the University of Leeds on the synthesis of such compounds. The most convenient synthesis used phosphoryl chloride as the starting material; but radiophosphorus is most readily available as inorganic phosphate, and the conversion of this to phosphoryl chloride is not easy. The difficulty might be overcome by the discovery of a suitable exchange reaction or by a supply of radiophosphorus in a more convenient form.

Dr. R. A. E. Galley urged that the nomenclature of the organo-phosphorus compounds should be standardized and favoured the introduction of trivial names for the better known phosphorus insecticides.

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[2] *Brit. Intell. Obj. Sub-comm.*, Final Report 714 (1947).
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'MECHANISM' OF *PHYTOPHTHORA*-RESISTANCE OF POTATOES

By K. O. MÜLLER and LOTHAR BEHR*

USING as a basis the American 'native forms' of the tuber-bearing species of Solanaceæ; German breeders during the last two decades have developed potato varieties with both leaves and tubers resistant to most of the known races of *Phytophthora infestans*. It is true that the origin and initial constitution of the material which was used for the breeding of these varieties is only imperfectly known; but we know that its refractory behaviour towards the *Phytophthora* fungus is associated with a gene *R* which is not found in any European variety of the old-fashioned type. Furthermore, according to K. O. Müller, this resistance gene has several associated alleles and is inherited independently of other economically important characters, such as yield capacity, time of maturity, and the like. Therefore it did not take very long to combine the *Phytophthora* resistance of these '*W* varieties' with the high yielding-capacity and other inherent qualities of the European cultivated potato. The first German commercial varieties resistant to *P. infestans* which appeared on the market were Erika (1941), Robusta (1941), Fruehnudel (1941), and Aquila (1942). There are at present in Germany ten new registered potato varieties, all of which possess this resistance gene.

Investigation into the mode of action of this gene, particularly as regards the tubers, led to the conclusion that the alleles present in the *W* varieties are not responsible for the resistant condition itself, but induce only a genetic predisposition of the tissues to 'acquire' a local immunity from infection when they come into contact with the hyphæ of parabiontic races of *P. infestans* (that is, those strains which kill the cells quickly, thereby arresting the further growth of the hyphæ). From the physiological point of view, whether or not a variety is resistant is not decided until the parasite has penetrated into the host tissues. Comparative investigations into the reactions of susceptible and resistant genotypes revealed that in both the host cell is destroyed after coming into contact with the protoplasm of the parasite. Judging by the morphological and physiological changes the final effect is the same—the cell collapses and loses its ability to serve the parasite as a host. But the rapidity with which this final effect is achieved differs in susceptible and resistant genotypes. While the cells of the former live relatively long —six to fourteen days at 19–21° C.—those of the latter die after one or two days, the process being too rapid for fructification of the fungus to occur; moreover, further penetration by the hyphæ is made impossible by the rapid reaction of the host tissue, which in the course of this reaction also loses its ability to serve as a host to eusymbiontic fungus strains (that is, strains which can invade the host tissue without killing it quickly). Other pathogenic micro-organisms, too, are unable to use such 'immunized' tissues as nutritive substrates. The reaction is therefore unspecific (K. O. Müller and H. Boerger).

Cytological-physiological investigations by K. O. Müller, G. Meyer and M. Klinkowski in 1938 showed that it is possible to distinguish in this 'defence-necrosis' at least five successive stages, the last two being characterized by cell collapse and by a heavy infiltration of the cell-walls and cytoplasm with phlobaphene-like compounds, which appear to arise, according to Meyer (1939), through polymerization of tannic substances. These can be demonstrated by the usual tannin reagents after infection in host cells which are still alive.

This close relation between the degree of resistance and the rapidity with which necrosis is brought about was demonstrated in extensive breeding material, consisting partly of previously unselected *F*2 families of crosses between 'resistants' and 'susceptibles'. It was also shown that the two extremes, 'highly susceptible' (the parasite penetrating uniformly through the entire tuber and fructifying luxuriantly) and 'highly resistant' (the parasite penetrating only a few cell-layers of the parenchyma of the tuber and then ceasing to grow without fructifying), are connected by at least two genotypically conditioned intermediate types. These are characterized by an intermediate reaction-rate and—in agreement with what has just been said—their tubers are penetrated to a considerable depth by the parasite (K. O. Müller).

Other investigations led to the conclusion that the 'defence necrosis' is induced by substances secreted by the parasite into the host tissue. Accordingly, the specific behaviour of the resistant genotypes would appear to depend on a specific sensitivity of the protoplasm, controlled by gene *R*, so that the highest reaction-rate would be associated with the greatest degree of resistance. Because the susceptible genotypes also react to these substances, there appears to be no fundamental, but only a graduated, difference between 'resistants' and 'susceptibles', and we conclude (paradoxical though it may appear at first) that the greater the sensitivity of the cells to the metabolic products of the parasite, the greater the resistance of the tuber. Further, the resistance gene *R* is not a factor *sui generis*; it only accelerates a reaction which the susceptible genotypes are also capable of producing. There is, however, a further conclusion. In the course of the reaction the host tissue loses the ability to serve as a nutrient substrate for the fungus. As not only *P. infestans*, but also other micro-organisms which can thrive on a wide range of organic matter (*Rhizopus nigricans*, *Aspergillus niger*, and others), are not able to exist in the necrotic, transformed tissue, the necrosis of the affected tissues must be accompanied by the formation or activation of a principle ('phytoalexin') which

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exercises a retarding influence on the penetrating parasite. In these circumstances necrosis would depend less on the absolute reaction-rate and more on the relation between the hyphal growth-rate (*Cp*) and the rapidity with which the principle forms (*Cw*). Hence with increasing *Cp* : *Cw* values the resistance would decrease, while with decreasing values it would increase.

From experiments in which the influence of temperature on the reaction in various genotypes was examined, valuable insight was obtained into the dynamics of the defence mechanism described above (K. O. Müller and R. Griesinger). At low temperatures (5–10° C.) the immunological behaviour of the susceptible genotypes strongly resembled that of the resistant ones. But on the other hand, no decrease in the capacity for resistance in the resistant genotypes was induced. This, however, was achieved in another way, and for this K. O. Müller and L. Behr started from the following hypothesis. If it were possible in the resistant genotypes to retard the necrosis of the tissues affected by the fungus by some kind of chemical or physical treatment of the tubers, the development of the fungus would be promoted simultaneously. The parasite would be able to penetrate farther into the tissues than in the untreated tubers, and at the same time would also fructify.

The experience of other workers (Salmon, Stakman, Bolle, Gassner and Hassebrauck), who have studied the effect of narcotics on the immunological behaviour of plants against fungal parasites, suggested the materials to use for such experiments. The tubers of resistant and susceptible genotypes were treated with methyl, ethyl, propyl, butyl, *iso*propyl, and *iso*butyl alcohol (water–alcohol mixtures), and were inoculated *after* treatment. Germination tests with sporangia of *P. infestans* in alcohol–water mixtures showed that the concentrations used did not check the development of the parasite. The results were as anticipated. The treatment of the tubers with the alcohols above mentioned prolonged considerably the life of the cells which had come into contact with the parabiontic strain of *P. infestans* (retardation of cell collapse, inhibition of phlobaphene formation). At the same time—and this is most important—a material increase in the development of the fungus followed, which in many cases went so far that the parasite developed thick aerial mycelium (with sporangia), even in the highly resistant types.

The accompanying table shows the effect of ethyl alcohol on the immunological behaviour of a highly resistant *W* variety.

That the tubers were *in toto* still alive after the alcohol treatment was shown by the ability of parenchyma cells to become plasmolysed and by the fact that non-inoculated tubers, treated with a 3–5 per cent alcohol–water mixture, could be stored for weeks at room temperature and high air humidity without showing any rotting symptoms caused by facultative parasites.

A comparison of the effects of the four normal alcohols showed that a close correlation exists between the length of the carbon chains and their effect in reducing resistance. Richardson's 'law of homologous series' has thus been confirmed. Furthermore, *iso*propyl and *iso*butyl alcohol were considerably less effective than the normal alcohols. This result, too, agrees with the findings of previous workers, who maintained that the greater the branching of the carbon chains, the less the narcotic effect.

Effect of ethyl alcohol on the immunological behaviour of the *W* variety *BRA* 23/31

Concentration of alcohol (% volume)	Reaction of the host			Development of the parasite	
	Necrotic changes immediately beneath the site of inoculation		Phlobaphene-formation within the tuber	Aerial mycelium (sporangia)	Within the tuber
	after 48 hours	after 120 hours	after 120 hours	after 120 hours	after 120 hours
0 (Control)	very strong	very strong	very strong, distinctly limited (2–3 cell-layers)	none	no mycelium outside the necrotic tissues
3	weak	strong	weak, diffuse, up to a depth of about 10 mm.	thin	parasite penetrated up to about 35 cell-layers
5	weak	strong	,,	thin	,,
10	very weak	very strong	very weak (greatly variable in extent)	thick	penetration up to about 15 cell-layers
12	very weak	very strong	extremely weak, diffuse	very thin	penetration up to about 7 cell-layers

The results of these experiments with narcotics have thus confirmed the original theory developed on a genetic basis : the greater the rapidity with which the host cells in contact with the fungus react to the metabolic products excreted by the fungus, the greater the resistance of the plant. The results also suggested a satisfying physiological explanation of the effects of narcotics in decreasing resistance, noted already by previous workers on other organisms, though differently interpreted. Details cannot be given without exceeding the scope of this short article, but our interpretation appears to have the advantage of explaining satisfactorily many effects, with only one premise to be recognized, namely, the greater the reaction-rate, the greater the resistance.

The effect of chloroform (applied as a vapour) has also been tested, and the result agreed with the above findings. This leads to the conclusion that the action of narcotics on the 'resistance mechanism' is an effect connected with the primarily unspecific changes in the physico-chemical structure of the host cell. As other observations point to the fact that the necrobiosis is connected with the respiratory metabolism of the host cell, it can be assumed that it is this metabolism which the narcotics affect first. This would also hinder the course of the defence necrosis and simultaneously postpone the material changes in the host cell, which we must consider to be the direct physiological cause of the 'defence' against the parasite.

Whether or not this attempted explanation conforms with the facts, we can deduce from the results that the necrobiosis of host tissue, induced by a parabiontic strain of *P. infestans*, is essentially an active response of the affected cells, very probably connected with an increased release of energy. If the defence necrosis of the resistant genotypes induced by fungal contact were the same as a passive dying, such as we can observe, for example, after the effects of supramaximal or inframinimal temperatures, it would appear incomprehensible why just those substances, which in our experience had only a paralysing effect on cell activity, should increase the longevity of infected cells.