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Presidential address : The impact of pesticides on biochemical defence systems of plants

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Professor Raychaudhuri, Professor Chattopadhyay, Dr. Ghosh, Distinguished Members of the Indian Mycological Society, Ladies and Gentlemen,

May I express my gratitude to the members of the Indian Mycological Society (IMS) for electing me as the President for the Session 1988-89. I have been greatly honoured to be the President of the IMS. You are well aware that this Society has been continuing to develop its national role in the scientific community by publishing journal, organizing symposia, instituting lectures in memory of pioneer Mycologists of the country and also by inviting eminent Scientists for delivering lectures apart from other routine business. This year we have invited Professor S.P. Raychaudhuri, an internationally known scientist and Professor A. Mahadevan, a distinguished Plant Pathologist and Diretor, CAS in Botany, University of Madras for delivering Dr. E. J. Butler Memorial lecture and Dr. S. N. Banerjee Memorial lecture, respectively. We are indeed grateful to them for kindly accepting our invitation.

Several new developments have taken place in the field of Mycology during the last few decades and it has become possible due to coordinated and sincere efforts of International Mycologists and Mycological Societies. We feel proud that our Society also with its limited resources has taken part in the developmental activities. I strongly believe that our distinguished members will continue to assist the Society for its further growth and development.

On the occasion of the 33rd Annual General Meeting of the Indian Mycological Society I wish to deliver a brief address on the Impact of pesticides on biochemical defence systems of plants. I have chosen this topic in view of its importance in agriculture and plant pathology in particular although it has received little attention of researchers so far. Pesticide-induced physiological stress often alters the natural resistance of plants to various parasitic diseases. But how they affect the resistance is a matter of conjecture. In many cases soil drench or foliar application of pesticides causes inhibition or death of both target and non-target organisms and consequently the plants escape disease. Generally the groups of pesticides are named in accordance with their intended use. For instance, "fungicides", "bactericides", "insecticides", "nematicides" and "herbicides" are used against fungi, bacteria, insects, nematodes and weeds, respectively. Although diverse array of effects of pesticides on living systems have been reported by several investigators I will restrict my address only to the impact of fungicides and insecticides. This aspect appears to be more interesting and relevant to crop disease management. Since phenol metabolism, lignin biosynthesis and phytoalexin production, accumulation are believed to be associated with the plant's defence responses, these aspects have been taken into consideration. Both these syntheses and degradation may be influenced by treatment with pesticides. Considerable amount of work has been done on these plant metabolites and several extensive reviews (Cruickshank, 1963, 1980 ; Farkas and Kiraly, 1962 ; Kuc, 1972, 1976 ; Purkayastha, 1971, 1973, 1985, 1986. 1989 ; Bailey, 1981 ; Keen, 1981 ; Sequeira, 1983 ; Ebel, 1986) have been published on their role on disease resistance during the last three decades but little information is available regarding the impact of pesticides on biochemical defence systems involving the aforesaid plant metabolites.

Pesticides are frequently and indiscriminately used by the crop growers without considering their long term effect on non-target organisms including host plants. Apart from killing activities these pesticides sometimes significantly alter the physiology of the host plant creating conditions favourable or unfavourable for the pathogens. In this process antifungal, secondary metabolite(s) may be formed or the level of any constitutive metabolite of host may increase which is inhibitory to the growth of the attacking pathogen. Sometimes plants may die due to toxicity of certain pesticides depending upon the magnitude of the impact or pesticides make plants vulnerable to attack by pathogens. However, some of these effects will be briefly discussed.

In 1963 Uehara and subsequently Oku and Nakanishi (1964) in Japan reported that some organo-mercuric and other synthetic organic fungicides induced phytoalexin production and also protected plants from diseases. This protective function was not only due to their antifungal activities but also due to their interference in the physiological processes of host plants. Similarly, sodium iodoacetate and sodium selenate were found to be the inducers of pisatin in peas (Perrin and Cruickshank, 1965). Reilly and Klarman (1972) tested 27 fungicides of which 15 induced detectable quantities of hydroxyphaseollin in soybean. Among the fungicides tested, Maneb showed two decomposition products, namely ethylenediamine and polyethylene (thiocarbamoyl) monosulphide. Both were active in inducing hydroxyphaseollin production in plants. Another interesting observation was that the butylamine, a non-fungitoxic portion of a common systemic fungicide, benomyl induced phytoalexin as effectively as did benomyl while the fungitoxic portion containing benzimidazole carbamic acid and methyl ester could not (Reilly and Klarman, 1972). It suggests that induction or elicitation of phytoalexin production may be more important in reducing disease than the fungicidal activity of benomyl. Besides Benomyl, Kitazin P, Tryphenyltin acetate, Triphenyltin hydroxide, Captan, Tachigaren (3-hydroxyl 5-methyl isoxazole), Triazine, Dichlone and Phenyl mercury acetate could also stimulate production of phytoalexin like pisatin in endocarp tissue of fresh pea pods (Oku *et al*, 1973). They have suggested that it is useful to search for compounds which accelerate phytoalexin induction for the development of harmless agricultural chemicals especially in the nature of bound fungicides for controlling plant disease. Although phytotoxicity of some phytoalexins have been reported by a few workers (Smith, 1982), it is not yet confirmed whether all phytolexin or any concentration of a phytoalexin is phytotoxic. However, some furanoacetylenes, isoflavonoids and terpenoids have been shown to be phytotoxic (rishitin and phaseollin). The possibility of chemical activation of defence in rice plants was conclusively demonstrated by Cartwright *etal.*(1977,1980). They showed that a fungicide 2, 3 dichloro-3, 3-dimethyl cyclopropane carboxylic acid (WL-28325) treatment could increase the capacity of rice plants to synthesize more momilactone A and B (rice phytoalexin) in response to infection by *Pyricularia oryzae* which causes blast disease of rice. Like momilactones, greater accumulation of glyceollin was detected in soybean hypocotyls inoculated with *Phytophthora megasperma* after pretreatment with a systemic fungicide Ridomil [DL-methane-N-2, 6-dimethyl phenyl) N-(2-methoxy acetyl) alaminate]. Ridomil was found to be associated with hypersensitive symptoms during the control of fungal diseases (Lazarovits *et al.*, 1980). In 1982, Moesta and Grisebach reported that application of 1-2-amino oxy-3 phenyl propionic acid (L-AOPP) 1 mM solution to cut ends of soybean seedlings (Harosoy 63) for 4 hrs prior to inoculation with incompatible race 1 of *Phytophthora megasperma* f. sp. *glycinea* (Pmg) resulted in 90% inhibition of glyceollin production and concomitant increase of infection. Hence they strongly supported the assumption that glyceollin played an important role in defence reaction of soybean. It is known that preinoculation treatmet of compatible soybean tissue with 20 ppm Metalaxyl (a systemic fungicide) solution resulted in glyceollin accumulation similar to that recorded for incompatible interaction (Borner *et al.*, 1983). Another systemic fungicide Fosetyl-AL was also identified as an inducer of phytoalexin. This fungicide induced greater accumulation of capsidiol in *Capsicum* and in tobacco after inoculation with *Phytophthora nicotianae* var. *parasitica*. These findings strongly suggest that some pesticides are phytoalexin inducers and they have considerable effect on the defence system of the host. It is not known exactly how many pesticides can degrade phytoalexins. It is not unreasonable to speculate that treatment of a plant with a fungicide or an insecticide having phytoalexin degrading ability may increase disease susceptibility of plant. Watson and Brooks (1984) showed that copper sulphate and silver nitrate were able to elicit capsidiol production in pepper and hence the plants were resistant to the pathogen. Accumulation of kievitone and phaseollin in different tissues of *Phaseolus vulgaris* in response to treatment with mercuric chloride was also reported (Goossens *et al.*, 1987). These two phytoalexins not only accumulated in cotyledons but also in hypocotyls and roots of HgCl-treated plants. It is interesting that production of these two compounds could be reduced by 10^{-4}M abscisic acid or it could be slightly increased by a 10^{-6}M solution. Induction of phaseollin and pisatin in bean and pea cotyledons respectively by $HgCl_2$ was recorded earlier by Hargreaves (1979). The rate of production or concentration of phytoalexin depends upon both duration of treatment and concentration of $HgCl_2$. Like $HgCl_2$, $AgNO_3$ treated bean seeds produced more phaseollin, isoflavone and kievitone than seeds inoculated with *Fusarium oxysporum* (Stossel and Magnolato, 1983). Similarly, $CuCl_2$ ($3x10^{-3}$ M) induced phaseollin and kievitone as reported by Dewick and Steele (1982). A number of abiotic agents have been identified which cause little or no stimulation of biosynthetic activity of phytoalexin (PA) but they could strongly inhibit the phytoalexin degrading activity. The ability of about 60 abiotic chemicals to stimulate glyceollin accumulation was correlated with their specific inhibitory effects on glyceollin degrading activity (Yoshikawa, 1983). It shows that increased amount of PA in treated plants is not always due to stimulation in biosynthesis but it is due to inhibition in PA degrading activity.

Pesticides not only affect the biosynthesis and degradation of phytoalexins but also markedly influence the phenolic metabolism of host plants. Aurora and Bajaj (1978) recorded a change in pattern of polyphenolic constituents of the hypocotyls *Rhizoctonia solani*-infected mung (*Vigna radiata*) plants after treatment with Vitavax (Carboxin). The increase in phenolic compounds suggests their role in disease resistance of mung. In addition to Vitavax, application of Metalaxyl to the surface of potato tubers medullary tissue prior to inoculation with *Phytophthora infestans* also stimulated the activity of polyphenol oxidase which conferred resistance to the pathogen. This compound activated the defence reactions in host tubers against all fungal parasites through enhancement of lignin biosynthesis. The tubers containing Metalaxyl residue were resistant to a number of fungi namely, *Fusarium sambucinum*, *F. culmorum* and *Alternaria solani*. The resistance was, however, lost gradually after 4 months of storage when the Metalaxyl level had decreased considerably (Borak *et al*, 1984). The elicitation of lignification response in wheat leaves by treatment with $HgCl_2$ (10^{-3}M) was pointed out by Pearce and Ride (1980). Lignification is undoubtedly one of the major defence responses of host. Some enzymes related to phenol metabolism such as PO, PPO, PAL and CHS (Chalcone synthase) may also take part in defence reactions. Treatment of tomato plants with ethephon (2-chloroethyle phosphonic acid) enhanced PO and PPO and increased resistance of a susceptible cv. to *Fusarium oxysporum* f. sp. *lycopersici* (Retig, 1974). It indicates that these two enzymes may be involved in disease resistant reactions. There is evidence that application of Benzimidazole derivatives reduced PAL activity in beans and barley. Generally PAL is considered to be a key enzyme in the production of basic molecule necessary for the biosynthesis of most phenolics including some phytoalexins and lignin (Agrios, 1988). It may be mentioned that foliar application of Anthio 25EC (0.1%) accelerated PAL activity and reduced anthracnose disease of jute significantly. Along with PAL, PO, PPO, total phenol and phenolic acids also increased in treated jute leaves (Mitra and Purkayastha, 1986). Similarly, Metasystox 25EC and Endosulphan 35EC also reduced anthracnose disease and enhanced total phenol contents of leaves (Mitra and Purkayastha, unpublished).

It appears from the above statements that some systemic pesticides could reduce fungal diseases by altering host metabolism. A few of them are capable of activating defence systems through elicitation of phytoalexin or biosynthesis of lignin or production of antifungal phenolic acids. All these responses may occur in pesticide-treated plants. Some insecticides appear to have dual functions. They kill insects and also enhance resistance of plants against fungal diseases. It is worth while to investigate as to how the systemic pesticides induce phytoalexin production or lignin biosynthesis in crop plants. The complete mechanisms by which the various pesticides control plant diseases are not yet clearly understood for most of the synthetic pesticides. The elucidation of these questions is fundamental to an understanding of the challenging defence problems.

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