have therefore devised all sorts of apologies for real measurements of populations; the production or dispersal of propagules, the frequency of vectors, the proportion of soil samples infective, or of plants, their leaves, roots or fruits that are infected. Such measurements are most important and most difficult early in attacks, when we want to predict how their increase depends on the supply of susceptible hosts and their environment. These tasks are conveniently included within the concept of 'epidemiology' - the study of diseases within populations - and a subject you will not expect me to ignore.

The ultimate in epidemiology is qualitative, through the exclusion, introduction or eradication of pathogens, sometimes as a result of introducing exotic crops, of reviving old ones or merely by changing crop husbandry. There often follows a 'happy honeymoon' before diseases become prevalent in the new circumstances. How long this lasts and what use we make of it often depends on the success of quarantine protection that I think we do not treat seriously enough or use fully. Too often quarantine and health regulations are regarded as bureaucratic nuisances rather than some of the best and cheapest controls in plant pathology. The British public more and more refuse to accept regulations that they do not respect. But, will they ever respect threats that are kept shrouded in secrecy rather than given the glare of publicity. Veterinary science both wields a fearful stick (as in the present anti-rabies campaign) and is allowed a slender carrot of compensation for early eradication (as with foot and mouth disease, bovine tuberculosis and brucellosis). Who, seeing this year's devastation of elm trees, would not wish we had tried rigorous felling with generous payment when first we suspected we harboured a new race of the causal fungus? Who among us has not heard wild rumours about new introductions that did nothing to enlighten but greatly exaggerated the real situation and obscured sensible official action that had already been taken. There is no need for vast cost, because compensation should be discretionary and be discontinued if the pathogen became widespread. Only stringent early eradication has hope of success.

Increasingly, agriculture and horticulture in European countries benefit from improved and healthier crops through planting certified stocks and seeds. There is increasingly a need to protect this material from trade in inferior material both within and between countries. Increasingly it becomes difficult to provide such protection as, for economic reasons, we make trade between countries easier and as plant breeders grow crops abroad to accelerate their work. By education and agreement we must find ways to prevent the harm and protect the improved stocks by some modified form of quarantine.

#### Increase and spread of pathogens.

Studying the later stages of epidemics had a hey-day fifty years ago, when the alternate hosts and long distance dispersal of the cereal rusts were first studied intensively. Each new epidemic renews a flurry of interest but little lasting

effort, so there is a need for prolonged studies of methods, principles and examples. Understanding the aerodynamics of dispersal or the methods of microclimatology are obvious starting points, but progress requires studying particular diseases, each so different that unique methods must be developed. Furthermore, many diseases involve annual crop cycles and longer climatic variations, so that replicating experience is painfully slow. Perhaps because of this slowness there has been a tendency to grasp any promising new technique, but most prove disappointing, as three examples will show. First, the controlled environment chamber, because pathogen increase depends not on constant environments, but on complex sequences favouring sporulation, dispersal, infection and growth. Second, microclimatology, because the more precise the measurements the more voluminous the records and the less possible it is to use them for agriculturally practical generalisations. Third, computer simulation for, as model makers always proclaim, their efforts revealed voids in our knowledge, but they were more prepared to fill them with doubtful guesses than to spend decades collecting real facts.

Similar difficulties apply to disease forecasting. Short-term forecasts aimed principally at timing individual sprays are most successful. Forecasts of annual disease severity would help farmers and especially the agrochemical industry much more, but long-range weather forecasts are impractical until meteorologists are cleverer at forecasting. Despite the derision their efforts often engender, there is real evidence of improvement. The time to begin collecting matching phenological data on crops, pests and pathogens is twenty years before meteorologists succeed, not just afterwards. Beginnings need not be costly, involving only nominating a few farms for co-operative collection of weather, crop and disease data. The network could later be expanded as crop physiologists, weed ecologists, environmentalists and others interested in crop protection realised the value of a sound data base.

Other factors also determine the development of plant diseases. I expect no-one here cherishes the delusion that agriculture ever has been 'natural', but many might agree that the ability of the cultivator to influence ecological processes has increased greatly. As much of the programme of these Conferences is devoted to the responsible development of chemical controls that are effective and unobjectionable, there is no need to labour that theme. Nevertheless, cultural changes such as some herbicides, direct drilling, straw-burning and machines like pea viners are introduced with little warning and may radically alter the survival of infected debris and therefore sources for later infection. Equally, machine harvesting may encourage wound pathogens on potatoes and fruit or on the trees and bushes from which it is picked. Host susceptibility is also changed through cultural practices and even more drastically by introducing new varieties. Some varieties last for many years, but recently the rise and fall of disease-resistant cereal varieties made the plant breeders and the farmers themselves dominant epidemiological factors in determining what foliage diseases we faced; agricultural practice indeed dictated what diseases were prevalent.

Several times I have qualified my comments on epidemiology by restricting them to foliage diseases. This shows the traditional but regrettable emphasis on pathogens that multiply and spread abundantly, cause dramatic symptoms and often show benefits for chemical treatment that merit advertising and publicity. Barley powdery mildew is now, I suppose, the classic example of a disease 'recreated' in the farmer's mind by demonstrating the benefits of new-found chemicals. By contrast, root diseases usually develop slowly, are seldom very evident and are often difficult or impossible to control chemically or by genetic resistance. Consequently the effects of root diseases may be more insidious, extensive and even more costly than foliage diseases. The fact that we cannot be sure whether the epidemic hare or the insidious tortoise costs more, implies it is time we concentrated on measuring the prevalence and effects of root diseases about which we know least.

## Measuring diseases and their effects.

Measuring the effects of diseases sounds simple and is certainly essential for deciding research and commercial priorities and the profitability of treatment, but it often proves one of the most complex and difficult problems in plant pathology.

The tuber troubles of potatoes provide examples ranging from slight blemishes to the rotting of whole stores. Even so, at today's inflated prices, losing a mere 1% of the national potato crop would theoretically cost £3-4 million (a sobering thought when few of our field experiments on potatoes detect significant differences smaller than 5%). I said 'would theoretically cost' advisedly because potato consumption is very constant, so any small difference from the requirement has a disproportionate effect on price and therefore on the contemporary cost of diseases. Conversely, acceptance standards for blemishing diseases like common scab are often adjusted to regulate the market in times of glut or scarcity. Diseases affect the production of healthy seed tubers quite differently; any occurrence of severe viruses, potato cyst eelworm or wart disease may prohibit production of the highest quality seed or even of growing potatoes. On the other hand, Scottish agriculture has benefited enormously from growing seed potatoes where aphid transmitted viruses are relatively easy to control. Such complex and contradictory factors could only be summated in monetary terms. In this age of 'accountability' perhaps we should encourage pathologists and economists to find how to do this, but I suspect their answers would be very ephemeral and would not greatly help me to answer that simple, but very embarrassing question, so often put to plant pathologists by administrators, farmers, plant breeders and representatives of the agrochemical industry: What is the importance of these diseases, actually and relatively?

Pathologists in different countries make very different attempts to measure diseases and their effects. Some make none; and certainly it is better for the few pathologists in a developing country to control some diseases rather than to measure some and decrease none. Others quote previous estimates until they carry conviction or make 'guestimates' to frighten their political masters into allocating more money. A few honestly decide that the cost of collecting accurate information is greater than the probable reward. The British approach has been more thorough and more logical than most; it attempts to relate disease incidence to effect, with the help of experiments. However, it tempts the risky assumption that incidence and effect are necessarily and constantly related. Whereas the harm that diseases do may not be proportional to the extent and duration of attack, but depend on the severity of a brief stress condition or the capabilities of the surviving healthy parts of the plant or crop. Furthermore, after the years necessary to collect enough data, the original question and the answer is often irrelevant because agriculture has changed; farmers have preferred other varieties, found other control chemicals or changed their methods to avoid early or severe attacks. Without being hypercritical, it is well to recognise that although our methods are inadequate and justify no self-congratulation, they have been valuable and informative not least by side-benefits from disease assessments. At a time when Bawden was one of its members, the Plant Pathology Committee of the British Mycological Society took the initiative with its descriptive key for potato blight. When put to use by E.C. Large this key forced pathologists to be quantitative and to begin to think of the effect of a disease on a growing crop; an experience that many found novel, salutary and beneficial. Since then we have become much more skilful in measuring the extent of foliage diseases of many crops. Now we should turn our attention to the much more difficult problems of root diseases and measuring the effect rather than the incidence of diseases.

### Yield capabilities of diseased plants.

To do this pathologists must recruit a corps of physiologists sufficiently 'bent' and disreputable to compare the metabolism and growth of healthy and diseased plants. I am certain that such a team could contribute much more to human welfare by defining the major limitations to growth and yield of various groups of pathogens, instead of continuing fashionable studies of dismembered organelles or, even worse, totally ignoring the effects of diseases that infect the plants they do study. They would, I am sure, come to insist on measuring the surviving yield capability of diseased plants rather than attempting, as most pathologists have done, to measure only the negative effects of diseases. Not only would this put us nearer to what the farmer wants to know, but it would emphasize the importance of stress conditions in determining damage. Recently several pathologists at Rothamsted have co-operated with the Letcombe Laboratory in trying to develop such methods. Radio-isotopes can certainly help define ion uptake and transport by diseased roots in the laboratory, but no satisfactory method for field use has yet been found. Nevertheless there are two hopeful indications: first, that soil moisture profiles might reveal drought stress periods that increase the effect of the take-all disease of cereals; and second, they might even derive a measure of the total root activity of wheat plants by measuring the amounts of silicon in their glumes at harvest.

### Controlling diseases without chemicals.

Only when the pathologist knows the pathogen, where it is and what it does, may he logically consider how to control the disease. However, the necessities of agriculture seldom allow such a leisurely course and priorities differ. The epidemiologist's inclination is first to decrease diseases by manipulating the ecology of host and pathogen, reserving chemical control as the high trump card to be played only when, as so often happens, other methods fail. In animal and human societies preventive medicine is embedded in instincts and customs. So far as we know, plants have no social taboos but agriculture has provided some indirectly. The traditions of cultivation, good husbandry and rotation are much concerned with hygiene and the health of plants. It is wise to challenge such dogma periodically, as the need to plough and for crop rotation is now being tested in cereals, with better herbicides and intensive cropping systems respectively. The latter seek to avoid the admission that the only way to control foot and root diseases is not to grow susceptible crops often.

Consecutive susceptible cereal cropping has shown that after reaching a maximum the take-all disease (*Gaeumanomyces graminis*) often 'declines'. No-one has yet proved why or how but the evidence suggests the cause is microbial. Certainly the increase of the disease is also affected by interactions within the complex of micro-organisms around the roots. Despite its mysteries farmers are able to utilise take-all decline, if not to manipulate it, and it may well be the most important form of biological control used today in British agriculture or horticulture. We may never arrive at a satisfactory definition of biological control, but it will remain an attractive possibility for decreasing disease: however, it is on practicality that most schemes founder. Nevertheless, hope springs eternal, so we shall watch anxiously current studies with clover rot, cereal cyst nematode and silver leaf disease of fruit trees. To the last, a new-found concern of mine, I shall hope to return later.

Diseases have been more effectively reduced, biologically, by constructing different host plants, either physically, by grafting together productive and resistant parts or by plant breeding to introduce or recombine genetic resistance factors. Genetic disease resistance has been most successful and durable among sedentary root pathogens, least so against prolific, readily-dispersible foliage

fungi that cause epidemic diseases among crop monocultures. Breeding against the epidemic diseases often becomes a race against the development of variants of the pathogens. Nevertheless some resistant varieties have greatly benefited agriculture, but temporarily and by using genetic resources at a rate, the eventual cost of which we still cannot define.

The 'forced evolution' that Wolf described, resulting from the race to breed sequences of varieties with specific resistance, leads not only to the familiar 'boom and bust cycles' in the popularity of crop varieties, but also complicates pathogen populations, exhausts genetic resources and quickly squanders many of the advances that breeders make, by inbuilding resistance that soon becomes obsolete. Most cereal and potato breeders now accept that we need more durable resistance, and that this will probably rely on more complex and diverse mechanisms, but neither pathologists nor breeders yet know them. If durability requires dependence on many genes, this will change the tactics of plant breeding and make it slower. To compensate we must make progress surer, perhaps by persuading breeders to concentrate first on selecting positively for characters like yield or quality that do not apply selection pressures, as does a resistant host to a pest or pathogen population. In other words, they should concentrate on characters that do not 'answer back'. Selection against susceptibility would still be necessary, but should be by crop performance rather than precise genetic constitution. There is hope that this might decrease the variety of pathogen genotypes common in populations, by substituting selection for general competitiveness instead of the intense screening for single genes by which a sequence of once-resistant host varieties adds pathogen strains to the population. Pacifying pathogen populations in this way should reopen possibilities of influencing population dynamics through ecological factors that are too subtle to modify the violent epidemics of the boom and bust cycles; for example, by adjusting cultural conditions, limiting sources and perhaps growing multilines or varietal mixtures.

#### Need for new chemical therapeutants.

These were the problems, especially concerning cereal powdery mildew and yellow rust, that had begun to sap the customary confidence of the plant breeders at this Conference in 1969, when by contrast many chemists were full of hope for the new systemic fungicides. Now that both are wiser and a little sadder, it should be the chemists' turn to pause for thought. We have almost passed through the stage of spraying semi-precious elements about our fields and orchards; we have learnt some of the consequences of excessive persistence in the environment; we have developed some fairly safe fungicides but often have to use rather much of them; we have improved their specificity and application to pathogens, but we are still not entitled to be satisfied with the chemical armoury.

We have no commercial viricide and until recently these received very little study or investment, perhaps because of the obvious difficulty of selectively disrupting the intimate relationships between the virus and the metabolism of its host. Nevertheless, it is encouraging that two English research institutes reported their first encouraging results this year.

Adequate plant bactericides are also lacking, but the success of so many in man and animals suggests that the difficulty may not be so much in killing bacteria as in finding sufficiently persistent bactericides that are harmless to plants and can be applied effectively. These do not seem such difficult problems as finding chemical viricides, so perhaps we also lack bactericides because the market on temperate crops, and as yet in the tropics, is too small to attract the investment necessary to develop new materials and prove them safe and effective.



The market for fungicides is much larger and many new ones have appeared. Even so, although downward translocated fungicides have been tested they have not yet entered commercial practice, so we can still do little to control root rots or wilts. Where we have produced effective fungicides and can apply them efficiently within extensive markets, we are often soon confronted with strains of fungi that are no longer killed by fungicides that were previously lethal. Like other British plant pathologists, I prefer to call such isolates 'insensitive' rather than 'resistant' or 'tolerant'. The two latter, although well established terms for comparable strains of bacteria or insects, already describe important types of plant reaction to pathogens, so it seems confusing to use the same words but in opposite contexts, so far as benefit to the farmer is concerned. Analogy with 'resistance' to insecticides and bactericides in animals suggests that several discrete insensitivity mechanisms may accumulate in the same increasingly impregnable isolate. If this is true, insensitivity to fungicides may prove at least as difficult, as important and as intractable a problem as is the breakdown of hypersensitive genetic resistance.

New classes of fungicide are produced neither easily nor cheaply, and if insensitivity mechanisms can accumulate, the search will become progressively more difficult. Therefore we must study how insensitivity develops and works. Meanwhile we need quick ways to identify and measure insensitivity and its distribution.

As with the complication of pathogen populations by genetic resistance, the development of fungicide insensitivity seems to be a consequence of our search for specificity of activity. Are there materials or methods that do not suffer these difficulties, One scientifically much acclaimed but, so far, agriculturally unrewarding approach is the study of phytoalexins. Plant pathologists may be forgiven for initially regarding these as 'failed fungicides' when plainly they do not prevent the diseases they are called upon to cure. Neither, when confronted with sick plants, will they be much relieved to be told that without their natural phytoalexins, the plants might harbour even more pathogens. Nevertheless, it is very legitimate to test whether phytoalexins might prove valuable fungicides against pathogens specific to alien hosts. Such thinking received support from the recent discovery at the Unit of Systemic Fungicides at Wye that 'sclareol', a compound present in tobacco leaf exudates (and not a phytoalexin), controls the rust (*Uromyces phaseoli*) on Phaseolus beans. Is this the reason why tobacco and other solanaceous hosts are little troubled by rust diseases? Could natural products of plants that are not hosts of the pathogens we wish to control, lead us towards useful fungicides? Might some be fungicides that the pathogen had been unable to breach with insensitivity mechanisms in millenia, whereas less than a decade seems ample for some fungicides we produce today?

#### Possible hazards of biological control.

The many poisons and drugs that we extract and use from plants should quickly dispel any myth that natural fungicides should be harmless to man and animals. However, the proponents of biological control often seem to expect their methods, like honey and wheat germ, to be especially blessed from suspicion of any harm. It was in this connection that I wanted to return to consider, as an example, some of the problems that could arise from Dr. Corke's Long Ashton experiments on the control of silver leaf disease. Orchard tests suggest that implanting wooden plugs impregnated with a selected isolate of *Trichoderma viride*, ameliorates the symptoms in more than 60% of fruit trees, a much better result than from any other method. However, experience of thalidomide, the aflatoxins and so on has properly made us all careful. We therefore sympathise with the suspicions and the unenviable position of officials responsible for public safety; for whereas our courts presume innocence until guilt is proven, the public now expects them to ensure life-long innocence at birth!

The details of the case of *Trichoderma* versus *Stereum* need not worry us, but it does suggest problems that could occur on food plants when we attempt to control a pathogen with another micro-organism. How logical is it to question the safety of the control agent without enquiring into the hazards of the pathogen? For entirely different reasons there was recently an enquiry about potential teratogenic effects associated with blight and gangrene of potatoes. The hypothesis was eventually withdrawn, but not before metabolites of diseases tissue such as rishitin and phytuberin were investigated. What do we know about the toxicity and metabolic consequences of *Stereum purpureum*, *Erysiphe graminis* or mild mosaic of potato? I suggest very little, and that we should take no comfort from knowing that any damage they might cause has not yet been detected. Supposing we knew we were introducing a toxigenic biological control agent into plums, wheat or potatoes; would it be enough to regulate the amount applied or to state that there was less than a threshold amount of toxin present at harvest? I doubt it; surely we might expect the agent to multiply after application, perhaps erratically and, worse, even from latent inoculum as the product matured during sale or storage. Endless complexities can be imagined of which perhaps few are likely; but, before we pin too many hopes or invest too much money in biological control, we ought to spend more time investigating possible hazards, necessary test procedures and deciding whom among the proponents of biological control should (or could) pay the price, especially if it is comparable to that of introducing a new chemical.

#### Future strategy for the agrochemical industry.

Despite my determination that it was time to remind the agrochemical industry of its dependence on the biology of plant diseases, I should be remiss not to comment on opportunities for the agrochemical industry. At present we can only attempt to control viruses chemically by killing vectors to limit virus spread. If we found direct viricides, the potential world market would be enormous, especially in vegetatively propagated and perennial crops. Bactericides may not be needed in such quantity, but would be important and there seems more prospect of success. Although fungicides have been made for nearly a century, it is easy to forget that little more than a decade ago their use in this country centred on little but seed dressings, potato blight, fruit and glasshouse crops. Few farmers were equipped to spray until they used herbicides; the values of cereals and forage crops were not thought sufficient to support costly sprays, and sprays could not be applied late because it was sacrilegious to run a tractor through standing crops. Demonstrating the damage caused by barley powdery mildew when better fungicides arrived, soon changed these agricultural conventions. Now farmers quite understandably demand a potion for every ill, and many can be provided, even if there are still so few effective in the neglected underworld of root diseases.

These considerations now make plant diseases seem the most rewarding target for the chemical industry. To maximise the opportunities, the industrial strategists must also pay attention to the biological 'writing on the wall'. Surely we must stop and think, when the high points of our technology in genetic resistance and fungicide chemistry are doing as well or better in producing fitter pathogens than healthier plants. We must not waste the fortunate simultaneous warnings from our search for specificity through the activity of single resistance genes and the equally precise action of some modern fungicides. For a time plant breeders have often been able to keep pace with the production of pathogenic variants, for example, of the wheat stem rust fungus. It may be neither possible nor economic to produce a comparable sequence of fungicides to replace those that lose efficacy, particularly as the search for new compounds would become harder as insensitivity mechanisms accumulate in populations.

I see no easy answer to these problems, but I am sure that pathologist and chemist must work together more closely to solve them. Certainly they will need to follow three paths of which we have heard much but know little, namely, durable protection, integrated control and deployment strategy. Each requires collaboration, the first to understand the mechanisms of fungicidal action and insensitivity, the second to fit together minimum amounts of widely different methods of control for maximum effect. Hence the need in this lecture to consider such widely different aspects as epidemiology, disease forecasting and biological control. Of the third course, deployment strategy, I have spoken little, but it may prove the most difficult because it must rely on unanimity through understanding. There is ample evidence to suggest that the period of useful resistance of single genes could be prolonged by introducing them in groups; equally, initial disease inoculum may be decreased by isolating young crops from old ones. By analogy one would expect similar principles to apply to plant protective chemicals. We should, therefore, be developing a strategy to delay the onset and spread of insensitivity. The task will not be easy because plant pathologists are as unprepared to suggest a strategy, be it based on integrated control or mandatory rotation zoning or mixture of materials, as are the trade, farmers or growers to accept it. Good bilateral co-operation exists, but the task will need multi-lateral and international co-operation, much more information and perhaps the sacrifice of some short-term commercial advantage before success is attained.

If I had to choose the piece of disease control that, in the last twenty-five years, had been most successful, most durable, least objectionable and least costly, it would undoubtedly be the production of King Edward VII potatoes free from paracrinkle virus by Bawden and Kassanis in the 1950's. As it happens, it needed to involve no chemicals, but relied on many of the types of biological understanding that I have described. I therefore have some confidence that, as my teacher, Sir Frederic would have approved part of my general thesis. Certainly he would recognise the important role of plant pathologists as the philosophers of disease control, and their need to work closely with the chemical technologists and the technicians in the field. Even in 1969 Sir Frederick clearly foresaw the possibility that pathogens might quickly evade chemicals that acted very specifically. By now, his thinking would be far ahead of mine and certainly we should not agree word for word because, as any who experienced Sir Frederick's hard-pointed pencil will recognise, he invariably improved my writing by removing many of my words. Would that he could have done it yet again!

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BAWDEN MEMORIAL STUDENTSHIPS

In memory of Sir Frederick Bawden, first President of the Council, the Bawden Memorial Trust awards a number of studentships each year to enable young people to attend the British Crop Protection Council's main Conference, held annually at Brighton. The awards are made on the basis of an essay competition, open to students and employees under 30 years of age, of any nationality, studying or employed in the United Kingdom.

In 1975, four studentships were awarded and the overall winning essay on "Ecological and Economic Aspects of Crop Protection" is presented in the following pages.

ECONOMIC AND ECOLOGICAL ASPECTS OF CROP PROTECTION

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INTRODUCTION

The objective of employing crop protection methods is to allow a crop or individual plant, to achieve its full potential for growth and development, resulting in maximum yield. Any agent which actively prevents the attainment of maximum yield must be considered a nuisance, and beyond a certain level may also be a factor causing economic loss. Soil and climate may also prevent maximum yields being obtained, but these are factors of a more passive nature, and, perhaps fortunately, can only be regulated to a very slight extent under practical agricultural conditions.

Considered as active antagonists in terms of crop production are organisms falling into three categories - weed competitors, diseases, usually of fungal origin, and insect pests. The other factors, soil and climate, closely influence these three "nuisance" categories, and may alter the dynamic equilibrium between them and the crop. Considered together they constitute the crop environment.

Weeds, diseases and insects may individually or together interfere with the crop, ultimately reducing yield, and thus under even the most primitive agricultural systems farmers take steps to prevent this yield loss. Such steps are methods of crop protection and have been carried on for thousands of years, although crop protection has a modern connotation of chemical control. Such a connotation is justified, however, since chemicals have revolutionised crop protection and played a large part in increasing crop yields, particularly in those countries with "advanced" agricultural systems.

WEED CONTROL

Weeds occur in all agricultural systems, irrespective of soil or climate. They compete with crops for light, nutrients and water, and depending on their competitive ability relative to that of the crop, may seriously reduce yield. Some weeds grow very rapidly and may choke out emerging crop plants. Others are suppressed by a well established crop, and cause little, if any, competition with the crop. Weeds may also cause economic loss by reducing the quality of the produce, increasing harvesting or drying costs, or contaminating seed crops.

One weed, all too familiar in the U.K., is the wild oat, Avena fatua and A. ludoviciana. This weed competes with cereal crops for light, nutrients and water, but is perhaps most important as a seed contaminant of grain, by which means it can be rapidly spread from one field to another, and from one farm to another. It displays the phenomenon of induced secondary dormancy which results in deeply buried seeds emerging each year for up to about seven years or more, all from a single

shedding of seed. A wide variety of figures could be quoted to indicate the gravity of wild oat infestations in the U.K., but fairly commonly accepted figures indicate that for example in 1972, some 800,000 acres of heavily infested cereals were sprayed for wild oat control. Yield losses on heavily infested land may be as high as 3 cwt./acre. If this yield could be saved, the cost of spraying would be recovered at least, and at the same time there would have been little or no addition to the reservoir of seeds in the soil, thereby helping to reduce the problem in the long term if treatments are continued.

This long term advantage is most important, as are the consequences of neglecting control, in which case grain would become unsaleable, the entire farm becoming infested, and at the same time losing value in the property market, all because of this noxious weed. In the case of wild oats there is a very clear case for advocating control measures, either chemical in heavy infestations, or hand roguing in light infestations.

In all arable systems relative freedom from weeds is necessary to production of a good crop, especially in the early stages of crop establishment, and as a result the cost-benefit relationship has become fairly well established and accepted, in favour of weed control.

#### DISEASE CONTROL

Disease pathogens are not as omnipresent as weeds, and are more affected by climate and cropping patterns.

All cereal crops may suffer great damage by diseases. The pathogens often spread rather spectacularly under the right conditions of temperature and humidity. Department figures indicate that in the U.K. in 1973 the barley crop suffered an overall yield loss of 18% due to foliar diseases, powdery mildew (*Erysiphe graminis*) alone accounting for 13% loss.

This disease is one of the commonest and occurs to some extent in most parts of the country, in most years. The simplest preventative measure is a seed dressing of a systemic fungicide, and in many areas this method of protection is adopted as a sort of "insurance" against loss. Bearing in mind the greatly increased cost of seed barley, and the relatively high price of harvested grain, the extra cost of such a seed dressing would now constitute a small fraction of the total input costs whereas its omission followed by a bad spread of mildew spores, could well lead to a 10-15% loss of yield, amounting to 3-4 cwt./acre valued at about £8. At conventional seeding rates, the seed dressing usually costs about £3/ac., so there is a clear advantage in using such a protection technique. When so much is at stake, omitting such a seed dressing would appear to be a false economy.

A similar case exists for many other crops and diseases. One example of fungicidal use is seen in the case of seed potato production, where quality seed has a high premium. A grower cannot afford to have his crop down-graded or rejected in the certification scheme, and so spraying against blight, *Phytophthora infestans*, and aphids spreading virus diseases, are usually repeated at fairly close intervals throughout the summer months prior to burning down the crop. The large price differential between seed and ware potatoes means that few farmers risk the chance of attack by the pathogens.

#### INSECT CONTROL

In the U.K., insect damage is less spectacular than in many other parts of the world, but despite this some insects are persistent pests on certain crops. In the case of aphids, diseases are also implicated, since the aphid is an efficient vector of viruses such as those causing potato mosaic, beet yellows and many others on a

wide range of crops.

In 1974 the weather favoured rapid development of aphid colonies on sugar beet crops, with the resulting spread of beet yellows virus over a large proportion of the crop. The colonies consisted of both Myzus persicae and Aphis fabae, and crop losses were estimated to vary from 10-20%. Other, external factors, increased sugar prices and made the U.K. crop even more important, and with slightly increased sugar prices many farmers doubtless wished that they had taken steps to avoid these losses by killing the aphids either with systemic seed dressings or, for longer term control, granular dressings in the soil, or sprays once the aphids were actually on the crop.

Insects, in common with diseases and weeds cause damage which once seen, cannot usually be remedied. The effects are by then often too widespread, or control measures might make matters worse by adding extra mechanical damage. In many situations the old adage must prevail - "prevention is better than cure".

#### ECOLOGICAL ASPECTS

Some areas however are sufficiently isolated as to be beyond the normal range of pests and pathogens. Clearly, preventative measures are wasted where a pest or disease is observed in only perhaps one year in ten. This is a costly waste, and in ecological terms it may also be dangerous, since chemical treatments may upset other components of the environment, such as predaceous insects which might prevent the occasional aphid infestation from becoming serious. This unnecessary chemical application may upset the ecological equilibrium against the interests of the crop.

Where insect or disease attacks occur more frequently, routine preventative measures still may be unnecessary in most years, and may be detrimental to other organisms in the environment. In such cases close observation of the crop is important, so that when a pest or disease is observed immediate action can be taken to prevent economic losses occurring.

The bean crop provides a good example of the types of control necessary. In the south of England Aphis fabae attacks are expected in most years and so control is largely preventative, preferably by use of soil-incorporated systemic granular insecticides which do not harm other insects such as bees. In the north of England attacks by the aphid are not very common and so no routine preventative measures are warranted, but if attack does occur, spraying of the crop can be done, aerially if possible but taking steps to ensure that pollinating insects like bees are unharmed. In Scotland, A. fabae attacks are rare, and not severe if they do occur. No control measures are needed therefore, since economic loss of yield is very unlikely.

If more information were available about likely spread of pathogens and insects, more accurate and more efficient use could be made of chemical treatments, so as to minimise both crop loss and environmental damage. In the case of weeds, chemical controls should not be for "cosmetic" weed-free cultivation effects, but should be done to eliminate competitive species in the particular crop being grown. Spraying of non-economic infestations may upset the ecological equilibrium, and may also lead to a change of weed flora towards other more aggressive, harmful species. Wherever possible selective chemicals should be used, so as to kill only the target species whether it be insect or weed, thus minimising ecological upset.

#### CONCLUSION

If, as seems to be happening, more attention is paid to the above points, there could be a continued use of crop protection methods which would contribute more to the already increased yields which have been obtained by development of effective chemical controls, and without detriment to the environment. Crop protection methods

are clearly an integral part of modern agriculture, and are likely to continue to increase productivity into the foreseeable future.



INSECTICIDE RESISTANCE IN APHIDS

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Summary Survey of resistance in three major species in Czechoslovakia. Resistance in the hop aphid (Phorodon humuli) has been reported since 1967, the year in which resistance caused a calamitous outbreak. Present knowledge on cross-resistance is reviewed including a series of organophosphates and some carbamates. Phosalone and methomyl have been found sufficiently effective and holding promise for future protection of the hop. From chemically treated hop gardens resistant aphid populations have spread rapidly to wild hop growths. From the point of view of ecological impact insufficient reliability of prognostical methods is pointed out: in the absence of natural enemies outbreaks of resistant aphid populations constitute an imminent danger in intensively chemically treated hop gardens. Greenhouse populations of the peach aphid (Myzus persicae) have proved resistant to many organophosphates and carbamates in all Czechoslovakia. Laboratory investigation revealed mephospholan, thiocarboxine, azinphosmethyl and pirimicarb as most effective, the last one deserving priority because of its mild effect on predators. First populations of the black bean aphid (Aphis fabae) showing tolerance or resistance to thiometon have been recorded in Czechoslovakia. However limited, this resistance means a warning of possible difficulties in the chemical protection of the sugar beet. In both sugar beet and potato cultures attention should also be paid to the green peach aphid, an important virus vector. Based on experiments with the green peach aphid, the spotted alfalfa aphid (Therioaphis maculata) and the black bean aphid, differences are pointed out in the effect of Insect Growth Regulators of the juvenile hormone type on resistant, and susceptible strains.

Résumé Aperçu de la résistance des trois espèces principales des aphides en Tchécoslovaquie. On dispose de données sur la résistance du puceron du houblon (Phorodon humuli) depuis l'année 1967 où la résistance a conduit à un surpeuplement. On passe en revue des connaissances sur la cross-résistance qui comprend plusieurs insecticides organophosphatés, mais aussi des carbamates. Phosalone et methomyle restent jusqu'ici suffisamment efficaces et prospectifs pour la protection du houblon. Les populations résistantes se sont propagées rapidement des houblonnières chimiquement traitées même sur le houblon sauvage. Dans l'optique de l'impact écologique, on met en relief le manque de fiabilité des méthodes pronostiques: dans le milieu de houblonnières traitées chimiquement de manière

intense, les populations résistantes libérées des ennemis naturels passent très facilement au stade de surpeuplement. Les populations de serre du puceron du pêcher (Myzus persicae) accusent une résistance envers de nombreuses préparations organophosphatées et des carbamates, sur l'ensemble du territoire de la Tchécoslovaquie. Selon des études de laboratoire, ce sont les insecticides méphospholan, thio-carboxine, azinphosmethyl et pirimicarb s'avèrent être les plus efficaces; une priorité est à réserver à la dernière des insecticides citées en raison de son effet ménageant envers les prédateurs. D'autre part, on a noté sur le territoire de la Tchécoslovaquie aussi les premières populations du puceron noir du pavot (Aphis fabae) manifestant soit une tolérance, soit une résistance envers le thiometon. Bien que la résistance ne soit pas élevée, elle nous fait penser à des difficultés éventuelles dans la protection chimique de la betterave à sucre. Quant à la betterave et aux pommes de terre, il faut réserver une attention également aux pucerons de pêcher qui transmet des viroses. Sur la base des essais effectués avec le puceron du pêcher, le thériocaphide de luzerne (Therioaphis maculata) et le puceron noir du pavot, on démontre des différences dans l'activité des régulateurs de la croissance des insectes du type de hormone juvénile sur les souches résistantes et sensibles.

#### INTRODUCTION

In undertaking a survey of the problem of insecticide resistance in aphids my task has been facilitated by the recently published review of Boness and Unterstenhöfer (1974) which summarizes basic data on the occurrence of resistance in aphid species. Resistance in aphids raises specific problems. Although evidence has been made that under selective pressure aphids are able to develop resistance, erroneous opinion is being offered repeatedly - though without the evidence of precise laboratory check - which claims that failures of treatment in the field have been due to faults in connection with the application of insecticides. Presumably the first confirmation of selective resistance in aphids was the finding of Boyce (1928) who pointed to the lowering of HCN effectiveness after sevenfold application to the cotton aphid (Aphis gossypii). It was assumed that parthenogenetically reproducing populations should be unable to adapt themselves to the insecticides then in use. Chemical control has been aimed almost exclusively at aphids on summer host plants, i.e. mainly at parthenogenetical generations. However, underlying the asexual type of reproduction there is, in aphids, a wide variability of morphological and bionomical characters. This variability may be based on endomeiosis as was found in three species of parthogenetically reproducing aphids (Cognetti, 1961).

Difficulties in the chemical control of the spotted alfalfa aphid (Therioaphis maculata) were among the impulses which lead to the modern conception of integrated pest control. Shortly after the introduction of this pest in the USA failure of parathion and malathion was encountered in Texas (Randolph, 1957) and one year later in California (Stern & Reynolds, 1958). Shortly after the general principles of integrated control were formulated by Stern, Smith, van den Bosch and Hagen (1959). The ecological approach

stressed in this conception has remained valid until today as the only possible guideline for effective management of pest populations including the resistant ones. Apart from the foregoing T. maculata, and A. gossypii resistance is known to be present in Aphis pomi, Brevicoryne brassicae, Chromaphis juglandicola, Eriosoma laniferum, Macrosiphum solanifolii, Myzus cerasi, Nasanovia ribisnigri, Neomyzus circumflexus, Pentatrichopus fragaefolii, Sappaphis piri, S. plantaginea and Toxoptera graminum. These species, however, are of less importance to Europe or their resistance has remained limited as yet on this continent. On the other hand, further species must be added to the list: the plum aphid (Hyalopterus pruni) the pest of the peach in Czechoslovakia (Hůrková, 1973) and three species belonging to the major pests in continental Europe and Great Britain: the green peach aphid (Myzus persicae), the hop aphid (Phorodon humuli) and the black bean aphid (Aphis fabae). In the present report I propose to summarize the findings on aphid resistance mainly obtained from Czechoslovakia and to concentrate on more recent data acquired by a unified method which therefore are fairly comparable.

#### METHODS

In our laboratory we tried to simplify the method of testing to make it workable for most agriculturally important species; it also was to be rapid and not too laborious but sufficiently accurate (Hrdý, 1975; Devonshire & Needham, 1974). The method is based on exposure of the aphids on an insecticide-treated leaf surface of the host plant. The leaves are sprayed with 1 ml insecticide emulsion in a sedimentation tower by a Potter's nozzle. Batches of 20-30 aphids (apterous females for insecticide testing) are confined in glass-rings smeared with fluon inside and fixed to the treated leaf surface. Usually a series of 5 concentrations is used ensuring that at least 3 of them cause mortality higher than in the control group, but less than 100% and as a rule, 2-3 replicates are used. Evaluation of the results adheres to the general principles recommended by FAO and to the specific FAO instructions for Myzus persicae (Needham & Dunning, 1956; Baranyovits & Muir, 1969, Anonymous, 1969, 1970). Normally, the base-line data, the LC50 values, and the slope of regression lines are computed. The majority of our data on the geographical distribution of resistant populations is based on measured Resistance Factor (RF). However for resistance monitoring the "single-discriminating-dose method" may also be used. The method for testing juvenoids is very similar (Hrdý, 1974).

#### RESULTS

##### Hop aphid - Phorodon humuli

In this pest resistance to organophosphorous insecticides was first reported from England (Dicker, 1967) and from Czechoslovakia (Hrdý & Zelený, 1968). Today all European hop regions seem to encounter difficulties with the hop aphid. Clearly resistant (R) populations have been currently found in England, both German states, France, and the resistance problem arises in Yugoslavia as well as in Austria and Poland. Relatively abundant data are available from Czechoslovakia (Hrdý et al., 1970; Kříž, 1973; Hrdý & Kříž, in print).

### Occurrence of R populations in Czechoslovakia

Resistance was among the principal causes of the 1967 calamity in Czechoslovakia. It was found that aphids from Žatec were five times more resistant to thiometon than those of Tršice area. Thiometon is still in use today for resistance monitoring. The following years saw a quick spreading of R populations not only to all hop-growing regions, but also to wild-hop growths (see Fig. 1 and 2). Thus in addition to the regular occurrence of R aphids in the Bohemian, Moravian and Central Slovakia hop-growing regions, R populations have also been found on wild hop in localities lying at great distances from hop gardens. Surprisingly, they also included populations with very high RF (Hrdý, 1972). Undoubtedly the rapid spread of R populations has been made possible by passive transfer of winged aphids over long distances (Taimer et al., 1975). Colonization and survival of R populations on chemically untreated areas can be explained by higher vigour in resistant aphids. Presently in Czechoslovakia we have difficulty in isolating sensitive (S) hop-aphid strain in the field, but there were still several localities in Slovakia (Slovakian Ore Mountains and in the foothills of the High Tatra) where S aphids can still be found on wild hop after spring flights.

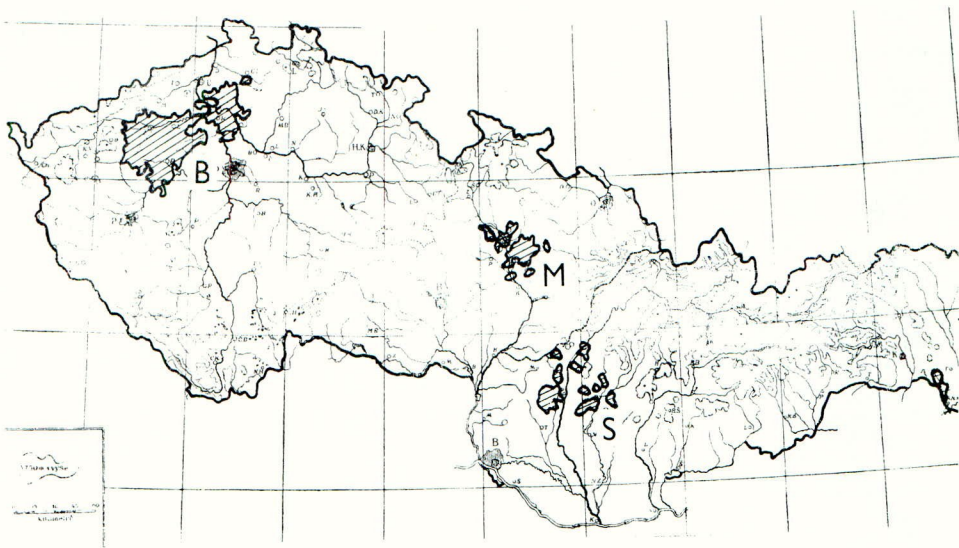


Fig. 1. Main hop growing areas in Czechoslovakia: C - Czech with centre at Žatec, M - Moravian with centre at Tršice and S - Middle Slovakia with centre at Piešťany.

### Cross-resistance

Resistance in hop aphid probably originated in our country as a result of the selective pressure of several insecticides. Originally, dimefox waterings were used in the years 1956-1965. Teration (O-methyl-O-ethyl-S/2 ethylmerkptoethyl/dithiophosphate+thiomethon+disyston) followed later (since 1963) for watering and thiomethon for spraying. In the meantime, as expected, populations in Czechoslovakia have become resistant to all foregoing insecticides (Hrdý et al., 1970). But in addition, cross-resistance was also found to two newly

synthesized carbamates never before applied in practice (Hrdý & Kuldová, 1970). Data on cross-resistance are summarized in Tab. 1.

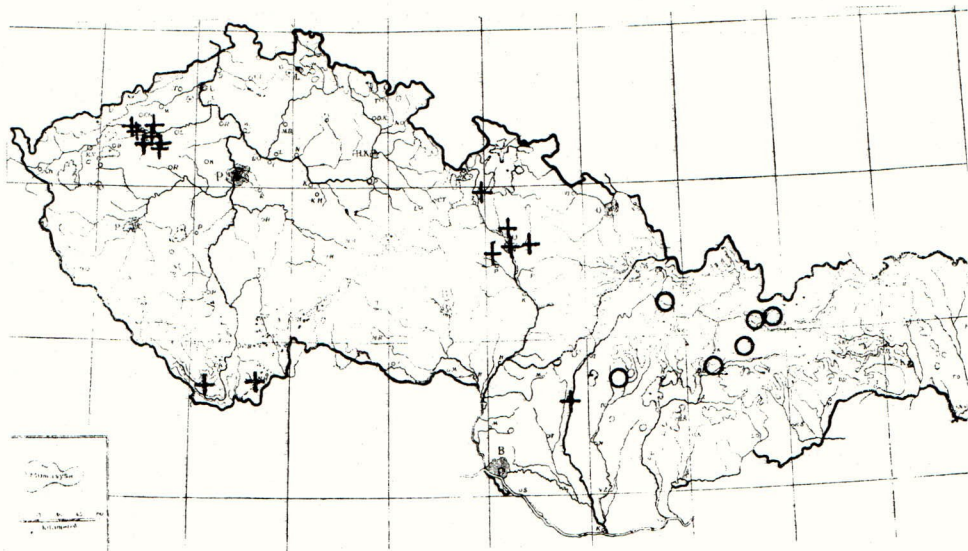


Fig. 2. Distribution of resistant (+) hop aphids according to monitoring in 1967-1971, and distribution of susceptible (o) populations on wild hop in 1971.

The insecticides phosalone and methomyl deserve mentioning among substances holding promise against resistant populations in our country. Presently, many compounds are being tested at the Hop Growing Research Institute. For instance, to rule out the risk of phytotoxicity, substitution of chlorpyrifos for mevinphos is under consideration. In recent watering tests (Kříž, 1973) dimefox invariably provided the best results despite the mounting resistance to this insecticide. From this it may be inferred that only little expectation can be raised in favour of other insecticides under evaluation (oxamyl, mephopholan, Teration, phorate, formothion).

#### Ecological impact

In spite of the elimination of thiometon from hop control the level of resistance to this compound has remained unchanged or has even been moderately increasing: in the Žatec area RF was ab. 5-6 in 1967, 6.5-6.9 in 1968 and 7.5-10.7 in 1971-1973. The increase due to suppression of originally sensitive populations is shown in Figs 3a,3b. At the same time the total effectiveness of chemical treatment has a decreasing tendency as shown in Tab. 2. This table presents annual average numbers of aphids on treated and untreated plots based on observations conducted in an experimental hop garden near Žatec. From the T/U index it is evident that since the calamitous outbreak in 1967 the effectiveness of insecticides has been decreasing until 1970-1971. The years 1968 and 1969, however, saw an extremely low population density of aphids and there is reason to believe that chemical treatment was in fact unnecessary.

Table 1

Cross-resistance in field populations of the hop aphid and in green-house populations or clones in the green peach aphid<sup>+</sup>

Insecticide	common name	Phorodon humuli RF at LC50	Myzus persicae RF at LC50
Phosdrin	mevinphos	5.5	40.4 Cl
Azodrin	monocrotophos	4.8	11.0 Cl
Bidrin, Carbicron	dicrotophos	4.6-3.1	
Dimecron	phosphamidon	6.8	
Ekatin, Intration	thiometon	7.6-10.7 <sup>+</sup>	20.0 St 5.6-91.3 St Cl
Thimet	phorate	(1.8)	(8.4)
Metation	fenitrothion	3.6	
Anthio	formothion	(4.3)	
Bi 58EC, Rogor	dimethoate	19.3-22.7	
Fitios	etoatemethyl	23.8	
Nogos	dichlorvos	1.6	
Teration	/1/	2.9 (27.4)	
Terra Sytam	dimefox	3.3 (11.4)	
Zolone	phosalone	1.0	
Cytrolane	mephosfolane	(2.5)	16.9 St, 1.4 Cl
Ultracid, Supracid	methidathion	3.8	39.3 Cl
Gusathion	azinphosmethyl	1.2	5.7 Cl
Dursban	chlorpyrifos	-1.8	
Folimat	omethoate	6.9	
Thiocron	amidithion	13.8	
Diazinon	diazinon		15.7-73.5 St, Cl
Fosfotion	malathion		2.3-50.6 St, Cl
E 605f	parathion		5.3-59.8 St, Cl
Furadan	carbofuran	1.1	
Lannate	methomyl	-1.3	
Vydate	oxamyl	(9.6)	
VUAgT 93	/2/	18.0 Cl	140.7 St
VUAgT 113	/3/	12.0 Cl	64.7 St
Talcord	thiocarboxine		1.7 Cl
Pirimor	pirimicarb		3.5 Cl
Thiodan	endosulfan	1.1	

/1/ O-methyl-O-ethyl-S-(2ethylmerkptoethyl) dithiophosphate + thiometon + disyston

/2/ 2-methyl merkptomethylphenyl-N-methylcarbamate

/3/ 6-methylmerkptomethyl-3-methylphenyl-N-methylcarbamate

<sup>+</sup> According to Hrdý & Kuldová (1970), Hrdý & Kříž (in print), Hůrková (1970, 1973).

<sup>++</sup> In brackets - systemic insecticides tested by spraying

St - Strain or population sample

Cl - clone

The original forecasting method with index number  $i$  determination was based on the premise of high density of outbreak distribution, when  $i = 0.1-8$  ( $i = p/v$ ).<sup>†</sup> Since about the introduction of organophosphorous agents the originally used scale has become unreliable a new one has been introduced: 1)  $i = 0-20$ , high density cover; 2)  $i = 20-40$ , medium-density cover; 3)  $i = 40-60$ , low-density cover; 4)  $i =$  above 60, very-low-density cover. When, during the past two decades,  $i$  was determined as a mean for the entire hop-growing region, the general rule applied that with  $i$  less than 15, occurrence of aphids was heavy, whereas with  $i$  greater than 100 it was very low.

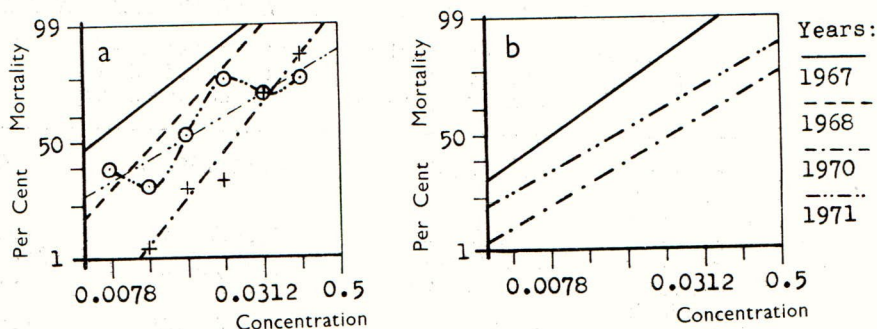


Fig. 3a Increase of resistance to thiometon at Tršice in Moravia. Fig. 3b Increase of resistance to thiometon on chemically untreated wild hop (locality Olomouc). + -.- + Calculated line, o -.- o Broken curve: mixture of R and S aphids in the population.

If, the mean was between 15 and 100, the estimate was inaccurate. Refinement of estimation through the study of aphids on primary hosts has become an absolute necessity. Repeated chemical treatment has engendered damaging effects on predators with the result of weakening the resistance of the environment. As a result, even very low initial population densities entail the risk of calamitous outbreaks.

#### Green peach aphid - *Myzus persicae*

From the point of view of resistance this pest belongs among species studied in most detail. Resistance in this species was reported for the first time from the USA (Anthon, 1955) and from Switzerland (Wiesman, 1955). In the USA, since ab. 1952-1953 (Landis & Shopp 1958; Shirck, 1960; Georghiou, 1963; Harding, 1973) this resistant aphid has been causing trouble on field cultures in warmer regions (California, Texas, Washington, Florida). In England (Dunn & Kempton, 1966; Russel, 1965; Wyatt, 1965) and similarly in other colder regions of Europe difficulties have been previously recorded only in the control of green-house populations. Actually, resistance to organophosphorous insecticides from sugar beet fields in England is reported (Devonshire & Needham, 1975). In the South of Europe, however, similar to the USA, R populations have been found on the peach and the tobacco (summarized by Boness & Unterstenhöfer, 1974). Ac-

<sup>†</sup>  $p$  = examined number of buds and buds sears on apical plum shoots  
 $v$  = number of hop-aphid winter eggs found

According to recent findings there have been difficulties in controlling virus vectors in sugar beet and the potatoes in milder regions. In green-houses in Czechoslovakia, extensive failure of several organophosphates and some carbamates was experienced and evidence was produced on high levels of resistance to these compounds in the aphid populations (Hůrková, 1970). Monitoring has shown the resistance of green-house populations to be a general phenomenon.

Table 2

Average numbers of hop aphid and the over-year insecticide stress

Year	Insecticides in use	Average number of aphids per leaf		T/U <sup>†</sup>
		Treated plot Winged/Apterous	Untreated plot Winged/Apterous	
1967	Teration thiometon mevinphos	0.42/29.75	0.60/168.22	5.65
1968	dimefox	0.02/0.34	0.06/2.44	(7.18)
1969	dimefox	0.10/0.44	0.48/14.87	(33.80)
1970	dimefox	9.22/12.69	13.25/70.76	2.44
1971	methidathion	4.29/58.00	4.53/89.08	1.50
	dimefox			
	methidathion mevinphos			

<sup>†</sup>T/U is the cumulative over year count of aphids on the untreated control in relation to the number of aphids from insecticide treated plot expressed as 1.

#### Cross-resistance

In all samples cross-resistance, or at least some degree of tolerance in the green-house populations under scrutiny was found in comparison with the sensitive standard (Tab. 1; Hůrková, 1973). According to a RF calculated at LC50 the highest effect on R populations was produced by mephospholan and thiocarboxine, followed by azinphosethyl and pirimicarb. In all cases however, the slope of regression lines was flatter in R strains (particularly with azinphosethyl, mephosfolane, pirimicarb) as compared with S strain. In this way predisposition seems to be established for further selective development of more specific resistance.

The study of M. persicae provided some degree of insight into detoxication enzymes considered at least partly responsible for the phenomenon of resistance (Needham & Sawicki, 1971; Beránek, 1974). Also, some data were obtained on the relationship between resistance and the bionomical-ecological forms (Eastop & Banks, 1970). These findings invite interpretation in terms of biotypes as coined by American authors. Evidently, the uncertainties are related to the still unresolved taxonomic problems involving both Myzus persicae and Aphis fabae as revealed by some authors in whose conception both are spoken of as group species.



### Black bean aphid - Aphis fabae

An important pest particularly on sugar beet in Europe, whose populations have been under heavy insecticide pressure for many years. This notwithstanding, there were no reports on developing resistance until recently. Only as late as 1974, Bonnes and Unterstenhöfer (1974) reported on samples from Arnhem (Holland) in which they found 3-15 fold resistance to parathion and demeton and 5-20 fold resistance to propoxur. In the current season in Czechoslovakia low resistance to thiometon in several populations from sugar beet fields was found (Hlináková & Hůrková, in print). The RF fluctuated between 1.5 and 4.8 at LC50. There have also been reports on insufficient effectiveness of thiometon in the practical control of this pest.

### Cross-resistance to juvenoids in aphids

Synthetically prepared compounds with juvenile-hormone activity hydroprene and kinoprene were tested on an R strain of M. persicae (RF=26 for malathion), on an S strain of A. fabae as well as on both susceptible, and slightly resistant strains of Therioaphis maculata (in the R strain the RF for malathion was 5.4). The results were discussed in more detail in a recently published paper (Hrdý, 1974).

Table 3

### Effects of juvenoids on insecticide resistant and susceptible aphids

Species	Strain	Develop. stage tested	Compound	EC 50 <sup>+</sup>	b <sup>++</sup>
<u>M. persicae</u>	R	Nymph 3	hydroprene	0.0027	1.07
<u>M. persicae</u>	R	Nymph 3	kinoprene	0.00043	1.14
<u>A. fabae</u>	S	Nymph 3	hydroprene	0.024	1.16
<u>A. fabae</u>	S	Nymph 3	kinoprene	0.0105	0.55
<u>T. maculata</u>	S	Larva 1	hydroprene	0.207	4.92
<u>T. maculata</u>	R	Larva 1	hydroprene	0.273	4.73
<u>T. maculata</u>	S	Larva 1	kinoprene	0.107	5.22
<u>T. maculata</u>	R	Larva 1	kinoprene	0.0855	4.04

+ Scored according retention of larval character of the cauda  
 ++ Slope of the regression line  
 Nymph - with wing pads  
 Larva - without wing pads

Cross resistance to particular juvenoids as evident on other species (Dyte, 1972 - Tribolium castaneum; Cerf & Georghiou, 1972 and others - Musca domestica) obviously will be equally present in aphids. This seems to be true for hydroprene whereas kinoprene showed a somewhat more marked effect on the resistant aphids as compared with those sensitive to insecticides (Tab. 3).

### DISCUSSION

It would be difficult to discuss the problem of resistance in aphids in general terms. I would rather concentrate on some particular aspects only. There is first of all the question of the conditions

underlying the development of resistance, and further the rate of this development. Thus for example we witness the expansion of resistance in green-house populations of M. persicae. Conclusive evidence has accumulated, almost simultaneously in several places, of the flood-like spread of resistance in P. humuli. On the other hand there have not been any complaints for a long time, on the part of the plant protection service, about A. fabae, nor was any laboratory evidence available on resistance in this species. There are, doubtless, biological-ecological factors coming into operation, such as those mentioned by Baranyovits (1973). Moreover, there are conspicuous differences in the slopes of regression lines corresponding to the original, notselected populations, of M. persicae and P. humuli. These regression lines are markedly flatter than the steep ones encountered with A. fabae which seem to have a much narrower variability in their response to insecticides.

It is of practical importance to be able to pick out the insecticide which holds most promise for future application against resistant aphids. By the example of the juvenoids, for which resistance was proved even before their introduction into practice, it was clearly shown how important it is that also essentially new insecticides be subjected to experimental check. P. humuli provided another example in revealing resistance to two carbamates which were still under development. In the field we find ourselves faced with an intricate situation. It is obviously possible for different populations of the same species to acquire resistance simultaneously in different places under totally different specific conditions (selective pressure by various insecticides). Moreover, active flight of aphids, or even passive transfer of resistant individuals seems to be a commonplace phenomenon furthering the selection of the fittest. Due to their specific ability these individuals are particularly capable of surviving in the new environment on chemically treated plants. This is bound up with the problem of vigour in the resistant aphid populations. It seems that in the so-called biotypes the biological-ecological characters favourable to these species are closely linked with some resistance, or tolerance to insecticides. The close relationship in aphids between change in resistance and change in bionomy, or ecology has been shown in studies on these aphid biotypes, for example the spotted alfalfa aphid (Nielson et al., 1970), and occurs probably in the green bug (Schizaphis graminum) (Wood & Starks, 1972). The implication in these species is evidently the combined impact of pesticide stress and environmental pressure which leads to the emergence of new, more resistant and more vigorous populations. More thorough study is necessary to elucidate the problem of whether under specific conditions there might be a chance in terms of breakdown of resistant populations that is a disappearance of resistance, which, in principle, ought to be possible in species with high one-gene specific resistance.

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TOLERANCE OF FUNGAL PATHOGENS TO SYSTEMIC FUNGICIDES IN RELATION TO DISEASE CONTROL

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Summary Although records of pathogen tolerance to systemic fungicides are common there is less information on tolerance associated with failure in disease control and yield loss. The best documented records are for pathogens tolerant to the benzimidazoles eg. Cercospora beticola, Cercospora arachidicola, Botrytis cinerea, Verticillium fungicola, Penicillium italicum and Penicillium digitatum. Tolerance and disease control failure has also been reported with the hydroxypyrimidines especially dimethirimol. Ways of avoiding tolerance have been investigated particularly the effects of fungicide programmes involving mixtures of materials with different modes of action.

Studies of tolerance and disease control failure must take into account the limitations of the detection method. Representative pathogen populations should be studied before making conclusions of the response of a pathogen to a particular fungicide. Disease control failure has frequently followed 2-5 years of high selection pressure on a pathogen which produces numerous air-borne spores. In some instances tolerance may have been predicted before the release of the fungicide emphasising the necessity for detailed pre-release work not only on the target pathogen population but also on the best use of the fungicide once it has been released.

INTRODUCTION

Since their introduction in 1968 many of the new systemic fungicides have been used on a world wide scale often resulting in dramatic decreases in disease incidence. Fungal pathogen tolerance before the introduction of systemic fungicides was rare (Georgopoulos and Zarcovitis 1967) with the exception of tolerance to some fungicides broadly classified as aromatic hydrocarbons and also to some mercury compounds. Plant Pathologists have had little experience with problems arising from pathogen tolerance and there is little information on the best way to use fungicides in order to minimise the chance of tolerant strains arising. Predictions of the consequences of the misuse of systemic fungicides have been plentiful (Spencer 1972, Dekker 1972, Wolfe and Dinooor 1973). The subject of fungal pathogen tolerance to the new systemic materials has been reviewed by various authors (Dekker 1972, Erwin 1973, Wolfe and Dinooor 1973, Greenaway and Whatley 1975) and the list of tolerant pathogens to one or other of these fungicides is increasing steadily.

For tolerant strains to become important and cause crop loss they must be:-

- a. Genetically stable and become dominant in the pathogen population.
- b. As aggressive and virulent as the sensitive strains.
- c. Equally competitive as the sensitive strains in their saprophytic phase.

- d. Have no limitations in their epidemiology, eg produce large numbers of spores and over-season effectively.

#### TOLERANCE AND DISEASE CONTROL FAILURES

Failure to control plant diseases may result in economic loss either as a result of reduction in yield or quality or because the presence of the disease necessitates changes in cultural practice. The development of tolerant strains can therefore have serious economic consequences both for producers and for fungicide manufacturers. Disease control failures attributable to tolerant strains have been reported for many of the major groups of the new systemic materials.

##### Carboxylic acid anilides

This group includes the oxathiins and the most commonly used fungicides in the group are carboxin and oxycarboxin. Tolerance to these fungicides has been induced (Grover and Chopra 1970, Y Ben-yephet et al 1974 and 1975) although there have been no reports of *in vivo* occurrences. Y Ben-yephet et al (1974-1975) working with laboratory induced tolerant mutants of Ustilago hordei showed that they were stable and when mixed with sensitive strains competed less well and were less aggressive but did not totally disappear from the population. Disease control was not successful when plants inoculated with the tolerant mutants were treated with carboxin.

##### Benzimidazoles and thiourea based fungicides

This group includes fungicides with a board spectrum of activity which have been used on many diseases throughout the world. The best known fungicides are benomyl, carbendazim, thiophanate methyl and thiabendazole. More work has been reported on disease control failure within this group than any other. Generally, cross tolerance occurs between the various fungicides in the group.

The work of Georgopoulos and Dovas (1973) on Cercospora beticola in Greece has shown how benomyl firstly gave excellent control of this disease, but within three years of its first use it was no longer effective. It was first used in Greece in 1969 and gave good disease control in 1970-71, but in late 1972 in Northern Greece large areas of benomyl treated sugar beet showed severe disease. Repeated benomyl sprays failed to control these outbreaks and a considerable loss in yield and sugar content occurred over 25,000 acres. Georgopoulos and Dovas showed that there was a close relationship between the number of benomyl sprays in 1971 and 1972 and the incidence of tolerance (Table 1). When tolerant strains were used in infection experiments, the disease was not controlled by applications of benomyl and cross tolerance was shown to other benzimidazoles.

Table 1

Relationship between numbers of benomyl applications and  
incidence of *Cercospora beticola* tolerance

(After Georgopoulos and Dovas 1973)

Field No.	Benomyl Sprays		% Tolerance
	1971	1972	
1	0	0	8
2	0	4	62
3	0	7	98
4	3	6	85
5	3	7	100

Ruppel and Scott (1974) reported a very similar situation with *C. beticola* in Texas. There was outstanding disease control with benomyl in 1971-72, but in 1973 some treated fields had severe leaf spot which was not controlled by additional benomyl applications. All the isolates of the pathogen from fields with one or more applications of benomyl, were tolerant. Subsequently, Ruppel (1975) showed that the degree of cross tolerance between benomyl, thiophanate methyl and thiabendazole varied with the isolates, fungicides and fungicide concentration. Benomyl was shown to be more toxic to conidia than hyphae and thiabendazole was more toxic to hyphae than conidia.

*Cercospora arachidicola* causes a serious leaf spot disease of peanuts in various parts of the world and in Southern Alabama the disease was well controlled with benomyl for three years (Clark, Backman and Rodrigues-Kabana 1974). However, in 1973 inadequate control was noticed in several fields when the recommended benomyl programme was used. Clark *et al* were able to demonstrate differences in incidence of tolerant strains according to the treatment of the sampled crop (Table 2).

Table 2

Incidence of benomyl tolerance of *C. arachidicola* in relation to  
crop treatment (after Clark, Backman and Rodrigues-Kabana 1974)

Field treatment	% growth (long term) of single spore cultures of <i>C. arachidicola</i>	
	FDA	FDA + 5 ug benomyl/ml
No benomyl	100	0
Benomyl 1973 and one other year - good disease control	100	1
Benomyl 1971-2-3 inadequate control	100	44

Working on the same disease in Georgia, Littrell (1974) showed in a plot experiment that the incidence of tolerance was related to the use of benomyl but although the percentage defoliation was considerable in the benomyl treated plots, this fungicide still gave the highest yield (Table 3).



Table 3

Influence of fungicide sprays on benomyl tolerant isolates of  
*C. arachidicola* in relation to defoliation and yield of  
peanuts (After Littrell 1974)

Fungicide	% Tolerance	% Defoliation	Yield in Kg/Ha
Benomyl	31.8	51.1	4532
Chlorothalonil	0	39.0	4440
Control	0	55.2	1922

In a survey in Georgia, Littrell reported tolerant isolates of *C. arachidicola* from 6 of the 13 locations examined.

Miller and Fletcher (1974) and Fletcher and Scholefield (1976) monitored the extent of tolerance in populations of *Botrytis cinerea* on tomatoes from one site following the introduction of high volume sprays of benomyl. They found that in 1972, '73 and '74, 0, 31 and 70% of the isolates examined were tolerant respectively. Whereas in 1972 and 1973 benomyl resulted in significant decreases in disease incidence and yield increases, it did not in 1974.

The benzimidazoles, and in particular benomyl have been used in the mushroom industry throughout the world giving excellent control of the major fungal pathogens (Holmes, Cole and Wuest 1971, Fletcher, Drakes and Talent 1974, Gandy 1972). In the UK benomyl was used from 1970 onwards, but in 1973 first reports of possible benomyl tolerance of *Verticillium fungicola* occurred. In 1974 a survey (Fletcher and Yarham in press) showed that tolerance was widespread and could be correlated with disease severity in that crops with the highest level of disease had the highest incidence of tolerance. Similar results have been reported by Bollen and van Zaayen (1975) for the Netherlands.

One of the first uses of the benzimidazole fungicides was for the control of citrus fruit rots. Thiabendazole has been widely used as a fruit dip and also incorporated into a wax which is used on the fruit surface. Harding (1972) reported strains of *Penicillium italicum* and *P. digitatum* from citrus pack-houses in California and Arizona following three years use of thiabendazole. He reported that dips of thiabendazole were effective against the sensitive strains he used, but not against the tolerant forms. Similar results were obtained with both fungi. Benomyl did not control the disease caused by the tolerant fungal strains but was more effective than thiabendazole. In Florida Smoot and Brown (1974) found that thiabendazole successfully controlled citrus fruit rots in pack-houses for 4-5 years. In 1973 a high incidence of green mould (*P. digitatum*) was found in two pack-houses and 65 of the 67 isolates tested were found to be tolerant to thiabendazole. The affected pack-houses had stored fruits for longer periods than normal which allowed infected fruits to sporulate. Similar results have also been published from Israel (Gutter 1975). Green mould (*P. digitatum*) was the dominant post harvest pathogen in Israel for many years, but recently blue mould (*P. italicum*) has become increasingly frequent. This trend has been associated with the use of the benzimidazoles, and Gutter has shown that these two pathogens differ in their sensitivity to these fungicides allowing the less sensitive *P. italicum* to increase.

Dennis (1975) found that the incidence of *Botrytis* rot of strawberries increased with the development of tolerance to benomyl, and Griffee (1973) reported benomyl tolerant isolates of *Colletotrichum musae* from treated bananas.

## Hydroxypyrimidines

The two most commonly used materials in this group are ethirimol and dimethirimol. Both of these fungicides have a narrow spectrum of activity and are only effective against specific powdery mildews. Ethirimol has been widely used on barley crops for the control of Erysiphe graminis both as a seed treatment and as a spray. King (1973) reported its use as a seed dressing on barley crops in England and Wales (acreage c.4.5 million acres) as 10, 14 and 24% for 1972, 1973 and 1974 respectively and 25% for 1975 (King personal communication). Methods for identifying tolerance depend upon the uptake of the fungicide by the test plants and the fungus isolates are then screened on leaf pieces from plants given known doses of fungicide (Wolfe and Dinooor 1973). There are reports in the literature of isolates of E. graminis which are less sensitive to ethirimol than others (Wolfe and Dinooor 1973, Shepherd and Bent personal communication), but it is not clear by how much these differ from the normal range of variation which might be expected within a population of the pathogen not exposed to ethirimol. Wolfe and Dinooor (1973) were able to show that there was an increasing incidence of less sensitive forms of the pathogen according to the treatment of the crop and its nearness to a treated crop. Shepherd and Bent found an increase in tolerance of isolates from treated crops when compared with isolates from adjacent untreated ones, but yields were improved with fungicide treatment in spite of this. Although strains of E. graminis, somewhat tolerant to ethirimol, commonly occur in treated barley crops, there is, as yet no positive evidence that these are affecting either the level of disease control or reducing the yield increase expected from ethirimol seed treatment.

In contrast, dimethirimol tolerance to Sphaerotheca fuliginea has been shown to be widespread and positively associated with disease control failures (Bent, Cole, Turner and Woolmer 1971). This fungicide was widely used in cucumber crops from 1968 onwards and in 1968-69 have excellent disease control. In 1970 in the Netherlands control was good in the early months of the year, but by April a number of failures had occurred. When isolates from 38 sites where control was unsatisfactory were examined, tolerance was found from all but two, whereas control was good on five of the six sites where sensitive isolates predominated. Similar results have been reported from England, but although the material has been used for some time in Israel, 1969-71, Spain 1970-71 and the Canary Islands 1970-71, tolerance has not been reported from these countries.

## N-substituted tetrahydro-1, 4-oxazines

Tridemorph and dodemorph are the most commonly used fungicides in this group and have a narrow spectrum of activity affecting mainly powdery mildews. Tridemorph has had widespread use on barley crops for the control of Erysiphe graminis in England and Wales since 1970 with 3, 7 and 11% of the barley acreage treated in 1970, 1972 and 1973 respectively (King 1973). In 1974 and 1975 16 and 11% of the acreage was treated (King personal communication). In spite of this there have been no reports of disease control failures, or tolerant isolates (Wolfe, Wright and Minchin 1974). Similarly, dodemorph has been used to control powdery mildews on a range of ornamental plants (Frost and Pattison 1971), but there have been no reports of tolerance either naturally occurring or induced.

## Triforine

Triforine has a wide spectrum of activity including powdery mildew of cereals, cucumbers, apples and various ornamentals as well as rusts on cereals and apple scab (Schicke and Veen 1969). Chloraniformethan, also in this group, has been used for the control of barley mildew. There are no records of fungal pathogen tolerance to these materials.

## AVOIDING TOLERANCE

Disease control with systemic fungicides will be effective in spite of pathogen tolerance providing ways are found of preventing tolerant strains becoming dominant in the population (Wolfe and Dinooor 1973). Recent researches on the behaviour of tolerant strains in fungal pathogen populations have been in two main areas:-

- a. Ecological studies involving competition between strains and the epidemiology of strains.
- b. The manipulation of fungicide treatments and the exploitation of fungicide mixtures.

### Ecological Studies

For a tolerant strain to become dominant it must be able to compete in the pathogen population with the sensitive isolates in its pathogenic phase and also in some cases as a saprophyte. This is particularly important for the survival of some pathogens between crops. Jordan and Richmond (1974) studied the competition of three isolates of Botrytis cinerea on strawberries growing in a polythene tunnel. Two isolates were benomyl tolerant and one was slower growing, although more freely sporulating on agar than the other. The sensitive isolate grew quickly and sporulated freely. They found that the amount of fruit rot with the three isolates was not significantly different in the absence of benomyl, but in plots where the fungicide was used there was more fruit rot where the tolerant strains predominated. The faster growing tolerant isolate was found in a number of plots including the uninoculated controls and those originally inoculated with the sensitive strain. Bollen and Scholten (1971) had previously reported unprecedented losses of benomyl sprayed cyclamen caused by a tolerant strain of B. cinerea.

Richmond, Pring and Tarr (1973) found that benomyl tolerant isolates of B. cinerea from strawberries were as pathogenic as sensitive isolates and able to survive in the field even in the absence of benomyl. Similar observations were made by D.M. Ann (personal communication) who examined strawberry crops following a number of seasons of different benomyl spray treatments. He found that tolerant strains of B. cinerea declined after two seasons in which benomyl was not used. Wuest, Cole and Sanders (1974) and Griffee (1973) have reported benomyl tolerant strains of Verticillium malthousei and Colletotricum musae respectively which were less virulent than the sensitive isolates. Whether such isolates are capable of causing epidemics in the presence of the selection pressure from the fungicide is not known. Fletcher and Yarham (in press) and Bollen and van Zaayen (1975) in the UK and Netherlands respectively have shown that tolerant strains of V. fungicola (syn V. malthousei) are associated with crop loss. A number of the tolerant isolates (19%) examined by Fletcher and Yarham were slower growing on agar than sensitive isolates, but these were not associated with lower disease incidence. Y Ben-yephet et al (1974) working with mixtures of tolerant and sensitive strains of Ustilago hordei and Harding (1972) working with similar mixtures of strains of Penicillium italicum and Penicillium digitatum found that in competition as pathogens in the absence of the fungicide the tolerant strains declined as a proportion of the population, but were not lost.

A further way of reducing the selection pressure is to limit the exposure of the pathogen to the fungicide. This principle has been applied with ethirimol for the control of Erysiphe graminis on barley (Wolfe and Dinooor 1973). The use of this fungicide on the winter barley crop has been prohibited in the U.K. in order to minimise the amount of less sensitive inoculum reaching spring barley. A break of this type may help to prevent the gradual increase in the degree of insensitivity of E. graminis to ethirimol.

## Fungicide Management

One possible way of preventing tolerance developing is to apply a mixture of fungicides with different modes of action in a programme of sprays. This reduces the selection pressure of one material and helps to minimise the build up of fungicide-specific tolerance. This system was successfully used by Littrell (1974) for the control of benomyl tolerance of Cercospora arachidicola. He found that only 5% of the single spore isolates from the benomyl plus chlorthalonil treatment were benomyl tolerant compared with 56% from the benomyl treatment. Fletcher and Scholefield (1976) working with benomyl tolerant Botrytis cinerea from glasshouse grown tomatoes found that combinations of dichlofluanid and benomyl did not reduce the incidence of benomyl tolerance in the plots. Tolerant strains were dominant throughout the experiment including the unsprayed control. Jordan and Richmond (1974) found that alternations of dichlofluanid and benomyl successfully controlled Botrytis cinerea on strawberries, although the isolates obtained from rotting fruits were benomyl tolerant. Changes in the proportion of the benomyl tolerant fraction of a population of Cercospora apii were recorded by Berger (1974) following applications of maneb. He found that changing the programme from benomyl to maneb resulted in a decrease in the proportion of tolerant isolates whilst the reverse change gave an increase in the tolerant proportion. These results are similar to those reported by Ebben and Spencer (1973) for Sphaerotheca fuliginea on cucumbers. They inoculated a crop with strains tolerant to both benomyl and dimethirimol and found that the disease could be controlled by applying the two fungicides either alternately or together.

## DISCUSSION

Disease control failure in the field following systemic fungicide application may be the result of a number of factors such as incorrect dosage, poor uptake, inadequate crop cover or degradation of the active ingredient, in addition to pathogen tolerance. All these factors must be taken into account in conjunction with tolerance test results when assessing the performance of a fungicide.

A pre-requisite for any study of tolerance is a suitable and reliable technique for its identification. The fungicide can generally be incorporated into the culture medium and growth, sporulation and spore germination measured with facultative pathogens. With obligate parasites, particularly powdery mildews, the method is more complex as it involves the testing of isolates on plant material pre-treated with the systemic fungicide. The problems of pathogen manipulation as well as uptake and translocation of the active ingredient makes such tests difficult and less reliable.

The results of the tolerance tests can only be satisfactorily interpreted if adequate data is available on the behaviour of the pathogen population to the fungicide. During the establishment of the spectrum of activity of new materials and the response of specific pathogens, many laboratory tests are done and ED<sub>50</sub> (median effective dose) values determined (Edgington and Barron 1967, Bollen and Fuchs 1970, Edgington, Khew and Barron 1971). Such work gives an indication of the normal response of the pathogen. This information is vitally important when monitoring the in vivo response of pathogens to particular materials. Inevitably, many of the ED<sub>50</sub> values stated in the literature are based on the examination of a small number of isolates, sometimes only one, and subsequent work has shown that they were not characteristic of the pathogen population as a whole.

The fact that tolerance would arise with some pathogens could have been predicted if sufficiently large numbers of isolates of these pathogens had been

tested prior to the release of the fungicide. Wuest et al (1974) were able to show that considerable variation in tolerance occurred in populations of Verticillium fungicola (syn. V. malthousei) well before the introduction of benomyl, and recent reports of epidemics of this disease in the Netherlands (Bollen and van Zaayan 1975) and the UK (Fletcher and Yarham in press) illustrates the consequences of continuously exposing this pathogen to benomyl. Variability has been shown to exist within populations of Erysiphe graminis to ethirimol (Wolfe and Dinooor 1973) but at present with a single application of the fungicide there has been no measurable failure of this material in terms of disease control or yield. A trend towards increasing numbers of applications of fungicides to barley could increase the selection pressure on the pathogen, particularly if sprays of ethirimol are combined with seed treatments.

The spectacular disease control obtained with some of the new systemic fungicides guarantees their widespread use. Effective materials with a wide spectrum of activity are likely to be used on many crops the world over. Although there have been numerous reports of tolerance, records of disease control failure associated with pathogen tolerance are less numerous. Failure to obtain disease control has often followed two or three years of frequent applications of the systemic fungicide, eg. Cercospora beticola tolerance to benomyl in Greece and the USA, Botrytis cinerea tolerance to benomyl in the UK, Verticillium fungicola tolerance to benomyl in the Netherlands and UK, Sphaerotheca fuliginea tolerance to dimethirimol in the Netherlands. Thiabendazole tolerance in the citrus fruit moulds (Penicillium digitatum and P. italicum) was found after three, four or five years use respectively in various parts of the USA. With all of these diseases the selection pressure on the pathogen has been considerable. But high selection pressure is not the sole factor responsible for the development of tolerant strains as shown by Tate et al (1974) for Monilinia fructicola on stone fruits in California and by Fletcher and Yarham (in press) for Lycogone perniciosa in mushroom crops. In both cases the pathogens have been subjected to many benomyl applications but tolerance has not been reported.

Tolerance and control problems have often been associated with pathogens that produce numerous air-borne spores. However some pathogens of this type have been effectively controlled for a number of years with systemic fungicides using single or a few fungicide applications, eg. ethirimol control of Erysiphe graminis and benomyl control of Botrytis allii. With the latter pathogen the fungicide has been used as a seed dressing since 1973 (Maude, Presley and Walker 1974) and tolerance has not yet been found (R. B. Maude personal communication).

With the present policy of fungicide testing and use, it is likely that many promising fungicides will be lost by the rapid development of tolerant strains. It has been suggested (Greenaway and Whatley 1975, Wolfe and Dinooor 1973) that their useful life may be prolonged by monitoring fungi to provide an early warning of the development of tolerance. Such monitoring of fungal populations is invaluable for the study of the effects of treatments on the incidence of tolerance (Berger 1973, Littrell 1974, Fletcher and Scholefield 1976), but it has limited value where alternative control measures are not available and cannot be put into practice once tolerance is found. Also, once a fungicide has become part of standard commercial practice, the industry may not accept limitations on its use when faced with epidemic attacks of diseases. The greatest long term success with systemic materials is therefore likely to be best achieved by well formulated policies by manufacturers so that when new materials are introduced, they are used in the best possible way to minimise the likelihood of tolerance developing. Serious consideration should be given to discarding all test fungicides with mutagenic properties. When fungicides are initially screened a representative population of target pathogen isolates should be used wherever possible to check whether tolerance is likely to arise or be present within the unexposed population. If tolerance is

detected at this stage the fungicide may not remain effective for long. Its satisfactory performance in commerce may be prolonged if the selection pressure of the fungicide on the pathogen is minimised possibly by use in combination with other fungicides possessing different modes of action.

Growers and farmers can assist by using fungicides at the recommended rates. Also by strict attention to hygiene the likelihood of tolerance strains becoming dominant in a population can be minimised particularly by preventing survival between crops.

The control of many plant diseases often involved a programme of sprays applied on a routine basis. Epidemiologists could help to prolong the life of systemic fungicides by obtaining information on the most effective use of these fungicides perhaps reducing the number of sprays and therefore the selection pressure on the pathogen population. This approach has worked well with the control of cereal mildew where it has been shown that single well timed sprays result in significant yield increases (Jenkins and Storey 1975).

It is essential that continued attention is given to the problem of tolerance and disease control in order to get the maximum advantage from the research and development work on new products.

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THE EGYPTIAN COTTON LEAFWORM - A UNIQUE CASE OF RESISTANCE

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Summary In Egypt, cotton leafworms and bollworms appear simultaneously during certain periods. Organochlorine insecticides controlled these pests effectively until the 1960s when the cotton leafworm developed resistance, and since then organophosphorus compounds have been used extensively. Chemical control has been complemented by the hand picking of egg masses by children from May onwards, and this has received more attention since the appearance of resistance to organophosphorus insecticides. The use of ovicides to replace partially hand picking has been tried recently for the control of leafworm and American bollworm. Since chemical control is aimed at several pest species, it is important to consider the performance of insecticides against all of these when making recommendations for treatment. Newer insecticides, including synthetic pyrethroids, are being investigated for the control of leafworm and promising results have been obtained.

INTRODUCTION

Egypt is a narrow fertile valley on which its ever expanding population depends for food, clothing and major industrial developments. Egypt has ideal climatic conditions for the continuous propagation of crops and agricultural pests.

The cotton leafworm, *Spodoptera littoralis*, is the major polyphagous pest in Egypt; it breeds continuously throughout the year, attacks cotton and most other economic crops and develops resistance to insecticides readily. Its propensity to develop resistance may stem from the large generation turn-over and its continuous exposure to pesticides over a wide area. Every effort is made to control this pest on cotton with as few insecticidal applications as possible to prevent rapid development of resistance. Thus only one application per season is used on cotton in the Quena district of Upper Egypt and 4-5 applications are used in the Alexandria district of Lower Egypt. This ability to develop resistance creates very serious control problems because the same insecticides are used to control the cotton leafworm and the three bollworms which attack cotton viz. the pink bollworm (*Pectinophora gossypiella*), the spiny bollworm (*Earias insulana*) and the American bollworm (*Heliothis armigera*). This last pest has become in recent years second in importance to the cotton leafworm.

History of the resistance to insecticides of the cotton leafworm

Widespread control of cotton pests with chemicals started in 1950. DDT was soon followed by a DDT-BHC sulphur mixture, which was replaced in 1956 by toxaphene, initially at 1.5 kg a.i./acre, and by 1959 at 4 kg a.i./acre. Toxaphene failed in 1956 and had to be replaced by carbaryl and trichlorphon (Maher Ali et al, 1962).

Soon carbaryl gave poor control in areas with toxaphene (Maher Ali et al., 1962) resistance and toxaphene has been considered to predispose the cotton leafworm to carbaryl resistance (Maher Ali et al, 1965). Laboratory tests showed that the toxaphene resistant insects also resisted endrin (Henein et al, 1964; Maher Ali et al, 1962) which had not been used in the field. Withdrawal of toxaphene led to the almost complete disappearance of resistance to this compound, but resistance reappeared very quickly when endrin was applied later in the 1965 season in areas where trichlorphon was losing its efficacy against late instar larvae of the cotton leafworm (Hassan et al, 1970). The 1965 control season was marked by the introduction of mixtures of organophosphates (OPs), organochlorines and cyclodienes to control the cotton leafworm. Mixtures of toxaphene/DDT/methyl parathion were used satisfactorily, but already by the following season the concentration of methyl parathion had to be increased from 5 to 7.5%. Organophosphorus insecticides with heterocyclic groups, e.g. cyolane, were introduced in 1967 to control the leafworms resistant to methyl parathion and chlorpyrifos was introduced in 1969. Monocrotophos used very extensively throughout Egypt from 1969 to 1972 to control the leafworm and bollworms early in the spraying season is now only used to control the bollworm in some areas of Upper Egypt, because the leafworm is very resistant to this insecticide (Tables 1 & 2). Much of the control of the leafworm now rests on leptophos which was imported in large amounts during 1971-74.

#### Development of resistance and insecticidal usage

There is close connection between the history of insecticidal usage, the development of resistance and increase in the population of the leafworm as shown in Table 3. The largest numbers of egg-masses were collected in 1961 (Fig.1) when toxaphene, and in 1971 when monocrotophos resistance were highest. High egg counts may be explained by either higher reproduction rates and fitness of the resistant individuals (Banks and Needham, 1970) or more likely by the ineffectiveness of the insecticides used to control the resistant individuals, as shown in the following example. Widespread use of monocrotophos on cotton was stopped in 1972 following the almost universal resistance of the cotton leafworm to this compound, but monocrotophos has been used to control bollworms in Upper Egypt in areas where the leafworm is less common. Thus in 1973 the Matay district was sprayed solely with monocrotophos and in 1974 three other districts, Abougurgas, Mallawi and Deir-Mouwass were also treated solely with this compound. In each case this led, as shown in Table 4, to a very large increase in the number of egg masses of the leafworm collected during the following season. The large decrease in the number of egg masses in the Matay district in 1975 was probably caused by the effective control of the leafworm in 1974 with different insecticides.

#### Cross resistance in the leafworm

The examination of the cross resistance of field strains or strains selected with insecticides in the laboratory (Ayad, 1973; Elsayed, 1973; Hassan et al, 1970; Maher Ali et al, 1962; Maher Ali et al, 1965; Toubgi et al, 1965) shows that cross resistance is usually restricted to closely related compounds. There is cross resistance between:

1. toxaphene and endrin.
2. dicrotophos, monocrotophos and trichlorphon.
3. formothion, methyl parathion, tetrachlorvinphos and demephion.
4. carbaryl and lannate.

However, selection with leptophos, parathion, EPN or curacron usually does not lead to cross resistance to may other compounds.

## Attempts to control resistance

A thorough knowledge of the biology and ecology of pests of cotton is indispensable in formulating a control programme aimed at decreasing the development of resistance in cotton pests, and the following measures have been taken in Egypt to decrease the use of insecticides against pests of cotton and in particular against the cotton leafworm and the American bollworm.

1. Irrigation of alfalfa is prohibited by law after 10th May. This checks the build up of populations of leafworm and *Heliothis*, which would move later in the season onto cotton.
2. Hand-picking of egg masses of the cotton leafworm by children when cotton plants are young is about 80% effective. It does not affect the predators of the leafworm and bollworm and prevents the build up and major infestations of leafworm in July.
3. Ovicides that give no cross resistance to insecticides and now tried out to control the leafworm and the American bollworm. These compounds may be used when there are not enough hand pickers, and should be less harmful to parasites and predators than the currently used insecticides.
4. Compounds to which the leafworm has become resistant should not be used for bollworm control. This practice only leads to build up of cotton leafworms. This point is particularly important to remember in areas where *Heliothis* has become a major pest. In those areas only insecticides effective against both pests should be used.
5. A continuous search should and is being made for new and effective insecticides against cotton pests. The new organophosphates (e.g. O-ethyl O-phenyl n-propyl phosphorate) seem very promising, and the light stable synthetic pyrethroids appear to be extremely effective. The effectiveness of these new compounds was tested against the 4th instar larvae of the standard susceptible strain, leptophos resistant strain (RF x 12) and strains reared from eggs collected on cotton in four governates (Table 5). Although the strains reared from eggs collected in the field were almost fully susceptible, there was measurable resistance to the new insecticides in a laboratory selected leptophos resistant strain. This indicates that resistance to these new compounds in the field in the future cannot be excluded and that these very effective compounds must be used with circumspection.

Table 1  
Resistance factor (RF)\* of the leafworm to insecticides

Insecticide	District	1970	1971	1972
monocrotophos	Benisuef	1	14	143
	Behera	5	43	143
	Dakahlia	5	5	19
leptophos	Benisuef	7	16	23
	Behera	2	14	7
	Dakahlia	1	9	5
phosfolan	Benisuef	4	3	4
	Behera	6	1	3
	Dakahlia	1	1	6
endrin	Benisuef	7	63	6
	Behera	36	6	3
	Dakahlia	7	7	46

$$*RF = \frac{\text{LC50 resistant leafworms}}{\text{LC50 susceptible leafworms}}$$

Table 2

## Resistance factor of leafworm to insecticides in Upper Egypt

Insecticide	District	1973	1974	1975
monocrotophos	Minia	1	9	
	Assiut	3	9	
	Souhag	1	9	
leptophos	Minia	2	7	20
	Assiut	5	2	6
	Souhag	11	2	8
methomyl	Minia		12	6
	Assiut		4	2
	Souhag		1	9
phosfolan	Minia	1	1	6
	Assiut	1	1	2
	Souhag	1	1	3
mephosfolan	Minia	1	3	6
	Assiut	3	2	5
	Souhag	1	2	3
chlorpyrifos	Minia	1	3	5
	Assiut	2	3	3
	Souhag	1	1	7

Table 3

## History of application of insecticides in Egypt

Year	Insecticide	Recommendation	Withdrawn (a)	Serious leafworm infestation (b)
1955-61	toxaphene	sole insecticide leafworms & bollworms	1961	1961
1961-65	trichlorphon	sole insecticide leafworms & bollworms	1965	
1965-69	methyl parathion & toxaphene mixture	leafworms		1966
1969-71	monocrotophos	major insecticide leafworms & bollworms	1971	1971
1972	monocrotophos	Upper Egypt only		
1973-75	monocrotophos	bollworms in U. Egypt		
1972-75	leptophos	major insecticide leafworms & bollworms		1974

(a) year when insecticide withdrawn due to leafworm resistance.

(b) year of serious countrywide leafworm infestation.

Table 4

Average number of leafworm egg masses collected per hectare per day  
during three growing seasons in the districts of Minia

District	Season		
	1973 (Sept-Oct)	1974 (June-Sept)	1975 (June-Aug)
Maghagha	258	265	38
Edwa	118	340	35
Benimazar	550	5093	45
Matay	103	8160	90
Samallut	103	253	23
Minia	28	270	45
Abougurgas	63	305	170
Mallawi	58	280	250
Deir-Mou wass	48	253	200

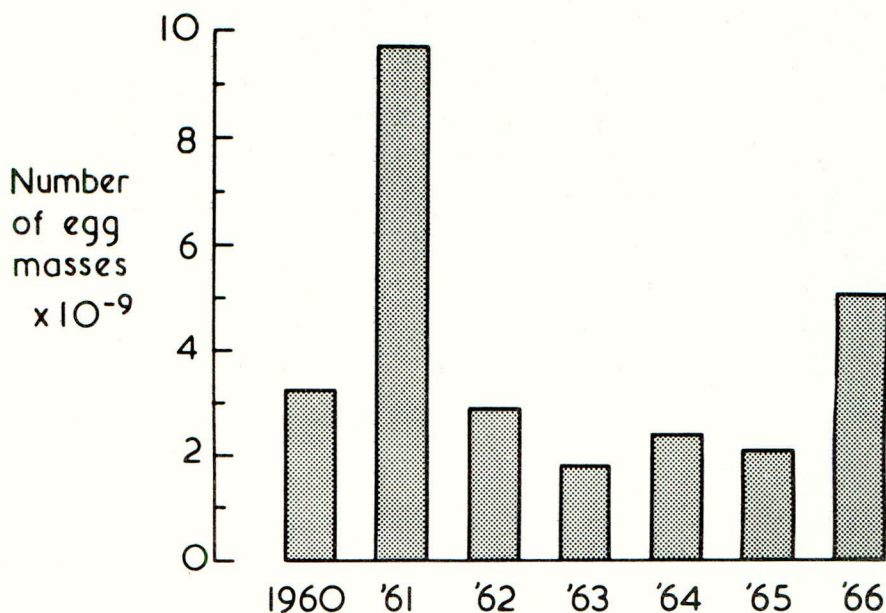
Table 5

Resistance factor of a strain of leafworms resistant to  
insecticides and of strains from several districts of Egypt

Insecticide	Resistant strain	District			
		Fayoum, Dakahlia, Sharkia, Benisuef			
SH 1479	3.3				
SH 1467	11.0				
RUP 951	2.5	1.9	1.6	1.8	
RUP 952	7.6	1.5		0.4	2.6
Permethrin	4.0				
Curacron	6.6	1.0		2.2	0.6

Figure 1

Leafworm egg masses collected from the whole country between 1960 & 1966



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