PART V

EFFECT of GLOBAL WARMING on PLANT DISEASES in UK.

Again, of local interest predominantly

Introduction

Plant pathologists are aware of the long time-scales associated with climatic change, but their concerns are usually more immediate, looking at the effects of microclimate on disease. Microclimate is however, ultimately determined by the macroclimate (Coakley, 1988), and long term changes in the latter will eventually be reflected in the range of plant diseases that occur on crops, and on their importance. There has been little research on climatic change and plant disease and the views expressed here are largely based on extrapolations from the mass of work done on microclimates.

The principal concerns raised by climatic change are:

1) Will the spectrum of diseases found in the UK alter?

2) Which, if any diseases are likely to become more serious?

3) Will disease control be more difficult?

Plant diseases are in the main caused by viruses, bacteria and fungi. Virus diseases receive separate consideration below since they are entirely dependant on vectors for spread. With bacterial and fungal diseases the effect of climate on the disease will be manifested in two ways: directly, through the effect of temperature and moisture on the distribution and life cycle of the pathogen and the etiology of the disease it causes; and indirectly through changes in the physiology of the host affecting its susceptibility or resistance to the disease.

V.1 The Spread of Disease.

Before a crop can be affected by a disease, inoculum of the relevant pathogen must be present. Some diseases that occur in Europe have not been seen in the UK or occur only rarely here, and it is possible that we have fewer pathogens. Others do not occur because their host is not grown, or is not widely grown, and if climatic changes allow hosts, such as sunflower, to be grown on a wider scale, then we should expect them to be afflicted by the usual range of diseases observed in other countries.

In any case, it is more than likely that inoculum of many important bacterial and fungal diseases which occur in continental Europe are either here already or will eventually reach here. Aphid vectors of viruses, and aerial pathogens such as rusts and mildews are carried by the wind and probably arrive from the continent in most years. If they do not cause significant disease losses at present, it is because the present UK climate, and the time of their arrival is not favourable to the establishment of early epidemics.

Pathogens, particularly soil-borne ones, which are disseminated in plant material, e.g. seed and cuttings, and others which are not transported on the wind over long distances, are subject to quarantine regulations designed to prevent their spread.

The inspection of imported plant material, and the exclusion of diseases that it may carry, is the responsiblity of Plant Health Inspectorates operating in the various parts of the UK. To support them they have scientific services, which can provide advice and assistance in detection and diagnosis, for which they employ a range of techniques e.g. microscopic examination, selective media, bioassays, immunoassays and nucleic acid probes. The principal centres for this work are the Plant Pathology Laboratory in Harpenden, DAFS Scientific Services in East Craigs, and DANI in Belfast.

These centres do excellent work, but their control over the entry of diseases on the quarantine list will be increasingly limited after 1992. In that year new EEC regulations for the control of the spread of diseases on plants will come into force, and it will no longer be the responsibilty of the importing country to keep out diseases. The onus will be on the exporting country to ensure that it is exporting healthy plants. The despatching country will be responsible for the issuing of health certificates, and the importing country will have to accept them. As a large importer of plant material the UK will be under particular threat. The increased movement of very large consignments of plants within the EEC will almost certainly result in the importation of new or uncommon diseases into the UK, or even threaten existing controls on indigenous diseases.

The apparent absence or relative unimportance of some plant diseases in the UK may not be due to the absence or infrequent occurrence of the causal agents, but may be a consequence of the present climate restricting the severity of the disease and its spread, e.g. *Verticillium dahliae* probably occurs in many agricultural soils in UK, but Verticillium wilt of potatoes is uncommon because summer temperatures are rarely high enough for symptoms.

It is therefore prudent in a scenario of climate change to assume that diseases previously unknown or rare in the UK, with the exception of some exotic diseases such as potato rust, are present, or soon will be, and that they will manifest themselves if climatic conditions alter in their favour.

V.2 Direct Effects of CO_2 and Other Atmospheric Gases on Plant Disease.

It is highly unlikely that the projected changes in atmospheric [CO_2] will by themselves have much effect on the amount or severity of plant diseases. Other atmospheric pollutants can pre-dispose plants to disease (Schoeneweiss, 1975), and can themselves do significant damage to plants, especially to trees. Nevertheless their principal effects will be through the changes in climate that they produce.

V.3 The Temperature and Moisture Reponses of Non-viral Diseases.

Infection by bacteria and fungi requires suitable conditions of temperature and moisture, and before considering the effect of the most likely scenario for climatic change on diseases caused by these groups, it is necessary to examine the effects of temperature and moisture levels in some detail.

In many cases the temperature range suitable for infection can be relatively broad: thus spores of *Pyrenophora teres* (net blotch of barley) germinate over the range 2°C to >20°C and 40% of infections occur within 100Kelvin-hours, once conidia are wetted (Shaw, 1986). For most diseases which at present are important within the UK, temperatures just above or at freezing prevent growth of the pathogen but are not generally lethal to it, so that the usual effect of 'cold snaps' is to arrest, but not eliminate pathogen development. Some pathogens from warmer climates are eliminated by low temperatures or have relatively high minimum temperatures for growth and infection, and consequently cause little damage in the UK as they are unable to overwinter. A classic example is *Phytophthora cinnamomi* which attacks a wide range of woody ornamental, and tree species. In the UK it is troublesome on protected nursery stock, but has been recorded only rarely on unprotected crops. It occurs on various tree species in France and Italy: in the former country it causes a serious root rot of oak trees in the Bordeaux region, but in Australia it has devastated millions of hectares of natural eucalypt forest in several parts of the continent. Weste & Marks (1987) state that the fungus is only active at soil temperatures above 10-12°C and at matric potentials greater then -0.9 MPa, a combination of conditions which explains its relative importance in the countries listed above.

The availability of free water is often a more serious constraint on dissemination, germination and successful infection than temperature. Motile bacteria and fungal spores such as *Erwinia* and the zoospores of *Pythium* and *Phytophthora*, are only motile at or very near zero water potential, and their ability to spread disease is severely restricted if soils are not at or near field capacity (Duniway, 1976). Other fungi only release their spores when in contact with water e.g. more than 50% of the pycnidiospores of *Septoria nodorum* (glume blotch of wheat) are released within ten minutes of coming into contact with water (Brennan *et al.*, 1986). Distribution of these spores then occurs by rain splash with intense rain showers distributing the inoculum further and higher up the plants than gentle rain (Royle *et al.*, 1986).

Most bacteria will not proliferate, and some fungal spores will not germinate and infect below 98-100% RH: the spores of *Pyrenophora teres* (see above) will only germinate in the presence of free water (Shaw, 1986). In many cases therefore, spread and infection will only occur in the presence of rain or dew. Coakley (1988) in reviewing the effect of climate variation on plant disease where vectors were not involved, found in five out of five diseases, losses were greater in years with above average precipitation during the critical infection period.

The ability of plant pathogens to survive in the absence of their hosts, usually in soil or plant debris, varies considerably for different pathogens but is in each case affected by climatic factors. Survival is generally better described by the frequency and extent of favourable and unfavourable environmental conditions than mean conditions, and frequent extremes of temperature and moisture will usually affect survival adversely e.g. the resistant zoospores of *Phytophthora fragariae* will survive for many years in soil at temperatures of 15°C or less and in moist soil, but rapidly lose viability at 30°C or in very dry soil (Duncan & Cowan, 1980)

In practice there will be an interaction between temperature and moisture levels which will affect the survival, infectivity and reproduction of a pathogen on its host, and the outcome of this interaction will determine the relative importance of any plant disease in any particular climate. Hepting (1963) considered the threat posed by *P.cinnamomi* to the production of Douglas fir in North-Western USA as small since soil temperatures were too low for zoospore production when the soil was wet enough for infection, and when temperatures were high enough the soil was too dry.

Water and temperature have other affects on plant disease. Waterlogging or drought, and high or low temperatures can stress plants making them more susceptible to disease (Schoeneweiss, 1975). The defense response of plants to disease involves the activation of new metabolic pathways with the synthesis of new enzymes and metabolic intermediates. This is almost always accompanied by an increased respiration rate, which is usually greater in the case of a resistant than a susceptible response. Aerobic metabolism is essential for the synthesis of many phenolic and sesquiterpenoid phytoalexins. Waterlogging reduces the availability of oxygen to the plant and interferes with these active plant defense mechanisms.

It can increase damage considerably: Duncan & Kennedy (1989) showed that raspberry plants could be killed by some *Phytophthora spp.* that were in normal circumstances only minor pathogens, and MacKerron & Jefferies (1988) found that short periods of waterlogging greatly retarded the emergence of potatoes, and that this was due in part to the effect of soil bacteria on the plants.

Drought can likewise affect the response of plants to disease. Tobacco can be predisposed to downy mildew (Rotem *et al.*, 1968), and safflower to Phytophthora root rot by water stress (Duniway, 1977). Once the stress is relieved the plants are extremely susceptible to infection.

Low temperatures can damage plants and make them more susceptible to disease, but in the context of global warming it is the effect of high temperatures on disease resistance mechanisms which may be of more importance. Disease resistance mechanisms are temperature sensitive: 'At 25°C wheat plants homozygous for the gene Sr6 are susceptible to cultures of *Puccinia graminis* to which they are resistant at 20°C.' (Vanderplank, 1978). In oats a similar situation has been observed with genes for resistance to oat stem rust (Martens *et al.*, 1967). Phytoalexin production by potatoes decreases with increasing temperature (Lyon, 1984) and is correlated with increased susceptibilty to *Erwinia* at higher temperatures. Vanderplank (1978) hypothesised that temperature changes affect hydrogen bonding between proteins produced by host and pathogen, these complexes being central to host/pathogen recognition in disease resistance mechanisms.

The above observations applied to the relatively short time-scales within single seasons, but there is evidence that similar stresses over longer time-scales can have profound effects on disease, and through disease on the distribution of some plants. Schoeneweiss (1975) listed seven diseases of trees in USA that were associated with an extended period of below normal precipitation in the 1930s, and concluded that host predisposition was one of the major factors involved in disease development.

V.4 *Effects of Global Warming on Non-viral Diseases in the UK.*

It can be seen from the previous Section (V.3) that aspects of climatic change with particular importance for plant disease will be the probable amounts, intensity and seasonal distribution of rainfall, and minimum and maximum temperatures during infection periods. According to the predictions of current general circulation models detailed in Section I.7, Scotland/N. England will become warmer and more temperate, perhaps with wetter, warmer winters and drier, warmer summers.

In such a climate one would assume that diseases in which infection took place during winter, or in spring before the intervention of dry weather, would in general be more severe than at present. Quiescent periods during the winter when temperatures were too low for infection/growth and when water was present largely as ice, would be fewer in number and shorter in duration than at present. Consequently the periods during which disease could develop would be longer.

By contrast warmer, drier summers could well mean a reduction in some diseases on spring sown crops, e.g. late blight on potatoes. However some caution must be exercised here in the case of cereal crops. There would probably be increased areas of Autumn-sown cereals due to the more favourable weather and these could act as 'Green Bridges' (see Section IV.4) carrying inoculum from one season to the next and perhaps infecting spring-sown ones earlier in the season. Powdery mildews and rusts of winter cereals where extensive colonisation could occur during mild, damp winters could become very serious in the drier, warmer summers that followed. Although summers might be generally drier, in a warmer climate a higher proportion of the rainfall might come from thunderstorms, the intensity of which might have a greater effect on development of some diseases than the actual amounts of rain delivered by them. Royle *et al.* (1986) ascribed sudden outbreaks of *Septoria* on the upper leaves of wheat to short, heavy rainstorms which elevated pycnidiospores up to 60cm through the crop canopy. Irrigation, the use of which would increase on high value crops in hot weather, could have similar effects on disease development.

Of soil-borne diseases only the snow moulds such as *Fusarium nivale* and *Typhula incarnata* which damage winter cereals under snow, would be ameliorated. Root rots of perennial crops such as Red core of strawberries (*Phytophthora fragariae*) and Root Rot of raspberry (*Phytophthora spp.*) where the damage occurs in the cooler, wet parts of the year but is not seen until the plants come under stress in early summer, would be more serious. Similar diseases would probably increase in frequency on other crops, notably trees.

Some diseases which are unknown or rare in Scotland would undoubtedly become important, especially in good summers. Leaf-spotting fungi often require moist, warm conditions for infection and these conditions do not occur for long enough in most years. Thus *Colletotrichum acutatum* which causes leaf and fruit blackspot of strawberry and which has recently arrived in England on imported plants, is unlikely to be a serious problem in Scotland under present climatic conditions. However in a warmer, wetter climate it could be very serious. Likewise the wilts caused by *Fusarium* and *Verticillium*: Verticillium wilt of potatoes is a major problem in warmer climates, where it is only held in check by a combination of fungicides, crop rotation, resistant cultivars and soil sterilisation.

Some bacterial diseases may be reduced in importance, others would be more serious. Potato blackleg (*Erwinia carotovora* var *atroseptica*) is favoured by cool wet conditions during summer and autumn (Perombelon, 1976) and might be less important, but other soft rots caused by *Erwinia carotovora* and *E.chrysanthemi* could be commoner. The present concern of regulatory authorities about high levels of blackleg in Scottish seed potatoes, might need therefore to be re-directed towards problems caused by related *Erwinia spp.* Conditions for the fireblight bacterium *E.amylovora* would also be more favourable, partly due to the climatic changes, and partly because a wider range of hosts would be grown. Leaf blights and spotting caused by *Xanthomonas* and *Pseudomonas spp.* would probably increase in frequency for similar reasons. Again these latter diseases are often reported to be worse after intense rainfall.

V.5 Effect of Climatic Change on Virus Diseases.

Many important plant viruses are aphid-borne, and as has been pointed out in the section on pests (Section IV.1b) the projected climatic changes should favour greater overwintering of aphid populations and earlier and more cycles of proliferation. This in turn will lead to earlier and heavier virus infection, and virus diseases should increase in importance in a warmer Britain. Of particular economic importance will be virus spread in seed potato stocks, and some of the more serious cereal viruses such as Barley Yellow Dwarf. The altered climate might also allow the establishment of vector species new to the UK, and these may bring new diseases with them.

Soil-borne fungi and nematodes are also vectors for viruses. The seriousness of the diseases transmitted by these organisms will probably depend on the time of year when transmission takes place. All the fungal vectors possess in their life cycle, motile stages which transmit the virus, and the importance of the virus diseases will largely depend on the effect of climatic changes on these stages. For the same reasons mentioned in the section on fungal and bacterial diseases (Section V.4) virus transmission by soil-borne fungal vectors may be more serious in overwintering crops e.g. Barley Yellow Mosaic in cereals, and less important in spring-sown crops e.g. Potato Mop Top in potatoes.

For a more detailed consideration of the likely effects of climatic changes on diseases of potatoes and cereals see Appendices F and G.

V 6. Conclusions.

1) The spectrum of diseases observed in the UK will alter for the following reasons:

    a) Cropping patterns will change.

    b) New diseases will arrive or be imported into UK, especially after 1992.

    c) Climate will render previously unimportant diseases more important.

2) Diseases of crops which overwinter, and diseases in which the host is pre-disposed by stress will be more important. A few 'cold weather' diseases will be less important.

3) Disease control may be more difficult where diseases establish earlier in the season e.g. fungal and viral diseases of winter cereals. Of particular concern is the effect of earlier build-up of larger aphid populations on virus diseases of seed potatoes.

V 7. Future Priorities

Any change in climate will create new disease problems and opportunities, which will affect future research priorities. Some thought should be given to relevant areas of research which could be looked at now, and to areas which may be of high priority in future.

1) Work on the early detection and diagnosis of disease should continue, as changes in legislation and trade are likely to increase the threat to UK agriculture from imported diseases.

2) Most disease forecasting is short-term and based on local weather data. Recent work in N.America has had some success, using longer-term climatic data, powerful computers and iterative techniques, to identify those climatic factors most highly correlated with the development of disease on crops (Coakley, 1988). Once identified these factors are used in multiple regression analysis as independent predictor variables, with measures of disease as the dependent variable. In this way the important climatic factors in any particular disease can be pinpointed.

    Once such factors have been identified modelling studies to show the effect of any change in them should be undertaken to determine how important particular diseases are likely to be in different climatic scenarios.

3) Modelling would require information on the effect of microclimate on disease, so there would be a need for some short-term forecasting work of the type already done at SCRI.

This might need to be expanded to diseases not previously considered important in the UK. A particular concern should be the effect of short periods of extreme weather on the progress of disease e.g. periods of drought or flooding. Coakley suggested that much work remains to be done on compensatory factors in plant disease. Rotem (1978) proposed that optimal states of some factors may compensate for the limiting effects of marginal ones. As perfect combinations of conditions for epidemics occur infrequently and are usually short-lived, such compensatory processes must be important in the progress of most diseases.

4) Most modelling of soil-borne diseases has been concerned with spatial and temporal aspects of epidemiology and there is little if any modelling/forecasting work. Yet the relatively stable temperatures encountered in soil should in some ways make modelling easier than with aerial disease.

5) There are also possibilities for some research which would underpin 'environmentally friendly' control techniques. Raising soil temperatures by mulching (soil solarisation) has been used successfully in Mediterranean countries to reduce inoculum loads of soil-borne diseases, and may even be effective in S.England in our present climate. In warmer summers, this technique may be applicable further north. Raised temperature is obviously the principal effect of this treatment, but it must interact with reduced availability of water, and consequently, reduced infection frequency. This could be one area for future research.

6) An altered spectrum of diseases will mean that the priorities of breeders will change in resistance breeding. Sources of resistance will be required for diseases, which previously received little or no consideration in breeding programmes.

7) Basic research on the biochemistry underlying temperature-sensitive resistance would be required, and would match efforts by breeders to find temperature stable resistances. Resistance of this type could be manipulated by the molecular geneticists. Partial resistance, which offers the best prospects for obtaining cultivars with durable resistance, is particularly dependent upon appropriate environmental conditions for optimum expression, and would receive a high priority in any programme.

In all the above possible programmes of work, SCRI is well placed to lead and participate, having the necessary expertise throughout its divisions and departments.

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