# MAJOR CROP DISEASES OF SRI LANKA AND THEIR CONTROL



*EDUCATION AND TRAINING DIVISON*

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# INTODUCTION

This booklet has been prepared for the participants of the National Workshop on Pest and Disease Surveillance and Management held at the In-Service Training Institute, Gannoruwa, Peradeniya from January 25 to February 12, 1982.

There is a strong need for publications on this subject particularly for use by extension staff, and in the Schools of Agriculture and training Centres. This publication describes some of the basic principles of plant pathology and other relevant information in respect of diseases, and their causal agents, in a simple manner. This would help the extension workers and trainers in developing a simple system of disease measurement, forecasting and long term strategies for management.

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Education & Training Division.

Department of Agriculture,
Peradeniya.
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# PLANT DISEASE MANUAL

(R. S. Singh)

The goal of agriculture is to optimize production of food, fibre and stimulants. Efficiency in the agriculture and forestry systems is a chief concern of plant pathologists who may be consid ered as efficiency experts. Crop destruction or disease loss being a biological phenomena as well as a social problem, and plant protection expert being directly responsible to society for solving this problem, the latter has to increasingly expand the selection and evaluation of disease management strategies to include short - and long-term loss management as a component of the broader field of crop management.

To achieve the goal the management specialist has to have a broad concept of what a disease is for the crop grower, what are the causes, how these causes behave and get in to the crop, what are their weaknesses and how these weaknesses can be exploited to manage their population to a level where they become an innocuous component of the ecosystem and do not rob the human beings of their labour to produce for subsistence.

Plant pathology is both a science as well as art. It is a science when we study the intricate mechanisms involved in disease development and planning for their control. It is an art when it comes to application of the science in farmers' fields for reducing the loss caused to their crops.

The information in this brief manual is compiled for the use of these scientists - artists to help them in demonstrating their art through education of the farmer. Only selected plant disease have been described at the end but the specialist can prepare his own description of other diseases on lines followed in this manual.

## What is disease ?

There are a large number of text books on plant pathology and there are as many definitions of disease. Few of the latest are :-

(a) Disease is a malfunctioning process that is caused by continous irritation which results in some suffering producing symptoms. The definition is accepted by American Phytopathological Society and the British Mycological Society.

(b) Disease is the sum of the normal chemical reactions that are inhibited, or the abnormal chemical reactions induced inside the cells and in the tissues of the plant as a result of the irritation brought about by the causal agent.

(c) Disease is an alteration in one or more of the ordered sequential series of physiological processes culminating in a loss of coordination of energy utilization in a plant as a result of the continuous irritations from the presence or absence of some factor or agent.

These definitions are good for a student of plant patholgoy. A practical plant pathologist can view a disease, the irritation induced by some factor or agent, in following way.

When a plant is diseased, it is at "dis-ease". It means the plant is uneasy due to some condition. (s) to which its system is not accustomed. When a man is in aun-easy position he usually loses his efficiency but sometimes he does not; he becomes accustomed to it and so maintains his efficiency. Similarly, when a plant is at un-ease it may lose its efficiency or may not. For a practical plant pathologist ( or the farmer) it is the loss of efficiency of the plant that matters. Simply being at dis-ease, without losing efficiency (production) does not matter for the farmer, or at least it should not. Hence, a realistic definition can be: a plant is diseased when its systems are not normal and therefore it is not producing as well as it should according to normal expectations of the farmer.

What causes disease ?

Any factor that puts the plant or its population to stress may result in disease. Thus the plant may suffer from physiological stress due to abiotic causes, due to biotic causes or such agents as viruses.

(A) Abiotic or inanimate causes:

(1) Imbalanced nutrition (N,P,K), micronutrient deficiency or toxicity due to excess.

(2) Deficiency or excess of water and light.

(3) Lack of oxygen or excess carbon dioxide and other gases.

(4) Abnormal soil pH, accumulation of toxic gases and metabolites in soil.

(5) Air pollutants including ozone injury.

(6) Misuse of agricultural chemicals like weedicides.

(B) Biotic or animate causes:

(1) Mycoplasma like organisms.

(2) Bacteria.

(3) Fungi.

(4) Algae,

(5) Phanerogams

(6) Nematodes, Protozoa, etc.

(C) Virus pathogens:

More than often, the symptoms produced by these agencies are overlapping and, hence, are not necessarily the identifying marks for the cause. The cause must be confirmed by laboratory tests. However, with experience in observations on pattern of incidence in the field, the nature of symptom development and often the appearance of the agent on the host surface, one can develop expertise in making on the spot identification.

For an infectious disease (biotic causes and viruses) there is always a chain of events which occur in an ordered succession to cause disease. This chain is known as disease-cycle or infection chain.

1. There has to be a source of inoculum from natural sources of the pathogen capable of inciting disease. This can be from soil-borne structures of the pathogen, internally or externally seed borne inoculum, air borne spores from other areas, inoculum from local native hosts, or from local contaminated crop residues (survival):

2. In order for inoculum to reach the infection court (the site of infection) it must be disseminated or carried to a susceptible host-;this can be accomplished by insect carriers, by air currents, by rain, by surface water, or by introduction of infected plant material (dispersal and landing):

3. After the inoculum has reached a susceptible surface, the environmental conditions must be favourable for the inoculation cycle which is comprised of germaination, penetration and colonization of the host by the pathogen (infection):

4. When this has occured symptoms of the disease, or disease expression, will be observed;

5. At this time multiplication of the pathogen will occur completing the primary cycle of host pathogen relationship (disease incidencae);

$. If environment favours, secondary infection cycle will be initiated which will go through the above steps (2-5); the disease spreads.

How the biotic causes survive ?

The knowledge of life cycle of a living disease causing agent is essential for a disease management specialist, because he will know when the population of the pathogen is going to multiply and when it will be at the lowest. It is easy to manage or fight a low population than a rapidly multiplying population. The disease cycle is often different from the life-cycle of the pathogen. It involves the host, the soil as well as other environments and tells us how the disease as a whole progresses with the changes in the pathogen population from low to high, at what stage these changes occur and which will be the right point in the chain when one should attempt to break it or interfere with it to manage the pathogen population and disease development. Example of a disease cycle is given for late blight of potato. The period when the pathogen is fast multiplying is indicated by double lines. Stages at which its population is dormant are shown by broken lines.

Survival of a pathogen is the first stage in the disease cycle. It can be active (population continues to be high) or dormant (population low, not multiplying). The sources of survial are :

I. Infected host:

1. The cultivated host - perennials-Fruit disease.
2. Wild hosts of same family- the collaberal host. Rice diseases are good example - Bacterial wilt of Solanaceae another example.
3. Wild hosts of other family - alternate host. Many viruses of legumes.

II. Sorprophytic active survival outside the host:

Soil and plant debris.

a) Soil inhabitants like Pythium
b) Soil invaders like Fusarium

III. Dormant organs of pathogens as source of survival:

Soil and plant debris - Spores, sexual fruits, Sclerotia etc.

Perennial host plant - mycelium, spores

Seed: Externally .- spores
Internally - dormant mycelium.

In citrus canker the bacterium is perpetually present on the infected tree in mild or vigorously active form. Brown spot and blast pathogens of rice are introduced in the standing crop (apart from seed and soil) by spores coming from earlier sown rice crops or suitable grass hosts. At these sources the fungus has survived in active form, thus, had a continuous infection chain. In such situations disease management becomes expensive. Same will be true for a large number of plant viruses which need living host to maintain continuity of the infection chain. In powdery mildew of cucurbits also the fungus survives on wild cucurbits. In all such cases of survival on infected host, removal of weed host, tree sanitation, resistant varieties and chemical protection of the host surface are management approaches.

In absence of a living host, the facultative parasites are capable of surviving as saprophytes. Soil and plant debris serve as media for this type of survival. Species of Pythium, Rhizoctonia, Sclerotium, Macrophomina survive as soil inhabitants for considerable length of time. Although in this type of survival resting (dormant) structures like oospores and sclerotia may play a major role, the ability of these fungi to attack and colonize dead plant materials enables them to remain active as saprophytes for some time, develop mycelium and produce more spores or sclerotia. However, due to competition from and antagonism of other soil microbiota this saprophytic survival is not very long. Ultimately these fungi will also survive only through dormant structures. The soil invaders like Cephalosporium, Fusarium and Verticillium usually live a saprophytic life only until they are having a pioneer position in the roots they had infected. When such roots are decayed and -

occupied by other microorganisms, these fungi are either displaced or form dormant structures. The root decay can be hastened by cultural practices.

The plant viruses and mycoplasma - like organisms have no resting stage and are transmitted through a continuous infection chain from host to host, mostly by vectors. The plant bacteria also do not produce resting or similar structures and therefore continuously live in active form in the host or on other substrates. Only fungi, nematodes, and flowering plant parasites are the major groups of pathogens that survive through some form of resting or dormant structures. Phanerogams produce seeds just like any flowering plant. Some nematodes maintain a continuous infection chain by living on a variety of hosts but majority of them survive through dormant structures such as eggs, cysts or galls formed from host tissues. Plant pathogenic fungi produce spores, sclerotia,thickened hyphae, etc. for dormant survival.

These dormant structures can be present in soil (soil-borne diseases), on seed coat or in the seed (externally and internally seed borne diseases), or they may be present on dormant or active host Some examples are :-

| | |
|---|---|
| Soil-borne: | Fusarium spp. (Chlamydospores) |
| | Verticillium spp. (Microsclerotia or resting mycelium) |
| | Powery mildew fungi (Perithecia) |
| | Rhizoctonia spp |
| | Sclerotium spp |
| | Sclerotinia spp |
| | Alternaria solani (Conidia) |
| Seed-borne: | Smuts of wheat, barley, sorghum etc. |
| | Bacterial diseases of cotton |
| | Anthracnose of beans, cowpea, cucurbits. |

Principles of disease management are usually based on the knowledge of survival of the pathogen. For instance, if the pathogen is carried, during off season for the crop, only on the seed (loose smut of wheat, covered smut of barley, grain smut of sorghum) it is easy to control it by giving suitable seed treatment (protectant or systemic fungicide, hot water, etc.). Effective management in such cases is no problem.Such diseases have a very slow rate of build up. When a pathogen is soil-borne, the control is somewhat difficult. One has to follow a number of soil - and crop management practices and even application of chemicals to soil to reduce pathogen population and maintain it at innocuous level. Very often the pathogens are both soil - as well as seed-borne and then the management becomes more complicated.

How pathogens are dispersed ?

Dispersal is a biological phenomena associated with every living organism. It enables it to find less crowded place to feed and also increase the changes of heterogenity and, therefore, survival through hybridization. For plant pathologists the dispersal of pathogens means trouble. There will be spread of disease, there will be new races or physiologic forms of pathogens and there may be new

hosts of the pathogen. Therefore, after tackling the survival the pathologist has to take care of the dispersal for a long-range management of pathogens.

Nature provides the pathogens the necessary mechanisms for exit from the host and move to new locations. In fungi production of asexual and sexual spores follows the active vegetative growth in or on the invaded tissues. These are dispersed mechanically in time and space by various means. In bacterial diseases, the bacterial cells come out on the host surface as ooze or the tissue may be disintegrated to such an extent that the bacterial mass is exposed and then dispersed by various physical and biological agencies. Viruses, which have no such organs, are transmitted by insects and man. Nematodes are themselves motile and try to move out of exhausted and overcrowded sites.

The mechanisms of survival of pathogens apply to dispersal also. As a matter of fact survival is "dispersal in time", i.e. the pathogen is dispersed from one active season through a period of unfavourable season to the next active season. In general, the seed-borne pathogens or diseases are dispersed by movement of seed (for which man is responsible), soil-borne pathogens through movement or displacement of soil and through root contacts, and those surviving on wild hosts (collaberal or alternate hosts) in active stage through wind-borne spores, other structures, and insects. If a pathogen could have only one method of survival or dispersal, the problem of its management would not have been difficult. But in majority of diseases a combination of mechanisms, aided by external physical and biological forces, operate in dispersal (as well as in survival). Movement of propagules of animate pathogenes through an external agency such as wind is common in even soil and seed-borne pathogens. The same pathogen may be carried by soil as well as seed and its transport may be aided by water, wind, insects, etc. The dispersal mechanisms can be classified as below :-

I. Autonomous: 1. Soil

(a) in soil - growth and movement.

(b) by soil - dispersal of soil by water, wind machines.

2. Seed

(a) Admixture: Dormant fungal structures, diseased plant debris.

(b) External : Dormant structures on seed surface.

(c) Internal : Under seed coat or in embryo.

3. Plant and Plant organs

(a) Vegetatively propagated: potato, sugarcane etc.

(b) Contaminated plants: Fruit trees.

II. Passive: 1. Dispersal by animals:

(a) Insects (b) Nematodes (c) Birds.
(d) Man.

2. Water

(a) Raindrop splashes
(b) Irrigation
(c) Mist.

3. Air

(a) Direct lift off

(b) Indirect through wind blown soil or mist.

How disease develops ?

After survival during off season, multiplication, where possible, and dispersal the pathogen lands on the host. If environmental conditions are favourable for the pathogen penetration and, finally, infection will occur. Environmental conditions include not only the physical environment (moisture, temperature, pH etc) but also the biological environment (susceptible host, presence or absence of antagonistic microflora on the host due to unfavourable physico-chemical as well as biotic environment). The infection leads to incidence of diseasee (the disease has appeared in the population). The pathogen may have a single cycle, i.e. what ever is the incidence that will decide the final severity of the disease In other words if from the primary inoculum (say 80% infected seeds giving 80% infected plants) a certain number of plants is diseased, the number will be final. The pathogen has no repeating cycle in the season. Epidemics of such diseases build up slowly and these are easy to manage. However, in majority of plant diseases, the pathogen has a repeating cycle also within the crop season. This means that the pathogen multiplies on the host, the spores are dispersed and new surfaces are infected. The rapidity with which the cycle is repeated determines whether the disease will assume epidemic form or not. The rapidity of multiplication and amount of inoculum produced for spread depends on weather and amount of susceptible area present in the field or locality. A favourable weather and extensive susceptible area means rapid build up of an epidemic. Such diseases usually pose complicated problems in management and often involve costly approaches directly or indirectly. Thus, plant disease development is a function of interaction between the host, the pathogen and the environment, the last affecting both of the former.

The severity of disease can be expressed by the following equation for the purpose of understanding approaches to management:

Disease severity = Inoculum Potential X Disease Potential

= (Pathogen inoculum denisity X Inoculum capacity) (Host susceptibility X Host proneness.

It is the disease severity which is responsible for loss through injury and damage of individuals. In the above equation there are four components involved in disease development leading to severity. Two are from the pathogen side and two from the host side. All the four are in totality governed by environment.

Pathogen inoculum density means the number of propagules available at the site for causing infection. While in some diseases one propagule (spore or similar structures capable of multiplying) is enough to cause infection, in majority a certain density of spores is essential.

Inoculum capacity is the energy available with these propagules to cause infection. Even if a high density is present it may fail to cause infection if the capacity factor is also not favourable. If plant parasitic nematode larvae are left to wander in soil for long without a host, they use up their lipid reserves for energy and soon become so weak that when a host is available they have no energy to cause proper thrust for penetration.

Host susceptibility (reverse of resistance) is a genetic character. Although often its expression is changed by environments (weather and climate) normally one cannot do anything with it in a standing population.

Host proneness is environment induced weakness in the host to make it prone to catching disease. The factors are called predisposition. Physical, chemical and biotic environments (including crop management practices) may be such that the plant becomes weak enough to be easily invaded by a pathogen. While proneness is easily expressed in susceptible varieties, even resistant varieties may become prone to disease if put under undue physical, chemical and biological stress. Breakdown of wilt resistance in many plants due to concurrent attack of certain nematodes is one example.

The management of plant diseases on the basis of above condition therefore involves four steps, viz reduction in inoculum density (cultural and chemical methods), reduction in inoculum capacity (mostly cultural methods to starve the pathogen), use of resistant varieties and avoidance of predisposing factors. If all the four components could be taken care of, there should be no disease. But it is normally not possible. Hence an integration of the four approaches is always recommended, as far as possible, so that there is reduction in disease severity to a level where there is no economic loss. The integration has two major advantages, viz: chemicals are used only where they are a must, and resistance of varieties is prolonged-; even tolerant varieties do as well as resistant varieties if not better.

## Role of Environment in Disease Cycle

As has been emphasised earlier the environment is the most important determinant of development of any disease and affects all stages of disease cycle (survival, dispersal, landing on host, the process of germination and infection, symptom expression and multiplication in and on the host). It is essential also for developing a system of forecasting. The major factors are temperature, moisture (humidity) and wind but other factors such as soil pH, host nutrition, sometimes light, may also become important.

The seasonal occurence of many diseases and their severity is known to be due to seasonal temperature variations. In warmer regions wheat suffers more from black rust while in colder regions brown and yellow rust are more common. The severity of an infectious disease being dependent on the number of repeating cycles the pathogen can complete within the crop season temperature determines, through its direct effect on the pathogen, how many cycles will be completed. Thus, the incubation period (time between infection and symptom development, i.e. multiplication of the pathogen) of P. graminis (wheat black rust) is only 5 days at 24°C, 9 days at 19°C and 22 days at 5°C. The incubation period of cotton will fungus (F. vasinfectum) is 12 days at 25°C and 58 days at 16°C. The root knot nematode (Meloidogyne javanica) takes 16 days to complete one cycle at 27°C but 80 days at 14°C. In rice blast plant age and nutrition may be of minor importance if temperature and moisture conditions are favourable for infection. In high humidity, best dispersal of spores occurs at 25-27°C while at 20°C the proneness of the host increases. At very low temperature (15°C) even resistant varieites may become susceptible. The late blight of potato is generally absent in areas where mean atmosphere temperature exceeds 25°C. At 12 - 13°C the fungus produces mostly zoospores while at 24°C mostly direct germination occurs. Hence at low temperature chances of spread are more. The onion bulb nematode is killed within a day if the infested soil or bulbs are exposed to 37.8°C.

Together with temperature, moisture is also important. Late blight of potato is a good example. Even if temperature (low) is favourable for disease, it will not appear or spread rapidly if the weather is dry. A humid period is necessary for spore formation, a relatively drier period for dispersal and again a wet period for germination and infection. In blast disease spores are not produced below 88 % RH. Best production of spores occurs between 4 and 6 a.m. when RH is above 90%. A minimum night temperature in the range of 20 - 26°C in association with a RH range of 90°C and above for a week is essential for disease development.

Macrophomina rot of collar and roots is favoured by dry soil, irrigation reduces the damage. Rhizoctonia, Pythium, Sclerotium, etc. are favoured by wet soil.

In disease descriptions given at the end of this booklet, the environmental relations are also specified.

Disease control or disease management?

There is a convincing rationals supporting the substitution of "Management" for "Control". The word "Control" is an improper term since it evokes the notion of finality, of having controlled or permanently settled a problem. But a disease is rarely finally disposed off. That is why we have a recurring schedule of operation every year to prevent the loss caused by the disease. Recurrence of disease after application of a "Control" measure is viewed by the farmer as a failure of the control system.

"Management" conveys the concept of a continuous process It implies that diseases are inherent component of the agroecosystem that must be dealt with on a continuous, knowledgeable basis.

Management is based not on complete or partial elimination of the pathogen but on the principle of maintaining the damage or loss below an economic injury level or at least minimizing occurrence of disease above that level. Management suggests the need for continuous adjustment in the system.

How diseases are Managed ?

A plant disease is the function of interaction between host, pathogen, and environment. Any approach to sustained management of disease depends on the management of all the three components (Fig. )

The philosophy and approach of the crop protection sciences have been influenced significantly during the past 15 years by the evolution of an integrated pest management (IPM) concept. Developed originally by entomologists as an integrated control approach for insects, this concept is equally applicable to diseases caused by all groups of pathogens.

Principles of Disease Management

The following principles of disease management are listed in the order of their application in developing a disease management programme.

(1) Identify the diseases to be managed: A problem disease is generally first seen by the farmer. If he does not recognize it he seeks the help of the specialist who must assemble the relevant information about ecology of the pathogen, the epidemiology (development of disease in the population, weather relations, mode of transmission, etc.) and also other relevant information.

(2) Define the management unit - The Agroecosystem : An agroecosystem comprises the total complex of organisms in the crop area, together with all aspects of the environment as modified by the various agricultural, industrial, social activities of man. A natural ecosystem is a basic functional unit of nature that includes both organisms and their non living environment, each interacting with the other and influencing each other's properties, and both necessary for the maintenance and development of the system. Both systems have no well defined boundaries but once defined, the connection between the system and inputs and outputs can be visualised. Agroecosystem is a very dynamic (changing) successor stage of a natural ecosystem. Cultivation, irrigation, fertilization and pesticide application break the normal ecological succession that would have proceeded in absence of these activities. There is always invasion of this system by a characteristic set of organisms that are determined by the quality of the agroecosystem and its surrounding environment. Many of these invaders become cohabitants and are beneficial (root nodule bacteria, mycorrhiza, rhizosphere non-parasitic migro-organisms) but some compete with, feed upon, or parasitize the cultivated plants.

The migrational capacity of these injurious organisms determines the boundaries of the agroecosystem that must be managed. If the migrational capability of the pathogens are limited (for example certain nematodes) the boundaries of the management unit may be restricted to a single field, but if these capabilities are high (for example air-borne diseases like cereal rusts or powdery mildews) the pathogen ecosystem may include most of a continent and consequently the management unit will be large, at least the entire region.

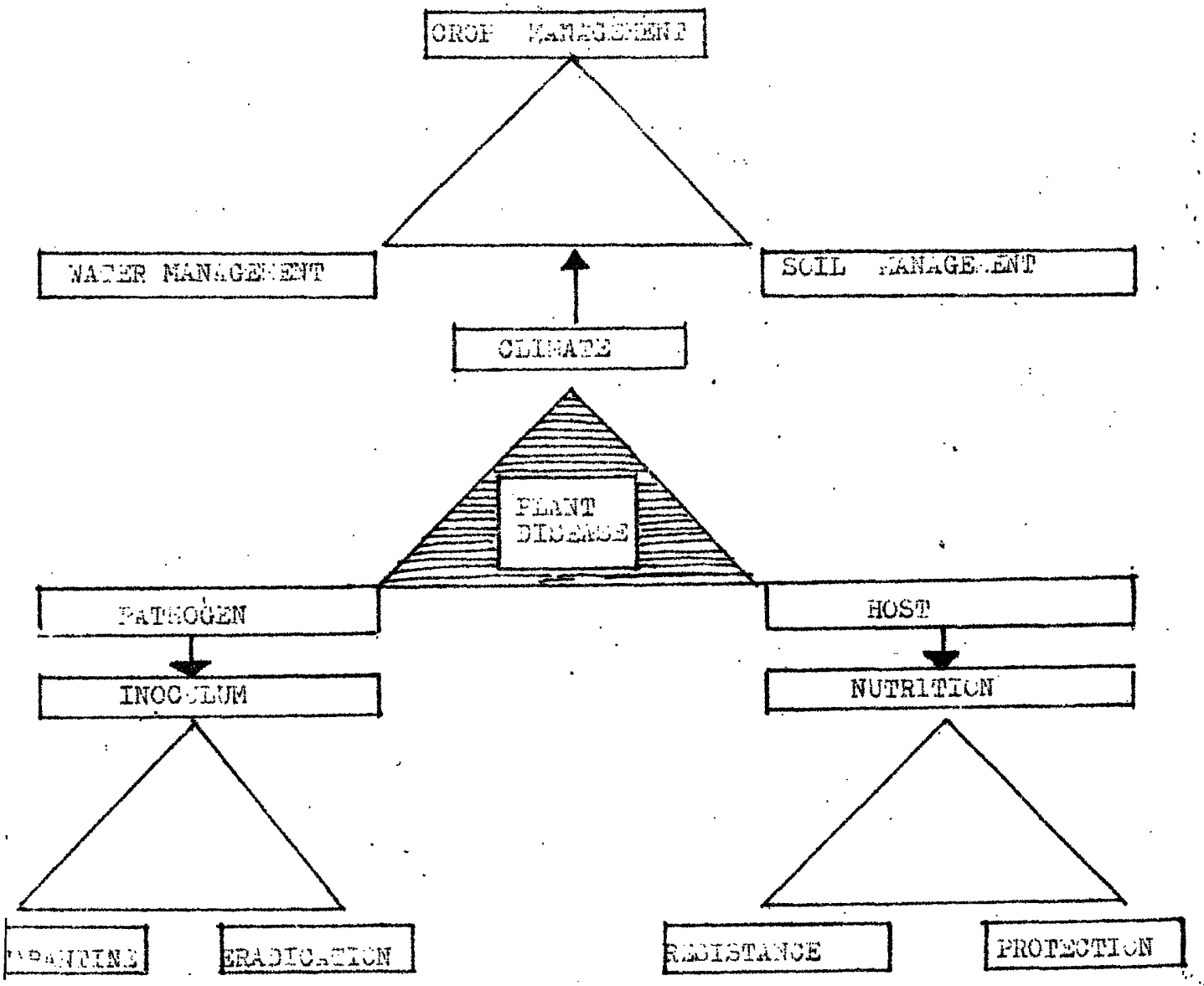
(3) Develop the management strategy: Some aspects of this have been covered on earlier pages such as the need for study of disease cycle, the components of disease severity etc. There can be a short term strategy and a long term strategy, the latter being more important for sustained management. Details of components of long term strategy, management of environment, associated biota, weeds, host genes etc. are given later.

In any strategy to manage a population, ecology of the organism (here the pathogen) is important. One must know how the pathogen responds to its environment (natural or modified by man) so that its weaknesses could be exploited for population check. At the same time knowledge of the nature of development of the disease in the plant population is also essential because it provides information on how a disease will progress from a known or detected amount of inoculum, under a set of weather or environmental conditions including the crop variety.

(4) Establish economic thresholds:Plant disease have been studied because they damage cultivated crops. They are controlled or managed to prevent the loss. If there is no loss from a disease, there is no need for any management or control practice. Since application of many practices involves cost which may go towards loss being suffered by the grower, there is always a side of costs benefit ratio in every production operation. The cost involved in the operation must increase yield to cover not only the expenditure but should also ensure some additional gain. Hence, in every disease management operation an idea of the possible loss, of the possible expenditure and possible gain is essential. However, even today there are few reliable estimates of loss. Since the development of economic threshold requires estimates of loss, most of the costly disease management operations are being undertaken without taking into consideration the economic threshold.

The economic threshold is that level of disease intensity that produces an incremental reduction in crop value greater than the cost of implementing a disease management strategy. Losses due to disease must be based on measurement of both the quantity and quality of the crop production. Only in this way can accurate relationship be established between amount of disease and amount of loss. Side by side, the relationship between the cost of alternative disease management tactics and their economic benefits must be established. These two determinations, plus willingness of the farmer to accept the uncertainties (risks) involved, constitute the basis for disease management decisions.

(5) Develop monitoring techniques: Finally,in every disease management programme there will be need for techniques to keep a watch on the crop (surveillance) and monitor the build up of inoculum and disease. This will enable the utilization of management tactics only when actual or predicted loss reaches the economic threshold level. However, it may be kept in mind that there are a large number of management tactics which do not involve cost to farmers (resistant varieties, cultural practices such as change in fertilizer, irrigation application etc). The former are part of the normal crop production technology and hence they are not covered by the constraint of economic threshold.


CROP MANAGEMENT
WATER MANAGEMENT
SOIL MANAGEMENT
CLIMATE
PLANT DISEASE
PATHOGEN
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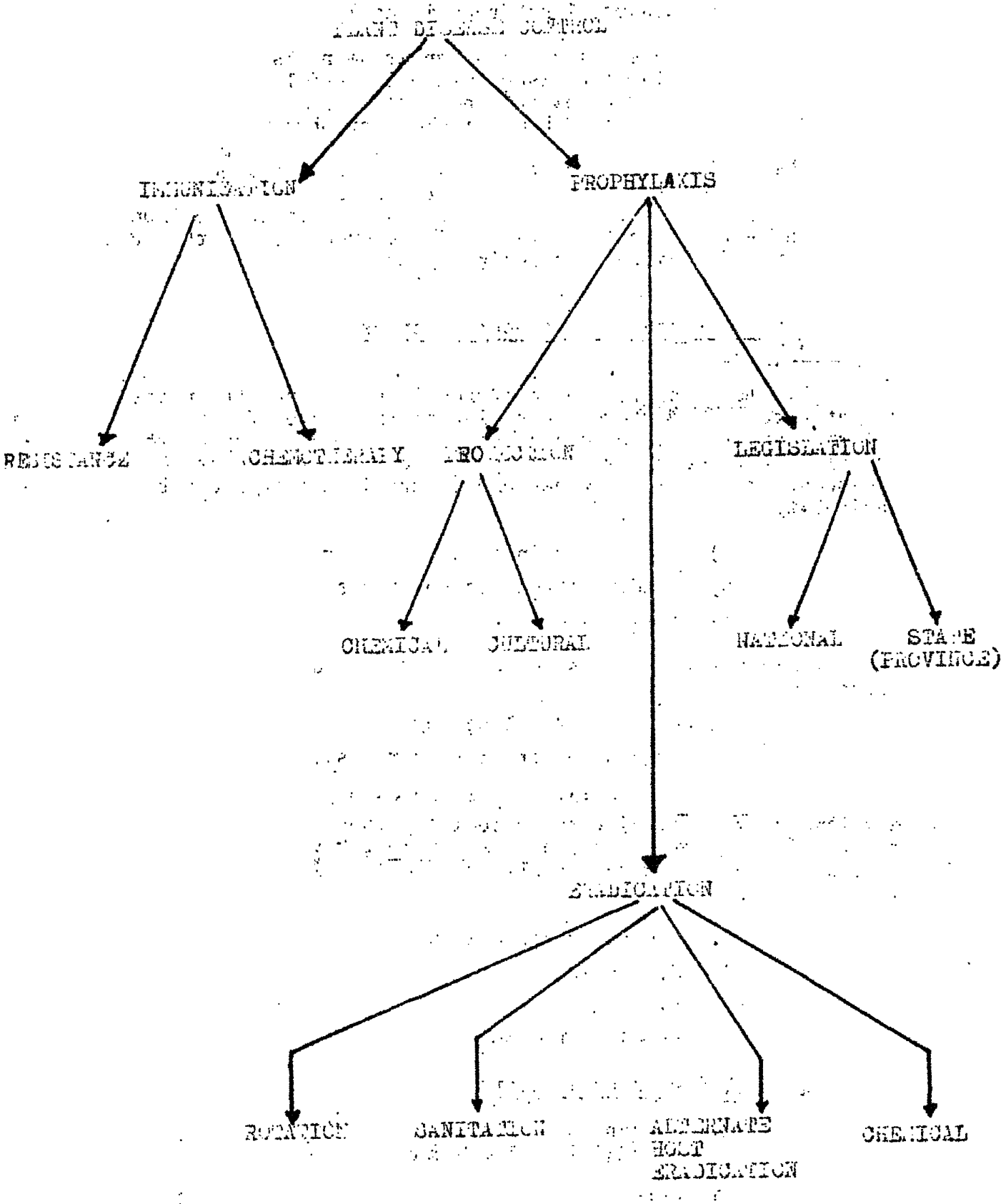
INOCULUM
NUTRITION
ERADICATION
RESISTANCE
PROTECTION

THE IMMUNIZATION - PROPHYLAXIS SYSTEM



## HOW TO MANAGE THE ENVIRONMENT

(To reduce loss from Disease)

As stated in earlier pages, environment is one major component determining the disease severity and loss. While it cannot be managed to the extent one would wish to do there are practices which can bring about significant physical, chemical and biological changes in the environment of the crop and the field. Emphasis on this approach can reduce dependence on chemicals at no extra cost. The approach is mainly based on the fact that there are discontinuities between the optimum conditions for growth of a plant and the growth and pathogenic development of a disease producing agent.

### Managing the physical environment of crops in the field:

1. Temperature: Temperatures in and around the plant and at sites of pathogen -host interactions are often the determinant of invasion and development of and injury by the pathogen. If necessary knowledge of temperature relations of a disease are available, the temperature in the crop can be manipulated by :-

   a) Alteration in date of sowing.
   b) Shading through cover crops, mulching etc.
   c) Irrigation.

Temperature can also be employed to reduce inoculum load in soil by:-

   a) Hot weather ploughing
   b) Burning of trash over the soil.

2. Moisture: Humidity is important determinant of development of many diseases especially those causedby Phycomycetes (Mastigomycotina) such as Pythium, Phytophthora etc. Moisture can be managed as well as used for disease management through :

   1. Row and ridge cultivation
   2. Irrigation,
   3. Flooding
   4. Spacing of crops.

3. Other Physical Factors:

   a) pH can be altered through liming or addition of sulphur

   b) Nutrition: Quantity and quality of nitrogen source; ratio between NPK; use of trace elements.

Managing Disease by Cultural Control:

1. Crop Rotation reduces activity, pathogenesis and survival of soil-borne pathogens through starvation as well as through changed biotic and chemical activities in soil.

   The type of soil-borne pathogens is important for effectiveness of crop rotation. Rotation is less effective tool against soil inhabitants. (eg. Pythium) than against soil invaders (Fusarium oxysporum, Verticillium) whose active survival in soil is restricted to the time during which invaded roots of host are present. It is also not very effective against pathogens which have a large host range (Sclerotium, Rhizoctonia solani, root knot nematode Meloidogyne). The choice of crops and length of rotation depends on the nature of crop host roots and survival ability of the pathogen. Host roots that take long to decompose warrant a long rotation Generally legumes after legumes or solanaceous crops after solanaceae are avoided even if they are non-host. This is because often the roots of non-host plants favour growth of the pathogen without being parasitised.

2. Sanitation: Sanitation is an important cultural practice which has the essential aim of reducing the initial inoculum (inoculum density). It includes such practices as removing diseased plants (roguing), pruning of infected parts of plants (surgery), removing or effectively treating plant materials containing inoculum (fallen leaves, twigs, fruits etc.) and preventing inoculum from finding suitable infection courts (preventing wounding, establishing barriers, defoliation).

Sanitation is particularly applicable to pathogens that do not spread from plant to plant in the field and that require a large amount of inoculum to develop an epidemic. It is particularly important when crops are grown in the same field for several years (legumes, potato, etc.)

Sanitation includes roguing also. In roguing we remove not only the diseased plants of the main crop but also the weeds or alternate hosts. Destruction of plant residue that may harbour inoculum constitutes sanitation which can be accomplished physically by burning, ploughing and flooding. Similarly pruning of diseased plant parts, digging trenches around diseased tress etc., are also sanitary precautions.

Sanitation serves as an important supplement to other disease control measures such as fungicidal sprays and even tolerant or resistant varieties. By removal of inoculum containing dead material of the crop host and by removing the weed hosts standing in the field along with the crop one can give fewer but effective fungicidal sprays and also prolong the life of a resistant variety which could otherwise break down under the pressure of high inoculum load.

3. Clean planting material: Management of the environment includes also the use of clean stock (cuttings, grafts etc.) and clean seed. The number of diseases carried or spread by this agricultural input is so large that almost all the countries in the world are trying to set up seed supplying units which could supply disease free certified seeds to grower. Seed certification does not include only the varietal purity and germination percentage but also the seed health. The seed should not carry

any disease causing agent that has the potential of developing in the field in the field and causing economic injury. The major steps in the production and distribution of healthy seed are : (a) Inspection and certification of seed plots as per certification standards fixed by the country on scientific basis,

(b) Where necessary laboratory inspection of seed samples to detect prevalence of serious pathogens and;

(c) Treatment of seed if necessary.

In absence of such a step the farmer himself has to ensure that he is using seed or planting material from disease free field/or any other source. He can do his own seed treatment with simple machinery.

Normally seed produced in semi-arid areas contains less load of pathogens than seed produced in cool humid areas.

4. Harvesting practices: Consideration of the life cycle of the pathogen is important in relation to avoiding contamination of the harvested crop by storage pathogens. To avoid tuber rot by the late blight fungus (Phytophthora infestans),potatoes should not be dug until the tops are either removed or killed. In the same crop, normally the hills that have shown any sign of wilting or viral infections are either rouged out during the crop season or are harvested separately to maintain cleanliness of the apparently healthy tubers. In rice,patches of plants showing symtoms of bakane disease and stem rot, can be marked out and harvested separately. In chillies, beans, etc. effort should be made to separate the pods or fruits bearing symptoms of anthracnose.

## MANAGEMENT OF THE ASSOCIATED MICROBIOTA

(To reduce losses from soil-borne root disease)

Management of the associated microbiota is the manipulation of micro-organisms associated with the pathogen, the host, or disease for the purpose of reducing disease. While this manipulation can apply to foliar diseases also (rusts, powdery mildews, virus vectors) it has been found highly useful for managing root rot, wilt and nematodes. It is a major form of "biological control" (host resistance, use of multiline varieties and use of trap crops are other forms of biological control).

The basis for management of microbiota is the fact that the agricultural operations bring about significant changes in the microbial component of the agroecosystem and these changes could be exploited to stimulate organisms which are harmful for active or dormant pathogens. The different elements in the theoretical base of this management principle are :

1. Reduction in Inoculum Density of the Pathogen through such activities as (a) rotation in which the pathogen is starved or weakened to the extent that it becomes an easy prey for attack of other organisms and is consumed, (b) direct attack by hyperparasites, (c) killing of dormant sclerotia and spores by fungi, bacteria, actinomycetes, amoebae, etc. (d) lysis of germ tubes or growing mycelium by action of bacteria and actinomycetes, (e) activity of nematode trapping fungi or parasites of nematodes.

2. Replacement of a Pathogen in Plant Refuse: This approach applies to THOSE pathogens that depend for survival on occupancy of plant residues during host free periods such as Fusarium udum (pigeon pea wilt fungus), Fusarium oxysporum f.sp. cubense (Panama wilt of banana), Fusarium oxysporum f. sp. lycopersici (tomato wilt) species of Cephalosporium, etc. The pathogens in such cases, due to their earlier possession of the host, occupy a pioneer position and use the debris as a place of shelter and food base. They resist intrusion by other microorganisms in the soil. This must somehow be nullified so that the inoculum surviving through crop refuse is reduced before next crop is planted.

The key to active possession of the crop refuse by the pathogen is its continued slow metabolism. The hold of the pathogen on the debris can be weakened by (i) a physical environment unfavourable to metabolism of the pathogen but not to potential colonists from outside, such as by changing soil pH, reducing or highly increasing moisture level, (ii) lack of an essential nutrient normally supplied by soil, such as withdrawal of nitrogen through application of high C/N materials to soil, (iii) stress on the occupant imposed by a sublethal dose of a fungicide or fumigant.

3. Suppression of Germination and Growth of Pathogens:

This form of biological control includes any effect that reduces or prevents germination of propagules (fungistasis), or if germination occurs, any effect that slows down the growth of the pathogen or cripples it (starvation, lysis, antibiosis, parasitism or predation.).

There does exist a natural and widespread soil fungistasis, that is, inspite of physical environment favourable for germination and growth and inspite of the spore being not under inherent dormancy, it fails to germinate in soil. There is evidence that this happens because in the soil both stimulatory as well as inhibitory substances are continuously produced by biodegradation of organic matter and when the inhibitory substances are more than stimulatory substances there is suppression of germination of spores. Therefore, by increasing microbial activity in soil through raising the organic matter status of the soil. chances of suppression of propagule germination are enhanced. However, this fungistasis can be broken down by root exudates of host or nonhost crops. In that case the high microbial activity may prevent growth of the pathogen through starvation by competing for food, lysis and such other antagonistic activities.

4. Protection of an Infection court: It means any action of the micro-organisms in or on the site of infection (infection court) that slows or prevents infection by the pathogen. The action may involve production of an antibiotic or other substances that may inhibit or injure the pathogen and also production and intake of substances by plant roots that may impart some sort of resistances. The action also includes the cases of crossprotection wherein a nonpathogen or a weak pathogen takes possession (pioneer colonist) of the infection court and adjacent tissues, causes little or no disease, but prevents a more potent pathogen from getting into the tissues. Mycorrhizas, and antibiotic producing organisms in the rhizosphere are examples of naturally occuring protectants of infection court.

How to Induce Heterogenity in Soil microhlora and fauna to manage microbiota:

A. Organic amendments: 1. Green manuring
2. Ploughing in of dry staw.
3. Oil-cakes of castor, margosa, Pongamia, etc.
4. Wood sawdust, rice husk etc. along with supplemental nitrogen.
5. Fish, blood meal etc.

B. Nitrogen Fertilizers: The source and quantity of nitrogen differently affects different pathogens and with proper knowledge can be used to manage disease. Sclerotium rolfsii is suppressed by 120-150 lb N/Acre through a nitrate source or urea. Same is true for root rot of beans caused by Rhizoctonia solani. On the other hand, nitrate nitrogen increases Pythium, Verticillium, and incidence of powdery mildews and rust of cereals.

C. Treatment with pesticides or soil fumigants: Pesticides or soil fumigants are not good alternatives so far as soil microbiota and pollution is concerned. However, most fungicides are harmless since they quickly get degraded in soil

(mercury compounds are exceptions). The fumigants are also harmless because of their volatile nature. Therefore, for strengthening of other measures their use can be recommended, especially at sublethal rates acting against the pathogen through associated soil microbiota than through a direct effect on the pathogen. Use of carbon disulphide, Thiram , DCP etc. against Armillaria mellea, Helminthosporium sativum or Verticillium are good examples. The root knot nematode eggs are usually protected by root tissues and most of the nematicides are larvicidal not ovicidal. When nabam is applied to soil,it induces hatching of eggs and if a fumigant is applied after this treatment, there is better kill of the nematodes.

D. Tillage provides a temporary improvement in aeration, accelerates drying and redistributes substrates. These, in turn, can expose the pathogens to antagonistic effects of the microbiota. Thus, the two main functions of tillage, in terms of biological control, are (i) reduced inoculum density and (ii) displacement of pathogens present in the crop refuse.

Tillage methods used to manage specific disease are- (i) early and deep ploughing to kill host roots and favour the saprophytes rather than the root pathogens like Fusarium udum, (ii) ploughing to bury the plant debris deep in the soil so that pathogens that require free air for production of sclerotia and vegetative growth (viz: Sclerotium rolfsii) are put to a disadvantage, and (iii) subsoiling to permit deeper root penetration and reduce root rot caused by such fungi as Fusarium solani. Loosening the soil also helps in escape of injurious volatiles that usually predispose plants to disease.

E. Irrigation and Flooding: Management of soil water offers two means to manage the associated microbiota: directly, through water potential, and indirectly through gas exchange of the soil. The biological control achieved is through reduction of inoculum density, suppression of pathogen growth, its replacement in the host refuse or protection of the infection court. When the fields are kept irrigated during tuberization, the potatoes escape infection of common scab (Streptomyces scabies) Irrigation may reduce water stress in some hosts, thereby preventing pre-disposition to root rots. Macrophomina phaseolina becomes a dominant pathogen only when plants are under stress, mainly water stress, and when the soil temperature is high. Irrigation not only reduces water stress it lowers the temperature also thus charcoal rot diseases caused by this fungus are suppressed. High moisture favours bacterial activity while low moisture is favourable for activity of actinomycetes. Both the groups have antagonists of pathogens. Therefore, depending on the ecology of the pathogen suitable adjustments in water regime can be determined for specific diseases.

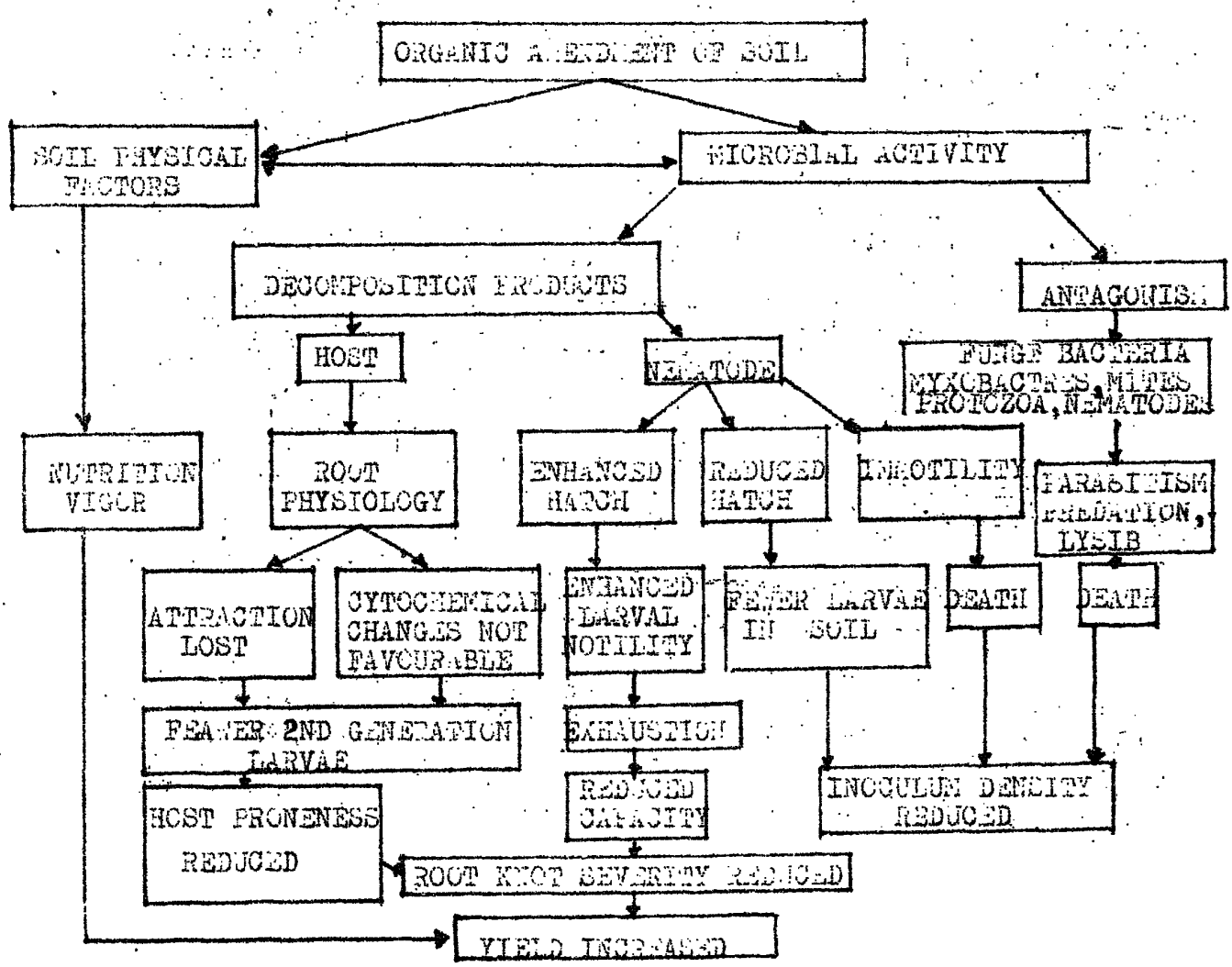
Flooding has been recommended for the control of Panama disease of Banana (Fusarium oxysporum f.sp cubense), sclerotia of Sclerotinia sclerotiorum and also root know nematodes (Meloidogyne spp.) The underlying mechanism is the weakening of the pathogen through lack of oxygen and production of toxic substances in anaerobic decomposition of organic matter. A period of dry fallow followed by irrigation or rain cause the sclerotia of Sclerotium rolfsii to leak nutrients, germinate, and rot due to microbial activity on or near them. When the soil is kept wet for 2 - 3 weaks at temperatures near 30°C there is rapid deterioration of sclerotia of Macrophomina phaseolina.

F. Management of microbiota with the crop: The crop (the entire vegetational cover) has a major influence on kinds and numbers of organisms present in soil, through root exudates while growing and through the residues left after harvest. Each type of plant species growing on a particular piece of land has its own chemical make up including the chemical constituents of the root exudates which decides the type of microflora and fauna in the soil.

Crop rotation creates hetergencity in soil biota due to change of crops one after another. This increases chances of propagule destruction through starvation as well as through antibiosis and other chemical and biological actions. Contrary to this, monoculture has a stabilizing influence on the soil microbiota. Although it may have adverse effect on crop growth, and sometimes health, there are examples where monoculture has succeeded in controlling a soil-borne disease (viz take-all of wheat, Phymatotrichum root rot of cotton, common scab of potato). This supression of disease has been termed as "disease suppressive soils" which is considered to be of biological ORIGIN.

G. Introduction of Antagonistic Microorganisms in to soil is theoretically a very sound practice but it can succed only when full ecological characters of the antagonist are known and the soil is receptive for it.

A FLOW CHART SHOWING HOW DECOMPOSITION OF ORGANIC MATTER IN SOIL CAN POSSIBLY PREVENT LOSSES FROM A DISEASE (EXAMPLE ROOT KNOT NEMATODE)



## MANAGEMENT OF HOST GENES

## ( THE RESISTANT VARIETIES)

Plant pathogens are part of the natural ecosystem, so their elimination from the system is neither possible no desirable. The association of pathogens with the host is also a natural part of the system. It is the imbalance of the host-pathogen association in favour of the pathogen which creates economic problems for us. Hence, we should ensure that the balance is in favour of the host rather than the pathogen. The management of environment and microbiota,discussed on preceding pages were only the first line of defence to ward off the loss caused by the pathogen. If this fails, then it is necessary to prepare the host to fight the pathogen. This is possible thorugh genetic or induced resistance in the host.

Details of terminology used to express types of resistance in plants, mechanisms of resistance, etc. can be read from any text book on Principles of Plant Pathology. Certain practical aspects are mentioned here. The term disease escape refers to a condition wherein the variety is otherwise susceptible but escapes the disease due to its other agronomic characters and practices(early maturing,erect shoot,etc). This is an important method of preventing loss because the farmer can adopt it without any investment or much change in his schedules of operations. Then, there are varieties called "tolerant", a term indicative of relative loss suffered. A population of host plants is defined as having tolerance if technically the variety is rated as suseeptible but is damaged less by the epidemics than another susceptible population. Although, by definition tolerant plants must look susceptible, practically such varieties are resistant and such resistance is of general nature. Sometimes a tolerant variety proves better than a truly resistant variety since the tolerant character lasts longer than true resistance. When aided by other management practices these varieties often give better yields.

RESISTANCE TO A DISEASE IS CHEAPEST AND CLEANEST METHOD OF DISEASE CONTROL.

But the major difficulty with it is that the pathogens go on changing their nature by producing new races or biotypes, especially when a resistant variety dominates the area. It is always advisable not to grow one resistant variety in the area over large acreages and for many years and always support the variety with suitable cultural practices against diseases.

## MANAGEMENT WITH CHEMICALS

Chemicals become essential to successful crop production only when cultural practices, host resistance, alteration of the environment or alteration of the associated biota are inadequate to suppress the pathogen sufficiently. Chemicals are generally used on high value crops. Low value crops have only chemical seed treatment. They are also generally used on crops grown in environments conducive to the pathogen.

### The need for efficient use of chemicals:

Losses due to disease might be lessened by greater use of chemicals but the high cost associated with chemical disease management must be weighed against the potential benefits and whether alternative methods can be used or not. The economic costs include the cost of the chemical and the cost of application. There are noneconomic costs that should also be considered. These include the risk of damaging natural enemies of pathogens, the risk to humans, to other animals and to the environment. Thus, the use of chemical should be decided on the basis of cost; benefit as well as risk; benefit analysis. Whenever it is decided to use chemicals, its efficiency must be assured through (I) timely application (2) proper dosage and (3) proper coverage of the plant, field and the whole area.

### PRINCIPLES OF PLANT DISEASE CONTROL WITH CHEMICALS:

These general principles are illustrated in the enclosed figure. The term "surface protection" means covering of a surface (leaf, stem, fruit, seed or flower) with a fungicide which will not permit penetration of the surface by a fungus against which it is meant. Amongst these chemicals there may be " seed protectants", meant mainly for seed treatment (for example Thiram) or " foliage protectants" (for example Zineb) meant mainly for spraying or dusting on the foliage.

Another category of fungicides has been specifically formulated to inactivate or prevent infection by soil-borne pathogens. These are known as "soil treatment products". These products may be fumigants (viz. D-D mixture) or granules (Furadan) or mix (viz. Terraclor or Brassicol).

Very few fungicides will "eradicate" or "cure" established plant disease infections. Those which can really kill a pathogen at the site of infection are "eradicant fungicides" but most act as protectants and hence should be known as plant or crop protectant or antipathogan chemicals. The chemicals which cure the disease or can prevent development of the pathogen inside the tissue are "Chemotherapeutants". Many fungicide chemicals neither kill the pathogen nor protect the tissue but do not allow the pathogen to produce spores, thus checking apread of the disease. They are "antisporulant chemicals".

A list of various chemicals, their dosage and diseases against which they are used, safe use of fungicides etc. are provided at the end of this section.

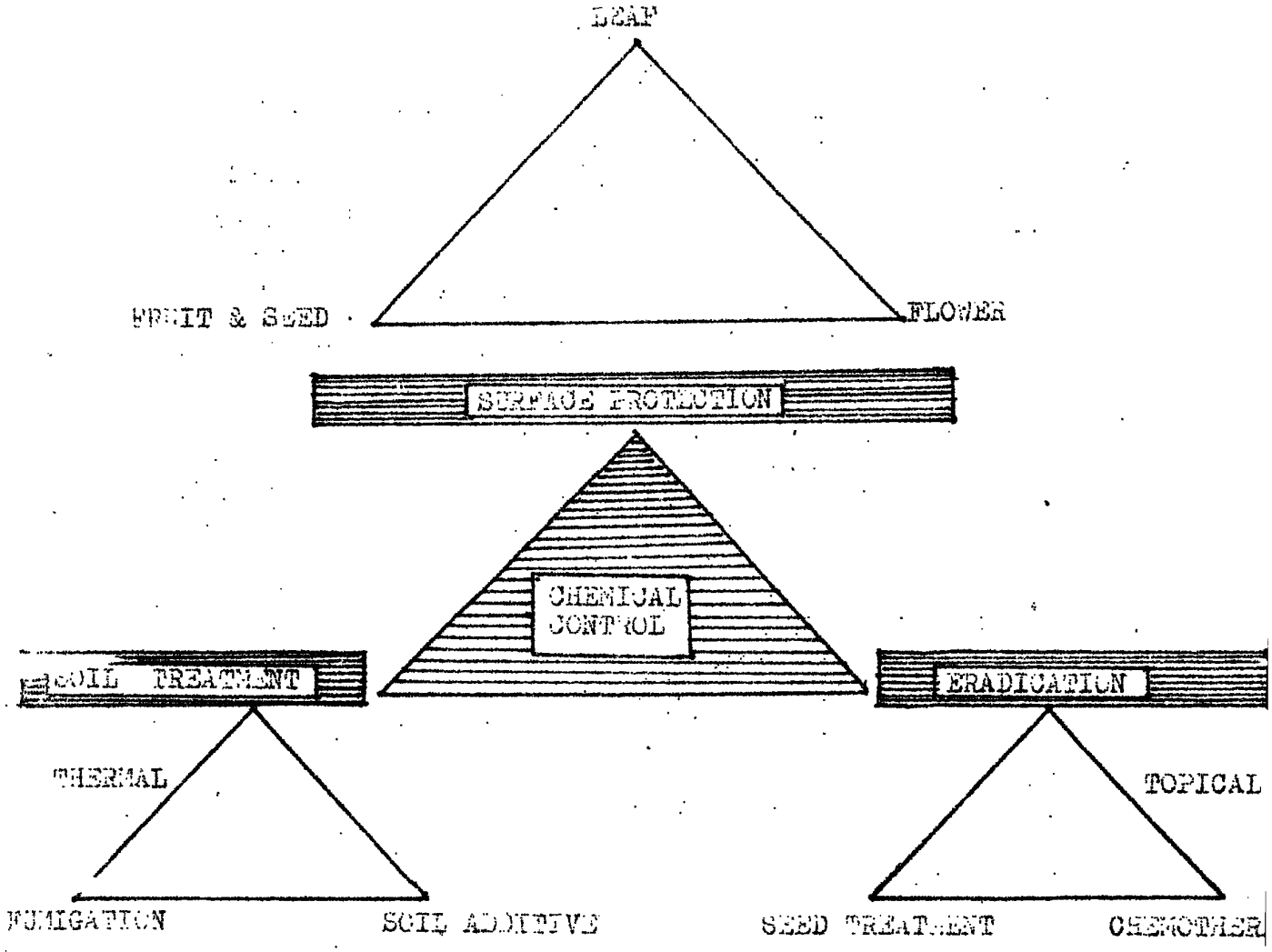
## THE IMPORTANCE OF CORRECT TIMING OF PROTECTIVE FUNGICIDES:

The correct timing of the application of a fungicide for protection of foliage is the fundamental key to its successful use. It must match with the life cycle of the pathogen or the disease cycle in such a manner that the disease cycle is broken with the first application and minimum subsequent applications are required. The example of potato late blight partly explains it.

The late blight starts from infected tubers, surviving mycelium in left over tubers in the field or from the fungus surviving actively on such hosts as tomato. The infection from seed tubers initiates spot formation on the same plant. Infection from left over diseased tubers may initiate disease on any plant in the field. Infection from existing hosts such as tomato may bring infection on a large number of plants in the field through spores. Obviously, the source of primary inoculum is variable and the farmer may not know of it. In addition to this, there is the problem of weather conditions favourable or unfavourable for spread of the disease. In these situations it is always better to givw a prophylactic spray of fungicides before conditions become favourable for disease apread, i.e, when the foliage has expanded enough to provide suitable humidity for disease spread or before the spores of the fungus get an opportunity to inititate their life cycle. If spraying of a fungicide is done at the right time, not more than 4-5 sprayings will be required to prevent an epidemic.

## THE IMPROTANCE OF UNIFORM COVERAGE:

A fungicidal protective coverage is effective only when it is not put to undue stress. If the inoculum is perpetually coming, any amount of fungicidal spray will be ineffective. Therefore, it is essential that the chemical protection covers not only the entire surface of the individual plant but also the entire field and the entire area susceptible to disease.




( FROM SHARVELLE 1979)

## Chemicals used in plant disease management

I. SULPHUR: Inorganic such as sulphur dust, wettable sulphur, lime-sulphur.

Organic such as Thiram, Zineb, Ziram, Maneb, Mancozeb, etc.,

II. COPPER: Bordeaux mixture
Copper oxychloride

III. MERCURY: Mercuric chloride
Organic: such as ethyl mercury chloride (Ceresan),

IV. QUINONES: Chloranil (Spergon), dichlone (Phygon)

V. BENZENE: PCNB or quintozene ( Brassicol, Terraclor, Avicol, Tritisan)

dinocap (Karathane, Mildex, Crotothane)
dichloram (Botran)
Daconil or chlorothalonil (Daconil, Bravo, Termil)

VI. HETEROCYCLIC NITROGEN COMPOUNDS:

glyodin (Glyodin)
Captan (Captan, Captane, Merpan, Orthocide)
Captafol (Difolatan)
Folpet (Folpan, Phaltan)

SOIL FUMIGANTS: Methyl bromide, ethylene dibromide (EDB)
D-D or Nemafume or Telone ( dichloropropane-dichloropropene), Nemagon or Fumazone (dibromochloropropane or DBCP),
Vapam (sodium methyl dithiocarbamate or SDMC)

SYSTEMIC FUNGICIDES:

OXATHIINS: Carboxin ( DCMO or Vitavax)
Oxycarboxin (DCMOD or Plantvax)

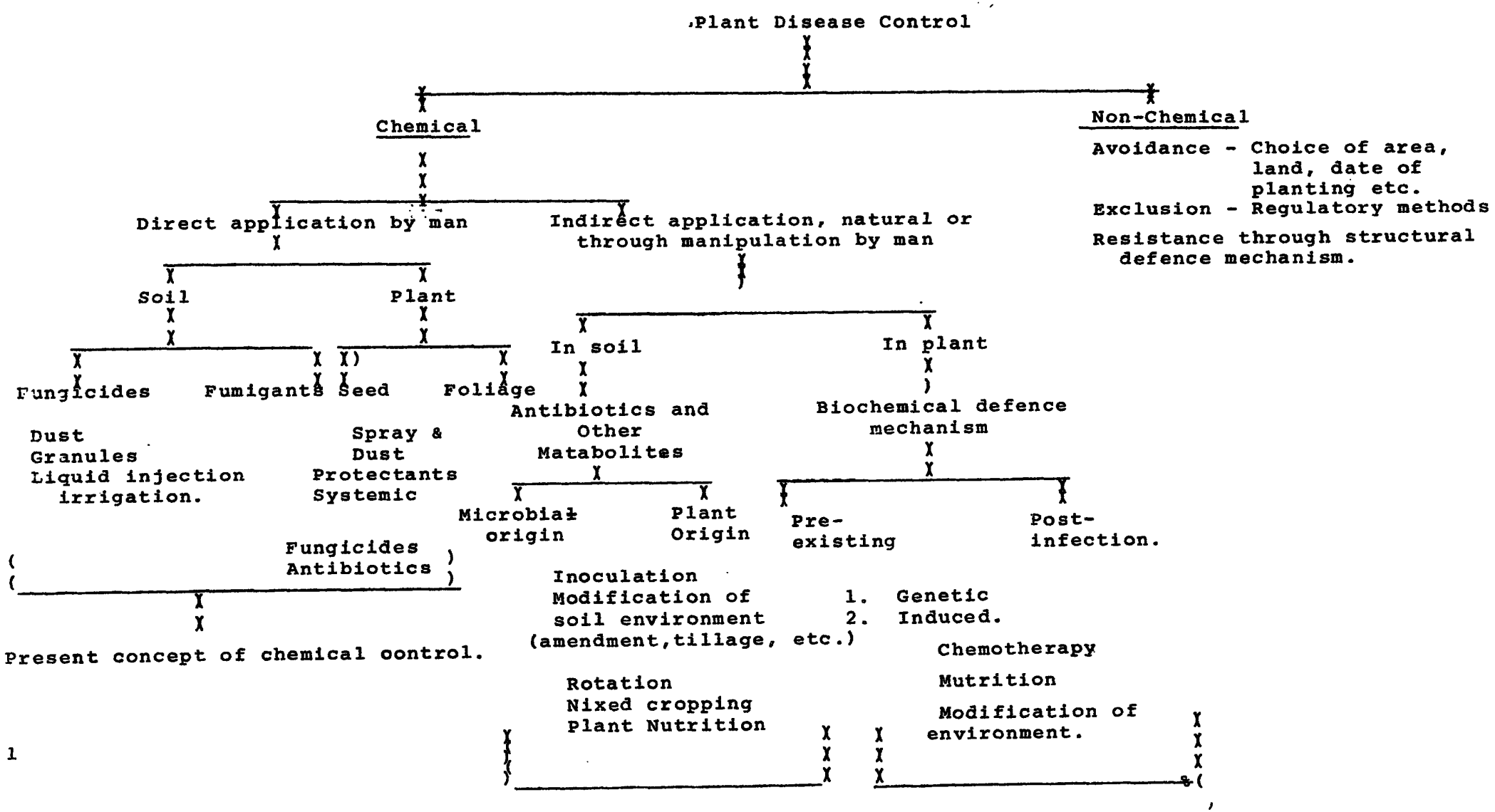
BENZIMIDAZOLES:
benomyl (Benlate)
M.B.C. (Bavistin)
Thiabendazole or TBZ, mecarbinzid, methyl thiophanate, etc.

MORPHOLINE DERIVATIVE:
Calixin

METHOXY BENZENE:
Chloroneb (Demosan)

ANTIBIOTICS: Streptomycin: Agrimycin, Phytomycin, Streptocycline

## A FLOW CHART SHOWING HOW MANAGEMENT MEASURES GENERALLY WORK




1

ACUTE ORAL AND DERMAL MAMMALIAN TOXICITY VALUES FOR SOME FUNGICIDES

| Fungicide | Oral LD (mg/kg.) | Dermal LD (mg/kg) | Toxicity class based on oral toxicity |
|---|---|---|---|
| Aspirin | 750 | 9850 | 3.Moderately Toxic(MT) |
| Acti-dione (Cycloheximide) | 113 | 8 | 2.Highly Toxic(HT) |
| Benlate (Benomyl) | 10000 | Lowdermal Toxicity | 5.Non toxic(NT) |
| Bordeaux mixture | NT | 1000 | 3.MT |
| Daconil | 10000 | 10000 | 5.NT |
| Lime Sulphur | Low | Moderate | 6.Harmless(H) |
| Morocide | 421 | Not known | 4.Slightly Toxic(ST) |
| Difolatan | 6.2 | Severe dermal toxicity | 5.NT |
| Captan | 10000 | Low dermal Toxicity | 5.NT |
| Carboxin (Vitavax) | 3820 | 8000 | 4.Slightly toxic |
| Chloroneb( (Demosan) | 11000 | 3000 | 5.NT |
| Chloropicrin | 250 | Severe | I.Extremely Toxic |
| Dichlone(Phygon) | 1300 | Severe | 4.ST |
| Telone (Dichloropcopene) | 250 | Severe | 3.MT |
| Karathane (Dinocap) | 980 | 9400 | 4.ST |
| Dithane d-14 (naham) | 395 | 1000 | 3.MT |
| Dithane M-22 (Manch) | 6750 | 100 | 5.NT |
| Dithane M-45 (nancozeh) | 8000 | 8000 | 5.NT |
| Dithane Z-78 (Zineb) | 5200 | 1000 | 5.NT |
| DNOC(Elgesol) | 30 | Moderate | 2.HT |
| Du-Ter (fen-tin hydroxide) | 108 | 50000 | 3.MT |
| Dexon(fenaminosulf) | 64 | 100 | 3.MT |
| Ferbam(Fermate) (Karban) | 17000 | 1000 | 6.Relatively harmless |
| Morestan | 2500 | 20000 | 4.ST |
| Oxycarboxin (Plantvax) | 2000 | 16000 | 4.ST |

| | | | |
|---|---|---|---|
| PCNB Brassicol | I700 | Moderate dermal toxicity | 5.NT |
| Streptomycin | 9000 | 6000 | 5.NT |
| Table salt | 3320 | - | 4.ST |
| Thiram Thylate | 780 | 8 | 4.ST |
| Vapam | 820 | 4640 | 4.ST |

Source: Sharvelle. I979

The above list is based on approval under the Federal Insecticide. Fungicide and Rodenticide Act of U.S.A. where facilities exist for making determinations of residual toxicity on edible crops. In absence of local studies it is safe to use this list.

Before usage of any fungicide be sure to read the label on the package or container, follow all directions carefully. It is against law not to follow these directions. In any case, do not spray any vegetable or fruit just before picking.

APPROVED FUNGICIDES FOR USE ON VEGETABLES AND FRUIT CROPS

Beets: Bordeanx mixture,Captan,Fixed Coppers,Thiram,Zineb, Vorlex,Vapam,D-D mixture,Telone

Cabbage: Bravo(Daconil),Captan,Fixed Coppers,Manob, Terraclor(PCNB or Brassicol), Thiram,Zineb,Vorlex, Vapam,D-D, DCP(Telone).

Canteloupe: Benlate,Daconil,Captan,Difolatan,Fixed Coppers, Karathane,Maneli,Thiram,DD,DCP,Vorlex,Vapam.

Carrots: Botran,Daconil,Captan,Fixed Coppers,Maneb,Mancozeb, Zineb, Thiram,DD,DCP,Vorlex,Vapam.

Cucumbers: Benlate,Botran,Daconil,Captan,Difolatan,Fixed Coppers,Karathane,Maneb,Mancozeb, Zineb,Thiram, Vorlex,Vapam, D-D,DCP.

Eggplant(Brinjal): Captan,Fixed Coppers,Maneb,Thiram,Zineb,D-D,DCP, Vorlex,Vapam.

Mustard: Captan,Maneb,Thiram,Zineb, DD,DCP,Vapam,Vorlex.

Onions: Botran,Daconil,(Bravo), Captan,Difolatan,Fixed Coppers,Maneb,Mancozei,Zineb,Thiram,D-D,DCP,Vapam, Vorlex.

Peppers: Chillies: Captan,Fixed Coppers,Maneb,Terraclor(PENB),Zineb, DD,DCP,Vapam,Vorlex.

Potatoes: Botran,Bravo(Daconil),Captan,Difolatan,Du-Ter, Fixed Coppers,Zineb, Maneb,Mancozeb,PCNB,DD,DCP, Vapam.

Pumpkin: Daconil,Captan,Fixed Coppers,Karathane,Maneb,Thiram, Zineb,DD,DCP,Vapam,Vorlex.

Radishes: Thiram,Zineb,DD,DCP,Vapam,Vorlex.

Rhubarh: Botra,Captan,Maneb,DD,DCP,Vapam.

Snap beans: Benlate,Botran,Captan,Daconil,fixed Coppers,Maneb, Terraclor (PCNB),Thiram,Zineb,DD,DCP,Vapam.

Spinach: Captan,Fixed Coppers,Maneb,Thiram,Zineb,DD,DCP, Vapam,Vorlex.

Sweet Corn: Daconil,Captan,Maneb,Manoozeb,Zineb,Thiram,DD,DCP, Vapam.

Tomatoes: Benlate,Botran,Daconil,Captan,Difolatan,Dyrene,Fixed Coppers,Maneb,PCNB,Thiram,Mancozeb, Zineb,DD,DCP, Vapam.

Turnips: Captan,Maneb,Thiram,Zineb,DD,DCP,Vapam,Vorlex.

Grapes: Benlate,Bordeanx Mixture,Botran,Captan,Karathane, Terbam,Phaltan,Lime Sulphur,Mancozeb,Sulphur,Zineb.

Pears: Benlate,Bordeaus Mixture,Captan,Karathane,Cyprex, Ferbam,Glyodin,Lime-Sulphur, Sulphur,Mancozeb, Streptomycin.

Strawberries: Benlate,Bordeaux mixture,Captan,Ferbam, Phaltan, Phygon,Sulphur,Thiram, Zineb.

## THE TWELVE GOLDEN RULES OF FUNGICIDE SAFETY

Although the modern fungicides currently in common use in most countries are relatively non-toxic,certain precautions should be followed when they are used.

I. Identify the plant disease problem and use the fungicide recommended and approved for its control.

2. Store fungicides in looked places, out of reach of children and domestic animals, with proper lable on the package. Do not take children to field when spraying is being done.

3. Weighing and preparation of fungicide should be done in an open area where ventilation is adequate. Never do it in an enclosed, unventilated area.

4. Never spray or dust on a windy day and stay out of the spray or dust drift.

5. Do not smoke when applying fungicides.

6. Wash bands,face,arms and other exposed body areas with soap and water after applying fungicides.

7. Change contaminated clothing after applying fungicides.

8. Clean fungicide sprayers or dusters thoroughly after use.

9. Never use spray applications that previously have been used for herbicide applications without careful decontamination. Preferably reserve these applications for fungicides only.

IO. Use fungicides in the amounts recommended by the manufacturer or the Department and weighout the recommended dose accurately, heither less nor more.

II. Dispose off empty packets or containers by burning or burying deep. Never use them for domestic purposes or throw them on roadside, in ponds or rivers.

I2. When in doubt contact a qualified Plant Pathologist, Extension Specialist or SMS.

# RICE BLAST

## (Fungus: Pyricularia oryzae)

Occurance:

The disease is found in all the rice growing areas of the world where average relative humidity of the atmosphere is above 88% and mean temperature during the season does not go very high(above 30°C). The disease appears in the crop in the seedling stage(nursery), at post transplanting tillering stage and then at the panicle emergence stage.Normally,the leaves develop resistance with age.

Symptoms:

Although the disease is a foliage disease,the symptoms are found on leaf sheath,rachis,the joints of the culm, and even the glumes. Major damage is caused by the culm or neck infection which leads to sterility of the panicle. If leaf infection is severe in early stages of the crop,there may be total destruction.

On the leaves, the symptoms appear first as small bluish flecks. In older leaves they remain circular but on young leaves they enlarge upto several centimeters long and about one cm broad. The centre of these spots becomes pale green and water soaked in appearance, finally grey or almost straw coloured. The outer rim of the spots is dark brown. Similar spots appear on leaf sheath and sometimes on glumes or seed coat.

Panicle infection does not produce well defined spots. The neck becomes shrivelled and covered with a grey fluffy myceium,appearing like a bluish patch. If this infection occurs before grain formation the ears are not filled and the panicle gives an erect white appeatance which may be confused with borer injury but the latter can be differentiated by easy separation of the panicle by pulling. If attack takes place after some grains have formed, the panicle hangs down, can break and fall off.

Environmental Relations and Pre-disposition

The plant is suceptible at seedling,tillering and panicle emergence stage. The disease is common where rice is grown from 9-45° N.L. It is not serious where average humidity is below 88% RH (areas of low reinfall and humidity). Abundant production of spores,which spread the disease,takes place when the relative humidity of the atmosphere is 90% or above. The conidia are therefore produced mostly in early morning hours. Dispersal of conidia also occurs during these hours when RH is 86-98%,temperature 25-27°C and wind is calm.

Resistance to blast is dependent on temperature. Even a susceptible variety will show less damage if grown in areas where night temperatures are in the vicinity of 30°C. At 20°C susceptible varieties show 3 fold increase in number and size of spots and few, small spots may develop in resistant varieties. At 15°C both susceptible as well as resistant varieties show high infection and large lesions. The increased susceptibility at low night temperatures is attributed to accumulation of absorbed nitrogen in leaves.

Based on above responses of disease to meteorological factors, the incidence of disease can be forecast. When the inoculum is present in the area (on weed or early sown host) the disease will apread fast if there is minimum night temperature range of 20-26°C in association with a hight relative humidity range of 90% or above, lasting for a week or more during any one of the susceptible stages of the crop. Timings for spraying can be fixed accordingly.

Host nutrition also affects rice blast severity, especially in susceptible varieties. Heavy nitrogenous manuring augments the disease proneness, especially in susceptible varieties and under conditions of low temperature. Greater the accumulation of N in leaf more susceptible the variety is. Under conditions of high N, there is less absorption of silicon which gives resistance. There is no direct effect of P and K. However, there should not be any P deficiency.

Survival and Host Range

The rice blast fungus can survive through seed as well as stubbles of diseased plants in the field. But if temperatures at the time of sowing or transplanting are above 30°C, these sources have no significance. The fungus, as such, has low survival ability in soil. The main sources of survival and then disperal by wind/rain are:

(a) The early sown rice crop in areas where there is not enough gap between planting of two crops, and

(b) Colateral hosts such as sugarcane, grasses like Setaria intermedia, Digitaria marginata, Panicum repens, Panicum proliferum, Brachiaria mutica, Leersia hexandra, Echonochloa crusgalli, Digitaria sanguinalis, Eremochloa ophiuroides etc.

Disease Management

Management strategies against rice blast will vary according to local conditions, especially the number of cropping seasons in a year. However, the use of resistant varieties should be a common approach for every region.

In areas where disease exists and rice seasons are almost telescoped, only keeping the inoculum level to the minimum should be the aim. In addition to seed treatment, field sanitation (removal of stubbles in a badly affected field) and destruction of weed hosts, it will be highly economical if a close watch on weather and development of the disease is kept. Trap nurseries planted with a highly susceptible variety can be maintained in strategic areas and as soon as blast is seen in these nurseries and if the weather is favourable (as explained earlier), farmers may be advised to spray the crop with a suitable fungicide (say Hinosan I.5 ml/litre of water). If correctly implemented not more than 3 sprayings (one each at the 3 susceptible stages) will be required. In absence of such nurseries close watch on the farmers fields may indicate the first appearance of the disease.

Disease Measurement and Loss Appraisal:

Since the loss to rice crop due to blast is governed by the stage of the crop when infection has occoured and whether leaf blast alone or node and neck blast also have appeared, the disease measurement and its use for loss appraisal requires quantitative measurement of both leaf blast as well as node and neck blast.

The growth stages and their numerical equivalents for the rice crop are: seedling/nursery (I), tillering (2), stem elongation (3), booting (4), heading (5), flowering (6), filling or milk stage (7), dough stage (8) and ripe/mature grains (9). Indicate the stage of growth through its numerical equivalent in the appropriate colum while preparing the disease measurement report.

Sampling will be crucial for recording of data. Any bias or inaccurate, round about judgement will not only vitiate the value of data for present use but will create inaccuracies and unreliability in future, also. Hence, the method of sampling should be standardized for comparisons in present and future observations.

The sample sites can be selected on hill basis, tiller basis or on area basis, For small plots of upto 0.01 acre size 10 tillers selected at random on a diagonal line can be used. Measure disease on all leaves. Record the method of sampling. For larger plots 50 or more tillers or hills or I sq.m. sections on one or two diagonals can be selected for measuring disease (both leaf blast as well as node or neck blast).

MEASUREMENTS:

(A) Leaf blast: With the help of area diagrams or keys record the leaf damage under following disease grades on a 10-point scale:

| Grades | Description |
|---|---|
| 0 | No infection on the leaf. |
| I | Small brown specks of pinhead size |
| 2 | Large brown specks |
| 3 | Small roundish or slightly elongated necrotic grey spots, about I-2 mm in dia with brown margin |
| 4. | Typical blast lesion; elliptical, I-2 cm long, usually confined to the area of the two main veins, less than 2% of leaf area occupied by these lesions. |
| 5 | 2% to less than 10% leaf area occupied by lesions |
| 6 | 10% to about 25% leaf area occupied by lesions |
| 7 | About 50% leaf area infected with typical lesions. |
| 8 | About 70% leaf area infected with typical lesions. |
| 9 | Almost 100% (whole) leaf area damaged |

The above grading will be done for all active leaves in the sample, indicating the 0-to 9 grade on the I0 - point seale. Now, tabulate the data as given below, for example:

| Disease Grade | Number of ratings (leaves) | Disease Rating |
|---|---|---|
| 0 | 60 | 60 X 0 = 0 |
| I | 30 | 30 X I = 30 |
| 2 | 20 | 20 X 2 = 40 |
| 3 | II | II X 3 = 33 |
| 4 | I2 | I2 X 4 = 48 |
| 5 | I0 | I0 X 5 = 50 |
| 6 | 6 | 6 X 6 = 36 |
| 7 | 6 | 6 X 7 = 42 |
| 8 | 2 | 2 X 8 = I6 |
| 9 | 0 | 0 X 9 = 0 |
| Total | I57 | 289 |

Disease Index = $\frac{\text{Sum of disease Ratings}}{\text{sum of ratings}}$

$= \frac{289}{I57}$

$= \frac{I.8}{\equiv\equiv}$

Disease Index % or Infection Index%

$= \frac{\text{Sum of disease ratings}}{\text{Total ratings X max.disease}} \times I00$

$= \frac{289 \times I00}{I57 \times 9}$

$= 20.2\%$

The above derived figures express that on a I0 - point scale covering the I00% leaf area the average severity of spots is I.8 or, in terms of percentage 20.2%.

The mathematical unit derived above can be for a field or for several fields in an area. If correctly followed, there will be comparability in figures thus arrived by different workers. Now, if one wants to prepare a map of disease incidence and severity for the whole district he has simply to plot average figures for different areas in the district. The overall disease index for the district or for a particular region can be prepared by using the formula:

$$\frac{\text{Field rating Class} \times \text{number of acres in class}}{\text{Total number of acres}}$$

(B) Node or Neck Blast: The following scale is used.

| Grade | Description |
|---|---|
| I | less than I% panicless infected |
| 3 | I-5 % panicles infected |
| 5 | 5-25% panicles infected |
| 7 | 25-50% panicles infected |
| 9 | 50-I00% panicles infected |

A panicle will be considered infected if more than I/3 of the spikelets are damaged.

## MAMMALIAN TOXICITY CLASSES FOR FUNGICIDES

| Toxicity Rating. | Commonly used term. | ROUTES OF ABSORPTION | | Probable Lethal oral dose for a 150 lb man. |
|---|---|---|---|---|
| | | $LD_{50}$ Single oral dose Rats mg/kg. | $LD_{50}$ Single dermal dose Rabbits mg/kg. | |
| 1. | Extremely Toxic | 1 mg or less | 20 mg or less | Simply taste or take a grain |
| 2. | Highly Toxic | 1-50 mg | 20 - 200 mg | A pinch. One teaspoon full. |
| 3. | Moderately Toxic | 50 - 500 mg | 200 - 1000 mg | 1 teaspoon - 2 tea spn. |
| 4. | Slightly Toxic | 500---5000 mg | 1000 - 2000 mg | 102 - 1 pt. |
| 5. | Practically Non-Toxic | 5000 - 15000 mg | 2000 - 20000 mg | 1 pt - 1 qt. |
| 6. | Relatively Harmless | Greater than 15000 mg | Greater than 20,000 mg. | Greater than 1 qt. |

Source: Sharvelle. 1979.

## POTATO LATE BLIGHT

### (Phytophthora infestana)

It is most serious of all potato diseases. It kills the tops and invades tubers causing dry or wet rot.

**Symptoms:**

The blight appears on the foliage as faded green patches which soon turn brownish black, enlarging rapidly in favourable weather until the whole leaf is killed. In wet weather entire leaf is killed in 1 - 4 days while in dry weather the spots do not enlarge and dry up. The killing of foliage involves petiole and stem also . . . On young growing spots, in wet weather or during morning hours a white haze (fungal growth consisting of spores) can be seen on the underside. Tubers get infected by these spores falling down and moving with soil water to young tubers. Affected tubers show brown to purple discolouration of the skin followed by a brownish dry rot.

**Survival and Host Range:**

In areas where post-harvest temperatures of the field soil do not go very high (30°C and above) the fungus can survive as dormant mycelium in the tubers left in the soil. In other areas the persisting mycelium in seed tubers stored at low temperatures is the chief source of primary inoculum. The fungus attacks tomato also and in many places the potato crop gets infection from earlier infected tomato. The spread of the disease is through sporangia or zoospores produced from sporangia and dispersed by rain or irrigation water, contact among leaves, mist, leaf eating insects and under drier conditions by wind (sporangia alone).

**Environmental relations and Pre-disposition:**

Build up of epidemics depends mainly on relative humidity within the crop canopy and outside and on fluctuations in temperatures. The sporangia germinate by a single germ tube, when humidity is low and temperature high while in high humidity (90%pH and at low temperatures (12 - 13°C) each sporangium produces a number of zoospores, each one of which can cause a fresh infection. Thus epidemics develop only when the latter conditions are present.

The disease normally does not occur in areas where the mean atmospheric temperature is above 25°C (77°F). The optimum temperature for germination of sporangia by zoospores is 12-13°C at 90% of higher RH. Optimum temperature for growth of the germ tube lies between 21-24°C. The living mycelium in the tiller is destroyed by exposure to 40°C for 4 hours or to 30°C for 65 hours. Optimum soil moisture for activity of spores is 15-20% saturation with low temperature.

On the basis of the above environmental relations forecasting of the disease has been possible if following weather exists (1) night temperature below dew point for at least 4 hours, (2) minimum temperature of 10°C, (3) clouds on the next day, and (4) rainfall during the next 24 hours of at least 0.1 mm.

Disease Management:

1. Field sanitation: debris of diseased crop should be destroyed.
2. Tubers for seed from a disease free crop.
3. Where necessary crop rotation.
4. Keep potato field away from tomato fields.
5. Use resistant varieties where available.
6. Give one prophylactic spray before expected time of disease appearance in endemic areas. If disease appears in the locality repeat spraying at 7 - 14 day intervals depending on weather and disease intensity. While spraying ensure that the liquid is applied to under surface of leaves, stems and also falls on the ground. The following fungicides are in common use: Mancozeb, Zineb, Daconil, Difolatan, Antracol etc. About 1.5 to 2.5 lbs/Acre chemical is needed according to the amount of foliage (stage of crop growth).
7. In order to reduce humidity, spacing and reduced nitrogen may be adopted in endemic areas.
8. Before harvesting, cut and remove the tops first and then dig out the tubers after 7-10 days in a clear sunny weather.
9. Keep only clean tubers for seed.

Disease Measurement:

In view of the fact that late blight may develop very fast under favourable weather conditions several methods are used for measuring incidence at different stages of plant growth. The following procedures have been recommended.

(A) Early stages of disease development:

Survey the crop for foci of infection (spots from where disease makes the start). Record the date of initial appearance. Estimate the average number of foci per acre and average area of the foci. Calculate percentage acreage affected (see example below) and also with a diagram key assess the percentage leaf area affected within the foci.

Example:

Average number of foci per acre = 5

Average area of foci = 1 yard$^2$

Average percentage leaf area infected within the foci = 1%

Percentage acreage affected (1 acre = 4840 Sq.yd). = $\frac{5}{4840}$ = 0.1%

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