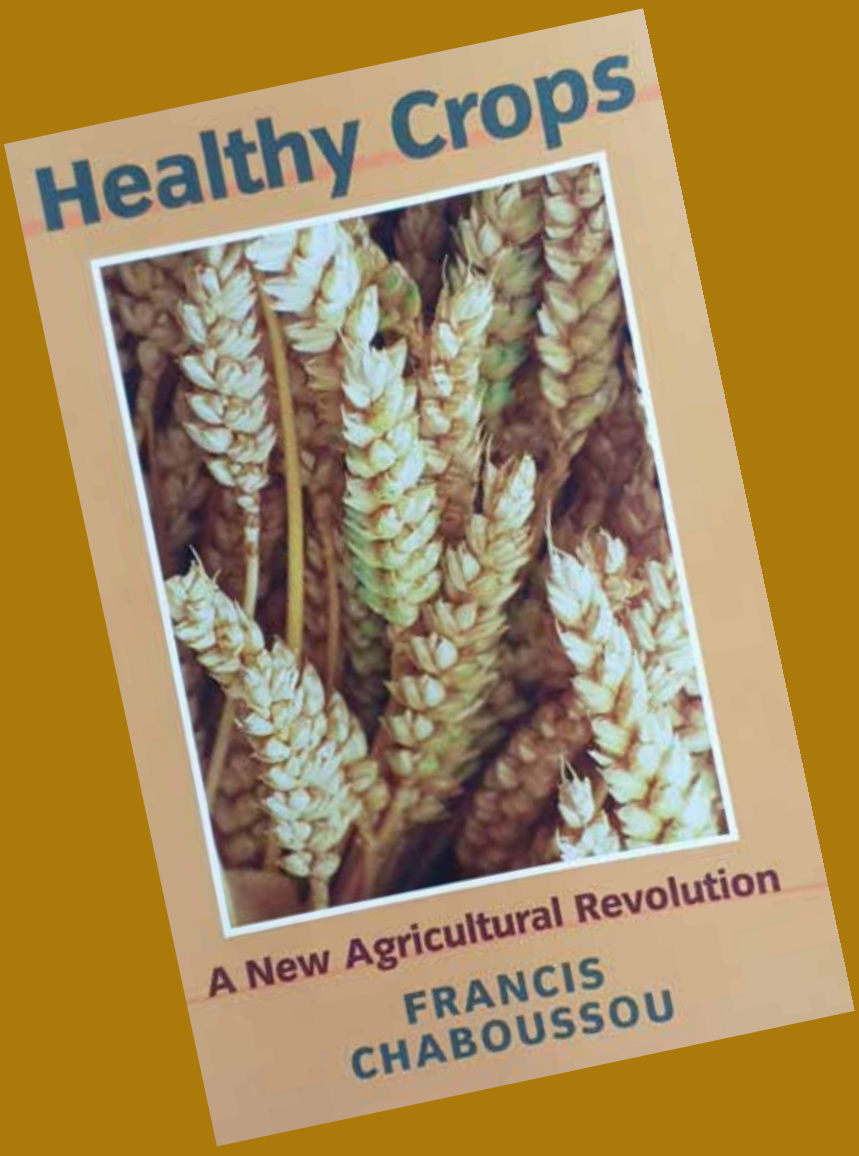
Healthy Crops

A New Agricultural Revolution



FRANCIS
CHABOUSSOU

Online version of the printed 2004 edition,
made available by The Gaia Foundation

This English translation is dedicated to the memory of
José Lutzenberger (1926 - 2002),
father of the environmental movement of Brazil.

Healthy Crops

A New Agricultural Revolution

FRANCIS CHABOUSSOU

Originally translated by Mark Sydenham, Grover Foley and Helena Paul

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Contents

PART TWO

Acknowledgements

JOSÉ LUTZENBERGER WAS PASSIONATE about this book. He believed that it was vital that it should appear in English, in order to counter the powerful pesticides lobby throughout the English-speaking world and provide a clear case for poison-free agriculture.

José Lutzenberger began as an agronomist working for the chemical giant BASF in Germany. In 1972 he decided to leave his job and return to Brazil after observing the terrible environmental impact of pesticides used in growing rice in his home state of Rio Grande do Sul. He became a tireless opponent of pesticide use and advocate of what he called 'regenerative agriculture', as an alternative to the relentless industrialisation of farming and the environmental degradation it causes. He based his methods on his careful observation of ecological processes, and believed that an understanding of ecological principles frees people to act because they learn to work 'in dialogue with nature'. He himself had little time for writing amidst the constant requests for his assistance and inspiration from all over Brazil and beyond, but much of the work that he was carrying out and advocating throughout the world was a practical application of the argument of this book. It is a great pity that he did not live to see the book published in English, but without his persistence it would not have happened.

Lutzenberger's daughters Lilly and Lara continue his work through the environmental organisation Fundacao Gaia and the ocmpany Vida Desenvolvimiento Ecológico, both located in Porto Alegre-RS, Brazil. The company focuses on industrial waste management and recycling and Fundacao Gaia receives people from all over the world to experience the application of regenerative agriculture and environmental restoration in transforming an abandoned quarry into a beautiful and diverse place.

The Gaia Foundation would like to thank Edward Goldsmith and Edward Posey for their personal commitment to the project, which has taken a number of years to complete. We are deeply indebted to the

Foundation Sauve for providing the funds required to publish the book. We would like to thank Ulrich Loening for his advice and his commentary for the book and Helena Paul who dedicated herself to ensuring that Chaboussou's message is at last accessible in English.

Commentary

by Dr Ulrich E Loening, biochemist and Emeritus Director,
Centre for Human Ecology, University of Edinburgh

CHABBOUSSOU HAS DISCOVERED a major new generalisation: by reviewing innumerable studies stretching over nearly 50 years, he demonstrated that the nutritional state of crop plants affect their susceptibility to pests and diseases. Like so many innovative ideas in biology, this work opens many new avenues and expands the horizon with new unknowns.

The theory that he developed from these observations, which he called Trophobiosis is a commonsense and essentially simple biochemical argument: that most pest and disease organisms depend for their growth on free amino acids and reducing sugars in solution in the plant's cell sap. Every farmer has experienced the increase in diseases after heavy fertilisation with nitrogen; the Green Revolution varieties are good examples in which rich fertilisation creates susceptibility to pests, requiring more pesticides to control. Chaboussou explains why.

Almost all conventional chemical agricultural technologies create favourable conditions for the growth of pest and disease organisms: this book shows how much of the problem can be explained through increases in soluble nitrogen, amino-acid and sugar concentrations in the plant cells. For example, heavy applications of soluble nitrogen fertilisers increase the cellular amounts of nitrate, ammonia and amino acids faster than can be used for the synthesis of protein. Similarly herbicides, even in recommended concentrations, inevitably affect the crop plant somewhat (perhaps not visibly), the main effect being a temporary reduction in protein synthesis - the most sensitive of metabolic processes. Spraying with almost any pesticide or fungicide has the same effect. These reductions in the rate of protein synthesis result in temporary accumulation of amino acids. Therefore, while the immediate attack by a pest may be reduced by a pesticide, the susceptibility of the crop is increased: when offered soluble free nutrients, pests grow better and multiply faster. In this sense therefore, agro-chemicals and poisons cause pests and diseases.

This conclusion suggests that pest and disease organisms cannot use protein and polysaccharides directly and require soluble amino acids an sugars for growth. Lutzenberger often argued that the various insects and even fungal pests lack proteolytic enzymes, (as implied in his introduction about leaf-cutter ants) but many insects and fungi and other organisms do in fact have them. Chaboussou's empirical evidence clearly demonstrates that pests and disease organisms grow and multiply faster when the plant contains more soluble free nutrients. In other words, the biochemical and physiological state of the plant is strongly affected by the methods of cultivation, and this controls whether pests can invade: one can avoid pests and diseases therefore by methods of cultivation that limit the amounts of soluble nutrients in the plant.

However, this important finding has been neglected. For example, a Royal Society Symposium on the Biological Control of Pests and Diseases (1987) held soon after Chaboussou's book was published in French, made no mention of the physiological or biochemical state of the plant as a factor in susceptibility to pests and disease. In other words, the idea that plants practice their own "Integrated Pest Management" is not part of modern thinking; yet this provides a way forward which is nothing less than "an agronomic revolution."

Of course there are other conditions, such as stress, drought, ageing, which similarly increase susceptibility. Good husbandry has always tried to minimise these. The balance of micronutrients is also important. Chaboussou brings these into his studies, including also much circumstantial evidence from trials designed for other purposes, as well as direct data that links onset of disease with reduction in protein synthesis.

It is clear that much needs to be done to establish the validity of Chaboussou's interpretations, by direct measurement of amino acid and reducing sugar pools under different cultivation regimes, correlated with infection or susceptibility. Few such direct trials have been done; it is a great pity that the extensive trials of the Boxworth Project near Cambridge, England, funded by the Ministry of Agriculture, Fisheries and Food from 1982 to 1988, did not address these questions.

The purposes of the project were to determine the ecological effects of different pesticide treatment regimes, the effects on crop yield and the identification of practical management problems that arise, such as from minimal pesticide treatments. Very small changes in the design of these trials would have allowed correlations to be made, which might support or deny Chaboussou's interpretations. Unfortunately it is not possible to extract the relevant data, either from the reports or from the original material.

This book therefore opens a fresh field for study and development of agricultural practices. Examples could include: a) other than the reducing sugars and amino acids, there must be many related factors that change according to treatment of the crop; a few are mentioned, but the scope for discovery of others and a deeper understanding is wide open; b) only a start has been made in the complex interplay of the micronutrients (or trace elements); c) in particular Chaboussou shows how the symptoms of viral diseases relate to those of micronutrient imbalances, and correction of the latter minimises the effects of the viruses; d) which of the many pests, fungi and bacteria, depend so strongly on soluble nutrients? And are there other factors of plant nutrient imbalances that can increase susceptibility? e) one might nowadays ask whether the new herbicide-resistant genetically engineered crops are similarly affected by herbicide: there are already reports of increased fungal infections in these; f) Lutzenberger mentioned in his preface, a number of substances that seem to prevent pests and diseases; do these work by helping the plant reduce its internal concentrations of soluble nutrients? g) and then one has the big challenge: could one grow the Green Revolution varieties under conditions that in place of increased susceptibility, promote resistance to pests and diseases? These are some of the challenges that should receive high priority for research.

Whatever the technical details, it is now obvious that close attention to the physiological and biochemical states of crop plants must become a vital part of agricultural practice. Such an approach constitutes a new biotechnology in a true meaning of this term. There is much to be done; Chaboussou has provided a powerful basis.

Preface

by J.A.Lutzenberger

BRAZILIAN FARMERS AND GARDENERS have a saying: 'Either Brazil eliminates the Saúva (the leaf-cutting ant, Ata) or the ant finishes Brazil'. This proverb cannot possibly be true, or Brazil and most of South America would have become a barren desert long before we humans even emerged. The many species of Ata came upon an efficient form of gardening millions of years before there were humans to even think of agriculture. Inside their caves in the ground, they make a kind of compost from pieces of leaves cut from trees, shrubs, herbs or grasses and the ants themselves feed on the fruit bodies of a mould they grow on this compost. They are incredibly efficient in collecting the leaf material. A big tree can be left bare overnight or a whole plantation destroyed.

The leaf-cutting ant is not only very efficient in its work, it is hardly selective in the plants it attacks. The species that it shuns can be counted on the fingers of a hand. I've seen it cut floating water-plants, where accessible. It also has a tremendous potential for reproduction. When the winged males and females fly out to mate, usually before a storm, the future queens carry with them the spores of their species-specific mould. So each one is equipped to start a new colony. There are many thousands of them in each nuptial flight. Ant colonies, like a people, are immortal, unless there is genocide. In my garden there is a colony that I have known and observed since my childhood. Sometimes it is big and causes great damage, then it may shrink again; it almost vanishes, but never disappears. So how come the leaf-cutting ants have not stripped Brazil of everything green? And why, in a preface for Chaboussou, do I mention a creature that he knew only from books?

I had the privilege to know him personally. He died, in l985, unexpectedly, of a heart ailment. He had been very pessimistic and depressed. As an important researcher in INRA, the French official agricultural research institution, he was under great pressure from the interests of the agrochemical industry. They did not like what he had discovered.

When I last visited him, on his personal farm in the region of Bordeaux, we did not discuss leaf-cutting ants. We walked mostly in vineyards and talked about the problems of European crops. But, even while talking about grapes, he gave me the answer to the question of why the leaf-cutting ant has not razed all that is green in South America.

To my knowledge Chaboussou was the first researcher to put order in the observations of dozens of other workers in agricultural plant pathology, including his own. He was the first to show that pests, such as insects, mites, nematodes and pathogenic agents, such as bacteria, fungi and even virus, only cause damage on plants that are somehow unbalanced in their metabolism and he gave us a working explanation of the biochemical mechanism. His theory of Trophobiosis is a revolution in plant pathology and is a mortal blow to agro-chemistry as commonly practiced in modern agriculture. But the chemical industry and the schools, research and agricultural extension that it has been able to coopt, do not even try to refute him. I've heard a guy on their side say: 'We will not even ignore him'. Unfortunately, with very few exceptions, Chaboussou is still unknown to most workers in agriculture, even in organic agriculture.

The theory of Trophobiosis has to do with how plant nutrition affects plant health, with what makes a plant susceptible or resistant to disease and to pest attack. Prevailing plant protection practices rest on the postulate, mostly unspoken but almost universally accepted, even among many organic farmers, that pests, such as aphids, thrips, caterpillars, scales, mites, nematodes and others; pathogenic agents such as fungi, bacteria and even virus, are arbitrary enemies that, when present, will furiously attack our crops and will only stop when there is nothing left. That is the philosophy behind the saying with which I began. So we have to fight them, eradicate them if possible, or keep them in check with our most efficient and potent weapons. The most aggressive weapons at our disposal are the synthetic chemical poisons, or pesticides, as the chemical industry likes to call them. But every farmer who observes his crops and environment carefully, knows that these creatures are not arbitrary in the way they harm our plantations and gardens and that, the more poisons we apply, the more diseases and pests we get.

For people not familiar with modern plant protection methods in agriculture, Chaboussou's book – with all the technical and brand names of pesticides, as well as Latin names of insects, fungi, bacteria, etc. - may be rather difficult to read. For me, as an agronomist, familiar with modern agro-chemistry, it was fascinating, I read it in two nights. He reviews and interprets dozens of other researchers' observations and experiments and is often repetitive to make matters as clear as possible. But it is a very important reference book and every agronomist should have it on his desk. That is why I insisted that we need a good English language version of it. We already have good German and Brazilian editions. Thanks to my friend, our great ecology guru Teddy Goldsmith, we can now present this edition. I hope we can now make the Theory of Trophobiosis known and applied in the English-speaking world. Too much time has already been lost.

For those who will not read the whole book but will use it as a work of reference, I will try to explain it in a very succinct way:

A healthy plant, a plant in metabolic balance, is either in repose, for example in hibernation or, in dry climates, in estivation, or it is growing as vigorously as it can, depending on the supply of nutrients, water and weather. When a plant is in repose the sap is poor in amino-acids; they were used up when preparing for the period of quiet. When it is growing vigorously the sap is also poor in amino-acids. Intensive proteo-synthesis uses up the amino-acids produced in the cell as fast as they appear. In both cases the plant is not nutritious for its parasites. They may just be able to survive, but they cannot thrive. They need a very high level of soluble food: not only amino-acids, to build their own proteins, but also sugars, the sources of energy for the synthesis of proteins, and mineral nutrients to help build their more complicated proteins, such as the enzymes. Other important ingredients are the nucleotides, the pieces of the genetic code. Plants that are not nutritious for the pests are also not palatable to them, the parasites ignore them. As to which particular pest or parasite will thrive, this depends on the proportion of the different ingredients.

We mentioned above that the leaf-cutting ant is not very selective concerning the species it attacks, but when we observe it carefully - something most modern agronomists don't do; they simply hurry for the insecticide as soon as they see the ant – we cannot help noticing that it is very selective as to the individual plants of any species that it demolishes. Sometimes the ant will only work on part of a plant, not touching the rest, sometimes it will limit its attack to a certain period only and then lose interest.

So, when is a plant nutritious for pests and disease causing bacteria, fungi and virus? When does it have an excessive level of amino-acids, sugars, nucleotides and minerals?

The way we manage our crops in modern agriculture means we frequently cause the plant to be metabolically unbalanced. One of the most fundamental changes compared to traditional peasant agriculture is the downpour of poisons. With very few exceptions, what the chemical industry calls pesticides (insecticides, fungicides, herbicides and a few others) are biocides. They are substances that harm living beings. Even those that are supposed to act only on the surface of the plant, always, to some extent, penetrate into the living cell. Harm may be slight, but it may mean inhibition of proteo-synthesis, therefore causing a build-up of the soluble nutrients the parasites need. Let's illustrate with a metaphor: a very small and momentary bottleneck on one of the lanes of a big expressway can cause enormous congestion. Beyond the congestion, traffic looks normal. So, my plant may seem to be growing quite normally, but there is a congestion of amino-acids in the sap. This can, of course, be verified by analysis, such as that carried out by Chaboussou and others.

Another cause of inhibition is the way we treat our soils. Intensive mechanical aggression, deep ploughing, disking, frequent harrowing to keep the soil as naked as possible, destroys soil micro-life. A healthy soil life is fundamental for a balanced uptake of mineral nutrients, especially of trace elements. Deficiency in trace elements also causes inhibition in protein synthesis, therefore a build-up of nutrients for the parasite.

Excess in amino-acids in the sap can also result when we force the plant to produce more than it needs, for instance, with high applications of ammonia fertilizers.

Inhibition can also be caused by adverse weather, such as drought, sudden heat or cold waves. On the property of Fundação Gaia, in a spell of unusual weather (something we are getting ever more frequently) a rather long spell of warm, damp days was followed by a sudden heat wave and drought. During this time a globular cactus, Notocactus ottonis, that is endemic to the region, therefore well adapted, was totally demolished by the ant, something we had never seen before. Only the thorns were left.

Many years before I met Chaboussou - I was still working for a multinational agro-chemical corporation, but I had intensive contact with organic farming - I knew that pests shunned healthy plants, as most observant organic farmers knew. But I didn't know how and why. Chaboussou was a revelation to me. Ever since, in my work with crops, in gardens and in intact nature, I see trophobiosis confirmed. Whatever disturbs a plant's metabolism - mechanical or chemical aggression, climate shock, destruction of soil life - may make it susceptible to attack by pests and to disease.

But why does Ata, our leaf-cutting ant, also choose unbalanced plants, when it doesn't eat them but turns them into something like a compost for its mould to grown on? We know the mould has no proteolitic enzymes. Somehow the ant also knows it and finds appropriate plants for the mould. Like all the other pests that I have been able to observe, the ant identifies the right plants from a distance.

When we start harvesting cabbages in our vegetable garden, healthy cabbages that had no pest attack or fungus disease, and we leave the outer, partially rotten leaves on the ground, it often happens that Saúva builds its road, coming from beyond the fence of the garden – they are also great engineers, and make beautiful roads, often dozens of yards long, radiating out from the nest to where they detect a harvest – and start harvesting the material on the ground without touching the remaining healthy plants. Of course, the cut leaves are beginning to rot, with proteolysis predominating over proteosynthesis.

There must be some emanation from the metabolically unbalanced plants. I wonder whether anybody has done research on this matter.

I dare say that Chaboussou's work is the most important discovery in agricultural chemistry since Liebig. It is a fundamental new instrument for a healthy, clean, regenerative and sustainable agriculture. The solution lies not in fighting arbitrary, wicked enemies of our crops, but in developing methods of keeping our plants healthy. All over Brazil we have already had great success when we substituted conventional pesticides with stimulating applications, such as whey, cow's urine, mature biogas slurry, ground rock rich in trace ele-ments and many other treatments that the farmer can prepare himself. But this is a subject for many other books.

I really hope that by making Chaboussou known in the English speaking world, especially in the so called Third World, we can make an important contribution to the fight against the uprooting of the peasant by the agricultural policies of transnational corporations. How sad Chaboussou is no longer among us.

December 2000

Introduction

WE WILL BEGIN with a brief summary of the argument.

The first of the book's three parts is entitled:

'Pesticides and Biological Imbalances'

The first chapter draws attention to the responsibility of chemical pesticides for the resurgence of diseases and pests. These are not just old or traditional afflictions but also new pathogenic agents that have attained the rank of pests. Among them are the psyllids, mites, numerous cereal diseases (including viral ones), withering diseases, and the viral diseases of fruit trees and the grapevine.

Such 'biological imbalances' do not arise merely from the alleged destruction of 'natural enemies' but, rather, from *disturbances of the nutritional environment* - that is to say, the biochemical state of the plant is altered by the intrusion of new chemical pesticides.

The basic conclusion is: *the relations between plant and parasite are above all nutritional in nature*. This is the theory of **trophobiosis**. It casts doubt on an alternative theory: that the resistance of plants is based on the possible presence of substances (phytoalexines) that are toxic or repellent to parasites. The trophobiosis theory contends, instead, that plants are made immune to the extent that they lack the nutritional factors that parasites require for their development. In short, what is involved is a deterrent effect, not a toxic action.

Moreover, our theory seems to agree with the nature of relations between plant and parasite. These relations are determined by various environmental factors such as, for example, *genetic factors* (varietal resistance), *the physiological cycle of the plant* (such as the state of flowering), *photoperiodism, climate, nature of the soil, fertilisation, nature of the stock* and, finally, *the effect of pesticides on the plant's physiology.*

Resistance and susceptibility depend on the level of soluble substances. A state where proteolysis predominates is linked to disease, while the predominance of protein synthesis correlates with resistance.

Current developments in pests and diseases stem from what are called 'intensive' agricultural practices. This term is applied to the misguided utilisation of chemical pesticides and fertilisers, particularly nitrogen fertilisers, which leads to inhibition of protein synthesis.

However, certain findings indicate that the initial mechanism of this susceptibility to pesticides may lie in the creation of various deficiencies. Therefore we entitle the second part of our work:

'Deficiencies and Parasitic Diseases'

This second part consists of four chapters, two of which are devoted to viral diseases. We have tried to analyse the effects of environmental factors on the development of these disorders and have found that they are not responsible for the different reactions of the various viral diseases we have reviewed. What is crucial is the physiological state of the plant, while the impacts of contagion and vectors seem to be only secondary. This conclusion means that we should probably change our techniques of disease control, above all if one also considers the indirect effects of pesticides on the plant being protected.

It is clearly no accident that the symptoms of viral disease are easily 'confused' with the symptoms of deficiencies. These deficiencies are the true cause of the disease. This also holds true for bacterial diseases. *The shift from strictly mineral products like copper and sulphur to chemical pesticides* could explain the recent and at first sight inexplicable outbreaks of various diseases, particularly bacterial and viral ones. Chemical pesticides have an opposite influence on the physiology of plants from the mineral ones.

We also thought it indispensable to dedicate one section to the work of Constantin Vago. Vago's research suggests there might be a general law applying to both the animal and the plant kingdoms, that would explain the phenomenon of 'disease' in the same fashion for both realms. With regard to outbreaks of viral diseases among insects, Vago has highlighted the existence of two factors that can unleash metabolic disorders, and that lie at the very root of disease: these are, just as with plants, *malnutrition and toxic effects*.

'Agricultural Techniques and the Health of Crops.'

Based on the theory of trophobiosis, the eighth and ninth chapters discuss the results obtained through the process of stimulating protein synthesis.

Here we review various examples of agricultural techniques, especially that of *balanced fertilisation* (a phrase used frequently but quite imprecisely). Such a balance implies *the prevention and correction of deficiencies*. Of course, such therapy ought to be based on still deeper knowledge of the physiology of plants, especially the role of various elements, chief among them the trace elements. Some extremely encouraging results have already been recorded. Since some of these findings stem from an empirical use of the principles of tropho-biosis, we feel justified in hoping that this therapy will prove all the more effective once it is applied with full knowledge of the factors behind it.

Balizac, July 20, 1984.

PART ONE

Pesticides and Biological Imbalances

Current concerns about saving the environment lead us to reduce pesticide applications as much as possible. It would be paradoxical if we should at the same time extend the use of these substances to areas where they have never been used before. We run the risk of paying a high price sometime in the future, for a temporary improvement of yields in a few special areas.

Émile Biliotti [1]

Chapter I

Resurgence of Disease and Pest Damage Brought about by Pesticides

I. Serious concerns about crop health

The arrival of artificial pesticides brought new problems for crop protection. Mites, for instance, only advanced to the rank of a 'major' threat to agriculture after 1945, as noted by the eminent acarologist, Mme. Athias-Henriot (1959). The same is true of the leaf suckers (Psylla)[2] nematodes, aphids, and even lepidoptera and coleoptera, and 'numerous outbreaks of disease,' that cannot be accounted for in the usual way.

One pesticide company expressed its amazement about the constant spread of diseases affecting cereals, and the appearance of new viral infections, calling them 'diseases of progress'. This same company adds, 'If certain diseases have, so rapidly, become so serious, there must be other factors involved beyond natural and climatic conditions.' (Publicity statement by Maison Procida.)

We wholeheartedly endorse this remark. Even though it disappeared from subsequent advertising, it sums up the situation perfectly. We shall come back to it later, in the chapter on cereal crops. With regard to viral diseases in vegetable crops, it is worth citing the somewhat disillusioned observations of a specialist:

> 'Since vegetable growers have become familiar with their crops' main parasites and have obtained effective fungicides, viral diseases have taken on far greater importance. These diseases are feared because their origin seems mysterious and their development insidious' (Marrou, 1969).

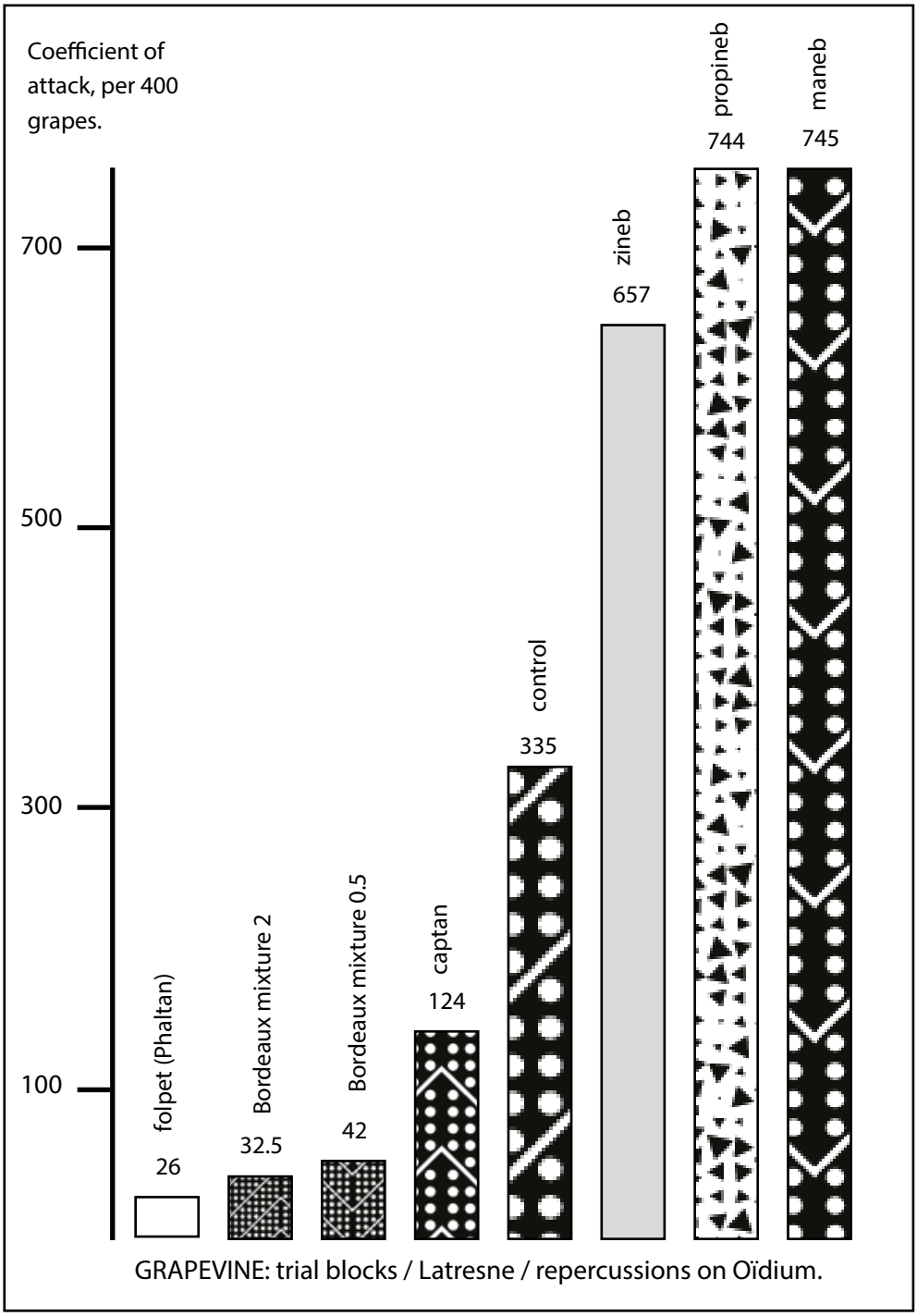

Fig. 1. Coefficients of the attack by grape *Oïdium* on grapes, according to the use of different antifungal chemicals employed against mildew. (Cabvernet-Sauvignon vine. Trials at Latresne, 1966.) Results of Chaboussou.

We would like to note at the outset that the 'effective' fungicides to which he refers are the new synthetic organic chemicals. Not only should we have reservations about their effectiveness; they also bring with them, as we shall later see, grave disadvantages.

II. Chemical pesticides are directly implicted in 'biological imbalances'

The authorities have been forced to take into account 'the side effects of anti-parasite products for agricultural use'. In France, a committee comprised of representatives of INRA (National Agricultural Research Institute) and the Crop Protection Department regularly draws up a list of a specific pesticide's 'disadvantages' in relation to one category of parasite or another. For the most part, the pesticides involved are chemical.

The vocabulary used to describe these repercussions depends on whether they are pest infestations or diseases. In the repertoire of 'side effects,' the term 'proliferation' is used when the subject is the spread of pests (mites, aphids, coccus,[3] etc.). In contrast, when dealing with the spread of disease (*Oïdium*, mildew, *Botrytis*, scab, etc.),[4] it is assumed that the pathogenic agent is showing 'resistance' to the pesticide in question.

The use of such different terms to designate these biological imbalances, whether in the animal or plant kingdoms, arises from the explanation that is offered for these phenomena.

The classical explanation for outbreaks of pests as a result of one pesticide or another is that the pesticide has destroyed the natural predators. With the suppression of the restraints exercised by phytophagous predators or parasites, proliferation seems to grow more serious.

This explanation disregards our own studies of mite outbreaks due to different pesticides (Chaboussou 1969, and other publications). It also rejects studies that show how aphid infestations may actually be caused by different pesticide products (Michel, 1966; Smirnova, 1965, etc.). We shall take up this subject again in a special chapter.

This is not to deny the noxious influence of pesticides on natural predators. But the different studies show quite clearly that these pest proliferations are chiefly the result of an increase in the biotic potential of the pests feeding on treated foliage. This includes, among mite populations and the like, an increase in fecundity, longevity, fertility, and ratio of females to males.

Many pesticides greatly increase the numbers of mites, despite being harmless to their natural predators. What is more, the chlorine-based products used to 'disinfect' soil actually increase the number of mites on potato crops grown on this soil.

The resurgence of old maladies or the development of new ones cannot be explained by the destruction of antagonists by the pesticides, since such antagonists are very rare. People speak of 'resistance' to the specific pesticide, but without genuinely demonstrating this. In reality, it is apparently more a question of a 'proliferation,' nutritional in origin, caused by the particular fungicide and its harmful effect on the plant.

Fungicides, herbicides and pesticides, as we shall see in the following chapter, have an effect on the physiology of the plant, above all when they are used repeatedly.

There may also be a selection of resistant or pre-resistant strains.

We have shown in experiments that, as compared to grapevines treated solely with pure water, treatments using various dithiocarbonates (zineb, maneb, propineb) brought about a significant increase in attacks of Oïdium. Vanev and Celebiev (1974) have also shown stimulation of the development of Oïdium (Uncinula necator)5 and grey mould (Botrytis cinerea) on grapevines by zineb-based chemicals (Fig.1).

It seems logical that these 'biological imbalances' brought about by chemical pesticides, whether in the animal or plant kingdoms, should have the same cause. Thus captan, though harmless to natural predators, produces not only a proliferation of mites, but also of Oïdium on the apple tree. In addition, it promotes development of the bacterial disease crown gall on the cherry tree (Deep and Young, 1965). These authors state that their findings do not coincide with the theory of antagonism. They suggest that other factors must be responsible for the growth of the tumour. Here again, everything points to the fact that it is the new physiological state of the plant, created by the pesticide, which has promoted the development of both crown gall and Oïdium. In a subsequent chapter on bacterial disease, we shall study the relation between plants and their bacterial parasites and show how the plants have been affected by various environmental factors, such as pesticide treatments.

III. Conclusions

The goal of this book is to explain the reasons for the failure of chemical pesticides, whether fungicides, insecticides or (above all) herbicides. The root cause is their impact on the physiology of the plant, a phenomenon that up to now has been overlooked by classical plant science. The following chapter will be specifically devoted to plant-parasite relationships, highlighting the great importance of environmental factors for the plant's susceptibility to its various parasites. We shall try to show the convergence of the effects of these factors, biochemical in nature, on the plant's susceptibility or resistance. The age of the vital parts, the developmental stage, the climate (temperature and photoperiod), the soil, and the variety all play their part. We will analyse the nature of the relations between the plant and its parasites, relations that are primarily *nutritional.*

We will look at *the nature of those nutritional elements that favour the biological potential of parasites, and thus identify the physiological state of the plant that will maximise resistance.*

Notes

1 Former chief inspector of the National Institute of Research in Agronomy (INRA), on the subject of protecting plant health in cereal crop production - Phytoma, No. 272, March 1975, pp. 21-23.]

2 E.g. Psilla mali (apple leaf sucker = psylla du pommier), Psylla pirisuga (pear sucker or pear psylla = psylla du poirier), or Trioza viridula (carrrot psyllid = psylle de la carotte).

3 Coccus: ; 'a spheroid bacterial cell' (Microbiology) [from Academic Press Dictionary of Science and Technology]

4 Tavelure = (1) scab (Fusicladium pirinum - pear scab, or Fusicladium dentriticum - apple scab, or (2) leaf spot of current: Pseudopeziza ribs).

5 Oïdium (French) = (e) Oidium = (e) powdery mildew = (dt) Faulschimmel. Powdery mildew is of three types: Sphaerotheca macular spp. Oidium spp. (Quercus) = Microsphaera spp.

Chapter 2

Plant-Parasite Relations: the Trophobiosis Theory

I. The influence of environmental factors on the resistance of plants

A plant's physiology and resistance are determined by environmental factors. We will review a certain number of these factors in order to establish the nature of the relationship between the plant and its parasites.

1. The influence of the physiological stage: the period of flowering.

Can the theory of trophobiosis explain how floral induction could change the behaviour of a plant? Floral induction can change a plant's behaviour, especially its susceptibility to disease, as we have already argued elsewhere:

> 'One fact seems clearly to confirm this view: the stage at which infections occur. According to the authors, this period of heightened susceptibility corresponds to the moment of flowering. It is in fact at the moment of flower formation that all the leaves lose their capacity for photosynthesis. They even undergo a certain amount of decomposition of their own proteins. This allows them to supply the reproductive organs with soluble substances.
>
> 'This means that in the flowering state, and still more during the development of the young fruit, the plant is affected by acute protein breakdown (proteolysis), with a very clear drop in the protein content of the mature leaves.'[1]

We also added:

'This flowering period therefore constitutes a primary period of vulnerability for perennial plants (as well as annual ones.'

We will now examine the influence of the age of the parts of the plant, as well as of the climate - each of which has an impact on the other - on the susceptibility of the plant to diseases, taking as our example the relations between the grapevine and mildew.

2. The Influence of the Age of Plant Parts on Susceptibility to Disease.

We will take several examples, highlighting at the same time several plants and various parasites.

a) The grapevine and mildew

The relation between the grapevine and mildew has been particularly well studied by Pantanelli (1921). We can sum up these studies in this way: an attack of mildew presupposes

- germination of the spore (conidium);
- attraction of the zoospores by the stomata, to allow them to penetrate.

Pantanelli observes that, while the humidity of the soil has a major influence on the opening of the stomata, condensation of water on the leaves has the opposite effect. So, ideal conditions for a mildew attack are created by a humid night following a hot dry period.

However, humidity constitutes a second important condition for the attack because it influences the nutritive exchanges in the leaf tissues. Pantanelli ascertained this by comparing the composition of leaves from vine plants grown in pots. While some were placed outdoors, others were put inside a humid and well-lit room. In the latter, the proportion of soluble carbohydrates was higher than that of starch (which had almost totally dissolved during the night). Thus, the proportion of nitrogen and phosphorous compounds was also raised, which encouraged germination.

As for temperature, it also has an important effect, not only directly on the fungus, but also indirectly on the metabolism of the leaf.

We know that spores germinate better if they have first been cooled to 9-10° C. This cool-ing also has an effect on the leaf: it produces an increase in the proportion of sugars, in comparison to the starch not yet dissolved. Moreover, there is a slight increase in the pro-portion of soluble nitrogen and phosphorous.

This whole process explains why infections are more likely to take place at dawn. This is the moment that combines the greatest breakdown of starch with maximum decomposition of proteins.

Both the influence of humidity and that of temperature combine to highlight the great importance of soluble compounds for mildew infections of grapevine leaves. The compounds do this through their nutritional effects. These effects are confirmed by analysis of the influence of the age of the leaf.

'The very young leaves,' as Pantanelli notes, 'do not become infected, because they contain a much higher quantity of albumin, together with an almost total absence of soluble compounds in the water. Older leaves become infected, but in adult leaves the development of the mycelium is very slow.'

As Pantanelli concludes, 'The most favourable conditions for a mildew infection are characterised by a high proportion of sugar compared to starch, and of soluble nitric and phosphoric compounds compared to insoluble ones (that is to say, in comparison to albumin, nuclein and proteins). The free organic acids do not seem to play a role.'

In a general way, this also explains the resistance of the mature leaves to infections: they have a low level of free nitrogen compounds, since the bulk of the nitrogen is contained in the protein (MacKee H. S., 1958).

Pantanelli (op. cit.) also studied the behaviour of the zoospores making their way towards the stomata. He was able to establish that

- neither the glucose nor the saccharose attract the zoospores, while peptone attracts them strongly.
- the free acids (tartaric, malic, citric, oxalic) repel them.
- alkaline salts (including sodium carbonate and potassium carbonate) and metallic phosphates repel zoosphores.

Finally, Pantanelli concluded that his findings: ' are sufficiently in line with what has already been established about a leaf's composition at the instant of attack: soluble nitrogen and phosphorous compounds appear to have a greater power of attraction than the sugars.'

Since no sugar or other carbohydrate is volatile, it is very likely that what attracts the zoospores are the amino and ether compounds of fatty acids, particularly amino acid compounds.

'In fact,' says Pantanelli, 'both peptone and cheese extract have a characteristic odour, mainly due to volatile amino compounds ... Sugar, which strongly attracts other fungi, has only minor importance for the zoospores of the *Peronosporae*'.[2]

Pantanelli's study of the grapevine-mildew relationship clearly shows the importance of soluble nutritional elements in contamination and infection by pathogenic fungus. This occurs in the context of a metabolism where protein breakdown predominates over protein synthesis. It implies a greater exudation of soluble compounds, namely nitrogenous substances and soluble sugars, on the surface of the leaf. Although sugars, as Pantanelli has demonstrated, appear to play only a secondary role in the case of Peronospora, this may not be the case for other fungi, which seem to require large quantities of carbohydrates.

It therefore appears that the impact of the nutritional ratio (soluble nitrogen elements to reducing sugars) on the contamination process varies according to the species. (We call this ratio, more simply, the C/N ratio.).

The C/N balance, as we shall see confirmed below, is dependent on various other environmental factors, such as climate soil, fertilisation, and – last but not least - the impact of pesticides. These factors will be discussed later on.

For the moment, we will study the relation between the age of the plant's parts and the parasite (which in this case is a viral disease).

b) Tobacco and mosaic virus

Viral diseases are no exception to the rule that the age of the leaves affects their susceptibility. Horvath (1973) showed that the susceptibility of the tobacco plant to mosaic virus depends on the position of the leaves on the stalk - in other words, on their age. There is an increase in susceptibility among the first senescent leaves at the base. This correlates with the rate of synthesis of proteins and RNA in the leaves, which is lower than the rate in other leaves.

If, therefore, a certain degree of senescence makes them hypersensitive, the phenomenon is suppressed in highly juvenile leaves. Hypersensitivity is characterised, as the term indicates, by extreme sensitivity of the host cells toward the parasite, whether this is a pathogenic fungus or a virus. At length, this hypersensitivity causes the cells to die. This halts the infection, because of the absence of nutritional elements. Even in this special case, there are still nutritive factors playing a role in the process of plant resistance to parasite intrusion.

Susceptibility of the plant's parts in relation to their age is equally important when it comes to noxious insects. Researchers have proven this, above all, in the case of sucking insects such as mites and aphids, to which we shall return later. For the moment, we take the leaf-hoppers (Cicadellidae) as an example.

c) The castor oil plant (Ricin) and the leafhopper. The mechanism of resistance of varieties

With regard to mites, we have shown (along with other authors) that their proliferation is linked to a diet rich in soluble substances such as free amino acids, along with reducing sugars (Chaboussou, 1969). The process is analogous with aphids, whose numbers at this time continue to grow as a result of the use of certain pesticides.

As with mites and aphids, the leafhoppers (Cicadellidae) and psyllids are both major threats to crops. Infestations are recorded on grape vines that have been treated with organo-pesticides (fungicides as well as insecticides) (Chaboussou, 1971).

In-depth studies have been carried out in India by Jayaraj (1966, 1967) into the nature of the relations between the castor oil plant's biochemistry and the attraction and reproduction of the leafhopper Empoasca flavescens. He touched on a factor that we have not considered up to now, that of determinism according to varietal resistance. This shows the influence of genetic factors. Jayaraj was able to establish that varietal resistance is expressed in terms of nutritional value in relation to infestations by leafhoppers. In varieties classified as susceptible or merely 'tolerant' to infestation, we find an accumulation of free amino acids and amines.

In contrast, the resistant variety contains more sugars than the susceptible or tolerant ones, while the latter contain, respectively, 113.5% and 42.3% more nitrogen than the resistant variety. The susceptible and tolerant varieties showed 12 and 7 times more free amino acids than their resistant counterparts.

Jayaraj's conclusions were very clear: 'The resistant varieties of the castor oil plant have a tendency to repulse E. flavescens due to their low nutritional level, as indicated by their low levels of total nitrogen and of amino acids and peptides'.

To summarise, a lack of nutritional elements is at the root of resistance to infestations by the leafhopper. We find similar patterns of resistance to other insects, such as aphids, as well as to pathogenic fungi (cryptogamic diseases), bacteria, and viruses. We shall look at the levels of these soluble compounds and their proportions, in terms of the C/N relationship.

The study of the relations between another leafhopper, Empoasca fabae, and the potato plant likewise shows the extreme sensitivity of leafhoppers with regard to the biochemical nature of the host plant. Miller and Hibbs (1963) were able to establish that, in the case of the potato plant, Empoasca fabae preferred unpaired terminal leaflets over subterminal-paired leaflets, the former receiving significantly more eggs. Such a selection of egg-laying location depends upon the biochemical characteristics of the leaflet.

This example shows that nutritional state determines resistance. It is the fundamental element, throughout this whole study, in the phenomenon of resistance. Resistance is a function of the variety (genetic factors) or of the age of the tissues. But, as we shall now see, resistance also depends on climate and the nature of the soil.

These results will help us to grasp better the mechanisms by which pesticides impact on the physiology of the host plant, promoting infestations of various pests and diseases.

3. The influence of climate

a) Temperature

This factor has already been touched upon in Pantanelli's studies on the grapevine and mildew. Although temperature has a direct influence on a spore's germination, it is equally dependent upon the nature of a leaf's

exudates, in other words their richness in soluble nutritional compounds, especially nitrogen. The nature and quantity of these exudates are strongly dependent on temperature.

As Dufrenoy (1936) writes: 'For each variety of wheat and each type of Puccinia glumorum, Gassner and Strait showed the existence of a critical temperature, above which the wheat is resistant. In general, lower temperature sets off an increase in quantity of soluble albuminoides in the first leaves of the wheat seedling.'

This would appear to confirm Fischer and Gausmann's results with regard to the parallels between 'the increase in soluble albuminoidal tissues and the increase in susceptibility to rust.'

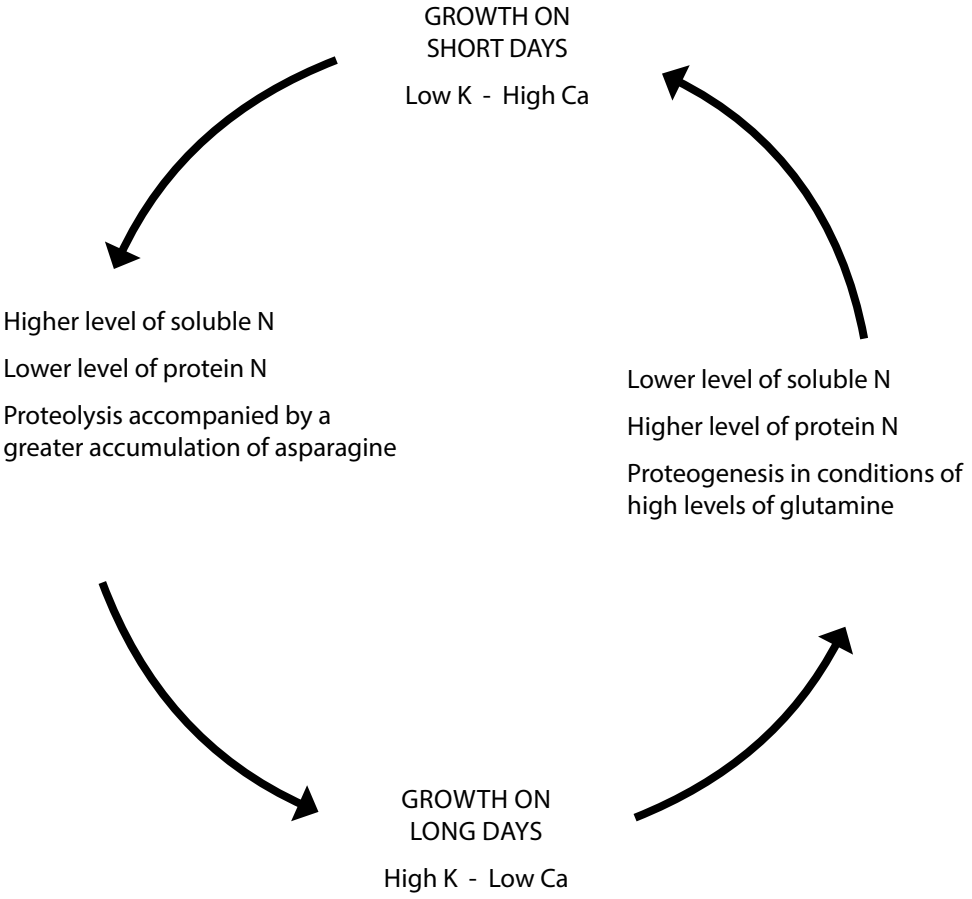

Fig. 2. Diagram showing the combined influences of the relation between the cationic ele-ments K and Ca and the phenomena of protein synthesis and proteolysis, as well as the in-fluence of the photoperiod on the level of certain amino acids in the leaves of Mentha piperita. (After Crane and Stewart, 1962.)

This sensitisation of the wheat plant to disease through a reduction in temperature, in other words by a slowing down of protein synthesis, fits perfectly with the nutritional basis of our theory of trophobiosis.

In passing we should point out that the increased susceptibility to rust brought about by a chlorine product such as DDT is the direct consequence of this pesticide's effect on the plant's physiology (Johnson, 1946). This would appear to be due to a similar nutritional process to that which lies at the root of DDT's encouragement of aphid proliferation. This involved inhibition of the process of protein synthesis and accumulation of soluble nitrogenous substances in the tissues (Smirnova, 1965). We shall return to this subject in examining intensive cereal production.

b) The influence of latitude and the photoperiod

Latitude and photoperiod have often been linked to crop resistance to disease. Young et al. (1959) noticed an increase in the maize plant's sensitivity to Diplodia zea when a hybrid of maize from a northern US state, such as Minnesota, was planted in a more southern state, such as Missouri or Oklahoma. This phenomenon arose from the fact that the lower latitude brings about a reduction in day length .

This may well be a general phenomenon. Umaerus (1959) found that the Sebago potato, fiercely resistant to mildew (Phytophthora infestans) in Maine, with its long daylight hours, was one of the varieties most susceptible in Florida, with its much shorter days.

Such facts can be explained 'biochemically.' Crane and Stewart (1962) showed that, in the case of Mentha piperita, the tissue composition of amino acids varied as a function of the photoperiod, but also according to the K/CA balance in the nutritive solution (fig. 2).

Crane and Stewart's results (op. cit.) can be summarised thus:

i) Short days accentuate the retention of soluble compounds in the tissues. Protein breakdown increases with the shortening of the photoperiod and is accompanied by an accumulation of asparagine, an amino acid that we shall often encounter as a fundamental nutritional factor for the development of pathogenic fungi.

ii) during long days the amount of soluble nitrogen is lower but quantities of protein nitrogen are higher. The plant is in a condition in which protein synthesis dominates, characterized by high levels of glutamine.

Crane and Stewart's results fully confirm the influence of the physiological state of the plant on its susceptibility to parasites, and also lead us to consider the influence of the plant's nutrition on the biochemical composition of the tissues. Here we touch on the influence of fertilisation on a crop's physiology and thus on its susceptibility to parasites.

Crane and Stewart (op. cit.), in studying the effects of the K/Ca balance on the nutritive solution in relation to tissue composition, show that these effects are additional to those attributable to the photoperiod. This particularly applies to the equilibrium between protein synthesis and protein breakdown (fig. 2).

Such effects on the resistance of plants explain those of a similar nature brought about by supplements and fertilisers, as we shall see later. However, fertilisers also have important repercussions for the nutritional value of the crop, and consequently for the health those that feed upon it: people or livestock. This aspect of the question leads us directly to investigate the influence of the soil itself on the plant's resistance.

4. The influence of the nature of the soil

a) The Colorado beetle and the potato plant

Together with the fruit fly and the silk worm, the Colorado beetle has rendered great services to science, thanks to the studies carried out on it. It has much to teach us about the influence of the nature of the soil on the potato plant's susceptibility to this celebrated Coleopter.

Chauvin (1952) was able to measure the feeding activity of the Colorado beetle on different varieties of potato, in relation to soil conditions. Two test sites were established, one an exposed site in Versailles (site A), the other on clayey soil, near Epernon (site B). (No further details of the soil type were given).

These tests showed major differences in the 'appetite' of the Colorado beetle, from which Chauvin attempted to deduce the ecology of the attracting substances. For each variety of potato, he was able to assign what he called its 'attraction units' for the beetle.

For each variety, the results showed in general that the potatoes on site B suffered fewer attacks than those cultivated at site A (Table 1).

Table 1: Units of attractiveness for the Colorado beetle, shown by several varieties of potato, in relation to the situation.

Situation	Bintje	Saucisse	Ackersefen	Parnassia
A	1.25	0.45	1.61	0.80
B	0.31	0.28	1.25	0.28

These studies on the Colorado beetle, including for instance those of Boskozska (1945), clearly demonstrate that the type of soil and thus of the fertiliser treatment, has an effect on the potato plant's physiology and thus its susceptibility to infestation by the Colorado beetle. It should therefore come as no surprise that certain traditional growing methods, such as the systematic application of manure and compost, encourage resistance to the Colorado beetle and even to disease, through the biochemical state that they create in the plant. We will return to this subject when we examine the effects of both fertilisation and the plants' needs on their resistance to parasites.

b) The disease 'resistance' of certain soils

There has been research into what has been termed 'a soil's resistance to disease,' of a similar type to that discussed in relation to the potato plant and the Colorado beetle.

A specialist on the subject, Louvet (1982), remarked that this resistance could be linked to the chemico-physical composition of the soil. Soils with a high pH, for instance, are resistant to club root,[3] a fungal disease affecting cabbages. In other cases, the basis of this resistance is fundamentally microbiological. According to Louvet, immunity is related to 'efficient microbiological barriers' which prevent the pathogenic agent from promoting the disease, even when it is present in the soil.

As Louvet states, 'This incompatibility between the pathogenic agent and the soil-plant interaction is the result of an active opposition; when these microbiological barriers are broken down, the resistance is destroyed. In the Durance valley of France, melons grown in soils fumigated or heated to eradicate corky-root end up developing stalk rot.[4]

In this example, interference has brought about a 'biological imbalance' similar to those provoked by synthetic pesticides, though in this case the processes are more complex. If, as is likely, these treatments of the soil lead to the destruction of the soil's microorganisms, then the central issue is the plant's nutritional process, and thus its resistance to disease.

This all leads to the conclusion that 'the incompatibility between the pathogenic agent and the plant soil system,' of which Louvet writes, is actually due to the physiological state of the plant. We are already aware of the importance of specific microorganisms to a plant's nutritional process, mainly through their impacts on nitration and nitrification, processes halted by fumigants which destroy the microorganisms.

Other examples help to demonstrate the complex relations that link the soil, a plant's physiology, and its resistance to various parasites. Through both field and laboratory research, Brain and Whittington (1981) demonstrated that high levels of manganese in tissues of the swede plant lowered the sensitivity of the plant's leaves to Oïdium. This confirms previous research that manganese is a vital trace element, essential for a plant's growth.

Any deficiency, especially in trace elements, leads to an inhibition of protein synthesis with a corresponding increase in free amino acids. It is known that manganese is crucial for the absorption of nitrates; manganese deficiency sensitises the oat plant to bacterial infections by inhibiting protein synthesis.

Everything would appear to confirm that 'soil resistance' is simply a special aspect of a plant's resistance. A balance of various elements in the soil contributes to a beneficial level of protein synthesis. This depends on the balance of cationic elements, most notably K and Ca, as we have seen above; it also requires the intervention of what have been called catalysing or trace elements, which we shall return to later.

However, the presence in the soil of one or another trace element is not sufficient: the plant must also be able to absorb this element. This is where pH comes in. Brain and Whittington (op. cit.) have shown that the higher the pH, the fiercer the attacks of Oïdium. Since these attacks are associated with a reduction in manganese in the plant tissues, it is the absorption of this trace element that is affected by the pH. The pH has an effect on the activity of the bacteria that assist manganese absorption: a deficiency of manganese in limestone soil results from the pH being too high to allow any activity by this category of bacteria.

As highlighted by Brain and Whittington (op. cit.), the effects of soil pH are extremely complex. Changes can occur, either by increasing the availability of certain elements, leading to toxic results, or by a reduction in uptake, leading to various deficiencies.

These indirect effects which link plant physiology with resistance, through the nutritional role of the living soil, clearly demonstrate the risks posed by the myriad herbicides, insecticides, fungicides, and nematicides - particularly artificial ones, as used on a huge scale in 'chemical' agriculture - and their effects on the soil's microorganisms. These arise above all from their impact in provoking deficiencies. We shall return to this point, which affects not just cereals but all crops.

As we shall see, the trace elements play a fundamental role in plant resistance. This is demonstrated by the repercussions of any deficiencies, and the prevention or cure of disease through the correction of these deficiencies.

The richness of certain soils in trace elements might explain why the plants that grow there are resistant to disease. This is particularly the case with 'volcanic soils'.

c) The disease resistance of rice grown on volcanic soils

Martin-Prevel (1977) noted that rice is more resistant to Piricularia oryz when growing on volcanic soil. When we consider the role of specific trace elements in soil, such as copper and manganese, we can reasonably ask whether this kind of resistance is due to the presence and availability of these trace elements issuing from deep in the earth.

To sum up, analysis of the effects of environmental factors on a plant's disease resistance - flowering period, age of the plant parts, climate, composition of the soil, as well as plant variety (genetic factors) - clearly shows their impact on the metabolism of the plant. This particularly applies to the tissue levels of soluble nutritional elements such as amino acids and reducing sugars, upon which parasites are dependent to different degrees.

Disease resistance in plant feeding insects provides further proof of the important role of plant metabolism and the ratio of protein synthesis to protein breakdown.

d) Resistance to viral diseases, in relation to the nature of the soil used to grow the deedling, in the case of certain lepidoptera.

Vago (1956) showed that it appeared possible to trigger a pronounced viral infection not only where there was no previous infection, but also when actively guarding against the entry of the virus. He showed that an infection could be provoked not just by a sub-lethal dose (for example, by the use of sodium fluoride) but simply by causing malnutrition in the insect.

In the case of Vanessa urtica (cabbage white), feeding the caterpillar on bramble leaves leads to considerable differences in the proportions of Nuclear Polyhedrosis Virus (NPV), depending on the type of soil on which the plant is growing (see Table 2).

Table 2: Proportion of NPV within *Vanessa urticae*, according to type of feed.

Feeding on bramble leaves growing on clayey soil:	18%
Feeding on bramble leaves growing on silty soil:	4%

This shows clearly the great importance of the nature of the soil and its fertilisation for the nutritional value of the plant, not just to domestic animals but also to man. We will return to this subject later.

The quality of a harvest resides in its nutritional value, which is not always easy to measure. If it can initially be measured in terms of its protein content (which is, as we have seen, a criterion of its disease resistance), this would explain why research into the stimulation of protein synthesis is the basis of a more rational approach to agriculture The health of the plant and the animals that feed upon it are directly linked.

II. The relationships between plant and parasites

1. The Relations between Plant and Parasite Are Nutritional in Nature

The various environmental factors that we have looked at - flowering stage, age of the leaves, the part of the plant, the climate (temperature and photo-period), the nature of the soil and the genetic factors that go with varietally determined resistance - have given us converging results with regard to the relationships between plant and parasite. Whether the issue is attack by a pathogenic fungus, an insect, a mite, or a virus, the relation to the host plant is a nutritional one.

The chapter on bacteria will show that the same is true of attacks by bacteria. This is a clear confirmation of the validity of our theory of trophobiosis, according to which:

> 'All vital processes depend on the satisfaction of the needs of the living organism, whether animal or vegetable' (Chaboussou, 1967).

In other words, this means that a plant will only be attacked when its biochemical state corresponds to the nutritional (trophic) needs of the parasite in question.

The theory also posits that the sensitising nutritional factors are found in the soluble compounds of intermediate metabolism: soluble nitrogen (free amino acids) and reducing sugars. This does not mean that all parasites have identical nutritional needs, but that they all draw from the same pool of soluble substances dissolved in the liquid of the cellular vacuole. Where the process of protein synthesis dominates over protein breakdown in a plant, disease resistance is strongest.

Thus it seems that the ratio of nitrogenous compounds to reducing sugars could serve equally well as a criterion for determining susceptibility to diseases such as noxious pests, depending on the nutritional needs of the pest. The following chapters will show that soluble nitrogen compounds are the main nutritional factors promoting the development of the various infections.

2. The presence of phytoalexines in tissues does not explain a plant's disease resistance

The classic theory of trophobiosis holds that resistance is linked to the presence in the plant tissue of 'phytoalexines,' substances that are antagonistic to parasites. So far, however, there is no proof at all of this theory. Numerous plant pathologists have expressed doubts, particularly concerning the supposed fungotoxicity of phenols and similar substances to pathogenic fungi. (Phenolic compounds and other tannins are the ones most frequently cited as being phytoalexines).

Tomiyama (1963) found that, although phenolic compounds may be fungotoxic, the level is not very high. As a result, we cannot answer the question whether peroxydase or phenolic compounds play any role in varietal differences in disease resistance.

In the case of the potato, it has been shown that the intracellular hypha of Phytophthora infestans (mildew) stay alive even after the epidermal cells of the potato have died. In fact, the hypha take ten hours or more to die after the cell's 'hypersensitive' death. The fungus, therefore, seems to die through lack of nourishment rather than through toxic effects. It might be the same for certain classic fungicides, such as Bordeaux Mixture (which has certain side effects that cannot be otherwise explained).

On the other hand, Tomiyama notes, 'The build-up of starch and increase in protein, phenolic compounds, and respiration indicate that the compounds circulating through the plant are linked to an accelerated metabolism in the plant tissue, which becomes resistant to parasite invasion.' If we have understood correctly, this corresponds to a metabolism where protein synthesis predominates, leading to a minimum of soluble nutritional substances, which explains the resistance.

Cruikshank (1936) expresses similar doubts: 'Testing in vitro the toxicity of some toxin or other cannot yield a complete explanation of its role in vivo because it does not take into account the dynamic nature of the parasite-host interaction.' As he comments, however: 'One cannot directly conclude from in vivo observations of the cessation of growth, that the phytoalexines are the cause, or that, even if they do not intervene here, they play a primary role in disease resistance.'

Such experiments cannot therefore confirm any toxicity of the various phytoalexines to pathogenic fungi.

As Kiraly (1972) points out, 'Hypersensitive necrosis in relation to the production of phytoalexine is only a consequence, not the cause, of resistance in potatoes, beans, and wheat to the different fungi (Phytophthora infestans) and to rusts.'

Kiraly adds: 'In other words, in the natural host-pathogen interaction, characterised by an incompatibility, it was not the necrosis of the host tissues which inhibited or prevented the development of the pathogen. Instead, it was one or more unknown mechanisms which inhibit or even kill the pathogen before the start of the necrosis.'

The 'relation between the host and the pathogen' is the crux of the problem when looking for evidence of toxic substances, as Dunez (1983) reminds us. .

Similarly, Obi (1975) found that numerous types of the maize plant's resistance to *Helminthosporium turcicum* are not due to the production of phytoalexines.

According to Wood (1972) (perhaps one of the researchers to have best analysed the mechanism of a plant's resistance to disease), 'A host's resistance can depend upon the absence or non-availability of the substances necessary for the parasite's continued growth, or upon unfavourable concentrations or proportions of essential nutritional substances'.

Wood (op.cit.) has also come up against the difficulties of explaining resistance through the presence of phytoalexines. In his view, we know precious little about the processes of disease-resistance. He points out, however, that as the tissues get older, the specific responses are replaced by a more general resistance, here called 'adult plant resistance'.

McKee (1956) noted that the mature leaves of the tobacco plant are characterised by an excess of protein synthesis over proteolysis. Similarly in the case of Bryophyllum, the ratio N proteins: N amino acids is 17.4 in very young leaves, 15.9 in young leaves, and 28.8 in adult leaves (Champigny, 1960). These examples confirm that a high level of protein nitrogen and correspondingly low levels of free amino acids can, in a general manner, explain the 'nutritional' resistance of adult leaves to their various parasites.

'The production of phytoalexines,' Smith (1980) recognised, 'constitutes only one of the components of the complex active mechanism that the plant has at its disposal.' Of the elements that potentially play a role, he cites (apart from the 'the release of previously formed antimicrobial compounds') 'the modification of the structure of the cell walls' and, finally, 'interference with the fungi's nutrition'.

In a study of the relation between Botrytis cinerea and the haricot bean (*Phaseolus vulgaris*), Garcia et al. (1980) concluded that 'there was no correlation between virulence and sensitivity to phytoalexines, or between virulence and capacity to break them down, the iso-lates being capable of metabolising the four phytoalexines'.

Certain researchers in this debate, however, cite the relation between resistance and processes that promote the synthesis of proteins. Thus Albersheim et al. (1980) highlight the role of a specific hormone which 'would appear to protect the plant by inducing the synthesis of proteins which act by inhibiting the digestive enzymes of insects and bacteria.'

Nothing convincing is presented to support this hypothesis, however, so we must still ask if stimulating the process of protein synthesis has a positive effect on resistance as a simple corollary of the decrease in soluble substances, principally held to be at the basis of the 'nutritional' sensitivity of the plant.

Fritig, who had the task of drawing conclusions from this discussion, and who took into account the fact that the various authors did not attribute a major role to the phytoalexines in the processes leading to resistance, drew attention to the fact that according to different authors 'the build up of phytoalexines was preceded by a general metabolic stimulus, implying an increased synthesis of mRNAs and then of proteins.' In other words, he confirmed our understanding of the resistance processes.

To sum up, the studies that we have analysed would appear to confirm that fungotoxicity is not the role played by phenols and other tannins, as alluded to by Cruikshand (op. cit.) and promoted by Dufrenoy. We are now going to see how this physiologist's concepts allow the various, and seemingly contradictory, hypotheses to be integrated with respect to the determination of resistance.

3. Nutritional elements are in balance with phytoalexines

All of the difficulties in establishing valid correlations between the plant's resistance and the presence of certain phytoalexines in the tissues are explained by the fact that the phytoalexines are often in balance with the nutritional elements to which resistance is attributed.

Here we must recall Dufrenoy's observations, that immunity to disease is linked to 'the concomitance of two antagonistic biochemical changes:

- on the one hand, the production and build up of sugars and amino acids in the vacuolar solution,
- on the other the production of phenolic compounds.'

Dufrenoy notes that 'the abundance of phenolic compounds and the paucity of amino acids appears to inhibit the growth or proliferation of pathogenic microorganisms. In contrast, an abundance of soluble sugars and amino acids guarantees growth and proliferation.'

Dufrenoy (1936) adds that 'circumstances which are unfavourable to the formation of new quantities of cytoplasm - in other words, circumstances unfavourable to growth in general - tend to encourage an accumulation of unused soluble compounds (sugars and amino acids) in the cells' vacuolar solution. This accumulation of soluble compounds would appear to favour the nutrition of parasitic microorganisms and thus reduce the plant's resistance to parasitic diseases.'

As Dufrenoy explicitly stated elsewhere: 'Recent physiological studies tend to confirm the results of the cytological study: that a plant's vulnerability to its parasites is a function of the abundance of amino acids and sugars in its vacuolar solutions. These amino acids and sugars are either present in the plant because of a nutritional imbalance or are the result of the protein breakdown or amylolysis brought about by the parasite'.

As we can see, our trophobiosis theory totally coincides with Dufrenoy's observations: the quantity of phenolic compounds, in correlation with the paucity of amino acids, brings about resistance. This resistance is not due to any toxicity to the parasites, but to a lack of nutritional elements. This is, for example, the case with the N / phenols balance, which explains the sensitivity of the maize plant to Helminthosporiosis. This sensitivity is related to the value of the numerator - in other words soluble nitrogenous products, the fundamental nutritional elements that we will encounter throughout this study, which encourage the development of various parasites.

4. Other environmental factors that have an effect on the metabolism, and thus the resistance of the plant

We have deliberately left to the end the cultural impacts capable of influencing plant physiology and resistance. Man has not always weighed carefully the consequences of his actions, when it comes to the resistance of plants. These consequences can be seen in the impacts of the new 'intensive' methods of agriculture, particularly in the cases of cereal production, arboriculture, and viticulture. The factors that seem to us most important are

- the use of new chemical pesticides;
- techniques of fertilisation.
- the technique of grafting (less important).

In the years since World War Two, the first two of these practices have undergone huge changes, with the arrival of new synthetic products for phytosanitary uses and the increased use of artificial fertilisers, notably nitrogen.

The introduction has already explored the role played by artificial pesticides in setting off 'biological imbalances' such as the apparently 'abnormal' development of disease and infestation of insects, mites, nematodes, etc that damage crops. We will therefore begin by examining in the next chapter the repercussions that pesticides have on a plant's physiology, particularly on the process of protein synthesis, which, as we have already seen, is fundamental to disease resistance.[5]

Notes

1 From Chaboussou: *Les Plantes malades de Pesticides*, Débard, Paris, 1980
2 These include downy mildew of cabbage (*Peronospora brassicae*), downy mildew of beet (*Peronospora schachtii*), and tobacco blue mould (*Peronospora tabacina*).
3 *Plasmmodiophora brassicae.*
4 *Fusarium* (L).

Chapter 3

The Repercussions of Pesticides on the Physiology of Plants, and the Consequences for their Resistance to Parasites

I. The participation of pesticides in the metabolism of the host plant

Apart from the phenomena of burns or of phytotoxicity, caused by certain pesticides,which prevents their being sold commercially, the less spectacular side-effects of phytosanitary products on plant physiology have scarcely been considered. There are several reasons for this.

First, the products used (whether fungicides or insecticides) were thought to act only 'on the surface.' Among these, for example, were copper-based products and 'ingestion' insecticides, such as arsenic-based ones. There was thus believed to be no need for concern about a chemical's penetration into a treated plant's tissues, or about any eventual reaction of the plant.

Nevertheless, with the development of 'systemic' insecticides and anti-fungal products, and the use of herbicides on a vast scale, the question of their effects on plants has become more and more urgent.

The previous chapter shows that the relations between plant and parasite are nutritional in nature. As Collectif (1979) has stressed, 'A parasite is pledged to the plant in the name of the nutrition offered it by that plant.' If we also take into account the case of herbicides and their 'reduced' selectivity, we need to become more and more urgently concerned about the impacts of pesticides on the physiology of plants.

We will study these effects chiefly with respect to the ratio of protein synthesis to protein breakdown. First, though, we will briefly consider how a pesticide penetrates into the plant tissues.

II. Pesticidal penetration into the plant

The use of folial fertilisers and herbicides, now commonplace in agricultural practice, shows that plant tissues can easily be penetrated by numerous chemicals. The 'overleaping' of barriers such as the cuticle and cell walls by liposoluble compounds is facilitated by the lipids in the cuticle and walls. This explains, for instance, how a metallic salt such as hydrated copper oxide (from applications of Bordeaux mixture) can penetrate into the leaf, as was affirmed long ago by Millardet and Gayon (1887) and demonstrated more recently by Strauss (1965).

This explains differences in sensitivity to pesticides, according to the plant species or variety. Penetration by sprays depends on the thickness of the cuticle as well as on the distribution and number of stomata. But perhaps it is the osmotic pressure inside the cells that chiefly regulates this penetration. When the cellular sap is isotonic or hypertonic in relation to the spray, there is no danger of burning, for penetration is impossible. When the sap is hypotonic, on the other hand, changes are more frequent, due to plasmolysis of the cells. For instance, Menzel (1935) was able to show that certain varieties of pear and apple tree with a high osmotic pressure (30-38 atmospheres) are almost totally immune to copper sprays, whereas other varieties with a low osmotic pressure (5.3 to 13.7 atmospheres) are severely affected.

This explains variations in pesticide penetration, according to the physiological state of the plant at the time of treatment. This state in turn depends on abiotic factors, tissue age, and the nature of the nutrition of the plant. Thus light, by promoting maximum opening of the stomata, increases the permeability of the leaf. An increase in temperature can have the same effect: in warm weather, for instance, nicotine treatment results in burning, whereas this now outmoded pesticide has no phytotoxic effect when applied in periods of lower temperature and weaker sunlight. The same applies to sulphur.

Likewise, 2,4-D penetrates much faster into leaves of the bean plant in daylight than in the dark (Sargent, 1964).

Finally, senescent plants allow greater penetration by chemicals.

Ultimately, of course, the chemical nature of the pesticide also plays a role. While, for example, organophosphates such as parathion disappear rapidly from the surface of leaves and penetrate them almost totally within just two days, substances such as chlorinated chemicals and carbaryl persist much longer. With respect to their relation with the plant, we can say in general that artificial organo-chemicals have a very special affinity for plant tissues. The radical C_6H_5 in a formula makes fats soluble (liposolubility). The radicals OH, COOH, SO_2 and NH_2 induce hydrosolubility. Cl, a constituent of many modern fungicides, is linked with persistence of the product. We shall return to this point later.

Another important point about pesticides is their circulation and diffusion through the plant. This can happen in two ways:

- the chemical can be transported to the cells by the apoplastic pathway. The chemical passes through the cell wall (the 'gap' between the wall and cuticle that gives solutes access to the living cell). This means that the pesticide is carried not only through the vessels of the wood, but also through the spaces between the cells. Numerous chemicals are diffused in this manner.
- transfer of the pesticide can just as easily take place from cell to cell. This is the symplastic pathway. It penetrates the plasmodesmes, those pores that allow easy exchanges from cell to cell.

All this can lead to disruptions at the level of cellular activities and will be discussed later.

As to the ways that pesticides gain entry, we can say that all the parts of the plant are involved. Penetration can in fact take place:

- *through the leaf*, during traditional treatments with insecticides, fungicides and, more recently, acaricides;
- also *through the roots*, as a consequence not only of possible disinfection of the soil against noxious insects or pathogenic fungi, but also of fall-out from folial treatments (herbicides, fungicides and insecticides). This fall-out has a much greater impact than one might at first suppose.

Researchers have been struck by the drastic consequences, such as sterilisation of the soil after cupric treatments, or even the elimination of earthworms by dithiocarbamates. But there are also more insidious repercussions, such as rendering a crop susceptible to parasites. The insidious effects take place:

- *through the seeds*, as a result of coating them against both diseases and insects that pose a danger to the seedlings. These indirect repercussions, through their effects on the physiology of the plant, can be either harmful or beneficial.
- *through the trunk and main branches* of fruit trees and, in particular, the grapevine. This is a fourth means of penetration, one not generally taken into account. Penetration here takes place during the application of both fungicides and insecticides during winter or pre-pruning treatments.

It has been shown that, even in winter, a fruit tree can absorb considerable quantities of a chemical sprayed on its bark. It is not surprising, then, that pesticides applied to a tree, even in winter, can have an effect on the physiology, and thus on the resistance of trees. What is more, Willaume (1937) was able to show that anthracene oils have a stimulating effect on tree growth. In certain dilutions, these anthracene oils can clearly accelerate the germination of grains of wheat. We can rightly ask whether a cupric application in late autumn against cryptogamic or viral diseases would not act in the same way. It could work indirectly, in other words, through the beneficial effects of the trace element copper on the physiology of the fruit tree. We shall return to the subject of how trace elements act on the plant.

For now, we will continue our investigation of the effects of the *growth substances* that we know fairly well. As we shall see, these are not unrelated to the effects of certain pesticides, although the latter are never used for the same ends.

III. The repercussions of growth substances

a) Growth substances and plant metabolism

Among the growth hormones we find:

- auxines, which have a specific effect on growth itself;
- cytokinins, which accelerate the cells' multiplication rate;
- gibberellins, which have multiple functions: acceleration of germination, increase of amylase in the endosperm, and activation of protease (which results in the formation of tryptophan, precursor of ß-indolacetic acid, or IAA).

IAA is therefore formed in tissues that are still growing, above all in the extremities of the roots. This is the auxine that causes roots to grow downwards.

As for the cytokinins such as kinetin, derived from adenine, these play a role in the growth and differentiation of cells, as well as in other interesting physiological functions such as the inhibition of senescence. The interactions of auxins and cytokinins control the differentiation of tissues previously undifferentiated in the roots and shoots.

Although until 1956 scientists were only aware of the existence of auxins, gibberellins, and cytokinins, they suspected the existence of another hormone, one that could balance out the effects of the others. In fact, in 1965 they discovered abscisin II in cotton, together with dormin in maple buds (which prevents their opening). These hormones have the same structure, a molecule that is widespread in the plant kingdom: abscisic acid or ABA.

In the majority of cases, ABA counteracts the effects of growth hormones: it is clearly the 'modulator' whose existence had been suspected. As a result, agronomists hope to be able to induce leaves to fall, prevent potatoes from germinating, encourage the ripening of fruit, etc. In fact, at a cellular level ABA activates certain amylases, bringing about the destruction of starches in amyloplasts. It also inhibits the synthesis of ribonucleic acid, leading to a considerable reduction in the quantity of ribosomes, which are required for the synthesis of proteins, these 'ultimate elements of the expression of the genes.'

ABA thus blocks the synthesis of proteins (while cytokinins have the opposite effect). This blocking causes a reduction in the total amount of cellular RNA. This confirms at cytological level that an excess of ABA leads to the disappearance of ribosomes.

To sum up, ABA has a regulatory role with regard to growth hormones and an antagonistic role (directly or indirectly) at the level of certain genes of synthesis. This role is mediated by enzymes.

Having briefly defined plant metabolism as the achievement, primarily, of a degree of equilibrium among antagonistic substances, we are better placed to understand the effects of the growth substances.

b) The effects of growth substances on the biochemical state of plants and on their resistance to parasites

Various growth substances, such as gibberillic acid, ß-indolacetic acid and 2.4-D, have been tested in controlling plant disease. They have been used against both pathogenic fungi and viral diseases. The results have been varied and often contradictory, without researchers being able to give reasons for this. We will attempt to clarify the mechanism of these effects. To begin with, we will analyse the results of attempts to control *Phytopthora cactorum* on the strawberry plant.

In their study entitled 'The Influence of Several Growth Substances on the Strawberry Plant's Susceptibility to Attack by Phytopthora cactorum,' Molot and Nourrisseau (1974) concluded:

- Under glass, dipping pre-refrigerated[1] plants in different growth substances, just prior to planting and inoculation with *Phytopthora*, elicits a variety of responses in terms of manifestation of symptoms. Thus, at concentrations of 10^{-6} or above,
- gibberillic acid, naphthalene acetic acid, and indolacetic acid promote disease;
- tri-iodo-benzoic acids, on the contrary, reinforce resistance;
- abscisic acid, at a concentration of 10^{-7}, increases the damage caused by *Phytopthora*, but ceases to have this effect as one moves away from this concentration.

The authors of this research were therefore led to the justifiable conclusion: 'A relationship seems to exist between the physiological state of the strawberry, subject to rather complex hormonal balances, and its response to *Phytopthora cactorum*.' Their analysis stops here, without giving any explanation of the diversity of responses brought about by the different substances tested. We will try to go farther toward explaining these phenomena.

With regard to gibberellin, Altergot and Pomazona (1963) emphasized that the effects of any pesticide or chemical whatever, when applied to a plant, depend on several factors. The most important are:

- *the dosage applied*;
- *the ecological conditions*: light, humidity, temperature, etc.;
- *the age of the plant* or its parts at which it is applied;
- *the plant's nutrition*, a concluding factor that has so far been largely overlooked.

The authors point out that in order for the gibberillin to have a positive effect, these conditions should correspond to a 'state of accelerated growth': in other words, a state of *optimum protein synthesis*. We shall encounter analogous phenomena with certain pesticides, for example DDT.

As Altergot and Pomazona (op. cit.) observe: 'We can conclude that when a plant has been stimulated by growth substances, it requires rapid complementary nutrition in those substances that are indispensable for the synthesis of its constitutive compounds.'

This is why various researchers working with the potato plant, such as Wort (1962), obtained an increase in yield only when they applied 2,4-D along with specific fertilisers, whether in the soil at the root level, or through a foliar spray mixed with the growth substance. Wort (op. cit.) used chiefly trace elements, such as iron and copper. These experiments showed, on various varieties of potato, gross yield increases of 5-45%, along with increases in the size of the tubers.

As Wort notes, in passing: 'While the causes of increase in resistance to disease are not as clear as the favourable effect on yield, it is clear that the general tendency is favourable.' This observation seems to agree fully with our own theory that resistance to disease is linked to stimulation of protein synthesis. In fact, the tubers of potatoes treated with a mixture of 2,4-D and trace elements have, in comparison with controls, shown lower levels of reducing sugars.

In the second place, like Altergot and Pomazona, Wort's findings show that the differences in effect correspond to the timing of the treatment. An interval of one month (July 23 - August 24) in treating potatoes with 2,4-D causes differences in the amounts of the different categories of sugars contained in the tubers. The reducing sugars become more abundant in potatoes treated later in the period, at the end of August.

We shall return to the importance of the stage of intervention with pesticide treatments, from the perspective of their effect on the plant's physiology, in relation to the physiological cycle of the plant. This inquiry is justified by the impact of the treatments on the balance between auxins and gibberellins. This balance can easily be upset, for good or for ill, by application of artificial growth substances or, as we shall see below, of any kind of pesticide.

We shall now attempt an explanation of the diverse responses to Phytophthora cactorum in the strawberry, as recorded by Molot and Nourrisseau (op. cit.), according to the different growth substances that were tested.

We shall first take the case of abscisic acid or ABA, which at a concentration of 10^{-7} increases the damage. The effects of this substance are well known: they act essentially as inhibitors of the synthesis of RNA. The outcome is a considerable reduction of the level of ribosomes. Now, as we know, ribosomes are directly involved in the synthesis of proteins. ABA blocks the synthesis of proteins, apparently by acting on the messenger RNA. This produces a corresponding increase in the levels of soluble substances in the plant tissues. This favourable nutritional effect on the pathogen explains the increase in damage by the parasitic fungus.

There is good reason to ask whether a similar process might not be involved when susceptibility to the disease increases after the plants are treated with gibberellic acid, naphthyl acetic acid, and indolacetic acid.

Likewise, we need to study the indirect mode of action of tri-iodo-benzoic acid. In contrast to other substances, it reinforces resistance. However, for each substance we must in each case take into account the physiological state of the plant. For example, a strawberry plant that has been in a refrigerator for a considerable period of time has to all appearances a slow metabolism, to some extent resembling a somewhat senescent plant. Under these conditions, therefore, tri-iodo-benzoic acid may show a positive effect on protein synthesis and thus our conception of resistance All the other substances, by contrast, yielded negative results.

We therefore can explain the often diametrically opposed repercussions of the substances that have been systematically tested - by their biochemical and nutritional effects, not their toxicity to the pathogenic agent.

With regard to gibberellin, Van Overbeck (1966) noted that opinion was moving to the idea that plant hormones act on the system of nucleic acids somewhere between the DNA and the messenger RNA. Such an influence would therefore control enzyme formation and, as a consequence, the biochemistry and the physiology of the plant.

Gibberellin would therefore, by its action on the synthesis of messenger RNA, stimulate amylase in accordance with the following diagram:

Action of the gibberellic acid

DNA ⟶ messenger RNA ⟶ α amylase.

Through its action on the proteolytic enzymes, gibberellin can cause the formation of tryptophan, the precursor to IAA. Auxin and gibberellin can act synergistically. On the other hand, we know that the functions of hormones overlap. It is, however, the initial state of the plant that appears above all to determine the nature of the effects of a specific growth substance.

Gibberellin, for example, stimulates the formation of reducing sugars in potatoes *in a dormant state*, according to Van Overbeck. This process promotes protein breakdown. We can therefore pose the question: to what extent might this same substance act similarly on the refrigerated strawberry plant, whose metabolism has been inhibited by the cold? Could it bring about an increase in the soluble substances which nutritionally sensitise a plant to *Phytophthora*?

If, on the other hand, gibberellin has positive effects on protein synthesis, depending on the initial state of the plant, the same is true for disease resistance. In this way, Rodriguez and Campbell (1961) were able to demonstrate that applying gibberellin to the apple tree, the bean plant, and the cotton plant both stimulated the growth of the plant and also reduced populations of *Tetranychus telarius*.

Applying doses of 10ppm of gibberellin to apple trees increased numbers of *Panonychus ulmi*, while higher doses had the opposite effect, decreasing numbers. These different population levels show significant correlations with the levels of total sugars in the leaves.

Populations of the two species, moreover, show a positive correlation with the nitrogen content of the leaves, which is in turn dependent upon the action of gibberellin at a given dose.

Likewise, Rodriguez and Campbell suggest that, generally speaking, there is an optimum level of reducing sugars for the development of parasites, when it come to a plant's susceptibility to attacks by disease and noxious insects. This situation can be slightly modified by taking into account the level of nitrogen and the interaction of these two nutritional substances, a point that was mentioned above and which we will discuss in detail later.

Hukusima (1963), looking at the proliferation of the aphid *Myzus malisinctus* on apple trees treated with varying doses of gibberellin, came to the same conclusion. While the maximum proliferation resulted from a dosage of 50 ppm, a considerable reduction in fecundity was registered with a dose of 100 ppm. This led the author to conclude that there is probably an optimum level of reducing sugars and total sugars for the development of aphids, as well as of mites. We shall return to this subject of aphid proliferation on cereal crops and the development of viral diseases. For the moment, however, we shall examine insect proliferation brought about by growth substances as a result of their effects on plant biochemistry. We will look particularly at the most widely used synthetic growth hormone, 2,4-D, and its close relative, 2,4,5-T.

Repercussions of 2,4-D and 2,4,5-T.

With regard to the development of disease, it has been found that 2,4-D has contradictory effects. After all that we have seen above, this should not surprise us, since these effects depend upon the initial state of the plant and its ultimate biochemical state. They also depend upon ecological conditions, the plant's nutrition, and the nutritional requirements of the specific parasite.

It has been shown that treating wheat with 2,4-D predisposes it to serious attacks of *Helminthosporium sativum*. In contrast, thanks to the same treatment of 2,4-D on the broad bean plant, Mostafa and Gayed (1965) obtained a net anticryptogamic effect against *Botrytis fabae*. It seems clear that this 'fungostatic' effect has its origin in the action of the chemical on the plant's metabolism. Analyses of tissue samples from treated plants, in fact, showed that 2,4-D had considerably reduced the sugar levels of the leaves. The authors concluded that this process seemed to interfere with the normal development and potency of the *Botrytis*.

We would add that this too has a nutritional cause, given that the fungus in 'grey mould' has particularly high nutritional requirements for sugars. This explains, besides, the inverse effects: dithiocarbamates such as maneb and zineb, which are well-known for promoting the development of *Botrytis*, particularly on the grapevine, produce an increase in the levels of sugars in the leaf tissues.

The explanation for these convergent phenomena lies in the fact that both dithiocarbamates and 2,4-D, applied repeatedly or at specific times, inhibit protein synthesis. This causes an increase of soluble substances in the tissues, which make the plant nutritionally susceptible to parasitic fungi.

The same processes may equally apply to proliferation of insect pests. Maxwell and Harwood (1960) were able to show that treating broad beans with 2,4-D produced a clearly elevated rate of reproduction in the aphid *Macrosiphum pisum*. This fact appears to be related to an increase in the sap of various free amino acids such as alanine, aspartic acid, serine, and glutathione. In this case too, reducing sugars appear to play a role in the aphid's fecundity. (We shall return to this point later.)

However, parallels appear between the repercussions of certain pesticides.

Smirnova (1965) found that the reproduction of *Aphis fabae* on flower stalks treated with DDT was at its greatest between eight and fifteen days after the application of the pesticide. According to the author, this increase in egg laying corresponded with an increase in non-protein nitrogen in the treated plants. Furthermore, the flower stalks treated with DDT showed an increase in sugars compared with the control plants.

The aphid larvae feed at the plant's expense where the sap has high levels of soluble substances (nitrogen and sugars), thus showing a balance that increases their fecundity. In other words, in this case (as in all other cases where the plant's resistance is undermined) the parasite proliferates where protein breakdown predominates in the host plant's metabolism.

The harmful side-effects of pesticides are not confined to sucking insects such as aphids and mites. They extend to other noxious insects that do not specifically feed off the sap. Ishii and Hirano (1963) showed that the rice borer *Chilo suppressalis* (a lepidopteran) multiplied on rice treated with either 2,4-D or DDT.

These researchers showed clearly that this was due to indirect nutritional effects. Treated plants increased their biotic potential by offering the larvae a better diet, *through a higher level of nitrogenous elements*.

Interesting research has been done on the related chemical 2,4,5-T (2,4,5 trichlorophenoxy acetic acid). The goal was to ascertain the optimum dosage, in the light of the increase in yield it produces. The research underscores clearly the great importance of the effects that a plant's nutrition has on its metabolism and thus on its resistance. Yurkevitch (1963) demonstrated that 2,4,5-T, applied to the tomato plant, reduced leaf loss and accelerated growth of the fruit. These beneficial effects, however, arise when there is optimum fertilisation. The best results are obtained by adding certain trace elements, such as boron or manganese, to the growth substance. Not only is there an increase in yield; in addition, treating the plant with a mixture of 2,4,5-T and trace elements such as molybdenum brings an increase in dry matter and vitamin C content in the fruit. In this way, an improvement is also achieved in the *quality* of the harvest.

The author insists on achieving an *optimum balance of the fertiliser* in relation to the stimulating effect of the growth substance. While tomato plants treated with a mixture of 2,4,5-T and boron produced a higher yield and better quality fruits, a concentrated treatment with just one of them suppressed the growth of the plants. We will have to return to this subject of trace elements. Suffice it to say at this point that they form part of the composition of enzymes and, because of this, they affect the direction of oxidative-reductive processes, promoting the transport of carbohydrates and acting on the respiration rate.

We should not be surprised if we encounter similar phenomena, whether beneficial or harmful (as is most often the case), accompanying the use of various phytosanitary chemicals such as insecticides and antifungals. Until now, little thought has been given to their effects on the physiology of the plant, as linked to its resistance.

c) The effects of maleic hydrazide

Drawing up a balance sheet of the effects of maleic hydrazide on the biochemistry of the treated plant, Wort (1962) states that when incorporated into the sand in which barley and wheat are grown, maleic hydrazide leads to a considerable increase in levels of free amino acids, particularly glutamine, in the leaves. Five days after the application, the treated leaves contain five times more free amino acids than the control plants, and after fifteen days, fifteen times more.

Similar changes occur with sugars. When applied to maize, maleic hydrazide reduces the levels of glucose, but it can increase levels of saccharose by as much as thirteen times, compared to controls.

In summary, although these repercussions of maleic hydrazide depend, as with all chemicals, on the initial state of the plant, they can be explained by a dual phenomenon:

- stimulation of photosynthesis, on the one hand;
- inhibition of protein synthesis, on the other.

Certain therapeutic effects have also been attributed to these biochemical effects. Horsfall and Dimond (1957), reasoning from the fact that maleic hydrazide increases the sugar levels of the leaves, concluded that this chemical should increase diseases requiring high sugar levels, such as rusts and Oïdium, and decrease those that tolerate low levels of sugars.

Maleic hydrazide does in fact decrease the susceptibility of the tomato plant to *Alternaria*. The same authors think that, in principle, it should also render the foliage less susceptible to *Helminthosporium sativum*.

In addition, Sprau (1970) showed that applying maleic hydrazide to potatoes inhibits the development of tumours produced by *Agrobacterium tumefaciens* and *Synchrytium endobioticum*. Sprau stresses the importance of studying the relations between host and parasite. With regard to the upsurge of bacterial disease, we shall see precisely what is the role played by the new chemical products in the harmful phenomena that occur after the use of these products.

Here, too, we shall find a confirmation of the relation: protein synthesis → resistance, while protein breakdown → susceptibility to attack from bacteria.

This is the process, nutritional in nature, that enables us to explain the results of Molot and Nourrisseau (*op. cit.*), obtained from *Phytopthora cactorum*, var. *fragariae*, using various growth hormones.

In sum, these results confirm that:

- the plant-parasite interaction is nutritional in nature;
- repercussions from the same pesticide will vary according to the initial state of the plant.

There is an inevitable interference, in fact, between the artificial growth hormones and the natural hormones that govern the plant's metabolism. This makes it vital to take into consideration two important factors relating to selection of the best therapy:

- the critical stages of metabolism, particularly the flowering period;
- the plant's nutrition, which depends not only on genetic factors but also on the climate and artificial factors, such as fertilisation. This can be mineral or organic, and must take account of the 'availability' of the trace elements.

It is the importance of these different factors, which has largely gone unnoticed, that has made the effects of the various commonly employed pesticides incomprehensible hitherto. Here we intend to begin this investigation, commencing with chlorinated chemicals.

IV. The effects of chlorinated chemicals, especially DDT

Because of their persistence and their transmission through the food chain, these chemicals are now banned from agricultural use in many of the industrialized countries..[2] However, like the growth substances we have just examined, they remain interesting because of their impacts on plant biochemistry and physiology, and thus on resistance to disease. Their impact on both is usually negative.

Based on studies carried out, it appears that DDT in particular acts as a growth substance for plants, comparable to natural growth hormones.

Chapman and Allen (1949) made use of this comparison to characterise the effects of DDT on the various plants they tested. These effects vary, of course, according to the botanical nature of the specific plant, and also according to the dosage applied. High concentrations have phytotoxic effects, such as stunting, deformities, chlorosis, and necrosis, while at low doses such side effects can disappear, to be replaced by growth stimulation.

Phenomena of 'remote' effects are also observed, although these must not be confused with 'systemic' effects (the transportation of the pesticide through the tissues of the plant). Chapman and Allen observed that applying DDT to the roots and lower leaves of a plant resulted in stimulation of the higher part of the plant. From their observations, the authors concluded that the effects of DDT on plants closely resembled those of certain plant hormones.

This explains why, with regard to both disease and insect attacks, DDT can have either positive effects or, more often, negative ones. In the course of the previous chapter we noted that DDT renders khapli wheat more prone to rust (Johnson, 1946). The explanation for this was given later by Forsyth (1954): 'DDT alters the wheat's metabolism in such a way as to make free amino acids and simple sugars accumulate in the leaves. This phenomenon appears to be due to an inhibition of synthesis of proteins and carbohydrates.' (Johnson, *op. cit.*)

This would appear to explain the swarms of various insect pests in the wake of treatment of crops with DDT or similar chlorinated chemicals. These outbreaks are not due, as the 'classic' view still holds, to destruction of the insects' natural predators.

For example, Huffaker and Spitzer (1950) investigated the validity of this last hypothesis. The outcome of their studies was that, on the pear tree, the natural predators proved more abundant on trees treated with DDT than on the controls.

In fact, as they concluded: 'The increased population levels brought about by DDT demonstrate that DDT can lead to an increase in the number of *Panonychus ulmi* greater than that on the controls. Although the reasons for this are unknown, it cannot be explained on the basis of natural predators.' (In certain experiments, the population of *P. ulmi* on pear trees increased twelvefold.)

For his part, Fleschner (1952) confirmed the findings of Chapman and Allen. He found that DDT applied to citrus caused changes in the leaves, making them less resistant to attacks by mites. This stage of reduced resistance persisted in the leaves for a period of more than seven months.

Moreover, Fleschner also showed that this reduced resistance is 'transmitted' from the lower, treated leaves to the upper, younger ones. At the end of 46 days the leaves above those treated with DDT carried a significantly larger number of eggs than the lower ones.

This suggests that in these circumstances, DDT behaves like a hormone. It seems to have hormone-like effects similar to those observed by Forsyth in wheat. It inhibits protein synthesis, leading to an accumulation of free amino acids and reducing sugars in the plant tissues. This phenomenon, by its nutritional effect, permits the mites to grow in number.

Similar results were obtained by Saini and Cutkomp (1956) in relation to proliferation of *Tetranychus telarius* when treating the runner bean with DDT. The authors conclude that this stimulation is indirect in nature, 'arising from a physiological change in the host-plant.' The increases in reproduction are produced when there is a certain balance of nitrogenous substances and saccharose.

We should recall that, for both *Panonychus ulmi* on the grapevine and *Tetranychus urticae* on the bean, we have shown that DDT, along with other pesticides such as carbaryl and certain phosphoric esters, has the nutritional effect of increasing the mite's biotic potential. It increases fecundity and longevity, shortens the evolutionary cycle, and distorts the sex ratio in favour of females (Chaboussou, 1969).

However, we do not wish to end this section on DDT without mentioning the case of soil treatments and their unforeseen side-effects. Klostermeyer and Rasmussen (1953) treated soil with various chemicals (DDT, lindane, HCH, chlordane, and aldrin) in varying doses. When they later planted potatoes, they found that plants growing in the soils that had received the heaviest doses were fiercely attacked by *Tetranychus bimaculatus*. In the end these plants were almost completely destroyed. After pointing out that the mites' predators were few in number, the authors concluded: 'It is likely that these differences in numbers of mite populations, due to the use of insecticides, had their source in the nutrition and composition of the plant.'

With regard to the physiological consequences of DDT, we note also that Kamal (1960) has shown how DDT, like 2,4-D, acts on the different forms of nitrogen (nitrogen ammoniac and amino nitrogen), according to the age of the leaf. This justifies the hypothesis, says Kamal, 'that using insecticides and fungicides on the metabolic activities of the tissues of the host plant can affect the resistance of the plant. It is also one of the possible causes of the outbreak of 'pear decline,' rampant at one point in the US state of Washington.' This decline is now being discussed in France in relation to proliferation of psyllids, which observers are unable to explain (Kamal and Woodbridge, 1960). We shall return to the subject of the effects of synthetic pesticides in the chapter dedicated to so-called 'intensive' cultivation of grapevines and fruit trees.

Now we shall turn to the impacts of organo-phosphates.

V. Side effects of organophosphates

Since phosphate pesticides were first put to use, various researchers have noted, apart from the phenomenon of burning, diverse impacts of a physiological nature on treated plants. Some of the repercussions are beneficial, for instance increases in yield. In the case of cotton, yield increases have been recorded as a result of weekly applications of a mixture of Toxaphene and DDT. However, the interesting point to note here (because it confirms a fact mentioned above) is that such positive effects on production only occur if the treatment begins with the appearance of the first flowers, but no later. This underlines once more the major importance of the initial physiological state of the plant for the final effects of the chemical applied, as a result above all of the auxin levels of the tissues.

In fact, as with growth substances and chlorinated pesticides, the bio-chemical repercussions of organo-phosphate products have their origin in the way they interfere with plant enzymes. Hascoet (1957) cites the work of two Japanese researchers who describe the physiological effects of certain organo-phosphate insecticides, which in their view are precisely comparable to those of 2,4-D.

Just as with growth hormones or chlorinated pesticides, tissue analysis reveals effects on the process of protein synthesis, which can be either negative or positive, depending on the circumstances. For the bean plant, for instance, Bogdanov (1963) notes that parathion and thiometon bring about increases in free and bound amino acids in the leaves. The levels of methionine, valine, and phenylalanine are significantly increased, as are those of tryptophan, whose importance to plant metabolism scarcely needs mentioning.

Pickett *et al.* (1951) found some negative effects of parathion on the peach tree, where it slowed down photosynthesis in comparison to the controls Similar results were found when treating apple trees (Red Delicious) with ethion and diazinon. These organo-phosphates reduced the photosynthetic potential (the net assimilation rate, or NAR).

Such effects may be compared with the results obtained by Nandra and Chopra (1969), regarding the effects of thiometon on 'groundnut' or 'ground pistachio.' The overall effect of thiometon was to increase nitrogen levels in the leaves of the treated plants, by comparison with controls. (Note that this increase in nitrogen levels is generally found with chemical products, particularly fungicides - or rather, anticryptogamics - and that this phenomenon lies at the root of a plant's sensitisation to its various parasites, as we have already pointed out above.) In the same way, and parallel to this inhibition of protein synthesis, thiometon increases the levels of reducing sugars.

This explains the apparently paradoxical proliferation of mites, brought about by what could be called a 'boomerang effect' of acaricide products. Several examples of this have been demonstrated in field and laboratory experiments. For example, Wafa *et al.* (1969) experimented on citrus with various acaricides (Thiocron, anthio, metasystemox), used alone, in a mixture, or alternating between the two. These brought about increases in the numbers of the mite *Eutetranychus orientalis*, an effect that lasted for long periods. The authors add:

> 'The harmful effect of the use of phosphorous acaricides, alone and in repeated applications, can be attributed to the effect these chemicals have on the composition of the plant.'

As for the biochemical mechanism of this phenomenon, the authors consider it to be linked to the following fact:

> 'In plants treated with these organo-phosphate acaricides - Thiocron, anthio, and metasystemox R - there was, in comparison with the controls, both an increase in soluble sugars and a decrease in polysaccharides in the leaves and stems.'

Once again, we see that the plant is made susceptible to mites by acaricides that inhibit the plant's protein synthesis. We have stressed in our book, *Plants Made Sick by Pesticides (Les Plantes Malades Des Pesticides)*, the full importance of the repercussions of the various acaricides used to treat fruit trees, as studied by Blagonravora (1974). The most effective products, as one might expect, were those with the most long-lasting positive effects on protein synthesis, such as dicofol and phosalone.

In the case of the grapevine, we have ourselves found that parathion, which brings about a reduction in proteic nitrogen and an increase in reducing sugars, provokes through a trophic effect the proliferation of 'yellow spider' (*Eotetyranychus carpini vitis*) at the end of the season (Chaboussou, 1969).

All these studies show that numerous organo-phosphates inhibit protein synthesis, especially since they are used in repeated applications and at sensitive stages in the plant's growing cycle. This is the cause of the plant's increased susceptibility, not only to sucking insects such as mites, aphids, aleurodes, and (so it seems) psyllids but also to diseases, fungal and otherwise, as we shall see below. Besides, these negative effects could also result from the use of 'fungicides,' as will now see demonstrated through a study of the effects of dithiocarbamates.

VI. The effects of carbamates and dithiocarbamates

Carbamates, derived from carbamic acid, and dithiocarbamates, derived from dithiocarbamic acid, constitute a large group of artificial organic substances. Among these are 'fungicides,' including carbamates (benomyl, carbendazeme, thiobendazole) and dithiocarbamates (zineb, maneb, mancozeb), to name but a few, as well as 'herbicides,' including carbamates (propham, chlorpropham etc) and dithiocarbamates (nabam, for instance, which is also a fungicide).

As with the categories of pesticides already examined, setbacks have been recorded. Polyakov (1966) - who was perhaps the first to make the link between the plant's metabolism, when altered by pesticides, and susceptibility to pests, noted that using zineb to protect potato plants against mildew led to the development of other diseases, particularly viral ones. This same carbamate, when used on apples against scab, stimulates the development of *Oïdium*. We have experimentally demonstrated this result on the grapevine (Chaboussou, 1968). It has also been observed on apple trees on several occasions in France, and was subsequently confirmed in Bulgaria by Vanev and Celebiev (1974). These tests, conducted with controls, show clearly that it was a question of proliferation of the pathogenic fungus, not its resistance or the effectiveness of the pesticide. This is a fundamental point to which we will be obliged to return.

Polyakov has also shown that a single treatment of a cereal plant against rust (*Puccina graminis tritici*) could induce a 'pseudo-resistance.' As Polyakov concluded, 'One of the principal factors determining a plant's resistance to cryptogamic disease is its physiological state.'

Similarly, dithiocarbamates such as nabam applied to tomatoes lead to the development of 'grey mould' or *Botrytis*, as does maneb also (Cox and Hayslip, 1956). This 'unexpected' result, as the authors called it, alsooccurs with strawberries. When these are treated with zineb or with nabam-zinc sulphate, much more Botrytis develops than on the control plants. This led the authors to concentrate more on this side effect of dithiocarbamates: that is, on the increase in the plants' susceptibility to *Botrytis*.

Analysis of the leaves has shown that some build-up of zinc is the only appreciable change in the composition of the leaves. In older leaves, after treatment, the zinc content is 13 times higher than normal, and in young leaves, 3.5 times higher. As the authors (Cox and Wainfree, 1957) point out, it is well known that an excess of trace elements can cause imbalances in plant metabolism. In a precise comparison with the control plants, significant differences in protein contents were noted: plants treated with either zineb or with a mixture of nabam and zinc sulphate had a low protein level compared with controls (Table 3).

Table 3: Levels of protein nitrogen in strawberry leaves following various treatments, in ppm

Controls	Zineb	Nabam + zinc sulphate
3.18	3.08	3.06

In other words, the higher rate of proliferation of Botrytis on strawberries treated with zineb and nabam is due to a decrease in protein synthesis. This offers yet another confirmation of our theory.

Such effects were also confirmed by Baets (1962). He found that even though dithiocarbamates such as zineb and maneb had positive effects on yields of tobacco, they lowered the levels of proteins. It is logical to ask, therefore, to what extent such biochemical effects might be linked to the development of certain parasitic infections of tobacco, that have also been recurring in certain years.

Thorn and Ludwig (1962), likewise, have assessed the repercussions of dithiocarbamates on host plants. They found that, in comparison with controls, treated beetroot leaves showed significant increases in glutamine, valine and leucine, along with the presence of tyrosine, which did not exist in the control plants. In short, we see here an inhibition of protein synthesis, which is the overall process that explains how carbamates and dithiocarbamates increase the susceptibility of plants.

VII. The effects of other pesticides: new fungicides and herbicides

We will look in greater detail at these repercussions, which have just as much importance for the host plant, when we study the particular types of cultivation, such as fruit arboriculture and cereal crop-growing.

These effects appear to be no less drastic than those of the chemicals we have so far studied. To take only the case of herbicides, it is perfectly clear that 'Selectivity is far from perfect.' Collectif (1979) has made the same point: 'On the whole, one can say that all herbicides are toxic for all plants.'

Such toxicity generally manifests itself by an inhibition of protein synthesis leading to susceptibility to various attacks of parasites. This is the logical conclusion of our examination of the effects of the various pesticides studied in the present chapter, whose results we shall now summarise.

VIII. Conclusions

As we can see, the conclusions we have drawn from the present chapter reinforce those of the previous ones. These are:

1) Confirmation of the nature of the relations, nutritional in kind, between plants and their parasites.

2) Confirmation that maximum protein synthesis correlates with maximum resistance, and that a plant's susceptibility is linked with a physiological state in which protein breakdown predominates.

3) Confirmation of the concept of a 'parasitic complex' that leads, in a plant with inhibited protein synthesis, to an outbreak of parasitism that may eventually become 'multiple.'

4) Impacts of hormonal substances, as well as of various pesticides, on the plant's condition and resistance confirm the great importance of the plant's nutrition, especially the balance of the various major elements and trace elements.

5) These results show the relation between the physiological conditioning of the plant by pesticides and its susceptibility to parasites. They clearly show the confusion that can be made between parasite proliferation, due to positive effects on the parasite's biotic potential, and the supposed 'resistance' of the pathogenic agent to the pesticide. This confusion occurs above all, if the supposed 'resistance' manifests itself only under field conditions. In reality, what we then observe is only the phenomenon of inefficacy. We will return later to this extremely important point.

6) These impacts of different pesticides, which by now are well attested, inevitably raise new questions. For example:

   - the delayed effects of pesticides: it appears that these side effects are only noticed at the end of a certain period. This makes it difficult to assess the relationship: pesticides → effects on the plant's physiology → susceptibility to parasite attack.

   -

- in the same category, the probable existence of cumulative effects of pesticides, especially in perennial plants such as fruit trees and grapevines. This could explain the apparent 'suddenness' of certain outbreaks of disease or insect pests.

7) To sum up, the results obtained in this chapter lead to the following, almost self-evident conclusion: '*The toxic effects of a pesticide on the parasite whose destruction is sought will also affect the host plant, making it more rather than less susceptible.*'

These are the difficulties encountered when the sole perspective of pest control is a chemical war to destroy the parasite - that is to say, the chemical war as currently practiced.

Notes

1 The plants have been previously placed in the refrigerator.

2 It should be noted that although DDT and other chlorine-based insecticides are banned in France because of their persistence in the food chain, numerous chemical fungicides contain chlorine. It has been shown that chlorine reduces the synthesis of amino acids and promotes the decomposition of proteins.

PART TWO

Deficiencies and Parasitic Disease

Behind the appearance of epidemics, which intervene like additional agents of destruction, lie hidden deficiencies. Such deficiencies also create individuals less resistant to parasites, predators, climatic variations, and the pressures of migration.

Maurice Rose and P. Jore d'Arces
(*Évolution et Nutrition*, Paris, 1957)

Chapter 4

The Failures of Chemical Pesticides in Arboriculture and Viticulture: Bacterial Disease and Plant Physiology

I. Similarity of phytosanitary problems in arboriculture and viticulture

From this point on we shall look (from a rather different perspective) at therepeated failure of chemical pesticides to protect fruit trees and grapevines.

With an understanding of the proven impact of pesticides on the physiology and resistance of plants, and the nutritional relationship between the plant and the parasite, we can see that a failure of one pesticide or another indicates more than just ineffectiveness. It also involves nutritional stimulation of the virulence of the parasite through modifying the biochemistry of the host plant. This is not so-called 'resistance' to the specific pesticide.

This phenomenon has been demonstrated in the case of the spider mite. However, as we shall see, the same holds true for aphids. Pear phylloxera, which thrives after treatments based on various pesticides such as demeton-methyl, azinphos-methyl, vamidothion, and omethoate, offers a particularly significant demonstration of this. The case of the psyllids (leaf suckers) probably arises from similar factors.

We should also note the difficulty of controlling these infections if, while we seek to destroy the vectors, the aphicides cause them to multiply. We will see this demonstrated later, in a special chapter on the control of viral diseases. In short, we must avoid aggravating the 'parasitic complex' – (plant, virus, and vector) through chemical intervention that has a negative influence on the physiology of the plant.

The term 'parasitic complexes' signifies the simultaneous existence on the plant of several different parasites that can combine against it. These include both plant and animal pathogenic agents: bacterial, fungal, or viral diseases and insect pests. One example, just described, is the coexistence of viral diseases and vector aphids. Others that we will discuss in the following chapter, involve viral diseases, cryptogamic diseases, and insect pests.

These 'parasitic complexes' compel us to question their common cause and even the very concept of 'disease.' We will be better able to address this issue when we have accumulated and examined the experimental data and have discussed the validity of the various hypotheses.

II. The exacerbation of parasite activity: 'resistance' or multiplication?

The exacerbation of virulence is similar in grapevines and fruit trees, attacked by the same parasites. Similar problems are encountered with increases of *Oidium, Botrytis*, and proliferation of spider mites. There are increases, likewise, in the 'resistance' of aphids to pesticides. Bacterial and viral diseases are continually increasing and, according to some researchers, will become the 'diseases of the future.'

We hope to show that these developments arise from a common cause, namely the modification of the susceptibility of the plant in response to chemical products.

With regard to grapevines, the incidence of pesticide failure, usually referred to by pathologists as 'resistance,' is common to a number of infections including Oidium and Botrytis and mildew: they have all increased in certain regions. This process, familiar from other diseases, has been called the 'boomerang effect'.

In the context of 'parasitic complexes,' acariosis, black-rot, Brenner (fire disease)and bacterial necrosis have increased in virulence and a new disease, eutypiosis has appeared.

If these were all cases of 'resistance', we would observe these phenomena of 'resistance' in abandoned grapevines which had not been treated with the specific chemical. Moreover, 'ineffectiveness' is found in the absence of treatment for more than three years. Finally, 'resistance' manifests itself in some areas before pesticides have been applied.

Certain observers, have therefore hypothesised for example that resistant root-stock may already exist and may have been transferred from one field to another and also from one region to another!

We think it more likely, that the pesticide or pesticides, by affecting the physiology of the plant, sensitise it nutritionally to the parasite, which then becomes virulent. We have clearly demonstrated this for Oidium, Botrytis, and spider mites.

This pseudo-resistance occurs with 'fungicidal' chemicals that inhibit protein synthesis. They have just as much impact on the host plant as on the pathogenic fungus. We will return to this point later in the context of controlling fruit scab.

This modification of the 'site' of the action in resistant root-stocks consists in a disruption of the plant's metabolism, that makes it more susceptible to attack.

This would also explain why the effects of treatments become much more harmful when they are repeated, since their effects accumulate in the course of applications from one year to the next.

Of course, the harmful effects of synthetic pesticides combine with a number of factors, outlined in the first chapter.

III. Bacterial disease and the physiology of the plant

a) The repercussions of pesticide treatment in relation to bacterial diseases. The influence of disturbing changes in the nature of phytosanitary chemicals.

More than twenty years ago, a specialist on bacterial diseases could already draw the conclusion: 'Current conditions of arboriculture have offered bacterial diseases a combination of circumstances that encourage their development' (Ride, 1962).

What are these conditions? The author refers specifically to so-called 'intensive' arboriculture. He adds:

'The abandonment of copper in certain operations over the course of the last decade in favour of various synthetic chemicals has led to renewed outbreaks of *Pseudomonas syringae*. (Ride, 1962).

He adds: 'Several aspects of the bacterial disease *P. syringae* have been confused with damage caused by fungi or with disorders brought about by deficiency diseases.'

The author thus draws a connection between symptoms of deficiency and those of bacterial diseases. We will encounter the same observation in relation to other diseases, both viral and fungal. To quote the same author again: 'Bacterial diseases occur most often in orchards where copper salts have been entirely replaced by chemical organo-fungicides.' And finally: 'Only copper salts (and, in the particular case of infection by *Xanthomonas pruni*, zinc sulphate) still command respect in controlling bacterial disease.'

This sums up, in a general way, the causes of the growth of disease, as a result of changes in chemical pesticides.

What is involved here is an indirect process, nutritional in nature, by which various chemical pesticides cause proliferation of spider mites. The same thing happens with aphids. Moreover, various dithiocarbamates drastically aggravate the development of Oidium on grapevines, in comparison with cupric products and even pure water (Chaboussou et al., 1968)

Similar harmful effects of chemical pesticides were also recorded with bacterial diseases. As Davet pointed out:

> 'The biological imbalance can spread just as easily to bacteria. Deep and Young (1965) note a recurrence of crown gall (Agrobacterium tumefaciens) in cherry trees treated with captan and dichlone to protect them against Chaloropsis and Thislaviopeis. They believe this effect should be attributed to the suppression of fungi that were antagonists of the bacteria' (Davet, 1981).

We must therefore consider the possibility that, with the appearance of chemical pesticides, two processes have played a role leading to the same result - increasing susceptibility of fruit trees to disease. These processes are:

- the abandoning of use of minerals such as copper, whose action on the metabolism of the plant could be beneficial;
- repeated use of synthetic pesticides, which have harmful effects on the physiology of the plant (because protein synthesis is disrupted).

If these effects are cumulative, and if such effects accumulate year after year in perennial plants, this would explain their increased susceptibility to disease, as well as the apparently sudden onset of disease.

We shall now turn our attention to an examination of the effects of growth hormones in relation to development of 'fire blight.' This example likewise seems to confirm that it is above all the physiological state of the plant, brought about by the indirect and insidious influence of pesticides, which promotes the development of the disease.

b) The effects of growth substances and new farming techniques

In regard to fire blight in pear trees, researchers have noted: 'The growth hormones used, especially to set the flowers, have increased the tendency to a second flowering period. This technique should be either better controlled or abandoned'. [1]

This confirms that infection of the pear tree by bacteria arises primarily through the flowers and only secondarily through the green shoots. That is to say, it attacks those parts of the plant where breakdown of protein predominates. Such a condition could be aggravated by certain chemical pesticides and particularly by growth hormones, as we shall now see.

Let us look, for example, at the repercussions of 2,4-D as described by some physiologists. They write:

> 'As an example, we shall take treatment by the well-known synthetic phytohormone, 2,4-D (2.4 dichlorophenoxyacetic acid). A few minutes after application, the cells of the leaf stalks touched by the substance grow longer, triggering a visible change in the position of the leaves. In hot weather blight may appear, not specifically through the effect of the phytohormone. After a few hours the 2,4-D has spread throughout the whole plant, and stimulation of 'growth,' together with deformities, will become evident over the succeeding days. Proliferation of cells takes place in the different tissues - in the cambium, the cortex, and the phloem - in a manner similar to the calluses that develop at the base of cuttings. These proliferations result in compression of the softest tissues and obliteration of the woody vessels and riddled xylem vessels. Death occurs at this stage, accompanied by bacterial or fungal invasions which destroy the soft tissues, that are rich in nutritional substances' (Collectif, 1979).

Such is the picture of the herbicidal effect of a growth hormone. Note the nutritional nature of the relation between development of bacteria or pathogenic fungi and the biochemical condition of the plant, following the physiological disruptions caused by the hormone. Of course here the death of the weed is intentional. However, similar though less drastic repercussions occur on the crop itself. This means that 2,4-D ('a good example,' say the same authors, 'of the complex action which we still only partially understand, in spite of many decades of work') can cause disorders in cultivated plants, particularly if it has been applied in a carefully considered manner, as in treatments of fruit trees. This is why it would not be surprising if (through a nutritional effect, arising from an inhibition of protein synthesis) the hormones could in this way promote the proliferation of parasites such as bacteria in the case we are considering.

We could even say that growth hormones act in this case as 'indicators' or 'demonstrators' of the importance of the physiological state of the plant in relation to its resistance to various parasites, both plant and animal.

As we have seen, disruption of the physiology of the plant, especially of fruit trees or grapevines, can be caused both by growth hormones and numerous pesticides, especially chemical ones.

It is undoubtedly no accident that Collectif (1979) could make the following observation: 'As a consequence, perhaps, of overly intensive agriculture, bacterial diseases are particularly difficult to control'.

We believe it is essential to consider two basic intensive farming procedures:

(1) the use of new chemical pesticides (with more appearing every year), often in multiple applications, without a serious examination of their effects on the physiology and resistance of the plant that the chemicals are designed to protect.

(2) excessive use of chemical fertilisers, most especially nitrate fertilisers. We know that they generally have the effect of enriching the tissues with soluble nitrogen, which makes the plant susceptible to bacteria, as well as other diseases. We will now see this confirmed by a detailed study of this process.

IV. The mechanism of effects of different environmental factors on the development of bacterial diseases. Importance of levels of soluble nitrogen in tissues

a) The role of nitrogen in proliferation of bacteria

McNew and Spencer (1939), experimenting on sweet corn cultivated in nutrient solution, have shown that nitrogen provided to young plants is taken into the xylem vessels by the bacterium Pseudomonas stewarti. Therefore, it is the quantity of nitrogen in the sap that determines its suitability as a growth medium.

The same authors have also shown the great importance of nitrogen nutrition for the virulence of different bacterial strains. If some of these turn out to be less virulent, this is due to their inability to make use of inorganic nitrogen. In other words, the virulence of a strain of P. stewarti is closely linked to its ability to assimilate inorganic nitrogen. 'This bacterium,' the authors conclude, 'depends on inorganic nitrogen for its existence as a parasite.'

In addition, McNew and Spencer (*op. cit.*) referred to Nightingale's statement: 'The invasion of the cortical tissues in apple shoots by Erwinia amylovora - the fire blight bacterium – is correlated with the quantity of organic nitrogen present. This organic nitrogen, moreover, may be related to the quantity of inorganic nitrogen furnished to the tree in the form of fertilisers'

Similarly, Gallery and Walker (1940) experimented on the tomato bacteria *Pseudomonas solanacearum*, which grows in the xylem. Cultivating the bacteria in a nutrient solution, they showed that while the bacteria grows more quickly in an environment containing 2% dextrose rather than only 1%, they multiply even more rapidly in a solution containing peptone than in an environment devoid of organic nitrogen.

Finally, we also look at two series of studies of the resistance of cotton to proliferation of Xanthomonas malvacearum. These studies have shown the great importance of the nutrition of the host plant for the virulence of the bacteria.

Through a study of the effects of 28 compounds which reacted positively to ninhydrin (the NHPC's), Lipke (1968) showed that analine, amino-butyric acid, and potassium were associated with the degree of severity of the bacteria. Potassium probably intervenes because of its well-known influence on the protein synthesis process. A deficiency of potassium results in an increase of soluble nitrogen compounds, providing a nutritional substrate that allows the bacteria to multiply. In other words, the cotton is sensitised because its tissues are predominantly in a state of proteolysis. This fact agrees well with the process postulated by the theory of trophobiosis.

Similarly, working on the same issue in regard to various types of cotton, Verna and Singh (1974) concluded: 'The susceptibility of a cotton cultivar depends on several nutritional factors. While there does not seem to be a direct correlation between the sugar content and the degree of susceptibility of the cultivar, it seems quite likely that the sources of nitrogen would be the limiting factor, playing a greater role than the sources of carbon in controlling the growth of the pathogen.'

It is also interesting to note that while alanine and glutamic acid are shown by other authors to be essential for the nutrition of X. vesicatoria, their utilisation by the bacteria could be hindered in certain cases, and the pathogen can die of starvation in resistant cultivars. This occurs as a result of the presence of certain amino acids such as serine. This amino acid is in fact known to hinder the growth of X. malvacearum, since it opposes the utilisation of glutamic acid by the bacteria. In this case too, what we find is resistance by the plant to the parasite, due to nutritional deficiency.

b) The role of the balance of cationic elements in the nutrient solution

As we saw in the course of the second chapter, while the role of potassium is important, it is quite clear that it acts on the metabolism of the plant in balance with the other elements, especially calcium. We recall that the balance between protein synthesis and proteolysis depends particularly on the K/Ca ratio. This is confirmed by the relationship between bacteria and the plant.

Experimenting with bean plants infected with Erwinia carotovora, Tejerina et al. (1978) cultivated beans in various nutrient solutions.

They kept the nitrogen content identical, but varied the balance between K, Ca, and Mg. They were able to demonstrate an interaction of effects between the host plant and the pathogen. Certain combinations of N, K, Ca, and Mg were able to render the bean completely resistant to the bacteria.

Above all, as the same authors stressed, 'The absence of bacterial growth when plants were treated with 1.5% Ca might be due to changes in the substrate, leading to a systematic imbalance of enzyme activity.'

They also added: 'A high level of Ca is able to counterbalance the effect of nitrogen, since treatment with 2%N, 1%K, 1.5%Ca and 1%Mg does not lead to virulence in inoculated beans which are growing normally.'

Thus, as Forster and Echaldi (1975) have shown in their experiments on the resistance of tomatoes to Corynebacterium michiganense, a supplement of Ca in the nutrient solution influences the content of other cations as well as of calcium itself. Moreover this is the reason why the ratio Ca+Mg / K+Na better expresses the negative correlation of the level of disease in the plants, taken individually, than does the calcium content alone. As a rough approximation, one could use the inverse ratio K/Na, which we have previously mentioned. This is often taken by physiologists as the metabolic criterion to characterise the cationic balance of plant tissues (Crane and Stewart, 1962) (Fig. 1).

c) The role of the photoperiod

As we saw above, the effects of the photoperiod superimpose themselves upon the effects of the balance of cationic elements. Both of these influence the physiology and therefore the resistance of the plant. As we likewise showed in the course of the second chapter, bacterial diseases are no exception to this rule. Concerning bacterial diseases, Gallery and Walker (1950) note that:

- on long days sugars and starch increase, as does insoluble nitrogen (in other words, a metabolism where protein synthesis predominates)
- on short days there is a decrease in carbohydrate content and an accumulation of soluble nitrogen (in other words, a state where proteolysis predominates over protein synthesis).

These variations in the virulence of bacteria according to the time of year are explained perfectly by Gallery and Walker (op. cit.) in the following way: 'On short days the plants contain both soluble nitrogen and carbohydrates, which are available to the pathogen. The plants are comparable to an environment composed of minerals, dextrose, and peptone.' They add: 'This is why on short days we can expect an increase in the development of the disease.' (We recall that what is involved is the bacterial disease of tomatoes brought about by Pseudomonas solanacearum proliferating in the xylem.)

d) The influence of the nature of the stock

The fact that the stock exerts a clear influence on the susceptibility of the graft to bacterial diseases underlines the great importance of the plant's physiology to its susceptibility - or if you prefer, its resistance - to these diseases. Ride (1973), for instance, notes that Bartlett stock is 'sensitive,' while Old Home is moderately 'resistant.'

However, we should point out that such a way of speaking could lead to confusion. If in fact a stock is reputed to be 'sensitive,' this means in reality that it makes the graft susceptible to the disease. The opposite is the case when it confers 'resistance' on the graft.

This is, moreover, a general phenomenon, one that we shall study in the chapter on the influence of the stock on the graft's resistance. This sensitising of the pear tree to 'fire blight' by Bartlett stock is reminiscent of the 'sensitising' effect of Brampton stock on apricot trees and certain varieties of plum toward the viral disease Sharka. In this case too the parasite's nutrition determines its proliferation. As we shall see in the course of the following chapter, this confirms the overall importance of the nutritional relationship between plant and parasite, regardless of the type of parasite involved.

On the same subject of bacterial diseases, Duquesne and Gall (1975) write:

'Considering recent publications on the relation between nutrition and susceptibility of trees to bacterial diseases (studies by the American, H. English), it is more reasonable to suppose that the stock affects the nutrition of the graft by causing it to be more susceptible when the nutrition is defective.'

According to our concept of trophobiosis, the only cause of such defective nutrition is a deficiency in protein synthesis. Protein synthesis in turn, as we have just noted, is linked to the balance of cationic elements in the tissues. Specifically with the pear tree, Blanc-Aicard and Brossier (1962) have shown that this balance in the graft, particularly the ratio of divalent ions to monovalent ions, is determined by the nature of the stock. Such a balance could also serve in this case as a criterion for the metabolism of the graft. It corresponds above all with the level of soluble nitrogen, which confirms the great importance of nitrogen for susceptibility of plants to disease.

The Passe-Crassane variety is notoriously sensitive to blight. Might this not be due to the fact that, apart from a possible intrinsic susceptibility of the variety, it is usually grafted onto Cognassier, a weakening stock?

To conclude: bacterial diseases are no exception to the rule that susceptibility is linked to a metabolism where proteolysis predominates, offering an abundance of soluble nutritional substances, especially soluble nitrogen. How then can we explain the particularly harmful effects of chemical pesticides? This is the issue we will now address.

V. Control of bacterial diseases involves taking into account the nutritional relationships between bacteria and the host plant. Development of bacterial diseases and the nature of the relations between plant and bacteria

We have shown that bacterial disease also demonstrates that susceptibility is linked to an excess of proteolysis, where soluble substances accumulate that promote the nutrition of bacteria. Tejerin et al. (1978) already arrived at such a theory in the light of their studies of bacterial disease in carrots. As these authors themselves noted:

> 'Not only genetic factors but also ecological factors are capable of controlling the pathogenesis of microorganisms. In this way, some fertilisers applied to the soil can influence the establishment of microorganisms in their host, because of physiological and anatomical changes produced in the plant tissues.

> The value for the bacteria of these nutritional elements proceeding from the tissues could be an important parameter controlling how they develop and become established in the plant, since the relations between pathogen and host plant are characterised by an exchange of chemical substances.'

We now intend to study the extent to which treatments with chemical pesticides are responsible for the exacerbation of susceptibility to bacterial diseases.

VI. The effects of chemical pesticides in the plant-parasite relationship

a) Effects of cupric chemicals: the copper-nitrogen balance

We begin by looking at the case of copper, which stands out in a certain fashion by its clear but still mysterious action on bacterial diseases.

Demolon (1946), in discussing the progress made in control of fungal disorders by the use of Bordeaux Mixture, stated:

> 'If progress has been made (against mildew) in regard to the timing and means of application, this is still not enough to explain the specific role that copper seems to play. Once research has explained the mechanism of the behaviour of copper, this might open new paths that could take us further and improve a situation which, however satisfactory it may be, is so costly for viticulture.' (Demolon, 1946).

In regard to the process of the anticryptogamic action of copper or Bordeaux mixture, can we claim that we are much further along today than forty years ago? How can we explain, for example, the fact that copper products, although ineffective against bacteria, display a far from negligible 'prolonged positive effect' against bacterial diseases? (From an INVUFLEC brochure on withering of the peach tree.)

If copper seems to lack toxicity to the bacteria, is it perhaps functioning in another way? Logically, there can only be one way: an indirect action, which can only consist of a beneficial influence on the metabolism of the plant, by stimulating its resistance.

Does the micronutrient copper have the effect of causing a decrease in soluble nitrogen in the tissues? This would perhaps hold true for other elements found in Bordeaux mixture, such as sulphur and calcium.

In fact, there is a 'delicate' equilibrium between N and the micronutrient Cu which has been demonstrated by authors such as Primavesi et al. (1972). In studying the conditions for the development of Piricularia in rice, these authors found that copper deficiency creates an excess of nitrogen, which unleashes the disease. The ratio N/Cu changes from 35.0 in healthy rice to 54.7 in diseased rice, through a deficiency in copper.

Primavesi *et al.* think it is 'the mineral imbalance which makes the plant susceptible to attack' (*op. cit.*). As they conclude in their essay, 'It is evident that contamination of seed, soil and water with 'Blast' spores has no effect on the health of the plant if there is a good nutritional balance. Even in susceptible varieties the disease cannot survive.' They specifically state: 'One must nevertheless admit that levels of 18ppm of Mn and 2ppm of copper are sufficient in the soils studied.'

We should note that, concerning the process of contamination and virulence, the inoculum is thought to play a secondary role. Only the physiological condition of the plant proves to be important. In our view the effect of copper and manganese (another micronutrient to which we have alluded) would be to stimulate protein synthesis. This is precisely what these various analyses seem to confirm. On the other hand, as we shall now see, chemical pesticides present a very different picture.

b) Effects of chemical fertilisers and fungicides: interference with micronutrients

W. E. Ripper (7th British Weed Control Conference) has commented: 'A slight increase in nitrogen content in plants increases parasite attacks.'

The same author has noted that plants treated with carbamates and chemical insecticides and herbicides show an increase in the level of nitrogen in their tissues.

The same conclusion was reached at a symposium on integrated control in orchards (Bologna, 1972). There it was clearly stated: 'Total nitrogen levels increase after any treatment with chemical fungicides.'

These findings seem to explain clearly why diseases, both viral and bacterial, are unleashed whenever chemical pesticides interfere with protein synthesis.

However, one may perhaps go one step further, by recalling the processes of interference they provoke.

It has been clearly proven that fertilisers, above all chemical nitrogenous ones, such as are often used in high doses to increase yields, cause interference with certain micronutrients, especially copper. (This is the source of the hypoglycaemia and dramatic drop in fertility in animals fed on copper deficient fodder.) This seems to demonstrate the importance of the copper-nitrogen balance in the metabolism of the plant. However, could it be that other balances also play a role and are disrupted by chemical fertilisers or pesticides?

One of the principal characteristics of chemical pesticides, given their origin, is that they all contain nitrogen, and many are also chlorinated. Chlorine compounds are well known to reduce synthesis of proteins and to promote their decomposition. This would seem to explain how applications of these chemicals cause increases in soluble nitrogen and the susceptibility of plants to parasites. However, might not the inhibition of protein synthesis result from the creation of certain deficiencies, such as copper or boron?

Mrs. Huguet (personal correspondence) was able to show that applications of nitrogen to the soil in increasing doses lead to a decrease in boron in the leaves of cherry trees (Table 4).

Table 4: The relation between applications of nitrogen fertilisers to the soil and levels of boron in leaves (of cherry trees)

Applications of N (kg/ha)	Levels of boron (ppm) in cherry leaves
0	24
100	14
200	16
300	15

In conclusion, Mrs. Huguet writes: 'The decrease in levels of boron grows more pronounced over the course of years, the more nitrogen is fed to the tree... We seem to have reached a level of boron which is no longer optimal for the cherry tree. However, this does not yet attain the threshold of a true deficiency.'

It is hardly surprising, therefore, that treatments with chemical nitrate pesticides (that is, nearly all fungicides and insecticides), repeated numerous times each year, can also and perhaps more drastically through a cumulative effect (of the kind observed by Mrs. Huguet in fertilisers) result in boron deficiencies after several years. This would explain these outbreaks not only of bacterial diseases but also of viral ones, which continue to mystify plant pathologists and farmers.

It is clear, then, why many observers are concerned about possible 'confusion' between the symptoms of disease, especially bacterial and viral disease, and those symptoms that can be caused by deficiencies, especially of micronutrients. As an agronomist specialising in micronutrients wrote twenty years ago:

> 'It is not impossible that a relationship exists between deficiencies and certain diseases, especially bacterial and viral diseases. Either these diseases promote the symptoms of deficiencies or these deficiencies promote the symptoms of disease. For example, trees with boron or zinc deficiency could be more susceptible to viral or bacterial diseases' (Trocmé, 1964).

According to our trophobiosis theory, this deficiency follows the sequence:

Deficiency → inhibition of protein synthesis → accumulation of soluble substances (especially nitrogenous) → then, through a nutritional effect → increase of the biotic potential and proliferation of various parasites (notably bacteria, and also viruses as we shall see in the course of the next chapter).

c) How anticryptogamics and antibacterials function. New perspectives on control, by acting on the physiology of the host plant.

We have intentionally begun by focusing on bacterial diseases. We did this partly because they are notoriously difficult to control, and partly because of the existence of an enigma. This enigma is the effectiveness of a classic product, copper (particularly in Bordeaux Mixture). Does this pesticide indeed act 'on the surface,' as the classic conception has it? The bacterial diseases themselves proliferate deep within the tissues. What then is the source of this effectiveness? This is the question we have posed, and which we answer by citing the beneficial action of copper on the metabolism of the plant.

Several facts, moreover, would seem to confirm these indirect effects. In particular, some 'fungicides' known to be effective *in vitro* against *Erwinia amylovora* turn out to be, in contrast, wholly ineffective against 'fire blight' in orchards (Ride, 1973).

Such facts seem to raise a question about how anticryptogamics work. Are they really capable of acting on the surface? Does it even make sense to try to achieve this? Various investigators have doubted this mode of action. Primavesi *et al.* (1972) emphasise the ineffectiveness of fungicides against *Piricularia* in rice. Parmentier (1959), for his part, contests the effectiveness of fungicides against *Oidium* in cereals. Finally, Soenen (1975), as a result of long years of experiments on fungicides in fruit cultivation, emphasises: 'The most recently developed anticryptogamics are not necessarily fungicides, yet they interfere in one way or another with the biochemical relationship between the fungus and its host.'

Likewise, we note that such conclusions seem to agree well with those relating to the mode of action of certain new fungicides. For example, a study of the action of fosetyl Al (aluminium tris(ethyl phosphonate)) leads the author to raise questions about the reaction of the plant. He writes:

> 'All these facts obviously suggest that there is a mechanism of action located at the interface between host and parasite, and not a direct action (of the pesticide) on the parasites themselves' (Bompeix, 1981).

Such conclusions, already suggested in our previous work (Chaboussou, 1980), also agree with those relating to control of bacterial diseases, since the bacteria cannot be reached from the surface of the plant. The aim is no longer to try to destroy the parasite - in this case, bacteria - but rather to prevent it from attacking and multiplying. If it turns out that such a treatment is effective, this is - as with fosetyl Al, the subject of special study - because it involves 'the participation of the metabolism of the plant' (Clerjeau *et al.*, 1981). What we want to know is whether this arises through a positive effect on protein synthesis, in accordance with our theory.

Not only do all the previous findings seem to confirm this, but so do some results obtained by means of certain special treatments. One such is protection by inoculation with hypo-virulent strains. Ahl et al. (1980) performed a preliminary treatment on tobacco by injecting the roots with live but incompatible saprophytic bacteria, or even dead ones. They succeeded in protecting not only against a second bacterial attack but also against possible infection with tobacco mosaic virus (TMV). This underlines the idea of a 'parasitic complex' which we shall discuss further, and which is closely linked to the physiological condition of the plant.

Moreover, this shows that this resistance brought about by contamination with hypo-virulent strains is accompanied by the synthesis of new proteins. The quantity of proteins is proportionate, to a high degree, to resistance to TMV, just as it is to a second bacterial attack.

Similarly, Staron *et al.* (1970) showed that the rapid recovery of peppers and tobacco affected by Stolbur, when treated with tetracyclines and their derivatives, is accompanied by an increase of proteins in the tissues. Such healing as in the foregoing case, through stimulation of protein synthesis, seems clearly confirmed by the fact that levels of proteins in diseased plants dropped by more than 50% at the leaf blades.

Finally, it is important to find out whether, by certain methods and precautions, it might be possible to stimulate protein synthesis without harmful effects, especially at particularly sensitive periods of the annual cycle of the plant. This would result in what we could call a nutritional type of 'deterrent' to the parasite.

VII. Conclusions: new prospects of control

Generally speaking, the failure of pesticides confirms once again the nutritional relationships between the plant and its parasites. This has been confirmed by detailed analysis of the relations between bacterial diseases and the host plant. This analysis also shows that susceptibility to disease is linked to proteolysis and, in particular, to the soluble nitrogen content of the tissues.

This also shows that the great majority of chemical pesticides, especially with multiple applications, act as inhibitors of protein synthesis. They render crops susceptible to various parasites, with viruses and bacterial diseases forming no exception to the rule.

To an overwhelming degree, chemical pesticides are based on nitrogen, from which they are synthesised. They are also chlorinated. This explains why they interfere with certain micronutrients, such as copper and boron.

This perspective is based on data from field trials: for instance, interference with copper by nitrate fertilisers. There is also the fact that, according to our theory of trophobiosis, the analogy between symptoms of deficiency and symptoms of disease, especially bacterial and viral disease, can be explained simply as cause and effect.

We shall return to this important question about the relationship between disease and deficiencies, particularly micronutrient deficiencies. In the first place, there is the problem of assessing how pesticides and especially fungicides actually function. In our opinion, for example, the effects of mineral products such as copper or zinc on bacterial diseases, as well as certain other diseases, can be explained by the reaction of the host plant. In other words, this would apparently involve a positive effect on protein synthesis.

This leads us to ask to what extent this process of effectiveness may be a general one, on the assumption that it occurs through a stimulation of protein synthesis.

In any case, in the present state of our knowledge of the plant-parasite and plant-pesticide relationships, we are able to make two points (taking account of the preceding findings, and from the perspective of sensible control):

1) The very objectives of control efforts are questionable. It is useless to try to destroy bacteria through toxic procedures. The toxicity of chemical pesticides affects the plant itself.

Such instances of 'poisoning', some of which appear to be benign, have the physiological effect of inhibiting protein synthesis. The first stage may be interference with elements such as copper and boron. The failure of these new chemical fungicides (anilids and others) can be explained by an initial step in metabolism that relates to deficiencies. This would also explain, at the same time, the similarity between the symptoms of deficiencies and those of disease.

The first measure that should be taken is to prohibit the use of all chemical pesticides (fungicides, insecticides, acaricides, and growth hormones) where the possibly harmful effects of their nitrogen and chlorine contents are not known.

This includes, in fact, all synthetic chemical pesticides, all the more so because we do not know the cumulative effect of these pesticides when used in multiple treatments throughout the season, particularly for perennial plants.

2) If there is a fairly direct relationship between the disease and one or two deficiencies, we should be able to detect possible deficiencies in order to correct them. This can be achieved by appropriate analyses, carried out especially during susceptible periods in the annual evolutionary cycle, such as the flowering period.

On this subject, we have some reliable figures on which to base our hypothesis. These relate especially to micronutrients such as boron, whose great importance we have seen, above all in its relation to nitrogen. Just as the deficiency can come from the soil's original composition, it can also arise from lack of availability due to the phenomena of interference by nitrogenous fertilisers, or through a deficiency in organic matter.

Apart from micronutrient deficiencies, we should also consider the balance of cationic elements, which as we have seen are also quite important. One criterion that seems fundamental is the K/Ca ratio. It is well known that the relationship of calcium to other micronutrients is very close. Boron is known to keep calcium in a soluble form, which is easily assimilated and therefore physiologically active.

On the other hand, just as with cationic elements, we can grasp the relations between the elements themselves. As D. Bertrand pointed out, boron is only active in combination with magnesium, manganese, and molybdenum. This appears to explain the attraction and effectiveness of the 'micronutrient complexes' advanced by some companies that produce phytosanitary products.

In grapevines with a boron deficiency, for instance, the practice of using foliar sprays with a micronutrient base has led, in two years, to an increase in the ratio of B/Zn from 11 to 47, along with disappearance of the phenomenon of failure to set fruit.

Moreover, it is normal to find (whether with sunflowers or with grapevines) that the beneficial effects of boron begin at the flowering stage. This is in fact, as we have seen, the sensitive period of the cycle where proteolysis predominates. This proteolysis can to a degree be arrested by applications of boron to the tissues.[2]

In the same way, this could explain the beneficial effects of cupric sprays in late autumn against various cryptogramic as well as bacterial diseases.

Finally, it seems that the results of research into correction of deficiencies, with the aim of stimulating protein synthesis and obtaining maximum resistance to various parasites, conform with a biochemical state that also characterises varietal resistance – in other words, the genetic effect.

Thus, in the case of apple scab, Williams and Boone (1963) recorded: 'The variety called Cortland, which is susceptible to all strains of Venturia inaequalis, contains asparagine levels of 1.969 (an amino acid necessary for the growth of pathogenic fungi), while the resistant variety, McIntosh, only has levels of 0.756.' This would seem to confirm clearly that, in this case too, resistance and a high level of protein synthesis go together.

However, we should also remember the statement: 'The gene can only express itself in relation to other factors in the environment.' We have seen this confirmed by climate, stock, and the physiological cycle of the plant, as well as with the soil, fertilisation (which we shall return to later), and finally the effects of pesticides. That is to say, genetic factors are only one element and their action can be thwarted by a whole series of others, most importantly by the effects of chemical pesticides.

It is conceivable that such factors, especially chemical treatments, could counteract varietal resistance, quite apart from effects on the plant's physiology. In contrast, control based on adequate 'nutritional conditioning' can only be advantageous. Through it, one would attempt to recreate a 'natural' state of physiological resistance. Results achieved in antifungal control by means of 'nutritional sprays' further encourage us to continue with this line of study for controlling bacterial and viral diseases. It becomes more and more clear that this is the only course of conduct that makes sense.

Notes

1 Chaboussou does not give a source for this quotation

2 F. Chaboussou unpublished results

Chapter 5

Plants and Viral Diseases

I. Environmental factors and viral diseases

Like bacterial and fungal diseases, viral diseases are again on the increase. Marrou (1970), for instance, wrote of diseases of market garden plants: 'Since market gardeners have become familiar with the main cryptogamic parasites and how to control them with effective fungicides, viral diseases have taken on a more important role. These diseases are feared because their origin seems mysterious and their development insidious.'

In the light of everything said so far, we may justifiably have certain reservations about the impacts of the new synthetic fungicides. To cite only one example, more than ten inorganic chemical anticryptogamics have proven ineffective against Phytophthora cactorum in strawberries (Nourrisseau, 1970). As we have seen above, this raises the question of the causal mechanism behind these failures. In other words, what are the relations between plant, pesticide, and parasite? At the end of the chapter we shall deal specifically with the issue of the relationship between plant and pesticide. As we have seen above, this is extremely important, although classical phytology has hardly even looked at it.

Furthermore, it has also been claimed as a characteristic of viral diseases that they are generalised, incurable, transmitted through the grafting, and show variations in the expression of their symptoms. Finally, it is claimed that plants have no defence mechanism against viral diseases.

We have tried to show that the inhibition of the pathogenic agent has its origin in the plant's deficiency in the nutritional elements needed for the pathogen's growth and reproduction. We shall now see how, in the case of viral diseases likewise, virologists dispute the presence of phenols in the tissues as a possible factor in 'eliciting' the virus.

We should also point out that variation in symptoms for the same viral disease clearly shows that the physiological state of the plant, and therefore the nature of the environment, plays a role. With regard to the effects of techniques of 'protection,' Limasset and Cairaschi (1941) could justifiably comment:

> 'When a strain of disease triggers off a protective mechanism in the plant against that disease (Tobacco ring spot) after a clinical cure, this represents an acquired tolerance of the plant toward the disease. The plant is cured because it no longer reacts to the infection. It is not the presence of the virus which constitutes the disease but the violent reaction of the infected cells.'

Symptoms are the expression of the plant's reaction. Their variability, therefore, signifies that a plant can in fact 'resist' the viral infection partially or even totally, depending on its conditioning by environmental factors.

This justifies the idea that in treating viral disease, 'We act upon the host, and not against the disease itself.' In the course of this chapter we will see new confirmations of this, which also give high priority to the relations between plant and virus. Three possible outcomes have been proposed by Lepine (1973):

1) 'the most frequently posed hypothesis: the alien nucleic acid imposes its own mode of production on the cell so that the cell, for the short time it continues to live, only produces viral proteins.
2) 'the cell, in refusing the alien nucleoprotein, metabolises it for the production of its own nucleoproteins. It will survive, it recovers, and this assimilation of an exogenous element triggers off the immunity of its tissues upon which the plant's acquired resistance is based;
3) 'without allowing itself to be invaded by the virus, yet unable to assimilate the elements completely, the cell integrates part of the nucleic acid of the virus into its own structures. In this way, the viral infection remains latent and unperceived, whether in vegetative form or in the form of an exogenous addition to the hereditary material or provirus.'

These considerations, applicable to viruses in animals or humans, can be perfectly extrapolated to viruses in plants. They underline the major importance of the factors behind the plant's receptiveness to the virus.

In the course of this chapter, we shall encounter the parasitic complexes to which we have already alluded: that is, the 'association' of both fungal and viral diseases on the same plant. What determines the 'sequence' of these infections? Is it first a viral disease, then a fungal one? Or could there be at the outset a factor common to both?

We should point out here that the heading of 'viral diseases' now includes several diseases related to specific microorganisms. Thanks to the latest investigative methods (using scanning electron microscopes), researchers have been able to discover various organisms, with names such as mycoplasmas, spiroplasms, rickettsias, and viroids. We note, moreover, that these enormous magnifications also have some drawbacks. At such a level of magnification, some researchers complain of difficulties in identifying the real or supposed pathogenic agent. For the agent/disease relationship still has to be proven.

This is what leads us to make a closer study of the effects of the nutrition of plants through fertilisation (that is, the composition of the soil) and, likewise, in relation to the genetic nature of the plant.

II. Inorganic fertilisation and the virulence of the virus

As Bawden and Kassanis (1950) note, 'Viruses seem to be parasites by nature. So one would conclude, therefore, that their ability to reproduce must be affected by changes produced in the metabolism of the host plant caused by variations in nutrition.'

At the time they were writing, the only study they could refer to addressed the way in which nitrogen tended to affect the virulence of tobacco mosaic virus.

These authors carried out a series of experiments on the tobacco mosaic virus and the potato X virus, which we shall return to later. As they concluded, 'The experiments concerning inorganic nutrition in plants can hardly hope to yield information about the mechanism of proliferation of the virus.'

In studying the effects of both organic and inorganic fertilisation on the virulence of the virus, we shall attempt to work out the reproductive mechanism, which the authors seem to link too closely to virulence.

We shall begin by examining the effects of inorganic fertilisers: first, because these have received the most study; second, because they have most often been blamed for making plants susceptible to viral diseases. This is particularly true of nitrogen.

a) Repercussions of nitrogen fertilisation

Martin (1977) bases his analysis of the effects of nitrate fertilisation on a number of different works, including those of Spencer (1941-1942), Bawden and Kassanis (op. cit.), Forster (1957), Chei et al. (1952) and Tomaru (1967).

As Martin (op. cit.) notes, 'Relatively little research has been done on the role of inorganic nutrition in viral diseases.' He suggests that the reason for this lies in the almost exclusively nucleoproteic nature of these parasites, which has led the great majority of researchers to examine the relationship between the virus and competition with the normal proteins and nucleic acids of the host plant. He states:

> 'The biosynthesis of the virus depends on the materials already synthesised by the healthy cell, with the aid of the enzymes of the host plant. The importance of the role of the mineral elements in these healthy mechanisms became secondary in the study of the infection as such.'

We may now query whether this is the proper way to pose the question. Martin notes apparent contradictions in the effects of nitrogen fertilisers,, pointing to the close relationship which seems to exist between nutrition and the appearance of symptoms. Thus, intensive use of nitrogen fertilisers leads to an increase in the symptoms of the YD onion virus. Yet the opposite is true in the case of beetroot yellows virus and in potato leaf roll. Some varieties of beetroot do not display any symptoms in spite of liberal application of nitrogen fertilisers, even though 100% of the plants may be infected.

These results give us an additional reason for carefully distinguishing the proliferation of the virus from the first evidence of damage, in other words, the symptoms.

With nitrogen fertilisers, generally speaking, it would seem that an excess of nitrogen could be beneficial for the growth of the plant, but at the same time for the proliferation of the virus. (The same holds true for other diseases.) The symptoms appear when the nutritional competition between virus and plant becomes detrimental to the plant. This is the perspective, it seems, from which we should view the ideas of Cervenka (1966), as reported by Rasocha (1973):

> 'Nutrition has a considerable effect on the state of health of the plant. Nutrition, and particularly the ratios of N, P and K, may affect not only the symptoms but also the resistance of the plant to disease, the resistance caused by age, and in particular the distribution of infection in the plant.'

Böning (1967) points out that isolated and high inputs of nitrogen fertiliser serve to prolong the vegetative stage of the potato, which leads to the appearance of symptoms of viral disease.

Nitrogen and other forms of fertiliser do not affect only the nutrition of the plant. They also indirectly affects the composition and life of the soil. We are referring here to effects on microorganisms and to the phenomenon of interference, particularlyregarding micronutrients, to which we shall return later on.

b) Repercussions of phosphate fertilisers

In contrast to nitrate fertilisers, alkaline phosphate fertilisers have a beneficial effect against viral diseases. By promoting maturity, they bring forward the stage of resistance in the plant brought about by age. This is a factor of resistance that we have already encountered in fungal diseases. Viral diseases, therefore, come under the general heading of factors of resistance.

Thus, referring to the resistance of potatoes to the X virus, Schepers *et al.* (1977) note that supplementary doses of phosphate show the opposite effect from that of nitrogen, which makes the plant susceptible to the virus.

The results of Rasocha (1973) on the resistance of potatoes to the Y virus seem clearly to confirm previous results. We think it worthwhile to provide some details here. These studies on the effects of NPK fertilisation were carried out under glass. They involved eight varieties and were carried out over three years, from 1966 to 1969. Their conclusions were the following:

'Considerable differences were noticed in the state of health of the plants, which received various nutritional treatments. As an average for the period 1966-1969, the greatest number of healthy plants was found among those receiving the 'nutrient free' treatment, or the treatment with extra P_2O_5 (Table 5).

Table 5: Percentage of healthy potato plants (virus free), according to type of fertilisation

Treatment	Percentage of healthy plants
Nutrient free	40.9%
Surplus P_20_5	32.2%
K_20 deficiency	25.1%
K_20 supplement	23.8%
Standard NPK sample	20.7%
N in excess	16.7%
P in excess	16.4%

Rasocha (*op. cit.*) also confirms that the health of the potato is affected to a considerable degree by the timing of the infection. The later this takes place, the fewer the tubers affected by the Y virus. Phosphates act in proportion to the maturity of the plant, and consequently at the stage where age related resistance intervenes. It is because of the plant's biochemical state (the balance of proteic nitrogen and soluble nitrogen) that excessive nitrogen fertilisation or an insufficient amount of phosphate causes maximum levels of disease. Strong and isolated treatments with nitrogenous fertilisers, which prolong the vegetative period, retard maturity and the resistance that is linked to tissue age.

In experiments on the potato X virus, Bawden and Kassanis (op. cit.) showed the following:

- P simultaneously stimulates both plant growth and concentration of the virus.
- K also increases plant growth but, in contrast, reduces concentration of the virus.

These results agree with each other as far as the potato is concerned, if one is careful to distinguish between the concentration of the virus and the health of the plant. It seems therefore clearly established - and of vital importance to agronomy - that the level of concentration of the virus cannot always be a valid criterion of its virulence.

Other studies were carried out on tobacco. Bawden and Kassanis (op. cit.) have studied the combined effects of NPK, since these major nutrients can only work in balance with each other, and not on their own. These authors also found that P increases both plant growth and the concentration of the virus in the sap. Furthermore, under the conditions of these experiments, N only stimulates plant growth when P is present. Similarly, N increases concentration of the virus in the sap, but only in the presence of P.

However, they add: 'Plant growth and the level of concentration of the virus are not in complete accord. While K has a positive effect on plant growth, it has a negative effect on the concentration of the virus.' Their final conclusions are as follows:

> 'It seems that in inoculated leaves, as well as in those which are systematically infected, the virus multiplies more quickly and extensively when the nutrition of the host plant is able to produce a vigorous plant than when growth is slow.'

This raises the question of the physiological definition of vigour. (We shall return to this later.)

Bawden and Kassanis (op. cit.) conclude from their experiments that their results do not seem to confirm those of Spencer (op. cit.). According to Spencer, nitrogen specifically stimulates the multiplication of the virus, and its effect is independent of plant growth. Bawden and Kassanis admit, moreover, that they cannot give any explanation for these differences. They observe, nevertheless, that they were experimenting on tobacco (Nicotiana glutinosa), while Spencer was working on beans (Golden Cluster variety).

It is highly likely, they remark, that nitrogen affects the infection in one host plant but not in another. It seems that the metabolism of nitrogen in tobacco (Solanaceae) could be fundamentally different from that in a legume such as the bean on which Spencer worked.

The latter suggests, moreover, that the process of multiplication of the virus is linked more closely to the simple forms of nitrate than to those where nitrogen has already been synthesised in the normal proteins.

Undoubtedly we will have a clearer picture as we study the effects of potassium more closely. We have already pointed out some of its physiological effects, above all in relation to the other major elements.

c) Repercussions of potassium fertilisation

Generally speaking, we can say that nitrogen, especially when applied excessively in fertilisation, tends to make plants more susceptible to viral diseases (just as is true for fungal and bacterial diseases). On the other hand, we find the opposite is true for potassium fertilisation.

In the case of tobacco, for example, Dufrenoy (1936) notes a greater susceptibility in plants cultivated in nutrient solutions which were relatively deficient in K (Fig. 3).

According to Rasocha (op. cit.), who maintains that nutrition of a plant has a considerable effect on its health, the NPK balance affects at one and the same time:

- the symptoms caused by the Y virus in potatoes.
- resistance to fungal diseases.
- the spread of the virus in the plant.

Looking especially at potassium, Rasocha notes that its effects vary according to the form of the potassium fertiliser. In every way, he sees potassium as the most effective of all the elements in controlling disease.

Working on beetroot yellows virus, Warcholowa (1968) studied the effects of different levels of K both on the disease and on the level of infection by the vector Myzus persicae. In the case of K deficiency, the symptoms of the disease both appear earlier and are more serious. They are, accordingly, at their weakest in plants that have had the largest dose of potassium.

Effects on the biochemical composition of the tissues are equally significant. While yellows virus increases the content in reducing sugars, potassium counteracts this effect of the infection, reducing the plant's sugar loss, while yield increases in parallel to the levels of potassium in the nutritive solution.

We see here the beneficial effects of potassium that stem from its activity in protein synthesis, mediated by its influence on sugar and phosphate metabolism. We know the important role played by phosphorus in the phenomena of potassium fixation. This gives it a fundamental role in the synthesis and structure of proteins.

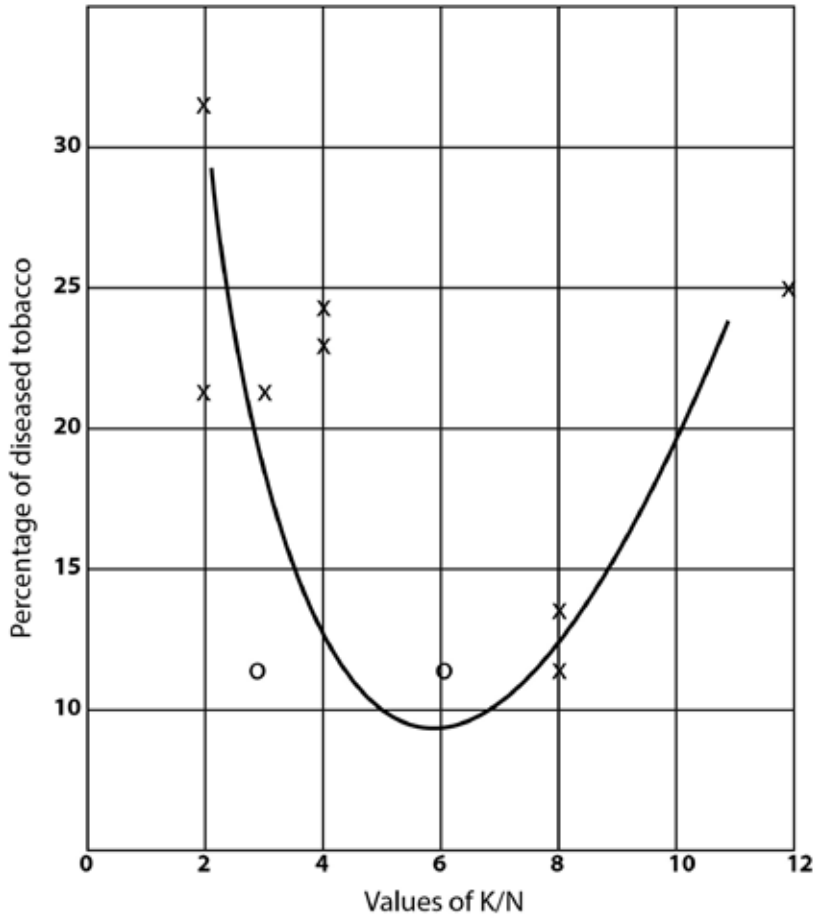

Fig 3: Percentage of tobacco affected by spotted wilt, according to the K/N ratio. (After Dufrenoy, 'Spotted-Wilt,' Ann. Epiphy., 1937.)

Potassium is linked to the migration of amino acids from the place where they are formed to the sites where they are used.

The analysis of the repercussions of potassium, according to its balance with the other elements, seems to show that its beneficial activity is linked to an elevation of the level of protein synthesis that it is able to produce in the plant.

This could explain the apparent contradictions that have arisen from some experiments. For example, when Bawden and Kassanis (op. cit.) did not obtain the same results when experimenting on tobacco and potatoes (two types of Solanaceae) as did Spencer (op. cit.) in experiments on the bean, it may be that some legumes have a completely different metabolism from that of the Solanaceae, especially with respect to nitrogen. Dufrenoy (1936) comments that while Solanaceae are sulfur plants, legumes such as alfalfa are chlorine plants.

Nguyen et al. (1972) studied this subject specifically, working on alfalfa. They also stressed that a good balance of NPK leads to:

- a decrease in the amino acid content of the tissues,
- an acceleration of their incorporation into proteins,
- an increase in yields.

These authors note in passing the importance of potassium for optimum protein synthesis. In alfalfa, it is the interaction of phosphorus and potassium that is especially marked. This shows the particular effect of nitrogen in legumes. It often happens that the same type of fertilisation can have very different effects on the metabolism of plants which are botanically distinct, and therefore on their resistance to disease.

With alfalfa, while nitrogen fertilisers obviously have little effect (which is logical enough because this legume is nitrogen-fixing), phosphorus causes an increase in serine and alanine in the tissues. The ratio of the following pairs of substances also increases:

$$\frac{\text{aspartic acid + asparagine}}{\text{glutamic acid + glutamine}}$$

In this case, as in the others, a potassium deficiency leads to decomposition of proteins, and therefore to increased susceptibility of the plant to various diseases, including viral diseases.

Still on the subject of the effects of potassium fertilisers, Russell (1972) experimented with two varieties of beetroot: Sharpes' Klein, sensitive to the BYV virus, and Maris Vanguard, a more tolerant variety. These two varieties were cultivated, using the same compost, with five applications of fertiliser. The results were as follows (Table 6):

Table 6: Harvest losses in beetroot according to the type of fertilisation

Treatment	Harvest losses in the roots due to the BYV virus	
	Sharpes's Klein	Maris Vanguard
No fertiliser	39.6	27.8
+ NaCl	42.7	47.1
+ KCl	35.9	36.4
$+NO_3Na$	48.0	34.1
$+NO_3K$	55.5	28.9

The authors came to the following conclusions:

> 'In Sharpes' Klein, the addition of nitrogen in the form of sodium nitrate or potassium nitrate increases losses due to BYV infection. Conversely, adding nitrogen does not affect the tolerance of Maris Vanguard to the virus. Chlorine ions seem to lower tolerance in Maris Vanguard but not in Sharpes' Klein.'

These varying results raise the issue of the 'availability' of the elements. This availability is determined not only by their balance in the fertiliser, but also by the regulatory systems of growth and development in the plant which govern the formation of proteins and protoplasm. This explains the importance of the other factors of nutrition. These are the micronutrients, growth substances, pesticides, herbicides, and finally, organic fertilisers.

d) Repercussions of micronutirents

Regarding bacterial diseases, we have seen over the course of the previous chapter that researchers sometimes associate two different things as part of the same process: deficiencies in micronutrients on the one hand, and the activation of viral diseases (whether bacterial or fungal) on the other. Trocme (1964) posed the question in terms of an alternative: a deficiency preceding and causing disease, or disease causing a deficiency.

We have responded by choosing the first hypothesis. We have based this choice on our theory of trophobiosis, according to which all deficiencies, including water deficiency, lead to a deficiency of protein synthesis. This creates an increase of soluble substances in the tissues and ultimately leads, by a nutritional route, to an outbreak of the pathogenic agent. The point, therefore, is to know whether viral diseases and other mycoplasmoses obey the same rule.

We must pay special attention to the convergence of symptoms between those of the deficiencies and those of the diseases. In the case of the grapevine, some observers who have paid close attention to the reactions of vineyards to fertilisation were able to conclude in the course of their trials:

> 'The vineyard, moreover, shows examples of numerous attacks of viral diseases. The effects they cause (discoloration, curling, changes in leaf form) have made the symptoms of deficiency more difficult to identify' (Delas and Molot, 1967).

This is hardly surprising if the deficiencies are the origin of viral and related diseases. There are many authors who have drawn parallels between the existence of certain deficiencies and the triggering of different diseases or even outbreaks of insect pests. As early as 1932, Labrousse noted that boron deficiency made barley more susceptible to attacks of leaf stripe, as well as mildew. Experts frequently note that beetroot plants can suffer from a boron deficiency, without recalling that this deficiency leads to a fungal infection, Phoma betae,[1] which disappears when boron is added to the soil. What we see here is how correction of a deficiency can cure a disease.

Another example is that of grape lead (Stereum), which can also be cured by applications of boron (Branas and Bernon, 1954).

Maromba disease, occurring in the Douro valley in Portugal, was due to boron deficiency and was effectively halted by the application of this element. It is also interesting, while looking at the grapevine, to compare the symptoms of disease-like deficiencies to the symptoms of the real disease. Drawing on the collective work , '*Viral Diseases and Similar Afflictions of the Grapevine*', we have drawn up in Table 7 a list of the symptoms of the deficiencies, along with the diseases with similar symptoms.

Table 7: 'Convergence' of the symptoms of deficiencies and those of viral diseases and other infections of the grapevine

Diseases	Deficiencies	Other damage	Other diseases with similar symptoms
Fan leaf	Boron Zinc	Blossom drop and formation of seedless berries	Aucuba mosaic Flavescence doree Bois noir
Leaf roll	Boron Magnesium Potassium	Mites	Flavescence doree
Corky bark	Boron		Bacterial necrosis (Bacterial blight) Excoriosis Maladie de bois strie
Flavescence doree	Boron		Pierce disease Leaf roll
Veinal necrosis	Boron		Esca disease

However, this is more than just a simple convergence between the symptoms of deficiency and those of disease. A cause and effect relationship emerges from this confrontation. We have cited some examples of diseases cured by a correction of deficiencies above and return to this in the third part of this work. We shall now concentrate on the relationships between the micronutrients and viral diseases and analyse various works on this subject.

Lockard and Asomaning (1965) studied nutrition in the cacao (Theobroma cacao L.) in relation to the 'swollen shoot virus.' They observed that certain swellings in the shoots resembled the blistering caused by the virus found in plants with copper deficiencies. From this they concluded that there is a possible interaction in cacao between nutrition and virus.

Applications of iron, zinc, or a combination of iron, zinc, and manganese were shown to correct the symptoms caused by certain viruses. However, the researchers noticed that copper is required for proliferation of the virus. There may therefore be nutritional competition for some of the metabolic products of the cacao plant between the 'swollen shoot' virus and the plant, resulting in symptoms of copper deficiency.

Lockard and Asomaning (op. cit.) carried out two types of experiments, one based on the effects of deficiencies or excesses of major elements, the other involving micro-elements. Each treatment was applied both to healthy plants and to plants infected with the specific virus.

After two months of cultivation, plants deficient in boron, zinc, and iron were on the point of disappearing. Since the experiment was thereby invalidated, the experimenters had to add a small quantity of micronutrients to the nutrient solution.

In the course of these experiments, the authors were also able to observe a worsening of the symptoms of the virus through a lack of light. A drastic reduction in growth accompanied these symptoms.

In these experiments with micronutrients, the effect of the virus is strongest in the leaves. There is a decrease in levels of Fe, Mn, Zn, B, Mo, and Na, and an increase in Al. In the roots, the virus leads to a reduction in levels of Fe, Al, and Na and an increase in the level of Mn.

These effects interfere with so-called 'nutritional' treatments that are superimposed on those of the micronutrients. This verifies some of the interference with levels of nitrogen in the stems and with P and Mn in the leaves and stalks.

The studies by Russell (op. cit.), of which we have already spoken, were carried out on viral diseases of beetroot. They are also related to fertilisation and the behavior of aphid vectors, to which we shall return. Russell notes:

> 'The manifestation of resistance to aphids and to inoculation with virus in sugar beet could be altered by various factors. These include the concentrations of major elements or micronutrients in the soil, along with the factors that affect the concentrations of sugars and amino acids in the leaves.'

Under glass, says Russell, 'the absorption of certain nutritional elements by the roots of the sugar beet could profoundly alter the manifestation of resistance to aphids and to inoculation with the virus.' The situation is the same with certain micronutrients:

- for example, while applications of Li, Zn, or Ni salts prompt the appearance of aphids on the leaves, boron actually discourages this;
- reproduction of aphids is accelerated by Ni and Sn salts;
- the transmission of BYV (beet yellows virus) is increased by Li and boron, and reduced by Cu, Zn, and tin.

Yet micronutrients which seem to encourage the establishment of aphids do not always promote transmission of the virus. Plants which have received treatments of cobalt nitrate are found, 48 hours after infestation, to have significantly more aphids per plant than those treated with iron. However, fewer plants treated with cobalt become infected with the virus.

Such results, the author observes, clearly confirm that resistance to the establishment of aphids and resistance to inoculation by the virus are not necessarily related. He adds: 'The concentrations of micronutrients in the leaves may affect the establishment both directly, by altering the composition of the diet, and indirectly, by changing the metabolism of the plant.' We see scant difference between these two suggestions, which both relate to a modification of the physiology of the plant through the action of micronutrients.

Russell (op. cit.) notes in passing a fact well known to agronomists, that the concentration of the micronutrients in the soil varies enormously from place to place. In the field, therefore, this leads to important variations in resistance to the virus. We shall return to these problems in the chapter on controlling viral diseases in cereals.

Russell's work, together with that of Lockard and Asomaning, shows the great importance, for resistance to viral disease and to other diseases, of the micronutrients' effects on the physiology of the host plant. These studies entirely confirm our position on the relationship between plant and parasite. This is primarily nutritional in nature, and dietary deficiencies constitute a fundamental factor in the susceptibility of plants to viral and other diseases.

In the course of the previous chapter, we have seen that pesticides used to treat fruit trees, or chemical nitrogen fertilisers incorporated into the soil, may lead to interference phenomena, especially for micronutrients. This could explain how such treatments assist the unleashing and growth of bacterial and fungal diseases. Given the preceding findings, one might assume that the same holds true for viral diseases. But before we address this aspect of the problem, we shall look at the effects of organic fertilisers.)

III. Organic fertilisation and viral diseases

Howard (1940) cites a certain number of ancient agricultural practices in India that helped improve the resistance of plants to disease. He devotes a chapter of *'An Agricultural Testament'*, to what he calls 'The Retreat of the Crop and the Animal before the Parasite.'[2] This ex-director of the Institute of Crop Protection at Indore, who was an agricultural advisor to the Indian federal authorities, acknowledges humbly that he is a pupil of the peasants in the Pusa region. As he observes, 'The products cultivated by the peasants were remarkably free from disease of all kinds,' in particular from the viruses with which we are currently preoccupied.

Looking particularly at tobacco, he notes:

> 'When care was devoted to the details of growing tobacco seed, to the raising of the seedlings in the nurseries, to transplanting and general soil management, this virus disease disappeared altogether.'[3]

Howard insisted particularly on the importance of the way the soil is cultivated. He noticed that attacks by certain caterpillars were localised in fields that had been flooded. He writes: 'Some change in the food of the caterpillar had obviously been brought about by the alteration in the soil conditions caused by the temporary flooding.'[4] According to Howard, 'Poor soil aeration always encouraged disease at Pusa.'[5] These phenomena, so well-observed by Howard, can be explained by the fact that flooding, by causing the roots to be asphyxiated, inhibits protein synthesis and therefore makes the plants which do grow much more susceptible.

Excessive dryness can have the same effect, since a lack of water increases levels of soluble nitrogen in the tissues, and particularly of asparagine and proline [Wearing and van Emden (1967), Slukhai and Opanasenko (1974), etc.]. Out of this, and always by a nutritional effect, comes proliferation of disease and pests, such as mites. [Wearing and van Emden (op. cit.).]

Conversely, soil that is adequately tilled to make it well-aerated, aids protein synthesis by oxygen, which it places at the disposal of the roots, yielding vigorous, healthy plants.

Howard (op. cit.) also highlights the beneficial effect of farmyard manure and humus on the resistance of plants to disease. He observes: 'It is a curious fact that in Indore there is no insect-borne disease or cotton fungus.' Furthermore, this remarkable observer appears to have fully sensed the important role played, in its resistance, by the plant's conditioning and biochemical state. With specific reference to viral diseases, he notes: 'In examining the affected plants, one may notice certain anomalies in the protein material, which leads one to suspect that the green leaves are not functioning normally. No build-up of proteins is taking place.'

It is interesting to compare the results of the methods practised by Hindu peasant farmers with those practised by their Chinese counterparts. Chouard (1972), on his return from a study trip to China, wrote: '

> Without going into detail, I can say here that I have been told of observations made by peasants about the most productive potato plants. These come from plants cultivated in the mountains or even from plants that were cultivated out of season.... The practice of the Chinese peasant farmers, as related to me, does not consist of destroying or eliminating the viruses. Instead, the aim is to reduce and sometimes to eliminate, through certain cultivation methods, the *display* of the viral symptoms. Low night temperatures and intense light have these effects. At present no-one really knows the rigorous scientific basis for the alleviation of the symptoms, without the virus itself disappearing.'

Apart from the beneficial effects of light and low temperatures, organic fertilisers are used extensively in China. We will now see how some rigorously conducted experiments may confirm the results of thousand-year-old empirical practices in agricultural methods, yielding evidence of the beneficial effects of organic fertilisers.

One might note in passing that there is sometimes a fine line between empiricism and science in the strict sense. As an example, we might take the use of chemical pesticides on a large scale, without paying attention to their effects on the physiology of the plant or the life of the soil. With chemical pest control as understood at the moment, we are dealing with pure empiricism. For instance, very little is actually known about how the products called 'fungicides' actually work.

Regression of a viral disease when organic manure is used.

In Tunisia, Mehani (1969) established the existence of a mosaic virus in the Provencal violet artichoke, whose effects are especially frequent in the second and third years of cultivation, and which result in a considerable drop in yield. In France, these artichokes never display symptoms of the disease. Yet if taken to Tunisia, they manifest symptoms of the disease and never really establish themselves.

This difference in behaviour has led the author to study the effect of manure on outbreaks of the viral disease. The studies were based on the fact that, over seven years, a 'well-cultivated' artichoke never displayed symptoms of degeneration. This seems to prove that, as in France, and due to certain cultural practices, the virus can remain indefinitely in a dormant state. (We shall return later to this idea of dormancy.)

Mehani's (op.cit.) experiments showed that the use of manure allowed a practical solution of the problem, by reducing the symptoms of degeneration in a fairly spectacular manner (Fig. 4). We should add that the effect of the organic manure is superimposed on that of inorganic fertilisers. It is the joint action of these two types of fertilisation that has proved to be beneficial for the resistance - or if one prefers, the tolerance - of the plant toward the proliferation and virulence of the virus.

The mechanism at work on the metabolism of the plant to give it resistance or tolerance is an organization of nutritive elements to promote better growth. In other words, a metabolism is created that is the opposite of the one characteristic of the viral disease. This disease has, in fact, been defined as a disease that stunts growth.

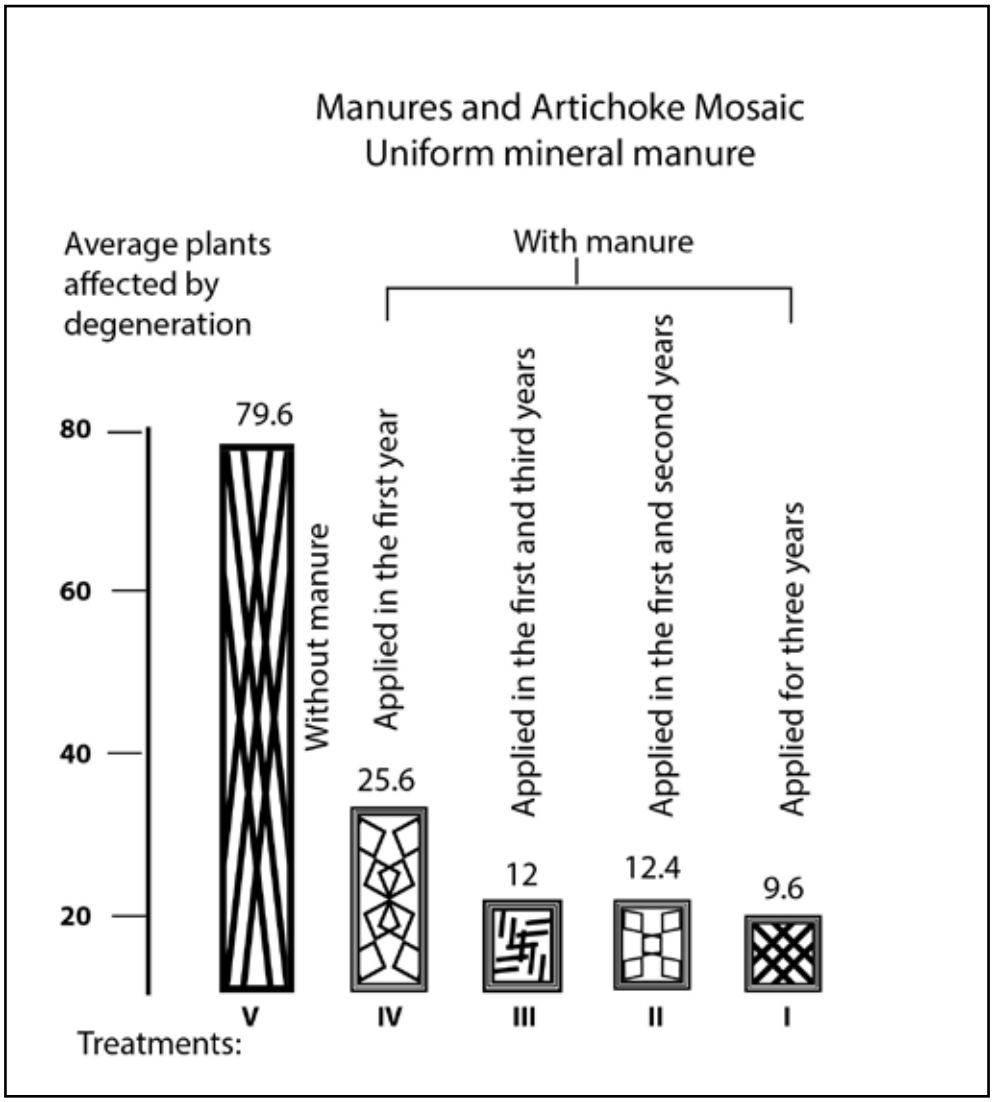

Fig.4: The influence of farm manure, together with mineral fertilisers, on outbreaks of artichoke mosaic. (According to the figures of Mehani, 1966.)

As for the deeper causes behind this effect of organic fertilisation on the growth of the plant and thus its resistance to disease, one can surmise that, through the improved nutrition of the plant, certain microorganisms occurring in this type of manure are brought into play. The situation is just the opposite with pesticides, whose effects we shall now consider.

IV. Repercussions of pesticides on viral diseases

Many now accept the view, regarding the control of viral diseases, that 'one treats the plant and not the disease itself.' (The one exception is chemical destruction of vectors, which we will discuss later.) As a result, a wide range of products have been tested which are capable of acting on the metabolism of the plant. This has met with varying degrees of success, apparently because it was not supported by a precise working hypothesis.

Limasset et al. (1948) carried out experiments on a small scale on tobacco, using 2,4-D against the X and Y viruses. As they concluded, 'The hormone spray did not prevent infection, nor did it destroy the virus. It simply showed a very strong inhibiting effect on the proliferation of the virus. In vitro studies, moreover, have shown that the product lacks an inactivating effect of its own, which fully confirms our point of view.'

However, this effect is temporary: plant stocks given the virus and hormone at the same time, and which do not contain detectable levels of the virus at that point, display symptoms in the uppermost leaves of the treated tobacco at the end of a few months.

In other words, the beneficial effect on the metabolism of the tobacco plant is only provisional. We should not lose sight of the fact that this reaction between plant and pesticide depends on the initial state of the plant - that is, on its physiological state and thus (as we have seen above) on its nutrition.

As to the effects of 2,4-D, to which we shall return, we should add that they are not always beneficial. In the case of maize, spider mites and corn borers increase, along with susceptibility to leaf stripe disease. We shall return to this when looking at cereal cultivation. (Oka and Pimental, 1976).

However, it is the herbicides that, through their specific and drastic effects, provide the greatest insight into the relationships between these three factors: the pesticide, the physiology of the plant, and the activation of viral diseases. McKenzie et al. (1968-1970) were able, by means of controlled laboratory experiments on resistant or semi-resistant maize, to show that susceptibility to the virus MDMV (maize dwarf mosaic virus) increases in proportion to doses of atrazine. A 100% manifestation of the symptoms is achieved with a dose of 20ppm of atrazine (Fig. 5).

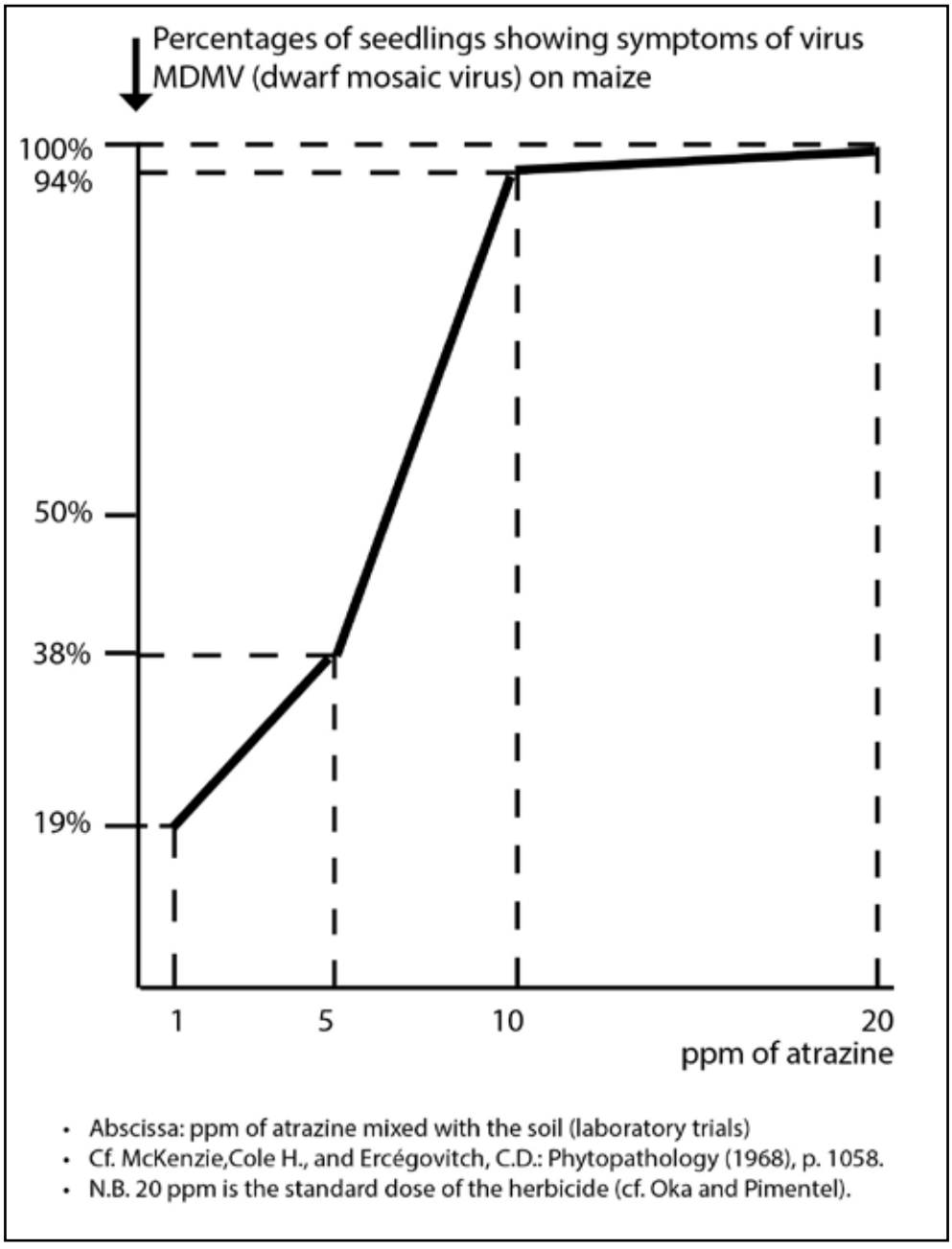

Fig.5: Repercussions of atrazine, according to soil levels, on the level of symptoms of MDVM (dwarf mosaic virus) on corn.

All the evidence shows that the sensitising of the plant is the result of physiological modification of the plant. Some research has been carried out on the subject of possible connections with proliferation of the virus. Misra and Singh (1975) have studied the effects of a number of chemical preparations such as giberellic acid (GA), 2 thio-uracil, and 2,4-D at different doses on a virus in chrysanthemum (the chrysanthemum stunt virus or CSV).

One important initial result is that all these chemical products cause nitrogen levels in the plant to rise, in comparison to controls. We now know that this is a factor of susceptibility to disease to the extent that the nitrogen is in soluble form. In regard to a connection with the virus, Misra and Singh (op. cit.) point out in fact that there is no asparagine in healthy tissues, but that, sixty days after infection, it is found in leaves infected with CSV. This suggests that the state of proteolysis could, as with other infections, be connected to viral diseases. This seems to be confirmed, furthermore, by the fact that proliferation of the virus decreases the amount of free amino acids, as well as levels of potassium.

This is also evident from the study of the effects of herbicides on plant physiology, where the cultivated plant is subject to the same attacks, and selectivity is in no way perfect. This is emphasised by Altman and Campbell (1977):

> 'The results, in the case of carbamate herbicides, indicate that the use of amino acids by susceptible species is inhibited by carbamate herbicides. Low concentrations of these herbicides are effective: 10ppm of CIPC represent approximately 5 x 10-5 molar. The inhibition occurs very soon after the herbicide comes in contact with the plant's tissues.'

As Altman and Campbell (op. cit.) observed, 'The inhibition of protein synthesis caused by herbicides may explain a number of their effects.' This is what seemed to be happening when diseases increased through the effect of pesticides on the physiology of plants, especially that of herbicides on cereal crops. Since this is in perfect agreement with our theory of trophobiosis, the issue may be summarised as follows: viral diseases, being diseases that inhibit growth, are promoted by any process that inhibits growth. This is especially true in the case of agents that cause deficiencies.

Since all herbicides are toxic for all plants - that is to say, inhibitors of protein synthesis – all may be causal factors in the current spread of viral disease. This is the case for atrazine, as mentioned above. It also seems to explain the conclusions of Altman and Campbell (op. cit.) in relation to herbicides:

> 'Since 1945, numerous reports have appeared of increased losses due to attacks of insects and pathogens, in spite of very great efforts to control them. It is difficult to calculate to what extent these are due to the environmental and biochemical impacts of herbicides. In a number of cases herbicides have been blamed for creating problems of parasitism in various plants.'

Confronted by such a spread of disease, one speaks of 'resistance.' However, it is now clear that what is involved is proliferation, caused by the plant's greater receptiveness as a result of the effects of herbicides on its physiology.

For the moment, we will note that Cors et al. (1971) showed that oxycarboxin, when applied to barley seeds, increases the level of the Brom mosaic virus in the leaves. This fungicide therefore enhances the proliferation of the virus by its action on the plant. Yet, as we have seen above, this is not an isolated example: the same is true of any chemicals that penetrate plant organisms and act on their metabolism, especially its resistance, which is linked to the level of protein synthesis.

V. Grafting and viral diseases. Case Study: the grapevine

However, we should say some words about another factor that also affects the 'susceptibility' of plants to viruses. This is the influence of grafting.

Souty (1948) concluded: 'It is not certain that trees which have been grafted are any more vigorous, productive, or longer-lived than those which grow from their natural roots.'

This author might also have added: 'or more resistant to disease.' As Wallace et al. (1953) note:

> 'Citrus stocks influence the size of the tree, the harvest, the quality of the fruit, and susceptibility to disease.'

However, certain combinations of stock and scion can turn out to be beneficial. We can see this from some results obtained by Sartic (1975) in observing Sharka, a viral disease of fruit trees that is currently on the increase in French orchards.

This author recalls that grafting may actually constitute a valuable means of protection not only against mycoses such as *Phytophthora parasitica* in citrus trees, but also against mycosis in the apple tree (*P. cactorum*), as well as against root rot in the peach tree (*P. cinamoni*).

The discovery of certain stocks that are 'resistant' to the Tristeza virus, and of various cultivars used as apple stocks resistant to or tolerant of so-called 'latent' viruses, enables us to envisage an elegant solution to the problem of disease control. Naturally, this is only possible where the use of these stocks is compatible with the other agronomic constraints (production, fruit quality etc.).

However, harmful effects of grafting in connection with viral disease are equally striking, and sometimes arise in a rather insidious manner, as Marrou (op. cit.) notes, in connection with viral maladies in crops for market gardening. This would also appear to be the case with the grapevine, where we have seen a potential relationship between deficiencies and viral disease.

The increase of viruses in the grapevine provides many lessons. It is very closely linked to another 'affliction,' the invasion of *Phylloxera* and the agronomic solution which has been found: the regeneration of the vineyards by grafting French plants on to American stocks - *Riparia, Rupestris*, and their crosses.

As Rives (1972) notes, 'The properties of the stocks are communicated to the scion or, at least, to the whole plant.' Rives stresses: 'From the results obtained from multiple combinations, there is no way that we can presume to differentiate between those which are due to the stock and those which are due to the virus.'

But is there no way of gaining an idea of the relationship between stock, scion, *Phylloxera*, and virus? Even in the context of 'susceptibility' to various afflictions and the biochemistry of the scion?

Here we encounter the role of vectors in the spread of viral diseases. They have been the subject of controversy. At the third international conference on winemaking and the grapevine. Father Branas denied ever having said that *Phylloxera* is an insect vector of viruses. However, as he stated, 'There is a direct relationship between the spread of *Phylloxera* and the spread of viruses.'

In response to this, Father Ciferri observed that this might be due to the use of American stocks which 'upset' the balance (probably an ancient one) between *Vitis vinifera* and the classic infectious viruses of degeneration. Ciferri maintained that such viruses do not cause obvious damage to European grapevines that are not grafted on to American root stocks. However, cultures with American root stocks, even those that have never been in contact with the viruses, unleashed the disease with the gravity seen in the last decades of the nineteenth century. From that point, they infected the parabiosis of the European grapevine grafted on to American root-stock.

Though *Vitis vinifera* is sensitive to *Phylloxera*, it proves resistant to chlorosis (that is, iron deficiency). Iron-induced chlorosis is the symptom of a disorder of iron metabolism, caused by limestone soils. Ungrafted grapevines were tolerant of chlorosis, either because of their low iron requirements or because their roots were able to make the iron in the soil easily soluble. 'However, after *Phylloxera* had destroyed the vineyards during the last century, it was necessary to graft the grapevines on to stocks (of American origin) which were resistant to the parasite. Yet some combinations of root stock and vine then showed themselves much more susceptible to chlorosis than vine varieties which were cultivated on their own roots. The iron requirements of the above-ground system of such combinations could not be satisfied by the weak ability of some root systems (of the stock) to extract iron from the soil.' (A personal letter from M. Delas, INRA, Bordeaux).

Basically, grafting led to iron deficiency. Could this be a sufficient reason for the susceptibility of the vine to a viral disease such as infectious degeneration? There is no reason why an iron deficiency should not be accompanied by several other deficiencies, such as boron or zinc.

In confirmation of the relationship between viral disease and deficiencies, Dufrenoy (1934) demonstrated experimentally that sprays based on zinc sulphate caused symptoms of grapevine fan leaf (infectious degeneration) to disappear. We shall return to this important issue of the treatment of diseases through correction of deficiencies in the course of Chapter 8

VI. The works of Vago and the concept of disease

In the introduction to his thesis 'The Progression of Diseases in Insects.' [6] on the subject of the general concept of disease, Vago wrote as follows:

> 'After more than two centuries of research in every domain of pathology, aiming to define pathologic processes as morbid entities, there is a new trend that acknowledges the difficulty of trying to explain numerous pathologic states on the basis of the isolated unit called 'disease'.'

A little further on, Vago adds: 'It seemed to us that the domain of insect pathology might possibly make a special contribution toward improving our knowledge of this new pathway.' There is a strange convergence between his conclusions and the notes that relate to the processes of disorders in plants. At this point we are persuaded to acknowledge a general law of biology that applies in equal measure to the reactions of plant and animal organisms: that is, the balance between health and disease.

Vago's work began with the study of a problem. In the years prior to 1950, jaundice of silkworms raged in a well-demarcated area within the Gard, Ardèche and Lozère regions. This area was bordered to the north by a chemical factory, and the gravity of the outbreak was directly due to the proximity of the factory and the direction of the prevailing wind.

Moreover, caterpillars reared on the leaves of the mulberry taken from this area clearly confirmed that this type of diet promoted the spread of the disease. In fact, only 2% *polyédrie* was recorded on a diet of leaves gathered outside the contaminated area, while this rose to 20% (a tenfold increase) with leaves collected in the contaminated area.

Another important point is that a diet of leaves entirely from the contaminated area yielded the following results:

- 13% *polyédrie* with leaves previously washed.
- 23% *polyédrie* with unwashed leaves.

Such a result suggests that pesticides in the form of residues, important though they are, are not the sole culprits. The 13% polyédrie resulting from a diet of washed leaves, but which had been treated with pesticides, stood in contrast to 2% when healthy leaves were used. This shows that the pesticide in question (NaF) had an effect after penetrating the plant tissues, by harmfully modifying the plant's biochemistry.

This jaundice was in fact provoked by the ingestion of sodium fluoride (NaF), the essential constituent of the emissions from the factory in question. Vago made comparisons with cattle fluorosis that raged in the same region, and with a similar case in Italy.

This is a case of the unleashing of a viral disease by poisoning. However, Vago's studies also deal with the effects of diet.

Thus Vago showed that, in the case of *Lepidoptera Vanessa urticae* L., at least two types of diet promote the development of polyédrie:

- a long-term diet of withered mulberry leaves, containing little chlorophyll and in the process of yellowing.
- a diet of nettles, which has different effects on the polyédrie, according to the type of soil from which the plants are taken:
- fed on nettles from clayey soil, the youngest larvae show levels of polyédrie of 15-19%.
- fed on nettles grown in an arable alluvial soil, young larvae only show a level of 4%.

Such indirect effects caused by the plant recall the differences we have already pointed out in the fertility of the Colorado beetle, or in the resistance of plants to disease according to soil type.

Vago confirmed, in his work with silkworms, the results obtained with *Vanessa urtica*. When fed on very ripe mulberry leaves the worms produce, from the first moulting, a high polyédrie, and the disorder persists throughout the whole larval stage.

Such experiments clearly underscore the great importance of diet and nutrition for the resistance of organisms to pathogenic agents. It is natural to draw a parallel with the effects of different types of fertilisers on the resistance of plants, both to insect pests and to diseases.

Provocation of viruses in silkworms in the absence of previous infection.

By taking many precautions, Vago was able to exclude any introduction of the virus. The experiment concerned a food that silkworms would not usually feed on: *Maclura aurantiaca* (osage orange or bow wood). Compared with their normal diet - *Morus alba* or white mulberry – the results were:

- less food ingested
- a higher mortality rate
- finally, development of a virosis.

From the third generation on, the polyédrie was intensified in the Maclura batch. During the fourth and fifth generations, the disease practically eliminated the whole batch through a process of nymphosis. Among the controls, however, mortality remained under 20%.

However, perhaps the most significant experiments were those with a diet including sodium fluoride. To trace these 'secondary' effects of the pesticide, it was essential to achieve only a sub-lethal action. For this reason the leaves of the mulberry were soaked in a 0.01% solution of NaF. (This was an experimental version of what had been observed in the natural environment.) These results were more conclusive:

- diet of contaminated leaves: 85% viroses.
- diet using healthy leaves: 8% viroses.

From the thirteenth day onward, the virosis became significant. The disease curve showed a rapid and total loss of the batch.

On the basis of these rigorous experiments, Vago's conclusions become so important that we reproduce them in full. As he says at the outset: 'It appears possible to trigger an acute virosis without previous infection and in the controlled absence of any sign of virus.' Vago adds: ' The underlying factors may be linked to diet, to poisoning by certain chemical substances, or to climatic and physiological conditions.'

Conversely, the factors behind non-appearance of the virus correspond to an optimum physiological state, free of any disruption of a pathological or parapathological nature. As Vago notes:

> 'These two considerations suggest that the factors behind the triggering of the virosis are physiological disturbances. In spite of the varied nature of their external effects, these may have in common a specific mechanism at the cellular level.'

In Vago's words: 'The unleashing of viroses by non-infectious factors seems to us to have the form of a complex. The first stage of this complex consists of various pathological processes, while the second stage is represented by the virosis.'

As Vago observes, 'Viroses are typically secondary in the case of metabolic disturbances.' He continues:

> 'The appearance of viroses following various primary processes which seem at first sight quite different, turns out to be very closely linked to deviations in cellular metabolism that follow these processes. Here the inner mechanism is set in motion by reactions of the intra-cellular enzymes, in relation to a pre-existent element with virus potential. The problem posed by this mechanism has been likened, in comparative pathology, to the induction of bacteriophages or 'dormant viruses' in plants.' (Nowadays we speak of 'latent viruses,' though the process is the same.)

Vago emphasises, moreover, that in this cause-effect chain 'metabolic disturbances → viroses' the primary effects follow upon either a diet that leads progressively to a state of imbalance or, instead, a toxic effect, in which the dose is decisive. Thus, to determine the complexes induced by sodium fluoride, we would need to establish a dose corresponding to each generation and each race in the study.

These findings about complexes of 'disease' in insects lead us to consider their general applicability . As we have just seen, Vago makes a case for 'dormant viruses.' Returning to this in his conclusions, he notes: 'Analysis of this complex (metabolic disturbances with delayed appearance of acute viroses) has introduced some elements now well-assimilated into comparative pathology.

Along with bacterial lysogeny, 'dormant viruses' in plants, and the Drosophila virus it raises the issue of viral induction without previous infection.'

According to Poljakov (1966), who also sees the physiological state of the host plant as one of the principal factors determining the resistance of cryptogams, dithiocarbamates are responsible for unleashing various diseases. He writes (op. cit.):

> 'We have observed an impact of organo-chemicals on the development of fungal diseases in apple trees and grapevines, where the use of zineb in the control of apple scab and mildew promotes the development of Oidium. Dithiocarbamic acid used over four years to control of Phytophthora in potatoes also provokes an increase of other diseases, especially viroses.'

These observations confirm our own research into the sensitisation of plants to Oidium in grapevines by dithiocarbamates (Chaboussou, 1966). The existence of a complex implies both an attack of pathogenic fungi and a viral attack.

However, we have seen that other pesticides, especially certain herbicides such as atrazine, may make plants more susceptible to viral diseases (McKenzie et al., 1970). Since such effects may be combined with those of malnutrition brought on by mistakes in fertilisation, such as excessive nitrogen, it is hardly surprising that there is currently an increase in diseases of cereals, such as Dwarf barley yellow.

The deeper causes of this phenomenon could lie in the manner in which deficiencies are induced. Vicario (1972) noted that pesticides with a nitrogen base (i.e. the majority of chemical pesticides), are cations. As such, they can displace other cations in the interchangeable complex, such as Ca, Mg, and Zn. Certain findings (Huguet, 1983) seem clearly to confirm this.

Clearly, then, the question is whether such a deficiency can alter the metabolism of the plant to the point of provoking a viral outbreak. We must not forget that a viral disease disease affects growth, and will therefore benefit from any factor which inhibits growth. Deficiencies fall into this category. We recall that Dufrenoy showed that in broad beans a boron deficiency causes a change in the core, to the point where it takes on a filamentous aspect in the meristems.

All this seems to show clearly, once again, the great importance of deficiencies in outbreaks of disease, especially viral diseases, in the context of a metabolism dominated by proteolysis. This view is supported, as we shall see in the eighth chapter, by the beneficial results of correcting deficiencies.

It also explains the difficulties in eliminating viral diseases if one does not take into account the complex of effects on the metabolism of the plant brought about by the use of chemical pesticides and fertilisers. We intend to provide additional proof of this in the next chapter.

Notes

1 Phoma betae
= Pleospora bjoerlingii
= Fr. Pied noir de la Betterave; maladie du coeur
= E: seedling rot rot, leaf spot, Black Rot
of growing roots, Heart rot of mature roots.

2 Sir Albert Howard, An Agricultural Testament (New York, London: Oxford University Press), 1943, 253 pp. [republished: Rodale Press, 1976, 253 pp.]: title of Chapter XI.

3 Howard, op. cit., p. 163. [Chaboussou has translated Howard rather freely.]

4 Howard, op.cit., p. 164.

5 Howard, op. cit., p. 162.

6 C. Vago, L'enchaînement des maladies chez les Insectes. (Thèse Fac. Sciences, Aix-Marseille), 1956, 184pp.

Chapter 6

Relations Between Viral Diseases, Vectors (Aphids) and Pesticides

I. What makes viral diseases exceptional?

It is frequently said that viral diseases are generalised, incurable, transmitted by grafting, variable in symptoms, and immune to any defence mechanism of the plant.

However, if we examine these different 'properties' closely, we shall see that they are not universally applicable. Certain plants of the same species, depending upon their age and stage of development, refuse to become contaminated.

Incurable? Yes, but only if one seeks to destroy the pathogenic agent, that is, the virus. Not if, as we shall see below, one takes care, through nutritional and protective measures, to prevent the virus multiplying or even replicating itself in the cell.

Transmitted through grafting? Certainly, but only if the 'combination' of rootstock and scion allows replication of the virus. This underlines the influence of the rootstock on the viral disease via the physiology of the scion. (We shall come back to this in Chapter 7.)

Variable in their symptoms? Yes, it has been pointed out that viral infections have few truly characteristic symptoms. However, while few symptoms can be strictly associated with them, there is a strong convergence between symptoms of nutritional problems, for example, and viral infections.

This is to be expected because nutrition and toxins both affect the physiological state of the plant, and thus its resistance.

Environmental factors play a role in viral as well as bacterial and fungal infections, as well as in attacks by pests. However, what seem to characterise viral diseases are the effects they have at the cellular level. These effects are brought about by metabolic problems, themselves the result of malnutrition or toxins, or by the genesis (or at least the multiplication) of the virus: DNA or RNA.

The usual approach to controlling viral diseases is to seek the destruction of the vectors (aphids, leafhoppers, nematodes, etc.) This is done without any regard for the physiological state of the plant that may condition its 'receptivity' to the virus, or for the negative effects of pesticides on the metabolism of the crops one seeks to protect. This lies at the root of the failure of chemicals to control the vectors. These chemical products are likely to cause them to multiply, and to aggravate the diseases by the same process, as a result of increasing the ratio of proteolysis to protein synthesis.

II. Chemical control of vectors

1. The failures of aphicide treatments

Numerous researchers have found that the chemical control of vectors is extremely disappointing. As Marrou (1965) notes, 'Direct control by insecticide sprays, though it destroys the vectors, does not in general protect the crop from viruses.' He points out that for the majority of viruses of the mosaic family, to be pierced once by an aphid is enough to infect the plant. Even if this first contact with the plant proves lethal for the vector, the virus has been passed on.

The facts, however, seem to be more complex. In particular, chemical control is blamed for deterring insects and so dispersing disease. However, the mechanism of these failures may lie deeper. After a brief period of effectiveness, many pesticides increase the numbers of aphids instead of destroying them. The mechanism for this proliferation does not lie simply in the destruction of natural enemies, as we mentioned in the course of Chapter II.

Münster and Murbach (1952), in treating the potato plant with the phosphoric ester 'pestox,' found that the largest populations of aphids were found on the most frequently treated plots. Moreover, the state of health of the plants harvested from the treated plots was not superior to that of the seedlings in the untreated plots.

The authors attribute these results to insufficient toxicity in the treated plants, and a greater mobility of aphids on those same plants. They were not able, however, to comment on the proliferation in proportion to the treatments.

Bovey and Meier (1962) likewise found that chemical treatments based on lime arsenate, carbaryl, DDT, and dieldrin led to an increase in the numbers of leaf aphids. They see the cause for this in the elimination of predators, or aphid-phages. But they also observe that one cause is insufficient action of the chemicals, because the aphids multiply rapidly on the lower surfaces of the leaves. This suggests another phenomenon, of an indirect and nutritional nature.

The authors advise keeping chemical use against the Colorado beetle to a strict minimum, in order, they say, to prevent a massive increase in viral infections.

Similar failures occur in the control of potato whorl by diazonon treatments, or *Myzus persicae* by schradan treatments. Two weeks after the treatment, the aphid populations in the treated plots were four times higher than those in the control plots. Here, the author thinks that the increase in disease and reduction in yield were linked to the upsurge, after the chemical application, of the population of winged and wingless aphids (Klostermeyer, 1959). But here too, another factor plays a part: the action of the pesticide on the potato.

In regard to controlling potato Y virus, Shanks and Chapman (1965) obtained equally disappointing results. They found one particularly unexpected effect: on plants treated with parathion, the aphids survived two to three times longer than on the control plants.

We believe this shows that the pesticide has altered the metabolism of the plant (in this case, tobacco). The result is a much greater impact on the viral infection, although the authors make no distinction between the role of the aphids and that of the plant's physiology. In line with the ideas of Russell (1972), one can assume, as a first approximation, that resistance to viral infection is made up of:

- resistance to the aphid vectors
- resistance to inoculation by the virus
- and finally, tolerance toward the virus.

All of these factors are integrated in the workings of the complex, Plant-Pesticide-Virus-Vector. We have gained an initial demonstration of this from the experiments of Roland (1953).

The aim of these experiments was to study the influence of repeated aphicide sprays, with the help of a chemical with persistent action, on the state of health of a potato crop, considered purely from a virological point of view. These experiments were carried out on the potato variety Ackerman, which was initially propagated in isolation in a greenhouse (after eliminating plants infected by whorl, the X virus, or theY virus). These were therefore not represented in the tubers subjected to the experiment. However, the potatoes were planted in two plots that already contained numerous plants contaminated with whorl and Y virus.

While one of these plots was treated on ten separate occasions with a 0.1 per 1000 solution of parathion, the other remained untreated, to serve as a control.

At harvest, two tubers were kept from each plant in the two plots. These were re-planted the following year, having been kept free of aphids during the winter. Two weeks after the first shoots appeared, the number of diseased specimens was counted. As a consequence, the disease could be attributed to the mother plant being infected by aphids in the preceding year. Table 8 gives the details of the results:

Table 8: Plants with Viruses in Relation to Treatments

Viruses	Treated plot		Control plot	
	N	%	N	%
Plants with viruses	33	83	21	50
Whorl	31	77	17	40
Mosaic	3	7	5	12

To the author's surprise, not only did the repeated parathion treatments have no beneficial effects at all on the state of health of the treated tubers, but there was also an increase in the proportion of whorl in the treated plot. Roland (op. cit.) deduced from this that the pesticide attracted the insects.

Shanks and Chapman (1965), who carefully studied the effect of various insecticides on the behaviour of *Myzus persicae* in relation to transmission of the potato Y virus, found that aphids stayed around two to three times longer on plants treated with parathion or phorate than on the control plants.

They concluded, moreover, that these experiments showed why insecticides were not able to prevent the spread of disease by aphids in the field, and why they can even be linked to an increase in infection.

The reason for this becomes even clearer, as we shall see later, when we consider that both parathion and mevinphos have a tendency, through their action on the metabolism of the plant, to lead indirectly by the trophic route to proliferation of aphids. This process is analogous to the one we were able to demonstrate with mites (Chaboussou, 1969).

Such aggravation of the development of viral disease, by pesticides such as parathion, recalls our discussion of 'fungicides' such as dithiocarbamates (zineb), which also affect potatoes (Poliakov, 1966).

2. Proliferation of aphids due to pesticide treatments.

Numerous authors have pointed out the responsibility of pesticide for the proliferation of aphids. As early as 1946, Michelbacher found that DDT led to the multiplication of *Chromaphis juglandicola* (Kalt) after treatment against nut *Carpocapse* on walnut trees. It should also be noted that DDT has the same effect on the caterpillar *Lecanium pruinosum* (Coquillet).

Pimental (1961) likewise notes that the use of DDT results in larger swarms of *Rhopalosiphum pseudobrassicæ* on the cabbage plant. Peterson (1963) observes an increase in *Myzus persicæ* on the potato plant, not only with DDT, but also with carbaryl, Gusathion, and Di-systom.

According to Granet and Read (1961), carbaryl is also responsible for increasing the numbers of *Rh. pseudobrassicæ* on the turnip plant, and according to Thurston (1965), for proliferations of *M. persicæ* on tobacco.

With regard to organo-phosphate insecticides, their promotion of mite populations have been reported many times. Fenton (1959) found that treating lucerne (alfalfa) with mixtures based on parathion, toxaphene, and demeton led to increases in populations of *Macrosiphum pisi*.

I. Redenz-Rüsch (1959) found that treating apple trees with parathion and demeton throughout the year resulted in larger populations of mites and aphids at the end of the growing season.

This is the more surprising as these pesticides are especially active against these particular pests, at least in the short term.

Steiner (1962), who made a close study of the effects of various insecticides, both mineral and chemical, on a whole range of noxious insects on apple trees, found very significant multiplication rates of aphids with products such as malathion, Thiodan, parathion, and DDT. Increases in Aphididæ were also found with demeton, Diazinon, and lead arsenate.

Until now, our discussion has focused on the effects of insecticides. Traditional theories - despite the demonstrations of multiplication by trophic means that we will discuss later - hold that proliferation is due to the destruction of natural enemies. This must be carefully examined when looking at the widespread proliferation of aphids, due to the synthetic pyrethrums used against Pyrales attacking maize. The phenomenon is equally prevalent with fungicides.

Repercussions of fungicides. Many fungicides are harmless for the predators and parasites of aphids. This makes their effects on aphid populations all the more interesting to study.

Steiner (op. cit.) recorded a significant increase in *Aphididæ* on the apple tree, as well as in psyllids. This occurred after treatments of captan and of thiram or (According to our research, captan is also a fungicide particularly prone to increasing mite populations.)

In line with these observations, Hukusina (1963) found on apple trees, particularly at the end of the season, a great abundance of mites and aphids, following the application of a series of treatments with chemicals that were not especially toxic to natural enemies. This was particularly the case with the combination: lead arsenate-captan.

It was, moreover, Hukusina and Ando (1967) who, following their precise observations, related the fecundity of the aphid *Myzus malisinctus* (Matsumuru) to the nutritive quality of the apple leaves. They were able to establish:

- that the aphids preferred the shoots of trees when the trees were in the growing process, in other words, when they were richest in total nitrogen;
- this richness in nitrogen explained the greater susceptibility to aphids of the variety Delicious in comparison with the variety Ralls Janet. (This helps to confirm that nitrogen influences susceptibility to attacks by different parasites.)

Results with plants grown in controlled environments, and thus beyond any possible influence of natural enemies, further confirm this hypothesis.

3. Stimulation of aphid fecundity by trophic means, following pesticide treatment.

Maxwell and Harwood (1960) are perhaps the first to have looked at the relation between plant fecundity /physiology, and the effects of pesticides. These authors found that the application, under laboratory conditions, of 2,4-D on the broad bean plant markedly increased the reproduction of *Macrosiphum pisum* (Harris). Similar results were obtained with *Melanoplus bivittatus*.

Leaf analysis has shown that where aphid development is greatest, the most notable biochemical changes are in the ratios of free amino acids. 2,4-D increases the free quantities of alanine, serine, and glutathione.

Smirnova (1965) was able to link the multiplication of *Aphis fabae* on the broad bean plant under the influence of DDT with increases in the tissues of non-proteic nitrogen and disaccharides. These changes occur some eight to fifteen days after application. The author concludes, 'The aphid population increases as the levels of non-proteic nitrogen increase.'

We find the same convergence of results and the same conclusions with Chan (1972). He has experimented with *Schizaphis graminum* on barley, examining the plant's resistance according to variety. As discussed above, the most vulnerable varieties of apple tree showed the highest concentrations of free amino acids. The same is also true of total soluble carbohydrates.

The results resemble those of Michel (1964) in regard to the effects of mevinphos and phosdrine on the multiplication rates of *Myzus persicae*, subsisting on treated tobacco plants. He records an increase in fecundity: from a rate of 25.09 on the control plants to 31.69 on the tobacco plants treated with mevinphos at a concentration of 1 ml per litre, and to 46.30 on plants treated with mevinphos at a concentration of 2 ml per litre (fig. 6).

In addition lifespan is prolonged according to the type of feed: 69.60 days on the control plants, 74.40 days for aphids living off plants treated at concentrations of 1 ml/l, and 87.43 for those on plants treated at a concentration of 2 ml/l. The results are quite similar to those we obtained with mites.

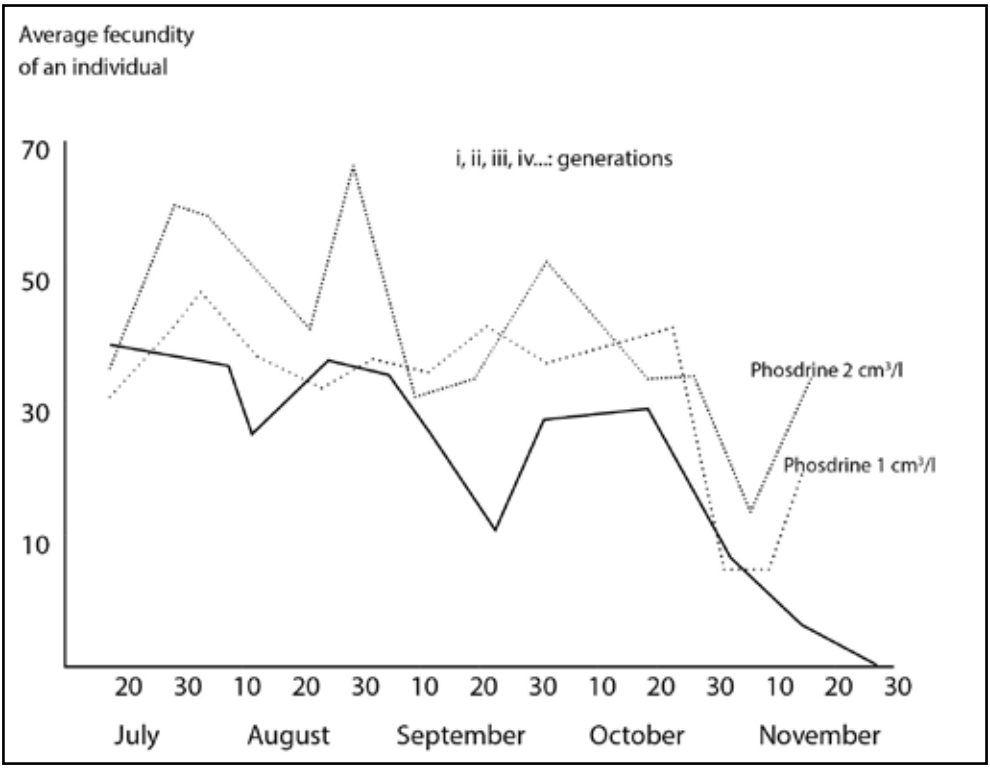

Fig.6: Fecundity and generational succession of Myzus persicae reared on tobacco treated with mevinphos (at two dose levels) and on controls. (Michel, 1964.)

Furthermore, the nature of an insect's nourishment has an effect on the rate of its life cycle. As a result of the alteration of the foliage by the pesticide, the aphids are likely to reproduce much earlier. From July to November, the result is the appearance of an additional generation of aphids, when they are fed on foliage treated with mevinphos.

These results also tally with those obtained by Kessler et al. (1959) concerning the effects of purines on the metabolism of nitrogen in the leaves, in connection with the plants' 'susceptibility' to aphids. Treating the leaves of apple trees with caffeine leads to a reduction in the fecundity of *Aphis pomi*. Caffeine does not have any direct effect through contact with the aphid, nor does it work through ingestion. It must therefore work indirectly via the changes it induces in the metabolism of the leaf.

Analyses have shown that caffeine lowers total nitrogen levels, particularly the soluble fraction and the DNA, while the protein fraction and the RNA increase in line with increases in the caffeine dose. The action of the caffeine and that of the age of the leaf in reality mirror one another: in both cases, the RNA/DNA ratio and protein synthesis rise, while levels of DNA and soluble nitrogen decrease.

The reduction in aphid development comes from these changes in the leaf's biochemistry, whether the changes are natural or artificially induced. The resistance of mature plants, as has already been discussed, is in effect explained by the increase in relative levels of RNA and proteins. With the approach of senescence, the opposite is the case: the proteolytic processes accelerate, together with decomposition of proteins and destruction of RNA and DNA. These anabolic reactions release considerable quantities of soluble nitrogen, a phenomenon linked to the renewed vulnerability of the plant to attack by aphids, as well as to their proliferation.

4. Nutritional requirements and fecundity of aphids

Considerable progress has been made over the course of recent years in determining the needs of aphids, thanks mainly to techniques of artificial rearing. We refer here to the works of Auclair and his colleagues (1957, 1960, 1964) on the pea aphid Acyrtosiphum, and of Mittler and Dadd (1963-65) on the subject of Myzus persicae. We must also mention Ortmann's studies (1965) on alfalfa and pea aphids, and those of Wearing (1967) on *Aphisfabae, Myzus persicae*, and *Brevicorne brassicae*.

It emerges from this collection of studies that aphids require different optimum diets, depending on the species and even the age of the individual. However, these show a certain balance between amino acids and sugars. On their own, amino acids prove incapable of inducing nutritional responses in *Myzus persicae*. On the other hand, they can act synergistically, in combination with sugars (Mittler and Dadd, 1964).

In a general fashion, however, as Auclair has shown with *Acyrtosiphum pisum*, the amino acids are in quantitative correlation with the growth, development, longevity, and reproduction of the aphids.

Mittler and Dadd (1964), moreover, note that the concentrations of amino acids and amides in the bean plant are generally lower than the minimum required for the healthy growth of aphids on a synthetic diet.

These findings would appear to be in line with the multiplication of aphids after pesticide treatments which, by inhibiting protein synthesis, enrich the plant in these nutritional elements. Such metabolic changes also seem related to the plant's increased power of attraction.

5. Factors determining the aphid's selection of its host

Here we encounter a fundamental problem. Lipke and Fraenkel (1956) quite justifiably point out that the aphid's preference for certain hosts constitutes the true heart of agricultural entomology. Kennedy (1965), for his part, while recalling old principles such as 'A healthy plant is an immune plant,' or 'The nutritional condition of the larva determines the imago's egg laying choices', counsels us to distrust over-hasty generalisations.

Aphid relations with plants, for instance, are complicated still further by the fact that different hosts are chosen at different seasons according to different stages of the insect's development. However, Kennedy's own studies on *Aphis fabae* imply that aphids choose their food in response to the nutritional factors made available by the plant. This appears to explain this species' preference for either young leaves or senescent leaves (a characteristic shared equally by *Myzus persicae*). In other words, it shows a preference for plant parts rich in soluble substances (saccharose and amino acids), which guarantee the insect better reproduction in relation to its nutrition.

Not long ago, this statement would have seemed to contradict the theory that was previously widely accepted, according to which the choice of food by phytophagic insects, when specific, was governed largely by responses to non-volatile chemicals such as various essential oils, alkaloids etc, acting as token stimuli.

While it cannot be denied that these elements play a part in attracting the insect to the host, they are in fact only by-products of a metabolism that assures the insect of the nutritional elements it needs for its growth and reproduction.

These views, which certain researchers distrust because of their apparent finalism, appear to be confirmed by relatively recent research that examines the response of aphids to artificial diets as carefully as the results - that is, their proliferation as a consequence of certain pesticide treatments of the host plant.

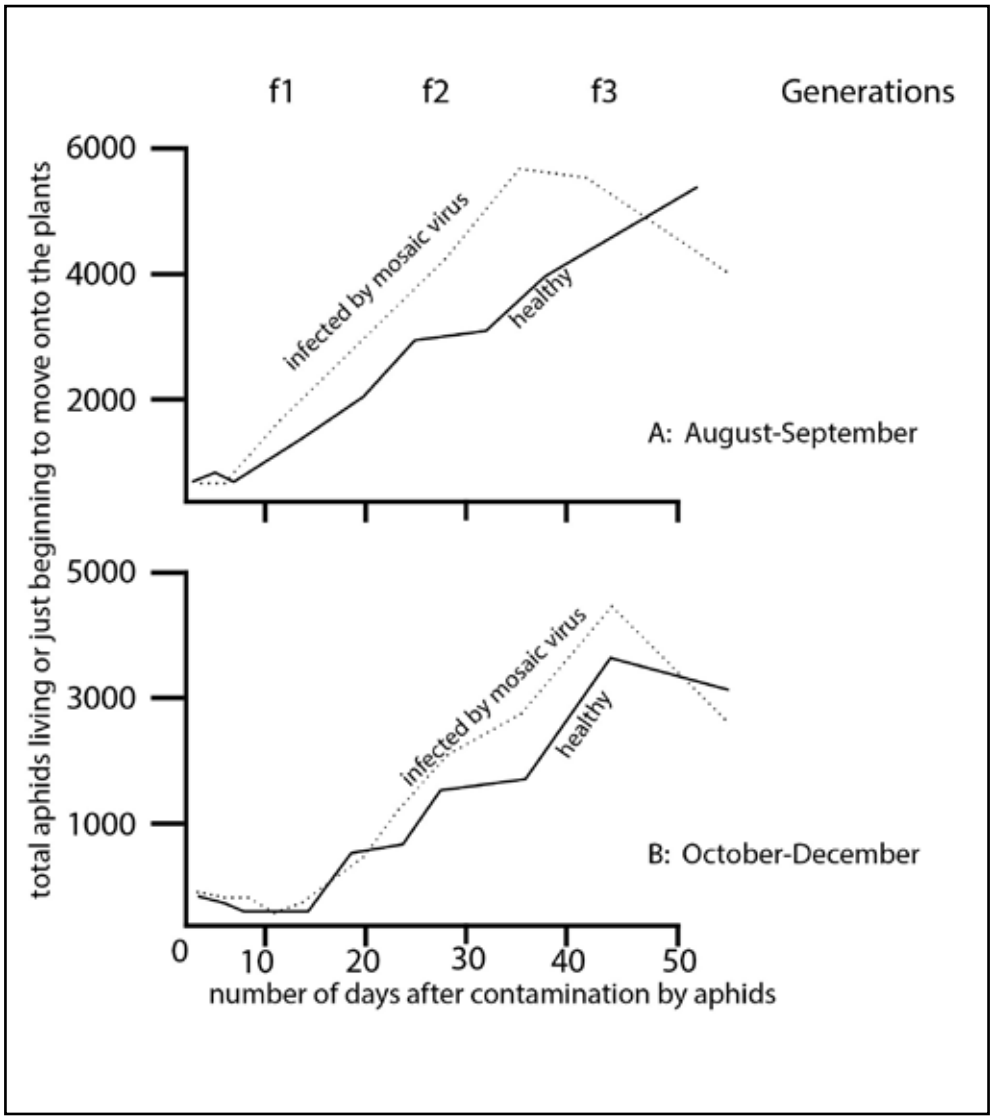

Fig 7: The development of *Aphis fabae* on the sugar beet, with or without mosaic virus.

A) On five plants of each kind, the mosaic was inoculated 18 days before contamination by aphids.
B) Four plants of each variety, with mosaic inoculated 12 days before contamination by aphids.
f2, f3: reproductive generations.

(after Kennedy)

Numerous studies have shown, in effect, that nutritional substances are linked to the mechanisms of phagostimulation in phytophagic insects, particularly for aphids. Auclair (1965) notes that preferences and fecundity go together in the case of *A. pisum* fed on an artificial diet. In this case preferences and survival are not correlated. There is a difference in behavior, in this respect, between the young insects, which prefer a diet rich in sugars (35% saccharose), and the adults, which choose a diet less rich in this substance (20%), but one which also brings them greater longevity and higher reproduction rates.

In consequence of their experiments in growing *Myzus persicae* with another artificial diet, Mittler and Dadd (1965) conclude that from now on, 'We can no longer consider Kennedy's views as absurd'.

But what happens when we introduce a new 'biological' factor, that of viral disease?

III. The complexity of the relations between viral diseases, vectors (aphids), and pesticides

1. The influence of viral disease on the attractiveness of plants to aphids

Experimenting with *Aphis fabae* on sugar beet, Kennedy (1951) observed a preference of aphids for plants infected with mosaic virus. The aphid's rate of reproduction is increased by 50% on mosaic-infected plants compared to its fecundity on healthy plants. Such a process takes place not only on leaves that are still growing or becoming senescent, but also on the mature leaves. These are more favorable for the biotic potential of the aphids when they show signs of disease than when they are healthy. In other words, according to Kennedy, 'The illness renders the entire plant, not just the mature leaves, more favourable to the vector.' (Fig. 7.)

Such a difference in behaviour is obviously linked to changes of a biochemical nature, of varying degrees of significance, that occur in the leaves as a result of the disease. These changes, however, are perceptibly different depending on the type of disease itself. Thus, 'mosaic'-type viruses lead to a reduction of the C/N ratio, which has the effect, as noted above, of inducing greater proliferation of *Aphis fabae*. With whorl or yellows-type viruses, on the other hand, no appreciable difference at all is found between the fecundity of aphids on diseased or healthy plants.

What is true for one species of aphid is not necessarily true for others, whose nutritional requirements may be slightly different. *Myzus persicae*, for instance, at the wingless stage of its development, prefers leaves infected with yellowing-type viruses to those suffering from the mosaic or 'curly-top' viruses (Mink, 1969).

Moreover, the aphid's reproduction is also related to the severity of the disease. This shows how closely the insect's biotic potential is linked to the plant's metabolism, Thus, on red clover (*Trefolium pratense*) that has been experimentally infected with the runner bean virus, yellow mosaic, Markkula and Laurema (1964) observed that the reproduction rate of *Macrosiphum* (*Acyrtosiphon*) pisum increased on plants displaying only moderate symptoms of the disease, while its fecundity decreased in proportion to the severity of the infection.

The same authors note that the concentration of free amino acids increases in diseased plants. On oat plants infected with barley yellow virus, the fecundity of *Rhopalosiphum padi* (L) increases in line with the availability of free amino acids in the plants. In the case of *M. avenoe*, however, reproduction rates remain the same. This would appear to be due to differences in the nutritional needs of the two species, as pointed out above.

Qualitative and quantitative analysis of the tobacco plant demonstrates important variations in the levels of free amino acids in leaves infected with the X virus, as compared with healthy specimens (Pojnar, 1963). Above all, marked increases are recorded in glutamic and aspartic acids, lysine, threonine, and alanine, as well as a reduction in glycine. Such phenomena imply changes at a nutritional level, and would appear to explain the repercussions on the behaviour and fecundity of aphids, according to their species.

Miller and Coon (1964) studied the influence of the virus itself on the biology of aphids. The virus involved was *Macrosiphum granarium*, feeding on barley infected with 'yellow dwarf' virus. The reactions of several groups of aphids were studied:

- aphids not infected with a virus, feeding on healthy leaves;
- aphids reared exclusively on virus infected plants;
- aphids fed for 48 hours only on infected leaves and then placed on healthy plants.

In the case of insects carrying the virus, the findings were:

a) an increase in the rate of development from egg to adult (a speeding up of the development cycle as shown by Michel (op. cit.) with Myzus persicae reared on tobacco treated with mevinphos),

b) increased longevity,

c) a longer period of reproduction,

d) as a result, a higher overall rate of multiplication.

According to the authors, these results show a 'biologically active' interaction between the virus and its vector. We believe that this interaction must be nutritional in nature.

Everything we have seen so far serves to explain the mechanism of the failure of the numerous chemical treatments used against the vectors. In fact, through their actions on the plant, the pesticides are liable, via trophic means, to increase aphid numbers. By inhibiting protein synthesis, they enrich the plant's tissues in soluble nitrogen, particularly free amino acids, which, in conjunction with reducing sugars, stimulate the aphids' fecundity.

The findings with regard to certain chemical pesticides are duplicated with plants infected by viral disease. But here a question arises:

IF THE APHIDS, PRESUMED TO BE VECTORS, GRAVITATE TOWARDS PLANTS ALREADY INFECTED WITH VIRAL DISEASES IN PREFERENCE TO HEALTHY ONES, TO WHAT EXTENT ARE THEY RESPONSIBLE FOR THE INITIAL INFECTION?

In other words, to what extent are plants that are healthy, or apparently healthy - that is, plants conditioned in a certain way - likely to become infected? This is the question that was already raised in Chapter V, to which we are compelled to return. We shall respond, at least in part, by briefly examining the case of 'parasitic complexes' in cereal plants.

2. Physiological conditioning of cereal plants and parasitic complexes

As we have seen, viral disease develops in the context of so-called 'intensive' agriculture. For us this term signifies the deployment of two

increasingly widespread techniques: large applications of chemical fertilisers, particularly nitrogen-based fertilisers, and (as a consequence perhaps?), ever-greater use of chemical pesticides, particularly fungicides, insecticides, and herbicides.

'In parallel,' there are considerable increases, at first sight inexplicable, in both viral and fungal diseases as 'parasitic complexes.' In these, according to researchers who helplessly witness this flood of infections, 'It is practically impossible to isolate the specific role of one constituent from that of the others.'

We have already shown how pesticides, most notably herbicides, are responsible for parasitic outbreaks. Here we would like to raise the question of fertilisation, that is to say, the nutrition of the plant. Together with any potential toxicities, a plant's nutrition is one of the principal factors in its physiology and, by extension, its resistance.

First point: differences in rates of multiplication in aphids in relation to the method of cultivation.

Kowalski and Visser (1983) recently studied the 'interaction of cereal aphids with winter wheat in relation to systems of 'alternative' agriculture.' They looked at this in relation to the amino acid content of the foliage.

'Conventional' cultivation, that is the part treated with agrochemical products, led to a much larger development of the aphid *Metopolophilus dirhodum* (Walk) than in the area cultivated 'organically.' The amino acid content of the leaves was also higher, particularly during July, which the researchers ascribed to an application of nitrogen at the beginning of April. But other chemical practices also appear to have played a role since, in their concluding remarks, the authors judge that 'an understanding of the plant's nutrition' plays a fundamental role in its resistance to insects, and thus in an integrated program of control.

Parasite populations were greater on winter wheat that had been chemically treated than where the cereal was cultivated organically. The latter suffered only minor infestation, due to greater resistance to aphid colonisation. As one factor in the population dynamic of the insect-plant interaction, Kowalski and Visser (op. cit.) stress the importance of free amino acids, particularly in their proportions relative to non-proteic amino acids.

Such results confirm the plant-virus-aphid interaction.

What are the effects of nitrogen fertilisers, especially artificial ones, on the provocation of deficiencies, especially deficiencies in micronutrients? Here we cite the following figures on copper levels in cows' milk:

- fields treated with nitrogen fertilisers: 14 microgrammes Cu per 100 ml;
- fields not treated with nitrogen fertilisers: 47 microgrammes Cu per 100 ml.

Second point: We must also address the effects of the type of fertiliser on the progression of viral disease and on the levels of aphid populations. Here we rely on the results of the highly original observations of Volk (1954) relating to plots treated with different kinds of fertilisers: potassium chloride (ClK), sodium chloride (ClNa),and calcium sulphate (SO_4Ca) . The crop was potato.

The plants bearing the most aphids grew on soil enriched with calcium sulphate. Nevertheless, the highest percentage of disease (leaf whorl) was found on chlorinated soils. This type of fertilisation was responsible for the highest level of viral infection.

The plants treated with calcium sulphate, in contrast, were relatively healthy, in spite of the higher numbers of aphids present.

Paradoxically, this would seem to confirm the absence of any relation between aphid infestation and the severity of the viral disease. In other words, the aphids would not have acted as vectors. However, the fertiliser's conditioning of the plant would be important. , As the author remarks, we should not forget the role played by 'chlorinated' soils.

We note that chlorine has a tendency to reduce the synthesis of amino acids, especially proteins, and to promote the decomposition of proteins. These are properties which, at the same time, sensitise the plants to their various parasites, including viral diseases.

Moreover, numerous chemical pesticides, especially herbicides, are chlorinated products. Under such conditions, we should not be surprised at either their poor selectivity or the sensitivity they create in the crop. This brings us finally to the 'parasitic complexes,' that can easily be explained if we take into account the effects of pesticides on the physiology of the plant being cultivated.

IV. Conclusions

1. Viral diseases follow the same rule as other infections: their severity, and even their existence, depends upon different environmental factors, mediated by their action on the metabolism of the plant.

2. In general, and in line with our trophobiosis theory, the criterion for this action resides in the value of the ratio of protein synthesis to proteolysis.

3. It seems that two principal factors can alter plant metabolism to the point of 'awakening' viruses that are 'latent,' or even inducing their formation at the very heart of the cell. These factors are the nutrition of the plant and the possible toxic effect of fertilisers or pesticides on it.

4. By ignoring such effects of pesticides, and aiming only at chemical destruction of the vectors (chiefly aphids), the deployment of pesticide intervention has led to failure. There are at least two reasons for this:

- it has not yet been proven that any particular aphid is responsible for the transmission of a virus, something that seems to depend above all on the physiological state of the plant;
- after an initial fleeting impact on the pest, pesticides can make the plant more susceptible to both viral diseases and aphids, through their effects on the physiology of the plant.

Here, as for other parasites, it turns out that sensible pest control against viral diseases resides in the adequate conditioning of the plant, either by achieving an optimum rate of protein synthesis, thanks to balanced fertilisation and correction of deficiencies, or by avoiding toxic effects, which seem to arise especially from the uncontrolled use of chemical (nitrogenous and chlorinated) pesticides. We will give some examples in the last part of this book.

Chapter 7

The Impacts of Grafting on the Scion's resistance

I. Objectives and Consequences of Grafting

In Chapters IV and V, we have already touched on grafting and the susceptibility of the scion toward disease. We now return to this, in order to look at the problem in a more general form and to establish an idea of the phenomena at the onset of susceptibility or resistance of the scion.

To recall the justification for this technique of grafting; it is necessary for the preservation of the variety and for its rapid reproduction. A stock adapted to the soil allows cultivation of a variety that would not otherwise be possible. This is the case, for example, for peach trees on soils that are not *a priori* suitable for them.

Moreover, while it very often happens that grafting sensitises the scion toward various parasites, it can also lead to resistance. For example, grafting against the viral disease Sharka.

Finally, grafting can be a last resort against a pathogenic agent through a process of 'tolerance,' if not destruction or dissuasion. This was the case, celebrated in agronomy, with the regeneration of French vineyards by the use of American rootstocks to stave off *Phylloxera*.

The physiology of the stock-scion composite

Grafting creates a new organism. It is a 'composite' or, to put it better, a 'chimaera.' Its properties are neither those of the rootstock plant nor those of the scion, but are distinctive to the new organism that has been created.

However, the scion supplies the roots with carbohydrates and other substances developed in the leaves, while the roots abstract mineral elements such as K, Ca, Mg, S, and micronutrients from the soil.

Moreover, the physiology of the composite proves to be dependent on two selections:

- at the level of the roots, chiefly through the uptake of cationic elements;
- at the joint itself.

But disease occurs mostly on the scion. This is the case with the famous apple stocks collected at East Malling, in Britain. The variety M1 displays great sensitivity to the environment and shows high need for potassium. At the same time it encourages the development of Oidium, scab, and canker attacks on branches of the Cox variety. Numerous stocks, besides, are infected with viruses.

In fact, we are faced once again with a 'parasitic complex.' We should not be surprised, then to encounter one on grafted vines. We now intend, to look at the repercussions of grafting on the grapevine, a technique that has been put to use on a huge scale.

II. The severity of diseases of the vineyard in relation to its reconstruction through grafting

One of the first diseases of the grapevine for which grafting has been held responsible was *Botrytis cinerea*. This is also in resurgence, as we have seen above, because of the use of certain chemical fungicides, such as dithiocarbamates.

> 'Grey rot has always existed in French vineyards with varying degrees of severity. But during the past twenty years, since the replacement of the whole country's vineyards by means of grafting, it has become frequent and extensive. It is only since then that we have begun to worry about fighting this disease.'

With these lines Perrier de la Bathe, a teacher at the Saintes Agricultural College, began an article on grey rot in August 1904 in Revue de Viticulture. Certain types of vine, such as 'folle blanche' are especially affected. When grafted, which makes it more 'vigorous,' this vine can hardly be cultivated any longer due to its excessive susceptibility to *Botrytis*.

Esca or apoplexy, caused by attacks of the fungus Stereum hirsutum, has also become far more virulent since the mass grafting of French vineyards. While this parasite had previously been considered as a pathogenic agent in older vines, it now began to attack young stock. This hypersensitivity was attributed by some to premature ageing of the rootstock and to production of tannin. Clearly there is a connection between attacks of *esca* and the alteration of the scion's metabolism by the grafting process.

There is also good reason to ask to what extent grafting may have been responsible for the severe outbreaks of mildew from 1880 to 1885, a few years after the re-establishment of France's vineyards. In the Roussillon region, for example, the first rootstocks were planted in 1883, and within six years the entire stock of vines had pretty much been replaced. But mildew raged - with the help of the climate - in 1885, 1887, and 1889, with losses reaching up to half the yearly average harvest.

Of course, some will say these are merely coincidences. But since 1873 Maxime Cornu - who name is forever linked to that of *Phylloxera* - had warned against the dangers of introducing American vines into France because in their own homeland they had been attacked by *Peronospora viticola*.

Was the American rootstock able to pass on to the scions its 'susceptibility' to disease, including mildew, as well as *Botrytis* and *esca* (not to mention the viral diseases, to which we shall return later)?. Is what we encounter here simply a 'parasitic complex' such as we have already seen in crops as varied as cereals and fruit trees?

We shall perhaps understand this mechanism better by looking at the physiology of *Riparia*, the rootstock most widely used for re-constituting vineyards, and still amongst the most widely used today.

III. The physiology of *Vitis riparia* and of certain 'composites' of *Riparia* and scion

The American vine *Vitis riparia* was, from the start, the most popular, with millions still growing in the South of France [1984]. One of *Riparia*'s characteristics is to give certain vines a bushy shape so that in French one says of these vines that they '*se riparient*' ('become bushy').

This bushiness is found in other plants, particularly the apple tree. It is called 'witch's broomsticks.' These malformations do not have a parasitical origin. They are caused by a calcium deficiency, which in turn may be caused by a boron deficiency.

This problem, also known in apples as 'proliferation' disease does not rely on diseased apples for its dissemination. It depends primarily on the soil and its composition (Bovey, 1971). However, levels of infection are higher with certain rootstock varieties, such as E. M. XI, than with others, such as E. M. XII. This clearly confirms the influence of the rootstock on the plant's nutrition, and with it the resistance of the grafted plant to this type of infection.

In vines grafted onto American vines, various kinds of deficiencies have emerged. For instance, grafted Merlot shows a clear deficiency in magnesium. In the branches of the plant, the K/Mg ratio varies according to the rootstock (Table 9) (Duval-Raffin, 1971).

Table 9: Variations in the K/Mg ratio of Merlot according to the nature of the root system.

Root system	Mg (mg/kg dry weight)	K/Mg ratio
Ungrafted merlot	2110	5.39
Merlot/riparia x berlandieri SO 4	765	14.27
Merlot/riparia x rupestris 3309	1009	11.28
Merlot/vitis riparia	912	17.09

In the case of grafted Merlot, therefore, we find a Mg deficiency, particularly when grafted onto *Vitis riparia* where the value of the K/Mg ratio is the highest. Duval-Raffin (op. cit.) concludes: 'Grafting in soil poor in magnesium increases the risk of deficiency of this element in Merlot.'

The same author showed that *Riparia* is also poor in calcium. This could certainly explain the 'riparisation' of certain plants, particularly in certain soils. Such deficiencies, especially of magnesium and calcium, are also found in fruit trees: walnut (Gagnaire and Vallier, 1968), citrus (Wallace et al., 1953), peach (Grosclaude and Huguet, 1981), etc. Blanc Aicard and Brossier (1962) consider that the stock determines the cationic balance in the plant, particularly the ratio of divalent ions to monovalent ions. As we have seen above, this ratio in turn governs the plant's physiology, especially the level of protein synthesis, or the ratio of protein synthesis to proteolysis. This is a criterion of the plant's receptiveness to its parasites.

All the same, the effects of grafting on the scion's metabolism are not confined to the elements N, K, Ca, and Mg. This would not be so, a priori, because we already know the relations between the major elements and the micronutrients, such as B, Zn, Mn, Mo, and I etc. In fact, various studies have shown the influence of the rootstock on the micronutrients. For example, studies carried out by Labanskas and Bitters (1975) on citrus trees, establish the variations of K and Ca, as well as of Cl, B, Cu, and Fe. This also includes sulphur.

Carles et al. (1966) have shown that on two different types of grafted vine (18,815 and 12,375 Seyve -Villard), grafting leads to pronounced differences in mineral levels, especially copper. In ungrafted vines, levels of copper are significantly higher than in grafted ones. Mn levels also vary, being notably heightened by Riparia X Rupestris 3309.

Deficiencies, viral and other diseases

We have also received a startling demonstration of a graft-induced deficiency through the appearance of chlorosis. It has been affirmed that in grapevines, chlorosis makes its appearance with grafting. In non-grafted vines, in fact, chlorosis remained subdued and did not prevent the cultivation of afflicted varieties, even in conditions which promote chlorosis (for example, vineyards in the Cognac region). The varieties were the same as the ones used nowadays - merlot, cabernet, sauvignon, semillion, etc. - but were tolerant to chlorosis.

This 'tolerance' could arise either from the plants' low iron requirements, or from the properties of their roots, allowing them to easily dissolve and absorb the iron present in the soil.

After the reconstruction of the French vineyards using American rootstocks, certain stock-scion combinations were found to be much more sensitive to chlorosis than varieties cultivated on their own roots. This is because the iron requirements of the aerial system of the combination were no longer satisfied by the poor ability of certain root rootstocks systems to mobilise iron from the soil.

According to our concept of trophobiosis, the grapevine's susceptibility to chlorosis through iron deficiency could also produce susceptibility to other diseases, particularly mildew. Is this perhaps how *Riparia*, which also causes deficiencies of calcium and perhaps boron, could transmit susceptibility toward mildew to the scion? It is certainly interesting to note that Millardet himself pointed out that treatments based on iron sulphate, when added to lime, gave excellent results in treatments against mildew.[1] This would lead us to admit that a chemical's effectiveness comes from internal action and through correction of deficiencies. This would constitute a revolution in the therapy for diseases, as we have pointed out in our previous work. We will now look at the relation of viral diseases to the technique of grafting the grapevine.

What are the relations between grafting and 'receptivity' for viral diseases and other infections? By 'grafting' we mean the modifications of a biochemical nature that are caused in the graft itself.

These same deficiencies, notably P and Ca, occur not only with *Vitis riparia*, but also with other rootstocks of the grapevine. Halmi and Bovay (1972) showed that *Riparia X Rupestris* 3309 - a widely-used rootstock, often used as a reference point - leads in the chasselas grapevine to nutrition high in N and K, average in Mg, and poor in P and Ca.

When European varieties are grafted onto the same 3309 rootstock, Mikeladze (1965) was able to show that:

- the process of synthesis in the roots of the rootstocks slows down, the quantity of amino acids decreasing in correlation with an accumulation of sugars that are not used in the transformations of the Krebs cycle;
- in the leaves of the scion we observe an accumulation of amino acids due to the slowing down of their conversion into proteins (and perhaps into other important compounds) required for the plant's continued growth and the circulation of ions.

In short, the physiology of the whole grafted plant, particularly the scion, reveals inhibition of protein synthesis. This reveals a metabolism with a heightened susceptibility to various pathogenic agents.

An experimental demonstration of this 'susceptibility' is provided by the difference in the rate of multiplication of mites (or 'red spiders') on the Merlot vine, according to the nature of the rootstock.

The biotic potential of the 'red spider' *Panonychus ulmi*, feeding off Merlot with *Vitis riparia* as the stock, is on average 70% higher than for Merlot grafted on Berlandieri x Riparia 420 A (fig.8A and 8B) (Carles et al., 1972).

Analyses of the foliage seem to show clearly that these differences in the mite's longevity and fecundity have their origin in higher levels of amino and amide nitrogen in red merlot grafted onto *Vitis riparia* (fig 8C).

A physiological state in which proteolysis predominates would seem to favour a certain 'laziness' in protein synthesis, itself induced by certain deficiencies such as those already shown in the case of calcium and iron (to say nothing of other possible deficiencies still unstudied, such as boron). This confirms what we have seen above, that deficiencies are clearly the origin of viral diseases that, like other diseases, are rampant in plants rich in soluble nitrogen.

We can see, therefore, why there were at first such dramatic outbreaks of diseases of the grapevine after the reconstitution of the French vineyards by mass grafting - for instance, the development of Botrytis - that captured the attention of the researchers. (We note in passing that the process of 'sensitising' the vine by the rootstock is parallel to that brought about by the use of the dithiocarbamates zineb, maneb and propineb against grey rot) (Chaboussou et al., 1966).

Manifestations of viral diseases are less striking, but no less real. Their spread has often been linked to *Phylloxera*, frequently accused of being the vector - for example, in the case of infectious degeneration. On this subject Branas remained sceptical, only noting that there was a link between the invasion of *Phylloxera* and the spread of the virus.

However, it seems that an important factor has been forgotten. As highlighted by Ciferri, this is the role played by the plant itself, serving as nutritional host to both parasites. Everything that was said before about the physiology of scions on American rootstocks shows that if phylloxera is responsible for the spread of viral diseases of the grapevine, this is only at a secondary level. It occurs because the European vine, to make it 'tolerant,' has been grafted onto American plants. The latter, as a result of the deficiencies and inhibition of protein synthesis that they induce, make the scion susceptible to viruses and other diseases, particularly to infectious degeneration.

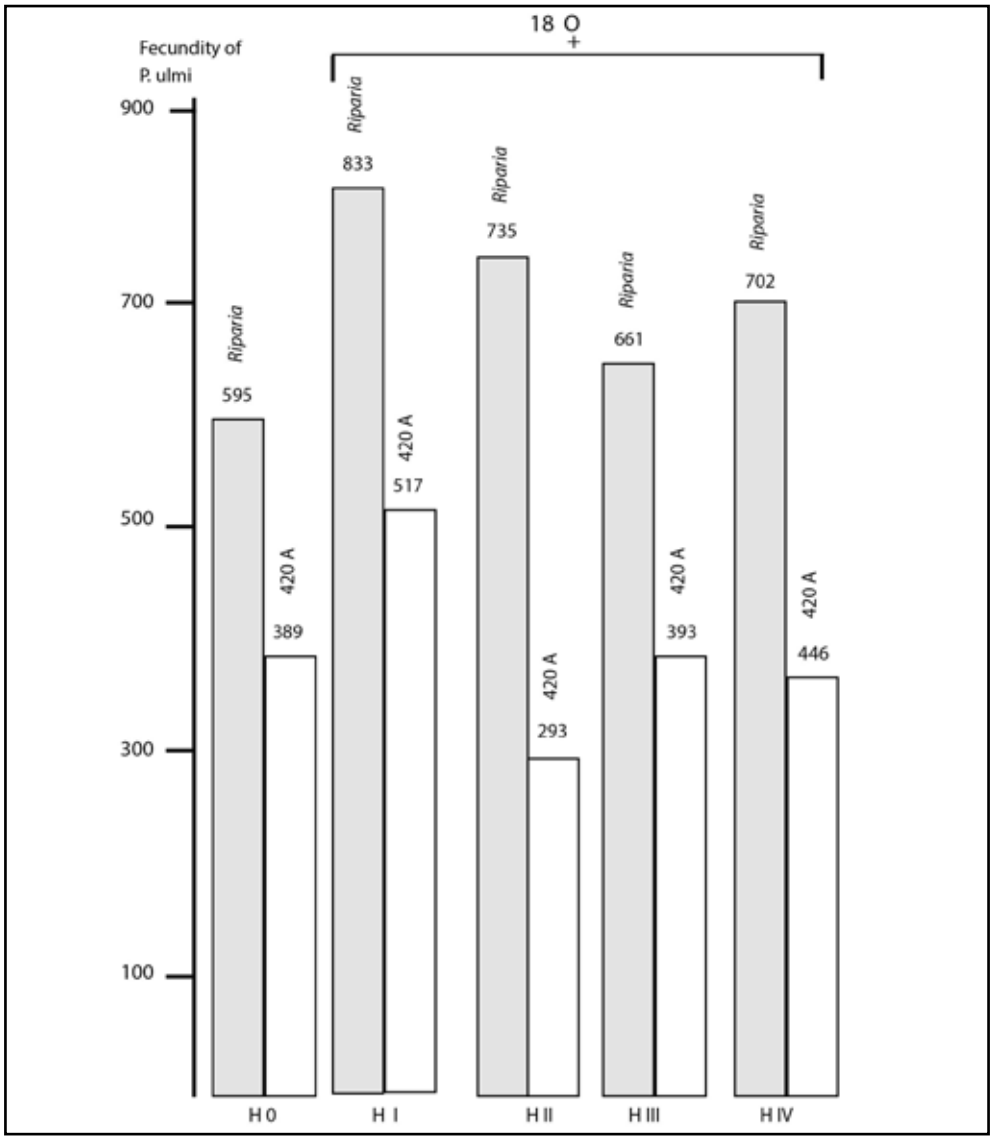

Fig.8: Effects of the nature of the grapevine stock on the fecundity of aphids raised on Merlot rouge grafts.

Differences in fecundity on the Merlot rouge vine, according to whether this vine is grafted onto Vitis riparia or onto Berlandiera x Riparia 410A.
N.B. - The bars O, I, II, III, IV indicate the periods when the aphids were raised, at intervals from mid-May to 20 July.
This graph does not show the results of the breeding period H V (20 July - 15 August) or H VI (5 September - 10 October), where the results are the inverse of those of the first four periods. (See the following diagram.)

We know, moreover, that Vuittenez has shown that the nematode Xiphinema index transmits infectious degeneration. Previous considerations, however, that rank the vine's physiological state as being of the highest importance for the plant's 'receptiveness' to disease, reduce the role of vectors to a secondary level.

We must in fact distinguish clearly between 'resistance' and 'receptiveness' to *Phylloxera* without, however, underestimating the interest of agronomy in grafting, which permits the cultivation of grapevines in '*phylloxeric*' soils. In this regard, Maillet (1957) observed that, in general,

> 'the plants with the greatest reputation for their 'resistance' to *Phylloxera* (*Vitis riparia, Vitis rupestris*) were the ones with the most abundant and most thriving *Phylloxera* populations, with a maximum of ovarioles leading to a maximum of egg clutches.'

We have already seen that the 'biochemical' and 'trophic' reason for this is the same as for the susceptibility of grafted vines to the 'parasitic complex' made up of *Botrytis*, mildew, viral diseases, *Phylloxera*, and mites. A similar complex is found in the apple tree, as pointed out earlier, where the rootstock MI. is considered as 'weak' or 'weakening' for the scion.

In short, in regard to grapevines, Maillet (op. cit.) could justifiably state that 'grafting has been and always will remain only a palliative; *Phylloxera* still remains unconquered.' Such a critique of grafting is all the more justified, as we have shown, because grafting can promote a complex of diseases.

Let us make it clear, however, that such effects stem from repercussions of the rootstocks employed at the time of the re-construction of the vineyards. Things could be quite different with new rootstocks, such as the one recently developed by the INRA at Bordeaux. This type, *fercal*, has neither *V. riparia* nor **V. rupestris** in its ancestry, and its genetic characteristics are *Berlandieri x Colombard* no.1 x 333 EM. We note above all that this variety has shown itself far superior in its resistance to chlorosis. We could therefore assume that it would have little or no iron deficiency, which might make is more resistant to various parasites.

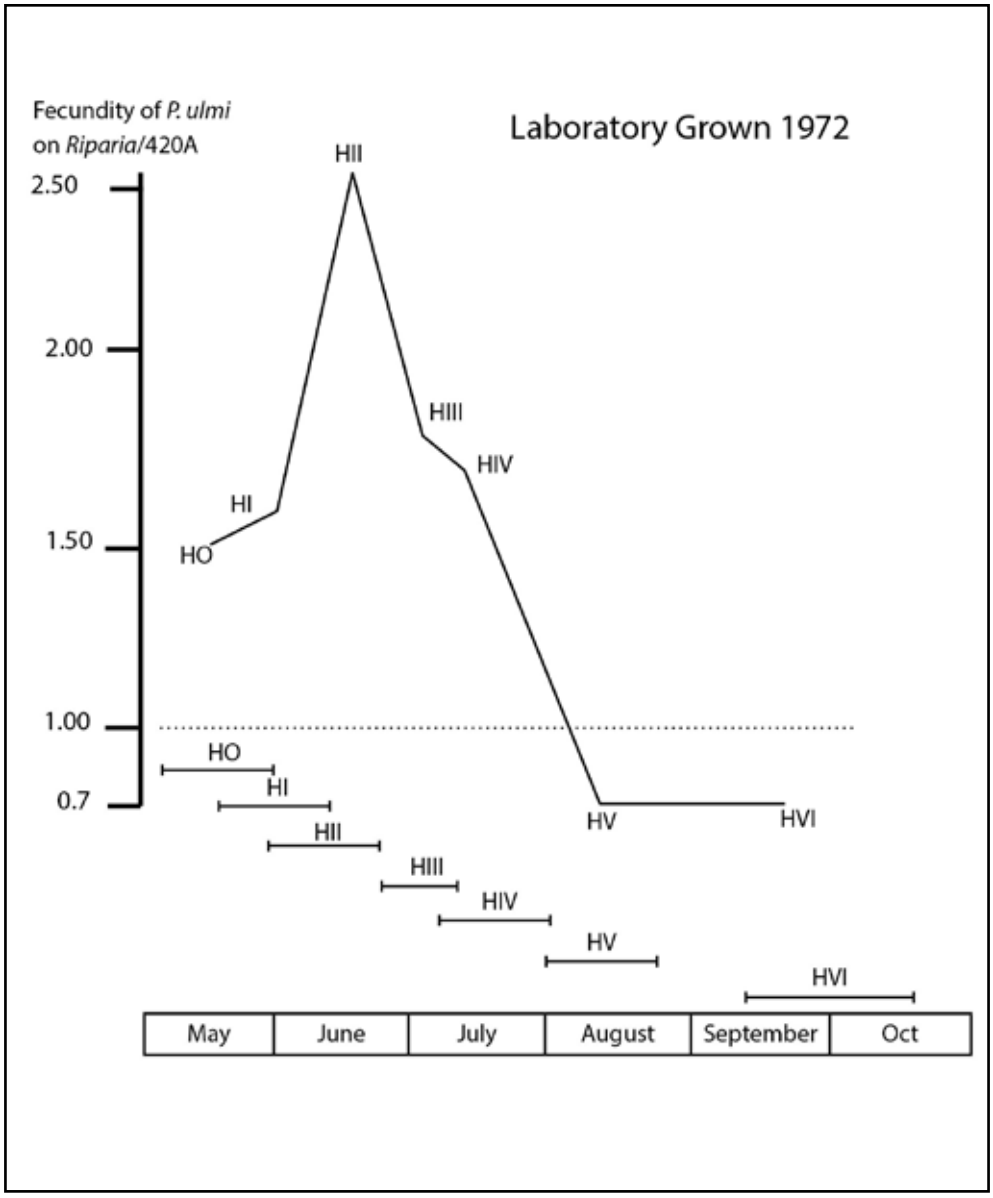

Fig.8B: Evolution, over the course of a season, of the relations of fecundity of P. ulmi on Merlot rouge, as affected by two types of stock: *Riparia* and *420A*.

N.B. - The reader will note (due to the biochemical evolution of the grapevine as it approaches senescence) the inversion of results, from the beginning of mid-July, for the last two groups of aphids.

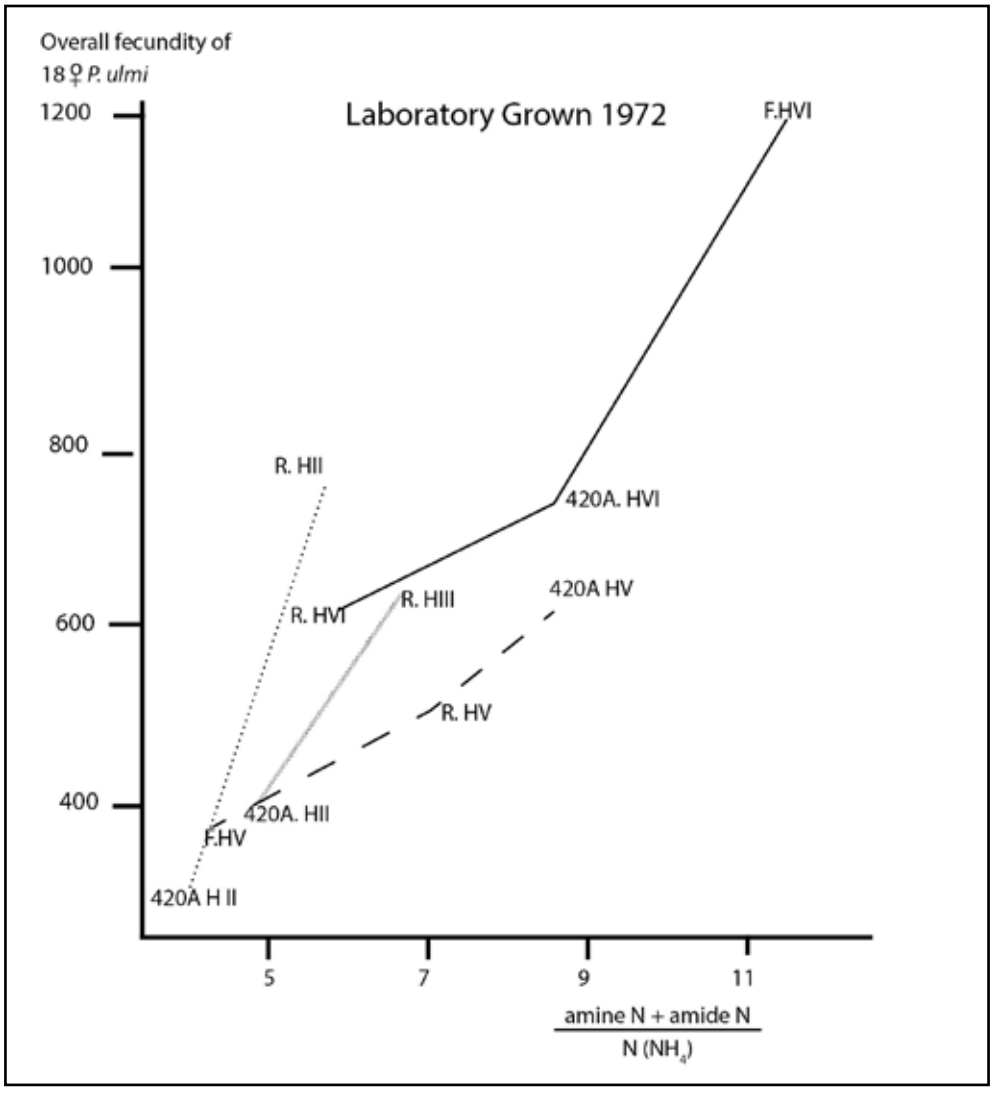

Fig.8C: Fecundity of Panonychus ulmi in relation to the ratio: amine N and amide N / N (NH_4), in grapevine leaves for the aphid populations II, III, IV, and VI.

N.B. - Apart from Merlot rouge grafted onto 410 A and Riparia, the populations VI and VI involve a third type of foliage - that of Merlot rouge on its own roots, hence the initial F (free).

The reader will note the very significant inversion of the results for the groups V (mid-July to mid-August) and VI (early September-early October), with respect to foliage F. The latter, resistant to aphids until mid-July, becomes susceptible at this stage as a result of senescence: the breakdown of proteins and the consequent elevation of amino acid levels.

IV. Rootstock-induced resistance in the scion

As we shall now show, certain grafting techniques permit transmission to the scion of better resistance to diseases, particularly to viral diseases.

Sutic (1975), who attempted to stimulate plants' resistance through grafting, found it was possible to combat various diseases ('neck rot' in citrus trees (*Phytophthora parasitica*) and apple trees (*Phytophthora cactorum*), and root rot in peach trees (*Phytophthora cinamoxi*)) by grafting onto appropriate rootstocks.

Likewise, rootstocks that seem 'resistant' to the Trustees virus in apple trees are at least 'tolerant' to 'latent' viruses, even to *Phytophthora*.

We have already seen with regard to *Phylloxera*, that what is involved is a tolerance. This allows the vine to survive and to reproduce, but also sensitises it to diseases, including viral ones such as infectious degeneration. This is the case, at least, with the rootstocks habitually used up to now.

Is it possible to find rootstocks capable of inducing a genuine 'resistance' in the scion, or rather, immunity to its principal parasites? We have seen that there is a need to distinguish clearly between the presence of the pathogen and the damage caused by 'disease'. If damage is avoided through a technique such as grafting, then we have a method of control even if the parasite to a certain extent survives.

There is one example of the deployment of rootstocks against the disease Tristeza in citrus trees, identified for the first time in 1955 in the Meyer lemon. Frezal did not find any symptoms of the disease on the Antillese Olimettier (*Citrus aurantifolia Christ*. Mexican variety) even though in 1967 these same trees were found to be carriers of the Tristeza virus (especially the *Owari* Satsumas). In other words, the physiology of the scion, modified by the special nature of the rootstock, prevented multiplication of the virus.

If this can be achieved with a virus, the same could be achieved against every other infection, for instance a mycoplasma, a cryptogamic disease, or even an insect pest. If, as we have shown above, certain rootstocks through their metabolic effects promote the establishment of veritable 'parasitic complexes,' they can create resistance to the apple tree woolly aphid.

Thus Soulard (1952) suggests that the rootstock's immunity is transmitted to the scion. In the case of the woolly aphid, 'the few feeble colonies which appear on it grow miserably and are radically destroyed by the first generations of its parasite, *Aphelinus mali*, before having caused any damage.'

In fact, 'grafting as a procedure for vegetative protection' was successfully used by Sutic (op. cit.) to control Sharka disease. In this case infected Pozegaca buds were grafted onto the crown of the Malevezka plum. Malevezka is particularly resistant to the disease, and the symptoms of Sharka disappeared after three to four years from the shoots coming from the infected buds. Symptoms did not re-appear later, either in the leaves or in the fruit of the shoots.

Analogous techniques have produced similar results. Sutic (op. cit.) reports that in Yugoslavia and Romania studies have confirmed the active role played by *Prunus spinosa var. dasyphylla* in the 'vegetative protection' of plum trees infected with Sharka. The same phenomenon occurs with apricot trees.

Such results should be compared to those obtained through affranchissement which, according to Bouché-Thomas (1948), protects the apple tree from canker, woolly aphids and cochineals. They also raise the question of the nature of the biochemical factors causing this immunity or 'resistance.' On this subject, the results obtained by Do Vale (1972) can, it seems, give us useful information.

Do Vale (op. cit.) carried out research on the biochemical effects induced by various rootstocks grafted to Lisbon lemon. He concluded that:

> 'The combinations giving good results produced low levels of soluble sugars in spring leaves, close to the levels found in non-grafted trees. At the end of the season, the sugars were significantly higher in the leaves of the scions having produced the best results, results as good as those in their non-grafted counterparts. Where performance was mediocre, high quantities of total sugars were found in the spring leaves.'

All this would appear to confirm what we have seen above with regard to metabolic differences between grafted and un-grafted trees, primarily manifested by deficiencies. In other words, the low level of protein synthesis in grafted trees yields only mediocre results.

These results are similar to other grafting successes in relation to viral diseases, such as those obtained by Morvan and Castelain (1972) in controlling chlorotic whorl in the apricot tree by grafting *Prunus spinosa*.

It would undoubtedly be interesting to be able to standardise plant metabolism by a biochemical criterion whose importance to the metabolic processes is known. For example, this could involve the link between the cations K + Na/Ca + Mg or K/Ca as a first approximation of the value of a rootstock. This would also show the potential resistance that it would be likely to transmit to the scion.

From another perspective, it would also allow the prediction of potential deficiencies in micronutrients.

V. Grafting and the quality of the product

Since grafting affects the scion's physiology, it must also affect the quality of the harvest. This question has not escaped the attention of viticulture specialists. The quality of wine is linked to the sugar level in the grapes at a certain stage in the vine's vegetative cycle. Since the sugar cycle and sugar levels depend on the nature of the rootstock, this also controls the rhythm of growth. An early halting of the vegetative stage, allowing the grapes to mature well and thus yielding a good quality wine, is best achieved in general with a weak rootstock. This is the case with *Vitis riparia*, a rootstock likely to promote disease, not to mention spread of *Phylloxera* in the roots.

So how can we reconcile wine quality with the plant's resistance? Could disease control reside in correction of deficiencies through treatments of a nutritional kind? This is what we shall examine in the last part of this work.

Notes

1 Using the formula: 4 kg of iron sulphate to 20 kg of lime. Cf. International Congress on Phylloxera, Bordeaux, 1881.

PART THREE

Agriculture Techniques and the Health of Crops

'How the devil can all these diseases, already catastrophic enough, blackmail cereal crops in such a fantastic, ceaseless way? What sort of curse has been placed on these fields? Is there no way out of this infernal spiral?'

(Publicity booklet of PROCIDA pesticide company, 1980.)

Chapter 8

State of Health in Relation to the Method of Cultivation

I. 'Intensive' cultivation of cereal crops and the xxplosion of so-called 'diseases of progress'

1. Parasite intensification: proliferation, not 'resistance' to pesticides

We will now take up the disturbing questions that farmers themselves are raising about the causes of the proliferation of parasites after treatment by certain pesticides.

It is only in the last fifteen years [to 1984] - that we have seen such a spread of disease in cereal crops. We must also remember that until recently many of these diseases were unknown, or their effects insignificant. This is the case, for example, with *Fusariosis* and *Septoriosis*.

Other diseases such as take-all in cereals and crown and sheath rot in rice (*Gaeumannomyces graminis*) and *Rhizoctonia* are also spreading beyond the norm. 'Barley is often subject to an attack by a parasitic complex (*Rhynchosporiose, Oidium,* take-all) in which it is practically impossible to isolate the specific role of any one of the constituent parts' (Harranger, 1982).

Here again we encounter 'parasitic complexes', whose origin we can assess only when we take the physiology of the host plant into account.

There is also the drastic spread of *Helminthosporiosis*, with the appearance of new species, and also of viral diseases such as Dwarf yellow in barley (which also develops in other cereals).

A researcher at INRA concluded that these developments 'appear to be linked to the increasing acreage devoted to the cultivation of this crop, and to the intensification of agriculture.' This researcher does not go into further detail.

Even the pesticide company whose discouraging statement we quoted at the start of this section bears witness to this uncertainty when it states: 'In fact, endemic diseases constitute a veritable baseline disease complex which must always be taken into account....If certain diseases have so rapidly become this severe, there must be other aggravating factors not accounted for simply by natural and climatic conditions' (Procida pesticide company).

In our view what is involved is *an increase in susceptibility* of the cereals through what is usually called the 'intensification' of agriculture. This term covers two essential processes:

- increased fertilisation, in particular the massive use of nitrogen fertilisers.
- the equally massive use of chemical pesticides, especially herbicides, and fungicides.

A researcher at ENSAIA in Nancy was able to establish that the main factors in the development of 'brown stains' (*Helminthosporiosis*) were:

- early and dense sowing;
- nitrogenous fertilisers;
- fungicide treatments applied during the course of vegetative growth.

It should come as no surprise that a non-persistent chemical acting on the surface as a fungicide can also alter the cereal's physiology, and thus its susceptibility to its various parasites. Moreover, some researchers are sceptical about the efficacy of systemic fungicides for cereal diseases (Parmentier, 1979).

A similar indirect influence via the physiology of the plant in this case, cereals led to a belief that the pathogenic agent (various fungi) had developed resistance to the fungicides. In the case of Pietinverse, some researchers speak of the existence of varieties 'resistant' to BMC (Benomyl, methylthiophanate, carbendazine). *But we have seen, particularly with respect to the grapevine, how much credit we should give these alleged 'resistances.' What is involved is a proliferation of the specific parasite through stimulation of its biotic potential, based on a nutritional effect.*

What is the mechanism of this susceptibility? Here *herbicides* apparently come into play, according to what we know from various studies, some of which have been analysed in the course of the chapters on viral diseases.

Only through effects on the physiology of the cereal could such developments of viral diseases be unleashed. References in the bibliography will back this up.

The work of Altman and Campbell is particularly impressive (1977). This gives a general survey of the role of herbicides in the development of plant disease. The authors recapitulate the many cases of disease brought about by pesticide use:

1) Inhibition of plant growth by application of a herbicide leads to an increase in susceptibility to disease: for example, beetroot treated with cycloate, a carbamate. As for the mechanism, Altman and Campbell (*op. cit.*) suggest that in fact one of the causes of development of disease could be an increase in the nutritional substances available to the pathogen.

2) Furthermore, treatment of soils with cycloate produces much higher levels of glucose and mineral salts in the plants that grow there. These nutritional substances are exuded through the leaves. They lead to susceptibility to pathogenic fungi that feed on the leaves, by an effect that is nutritional in nature. This is an inhibition of protein synthesis, which enriches the tissues in soluble substances that make the plant susceptible. The chief substances are amino acids and reducing sugars.

Some pesticide companies seem to be aware of these major disadvantages, and also of those that could result from further expansion of the market in herbicides. Rather than renouncing them, they appear to be trying to counter the disadvantages by bringing out 'antidotes,' substances that would allow a herbicide to be selective in relation to the crop to be weeded. Even if this development should by chance prove successful, it will not address the impact of herbicides' on the life and composition of the soil, which we shall discuss further on.

2. The effects of atrazine and 2.4D on the development of various parasites.

McKenzie *et al.* (1968) showed that with corn resistant to 'dwarf mosaic virus' (MDMV), severity of the symptoms increases with the dosage of atrazine. With a dose of 20ppm of atrazine applied to the soil, symptoms increase by up to 100%. (Fig. 5).

These results were confirmed subsequently (McKenzie *et al.*, 1970). They also show that atrazine profoundly alters the biochemicalcomposition of the foliar tissues, particularly in P, K, Ca, Fe, Cu, B, Al, Sr and Zn.

In addition, Gramlich (1965) showed that treatment in the field led to a much higher percentage of nitrogen in corn and sorghum halepense than in the controls. This was the case with *weak doses* of atrazine. This explains the increased susceptibility to parasites brought about by this chemical. This becomes even more apparent when one considers that, with grasses, simazine increases the effectiveness of atrazine. This increases the risk of problems in crops through the persistence of this chemical, along with its negative effects on protein synthesis. These problems become more severe not only through phytotoxicity but also through susceptibility, by means of mechanisms that are now under discussion.

Altman and Campbell (*op. cit.*) studied the effects of simazine on wheat. They found that, in comparison with controls, it increased nitrogen levels by 30%, slightly increased the levels of the amino acids threonine and valine, and very clearly increased the levels of asparagine. As we shall see confirmed later, this amino acid is particularly 'effective' in *nutritionally* promoting the biological potential and proliferation of parasites, especially pathogenic fungi.

From their studies of the literature, moreover, Altman and Campbell conclude that the inhibition of protein synthesis by these herbicides can explain many of their harmful effects. Carbamate herbicides can in fact inhibit utilisation of amino acids by vulnerable species. These authors also note that these chemicals have a structure in common with proteins – that is, CO-NH. They conclude:

> 'Since 1945, numerous reports tell of increased losses through insect and pathogen attack, despite ever greater efforts at pest control. While it is difficult to calculate to what extent these increases are due to the biochemical and ecological impacts of herbicide use, herbicides have been blamed in numerous cases for causing problems of parasitism in the treated plants.'

This loss in plants of 'resistance' to insects and disease can be explained through the availability of improved nutrition for the parasites. This occurs through inhibition of protein synthesis, as well as through enrichment of the tissues with soluble substances (amino acids and reducing sugars).

In concluding their study, Altman and Campbell pointout: 'It is possible that the use of herbicides can alter the susceptibility of the plant being cultivated.'

The examination we have made of simazine which also leads to proliferation of leafhoppers on maize - is also relevant, as we shall now see, to the artificial hormone *2,4D*. Oka and Pimental (1976) in fact showed that treating maize with *2,4D* leads to:

- proliferation of maize aphids, in proportion to the dose used;
- swarms of maize *Pyralidæ* (*Ostrinia nubilalis*).

The statistics are the following (Table 10).

Table 10: Effects of *2,4D* on aphid and pyralidæ populations on maize

Doses of 2-4 D	Aphids (in September)	Pyralidæ - % of maize attacked
Controls	618	16%
0.14 kg/ha	1,388	24%
0.55 kg/ha (standard dose)	1,679	28%

These results came from studies carried out in 1973; and repeated in 1974 with similar results. As to the average weight of *pyralidæ* pupae on maize plants, treated respectively with doses of 5.2 and 80ppm of *2,4D*, this was found to be significantly higher than on the controls. This fact seems to confirm the nutritional advantage for pyrales of maize treated in this way.

Through other tests, furthermore, Oka and Pimentel (*op. cit.*) also showed that the susceptibility of maize to *Helminthosphoriosis* was increased by use *of 2,4D*. This appears to confirm the idea of a 'parasitic complex,' in which noxious insects, pathogenic fungi, bacteria, and even viruses can co-exist on the same plant.

The results of Oka and Pimentel explain the observations made by Adams and Drew (1965) with aphids. These showed that proliferation of *Rhopalosiphum padi* and *Macrosiphum avenae* on oats and barley was associated with the application of *2,4D*. One can therefore imagine the difficulty of interpreting results when an aphicide is applied simultaneously with a herbicide, to restrain from the outset the development of aphid populations.

However, whenever the herbicide is applied on its own, we see that aphid populations, which vary from year to year, are always higher than on the controls: 108% higher in 1962, and 205% higher in 1963. The authors put these increases down to the negative effects of the herbicides on ladybirds. But there are grounds for surmising indirect repercussions, through effects on the physiology of the specific cereal.

It should come as no surprise if, by an analogous process, the Pyrethrum-type insecticides used against maize *Pyralidæ* should also make aphids proliferate. Based on what we have already seen, these increases in *Pyralidæ* would appear to be linked to herbicide use, particularly in the central and Parisian basin regions of France. Significantly, it is the agricultural experts themselves who end up wondering about such 'abnormal' proliferation and what extent it might be linked to an increased susceptibility of maize, due to 'a modification of the physiology of the plants which would make them more appetising'(*Agri-Sept*, 17th April, 1981).

Such close inquiry is encouraging. In fact, it shows that these 'biological imbalances' cannot be explained by the (real or, more often, alleged) destruction of natural predators. An entirely different explanation is called for.

To sum up, these proliferations of parasites on cereal crops result from an alteration of the plants' metabolism by the use of chemical pesticides. Most often these are herbicides, but insecticides and fungicides can also be involved. So too can chemical fertilisers.

Apart from *Pyralidæ* and aphids, other pests can proliferate on cereal crops for similar reasons. This is particularly true of nematodes. While in the past, diseases caused by worms have been neither numerous nor serious, four types of nematodes that damage cereal crops have recently been recorded. A nematode specialist at INRA was led to acknowledge, 'The evolution of agricultural methods has encouraged the appearance of species not previously suspected or introduced from elsewhere.' (Ritter, 1981).

It is strange that, amongst these 'modifications' in agricultural methods, there is no mention of either the new and intensive use of herbicides or the intensified use of fertilisation, particularly of nitrogen fertilisers.) However, as was demonstrated long ago: 'On the oat plant, *2,4D* stimulates the development of *Ditylenchus dipsaci* by modifying the biochemistry of the plant, rendering it more suitable for the parasite's diet' (Wester, 1967; Rambier, 1978).

Various fungicides cause proliferation of nematodes, particularly on strawberries and onions.

It has been shown that levels of amino acids and amides in the roots of nematodeinfected plants are always higher (from 17% to 316%) than those in healthy plants (Hanks and Feldman, 1963). This seems to clearly explain the 'integration' of nematodes in the 'parasitic complexes' recorded since about 1969 or so - in other words, since the intensive use of herbicides began.

It also seems likely that the effects of fungicides and the type of fertiliser intervene in such a process. These are mainly nitrogen fertilisers, heavily used to increase yield. The reasons are always the same: enrichment of the tissues in soluble nitrogen. Thus some experiments can lead to conclusions like this:

> 'Fertilisers, particularly those over-rich in nitrogen, lead to an increase in damage caused by parasites. Thus, in research on wheat carried out in 1972 by the I.T.C.F., in the northern half of France, the use of a fertiliser containing 240 units of N per hectare led to a decrease in yield of 250kg/ha, due to increased damage caused by parasites.' (BTI, 'Protection of crop health by cereal rotation,' 1975).

Parmentier (1979) repeatedly warned of the dangers posed to cereal crops, especially by *Oidium*, through the indiscriminate application of nitrogen fertilisers.

3. The 'secondary' effects of fungicides on cereal crops

We shall now analyse some special studies of the effects of Benomyl on cereal crops.

Fungicides too are being called in question. As usual when any pesticide proves to be ineffective, the cause is said to be a possible 'resistance' by the pathogen to the specific fungicide. However, what we have seen of the relationship 'cereal←→herbicide', or 'nitrogen fertiliser←→parasite', should lead us to examine the issue from the perspective 'plant←→parasite,' according to the 'conditioning' of the cereal.

In Great Britain, the two strains strains of eyespot (*Pseudocercosporella Herpotrichoides*) that are said to be resistant to benzimidazoles are found in parcels of crops treated with Benomyl and carbendazine. These treatments may well have made the cereal susceptible to eyespot. But the other possibility is that the plant has already been made more susceptible to attack by previous applications of either herbicides or fertilisers high in nitrogen. This would render fungicide treatments ineffective. A scrutiny of the effects of Benomyl appears to confirm this hypothesis.

According to an eminent plant pathologist, in the 1960s this fungicide promised 'to fulfill everyone's dreams of a successful treatment' (Ponchet, 1979). It has now been the subject of an indepth study of its effects on cereal crops (Poulain, 1975). Here is a brief analysis of the outcome. This research, which was the subject of a degree thesis, was carried out both in the laboratory and in the field.

In the laboratory, Benomyl did not always act as a fungicide. *In vitro*, the presence of Benomyl did not prevent the germination of *Fusarium roseum* spores, even at relatively high concentrations (10ppm). Benomyl even stimulated growth of the mycelium at doses only slightly lower than those that inhibit its growth totally.

In a similar way, Benomyl shows favourable effects on *Septoria nodorum*. This occurs on two levels:

- it leads to an earlier appearance of pycnides.
- it increases the percentage of colonies forming pyrenoids.

Three strains that were in the process of degenerating were regenerated by the application of Benomyl.

It is not therefore surprising to find that three Benomyl sprays applied on rye growing in the field led to a major increase in attacks by *Rhizoctonia solani*. This shows clearly that what is involved is proliferation caused by the fungicide, and not a 'resistance' of the fungus toward the chemical.

The field results obtained by Poulain (*op. cit.*) are no less interesting and thought-provoking. First of all, they confirm the positive effects of nitrogen fertilisers on diseases. In tests on Capitole wheat, disease spreads slowly in the period before June 25, at the beginning of vegetative growth, but explodes soon afterwards, whether or not the plant has been treated with the fungicides. 'The fungicide treatment does not stop the spread of the disease but merely delays its progression for two to three days'.

To recap: despite this 'qualifier,' Benomyl has not proven itself to be a 'fungicide,' as far as *Septoriosis* is concerned. In confirmation of what we previously discussed, one of the reasons for this failure could be the physiological 'conditioning' of the cereal, mainly by fertilisers. Poulain notes that nitrogenrich fertiliser (220 U) applied to hardy wheat 'has probably prevented the fungicidal treatments from acting normally.' The author adds: 'Alone among the diseases of the roots, Rhizoctonia showed a major spread *which the treatments appeared to promote*. A single application of the fungicide, in fact, leads either to a greater number of infected plants or to an aggravation of the disease.'

Proliferation occurs in these circumstances because pesticides make the cereals susceptible to these disorders. The mechanism behind this could be the following:

Toxic effects (of herbicide or fungicide)
Malnutrition (excess of nitrogen fertiliser) ⟶

→ Metabolic problem (inhibition of protein synthesis)
(triggering of deficiencies) ⟶

→ Sensitisation (to fungal disease)
(soluble substances) (to animal pests) ⟶

→ alteration of cell nucleus → onset of a virus

One last indication of the profound and long-term effects that Benomyl has on plant physiology: as Poulain notes, 'In every case, the germination potential of batches of plantstreated with a fungicide during the vegetative phase was lower than that of untreated batches.'

4. Herbicides and weeds

Chemical control has not been able to win the battle against weeds any more than the battle against plant diseases. Here too, the so-called problem of 'resistance' arises. Everything we have so far said challenges the validity of this term, particularly if the so-called 'secondary' effects of herbicides in some way stimulate the growth of plants that are considered harmful.

Beyond their direct effects on plants during application, *herbicides can modify the life of the soil.* Through their harmful effect on microorganisms, they are capable of affecting the processes of nitrification and the activity of enzymes such as phosphorylase and dehydrogenase. This can lead to malnutrition of the plant, resulting in the susceptibility that we described above. McClurg and Bergman (1972) were able to show that various herbicide treatments can lower calcium levels in plants, resulting in major damage. They simultaneously reduce both resistance and crop yield.

We can also draw a parallel between the effects of herbicides and those of nitrogen fertilisers. As mentioned above, a certain dose of fertiliser can block trace elements such as copper[1] with all the consequences of such a deficiency such as effects on the plant's physiological state and its resistance to parasitic attack.

It has been pointed out that pesticides that contain nitrogen –practically all chemical pesticides - are cations. They can replace cations such as Ca, Mg, and Zn from the exchange complex. The same is true of chlorine. Huguet showed clearly that repeated applications of nitrogen fertilisers result in inhibition of boron in the cherry tree.

The same process seems to explain the disappearance of calcium after prolonged treatment with NPK (Betaeghe and Cottenie, 1973).

We should also point out the reduction in phosphorous levels in soil treated with MCPA and malathion (Vicario, 1972).

We could justifiably ask whether such disturbances in the soil might explain failures in the use of herbicides. These disturbances result either from certain fertilisers (mainly nitrogen ones), or from nitrogenous and chlorinated pesticides (herbicides, fungicides, insecticides). Deficiencies in P and low levels of Ca and Mg promote the development of dicotyledons. Recently there has been a spread of self-sowing grasses: for instance,

foxtail and wild oats, as well as dicotyledons such as chamomiles, knotgrass, and speedwell. The spread has been ascribed, erroneously, to 'resistance.' (Barralis, 1978). Might this not represent an analogous phenomenon, due to disruptions in cationic elements?

Here, once more, we are dealing with proliferation due to changes in the nutritional environment offered to the self-propagating plant. This could explain how some insecticides used against maize *Pyralidæ* (*Ostrinia nubilalis*), which inevitably fall on the soil, can influence the nature of the flora. One study, for example, notes the stimulation of various species of 'weeds' in maize fields treated with diazinon against *Pyralidæ* (Gojko A. Pivar, 1967). As the author concludes in his article: 'These results show the necessity of continuous and systematic study of the appearance and spread of weeds, after pesticides have been applied to protect cultivated plants.'

As one crop expert recently stated, with regard to weed control in maize cultivation: 'The continual use of concentrated doses of atrazine creates new problems. What is noticeable is *the ever increasing difficulty in eradicating summer grasses, but without the appearance of true resistance*' (Morin, 1981).

Other examples include the failure of triazines against knotgrass. In Villiers-Bonneux in Yonne, south-east of Paris, farmers were having difficulties eliminating knotgrass (Polygonum lapathifolium) which had chloroplasts resistant to atrazine, a pre-emergence herbicide. Maize and corn had been grown in rotation on these fields since 1964. The 'resistance' of the knotgrass appeared after five or six applications of atrazine (with the formula, $C_8H_{14}ClN_5$). According to noted researchers, however, the spectacular spread of the knotgrass 'points to the conclusion that numerous resistant plants were selected from the very first treatments and then proliferated greatly, profiting by the selective advantage the atrazine gave them' (Darmency, Compoint, and Gasquez, 1981).

However, it seems rather unlikely that selection could have manifested itself from the very first treatments. Selection is normally a lengthy process. The authors seem, quite right to be astonished. Yet, it does not seem so unlikely that after five or six years of treatment with simazine, this chemical would be able to modify the soil in such a way as to promote the growth and proliferation of knotgrass. Besides, it does not seem to be mere chance since, as certain observers have noted.

> 'In agricultural practice, one speaks of 'resistance' to atrazine when one is helping with the massive survey of weeds after the application of atrazine' (Morin, 1981) .

Such a manner of speaking suggests the existence of an immediate process of proliferation, not just a later difficulty with eradication after a series of interventions with herbicides.

These remarks seem to confirm that, as in the case of the 'biological imbalances' resulting from treating foliage with pesticides, two essentially different processes are commonly confused: genuine proliferation and alleged resistance to a pesticide, which is much more rare.

It would also appear to explain why it is ineffective to try to overcome this 'resistance' to herbicides by increasing the dosage. Such a practice can only end by increasing proliferation. These indirect repercussions of herbicides confirm the position of those who think it 'is totally mistaken to release complex molecules into nature, in a complex environment, without having at least sketched out a way of controlling them' (Bouche and Fayolle, 1981).

II. The state of health of cereals using traditional techniques: 'organic agriculture'

A publication from IRAAB (Institute for the Research and Application of Organic Agriculture) looked at nine farms that were practicing organic farming - in other words, without using artificial pesticides and fertilisers. On some of these farms, where 'classic' or 'chemical' agriculture had previously been practised, the farmers were faced with the spread of weeds which systematic use of chemical herbicides had not been able to prevent. Perhaps, as we saw above, the herbicides even encouraged the weeds (particularly in the case of couch grass, thistles, wild oats, and foxtail).

In contrast, re-conversion to 'organic' led to the disappearance of these weeds, particularly wild oats and sorrel in sandy soils.

References involving improvement of the soil in this respect are found in four farms out of the nine reviewed. What accounts for the disappearance of the weeds?

If we examine the new growing conditions that the farmers adopted, we find that they fall into two categories:

- On farm number two, where there had previously been two weed-killer applications, two treatments against fungal disease, and one treatment against aphids, both wild oats and sorrel have disappeared.
- It also seems clear that other practices help a great deal. Firstly, some deep ploughing has led to the eradication of couch grass. Greater emphasis on leguminous plants (or companion planting: leguminous plus grain plants) allows for the cleaning of the soil and its enrichment in organic nitrogen and calcium. This effect is the inverse of what happens with fertilisation using synthetic chemicals or with the utilisation of chemical pesticides (especially fungicides and herbicides).

Moreover, according to the farmers themselves, this has not only solved the problem of weed infestation. It has also:

- shown that halting pesticide treatments (as we saw regarding aphids) seems to lead to an excellent state of health in the crop.
- led to better health in livestock, by the elimination of septicaemia, mastitis, etc. (It even leads to the elimination of ticks - a point which it would be unreasonable to neglect, if this shows the existence of a certain state of 'physiological resistance' in animals.)
- demonstrated, finally, important energy savings. The study showed that 'organic agricultural methods for cereal production (maize, wheat, barley) consume around two to three times less energy than do conventional agricultural methods.'

Such results could therefore be considered as a confirmation of the harmful effects of synthetic chemicals through their dual action on the plant: a toxic effect by direct action at the moment of treatment, and a rather pronounced alteration of the nutrition mechanisms of the cereal crop. The results therefore seem to confirm our theory of trophobiosis, particularly through the techniques of this traditional agriculture: stimulation of the resistance both of plants and, via nutrition, of the animals (including humans) who feed on them. All this comes together in *improvement of the nutritive value of food, according to the nature and level of protein synthesis*. This comprises a vast and engrossing subject of research for physiologists and other nutritionists, one that has only partially been attempted before (Schuphan, 1974).

A question of yield: 5,000 kilograms per hectare a reasonable objective or a dangerous myth?

Yields of 5,000 kilograms per hectare have been recorded on certain sites, particularly in France. But is this a reasonable aim in all agricultural situations? A primary obstacle to increasing yields, on which everyone is agreed, is *climate*. Mild and wet winters, for instance, lead to plants putting out too many suckers, resulting in a number of inferior ears of grain. On the other hand, a drought during the month of June can be equally damaging. However, there have been hardly any publications warning farmers against reduced yields brought about by these farming errors: namely, unbalanced fertilisation and pesticide intervention (herbicides, insecticides, and fungicides. Progress is equated with chemical control. And the serious repercussions of pesticides on the 'health' of plants, especially cereal crops, can be masked by other factors. It is the farmers themselves and their common sense that can help us answer the questions posed above.

Obviously, a yield of 5,000 kg/ha can be achieved through 'intensive' agriculture, with high levels of nitrogen fertiliser and repeated use of herbicide. But such techniques render the cereal susceptible to diseases, especially viral diseases, confirmed by the need to extend fungicide and insecticide treatments.

On this subject, the 'Letters to the Editor' in corporate newspapers such as *Agri-sept* have much to teach us, if we only read between the lines. One letter runs as follows:

> 'Too many farms of the Loiret have been blinded by the cereal mirage. The Loiret is a heterogeneous *departement* where we find the rich Beauce region producing 4,000 kg/ha of wheat alongside less productive regions such as Orleanais, Sologne, Giennois, and Berry. From 1970 to 1974, good harvests and high prices encouraged a large number of farmers to plough up their pastures and sell their livestock in order to buy powerful tractors.
>
> 'The good years were often followed by bad ones. The satisfactory yields of 19721973 (2,000 to 2,250 kg) dropped from 1974 onwards. At the present time, if we take the average of the last seven years, the yield is only 1,650, 1,750 kg. This is clearly insufficient, because it hardly covers costs' (*Agrisept*, May 2, 1981).'

So what brought about this drop in yields? Is it not due to outbreaks of disease, the inevitable corollary of 'intensification' of agriculture? In other words: excessive nitrogen fertiliser, plus the harmful effects of herbicides and then fungicides? When someone spoke of 5,000 kg/ha, a farmer in the Toulouse region laughed and said: 'The wheat got attacked by dwarf yellow, and the average was only 3,050 to 3,100 kilograms a hectare.'

Another farmer said: '5,000 kilograms? I don't think that's possible or sensible. The point isn't producing more, but producing at the lowest cost. In a drought year, when you've put on your treatments and put down nitrogen, there are nights when you don't sleep too well.'

These farmers had not yet had the chance to draw a connection between their farming methods and the outbreak of what are (hypocritically?) called 'diseases of progress.' These diseases naturally need to be treated with more chemicals, thus further increasing the cost of growing the crop, in a vicious circle. However, since it is becoming clear that one cannot control viral diseases 'chemically,' might this not lead people to ask about the real cause, or even begin to blame the pesticides themselves?

By way of contrast, the example of the results of traditional (organic) agriculture has shown us that certain agricultural techniques, particularly a balanced fertilisation, can stimulate the resistance of a plant to its parasites. Rice growing offers a further example when we study it from this perspective.

III. Rice growing: health problems and the results of a balanced fertilisation

1. Rice parasites

The principal enemies of rice include:

a) a fungal disease, 'blast,' caused by *Piricularia oryzae* (rice blast). This affliction does major damage, especially in India and Brazil;

b) a bacterial disease caused by *Xanthomonas oryzae*. Every year, this accounts for thirty percent of the loss in rice crops in Japan and South East Asia;

c) various insects, chiefly the following:

- a leafhopper, *Nilaparvata lugens*, or Brown rice hopper. Its attacks lead to a blackening, and then dying off of the rice. Chemical pesticides have not been able to address this. According to *New Scientist*, the cause of these outbreaks of 'hopper burn,' which was never so harmful in the past, is 'certain fundamental changes in agricultural practices.' We will take up this point later on.

Among the other noxious insects, we should also mention:

- the classic 'borer', or *Chilo suppressalis*, a microlepidopteran;
- the rice stem borer, *Tryporyza incertulas;*
- the whorl maggot, *Hydrellia sesakii;*
- the 'green leaf hopper', *Nephotettix virescens*,\ one of the Cicadellidae;
- the 'leaf roller', *Cnaphalocrosis medinalis.*

To complete the list, we should mention the rice weevil, *Sitophilus oryzoe*. Its toll is growing, like that of other insects, with the growth in use of nitrogenous fertilisers.

2. Some harmful effects of herbicides on the resistance of rice to its parasites

Ishii and Hirano (1963) were perhaps the first to warn against the harmful effects of *2,4D*, when used to control weeds in rice plantations. This synthetic hormone causes more intense attacks by the borer *Chilo suppressalis*. According to these researchers, proliferation of this insect is more rapid in rice stems treated with *2,4D,* due to higher levels of nitrogen in the treated plants (+25%) compared to controls.

For the same reason, treating rice seed with *aldrin* increases the number of larvae of the rice water weevil, *Lissorphoptrus oryzophilus* (Bowling, 1963).

The mechanism of this sensitising of rice arises from an increase in soluble nitrogen in the rice tissues, as Ishii and Hirano (*op. cit.*) have shown. This process is identical, we shall now see, to that caused by nitrogen fertilisation when it is not sufficiently balanced with potassium.

However, pesticides and especially herbicides may be more to blame than misguided fertilisation for the development of various parasites, due to the inhibition of protein synthesis caused by these chemicals.

It has been shown that rice, along with other species of aquatic plants, absorbs and concentrates chlorinated products (pesticides and polychlorophenols) in the aquatic environments of the Camargue. These plants absorb and concentrate the chemicals (alpha and gamma HCH, DDT and PCB). Measured in dry weight, the levels of concentration in relation to levels in the water vary from 16-fold to 20,000-fold, depending on the species and the chemical (Vaquer, 1973). It is therefore not surprising that such concentrations cause acute changes in the metabolism of the treated plant (in this case, rice).

2,4D is obviously not the only herbicide used in rice growing. We will single out, for example:

- fenoprop or *2,4,5TP* with the formula $C_9H_7O_2Cl_3$, used after germination at the 34 leaf stage, when suckers are being put out;
- propanil ($C_9H_9NOCl_2$) applied as a pre-emergent;
- and finally, nonchlorinated molinate ($C_9H_{17}NO_4S$) derived from carbamic acid. It has been shown that the nitrogen content of plants treated with carbamates whether herbicides or insecticides increases compared to that of the controls, and that they often become susceptible to various parasites.

As to chlorinated products, it is well known that they reduce amino acid synthesis and promote protein decomposition. Because of these properties, the chemicals can often act as herbicides. However, the same phytotoxic properties affect the cereal crop being weeded. No doubt this effect is much less marked, but such an inhibition of protein synthesis can render the crop susceptible to a whole range of parasites, including viral diseases.

In regard specifically to rice, Trolldenier and Zehler (1976) found that a large number of pathogenic organisms depend, for their nutrition and growth, on the soluble constituents of cells such as sugars and amino acids. (This confirms the view of Dufrenoy and our own concept of trophobiosis.) Such substances are found in high concentrations in plants containing abundant nitrogen, particularly those that are deficient in potassium. This explains the harmful effect of a high N/K ratio and too much nitrogen-rich fertiliser on a plant's level of resistance. We will now see this confirmed in the case of rice.

3. Fertilisation and the resistance of rice to its parasites

A. Harmful effects of nitrogen chemical fertilisers.

Having seen how herbicides make rice susceptible to its parasites, we should not be surprised by the effects of nitrogen fertilisers. Eden (1953) shows that the damage caused by the rice weevil *Sitophilus oryzoe* increases in proportion to the quantity of nitrogen in the fertiliser. We have seen that the same mechanism was at work in the proliferation of the rice borer *Chilo suppressalis*, caused by *2,4-D*.

However, the effects of nitrogen fertilisers are perhaps most clearly and spectacularly seen with 'blast', or *Piricularia oryzae*. Through experiments using ammonium sulphate, Sridhar (1975) was able to establish that a high level of soluble nitrogen in the leaves of rice promotes the spread of the disease. *Piricularia oryzae* uses various amino acids as sources of carbon. In plants fertilised with these chemicals, glutamine and asparagine levels increase. Sridhar concludes, therefore, that high concentrations of nitrogen in the host provide the pre-requisites for the development of the parasitic fungus.

This excess of soluble nitrogen in the plant tissue can result in part from a dis-equilibrium with the other elements, inhibiting the condensation of the amino acids into insoluble proteins which are of no nutritional use to the various parasitic organisms. This is often the case with potassium deficiency. We shall see that rice conforms to the general law of the importance of the N/K balance for a plant's level of protein synthesis, and therefore its resistance.

B. Beneficial effects of potassium fertilisers on the resistance of rice to its various parasites.

Clearly, the application of fertiliser does not call forth the same reactions from all strains of rice. Ranga, Reddy and Sridhar (1975) were able to show that, depending on the variety of rice, identical applications of potassium did not have the same outcome with respect to the bacterial disease caused by *Xanthomonas oryzae*. While no improvement in resistance was in fact noted in the highly sensitive variety T (N) I, the application of potassium halted the disease markedly in the less sensitive variety IR8.

However, confirming the general rule, the leaves of the sensitive variety T (N) I showed higher levels of phenol, reducing and nonreducing sugars, and amino acids, than those in the less sensitive variety IR8.

The results clearly show that the phenol content has no link with the process of resistance (according to the idea of a possible antagonistic effect). It is the presence of soluble nutritional substances - reducing sugars and amino acids - that makes rice susceptible to the disease.

In the end, according to these authors, it is insufficient application of potassium that promotes the buildup of these substances in the leaves of these two varieties, by comparison with plants provided with a sufficient amount of this element.

The beneficial influence of potassium fertilisers in promoting resistance to fungal diseases is also shown by the resistance of rice to various noxious insects. This fact emerges particularly from recent research carried out by Vaithilingam (1982) on resistance of rice to various insects, brought about by potassium fertiliser.

Thanks to both field and laboratory trials that involved numerous analyses of leaves, Vaithilingam (*op. cit.*) established that potassium leads to a notable degree of resistance to a series of insects harmful to rice, when applied as a fertiliser to plants. These insects are:

- the brown plant hopper or BPH (*Nilaparvata lugens*). This result is all the more interesting, since chemical control of this insect is totally ineffective.
- the green leaf hopper (*Nephotettix virescens*), a Cicadellidae.
- the leaf hopper, *Cnaphalocrocis medinalis.*
- the rice stem borer (*Tryporyza incertulas*), whose numbers are also significantly lowered in rice fertilised with potash.
- finally, *Hydrellia sesakii.*

The author concludes'Against the five principal insects observed, a maximum dose of 150 kg of K_2O per acre proved so effective that not a single application of insecticide was needed.'

For Vaithilingam, this resistance process results from the plant's increased utilisation of amino acids for the buildup of proteins. On the other hand, with a deficiency of potassium, there is a build up of various amino acids such as valine, alanine, aspartic acid, glutamic acid, arginine, etc.

This is why, as the author points out with reference to nitrogen, one must be careful to distinguish clearly between protein and nonprotein nitrogen. Resistance is linked to an absence of ammonium nitrogen in the tissues.

Potassium fertilisation should also, it appears, increase resistance to diseases such as *Piricularia*. We will discuss this disease later, in relation to the role of the trace elements. Other cationic elements also play a role. Primavesi et al. (1972) note that the ratio K/Ca is 7.6 in healthy rice plants, as compared to 2.9 in rice suffering from *Piricularia.*

The beneficial results should also show up in relation to viral and bacterial diseases. This is a result, as we have seen above, of the common mechanism at the root of the 'parasitic complexes' which stem from a lack of protein synthesis. However, according to current knowledge, achieving an optimum of protein synthesis depends both on the nature of the available potassium and on its balance (as we reported above) with the other metabolic elements, chiefly the trace elements.

In this respect we should note that, according to Xie JeanChang et al. (1982), absorption of potassium by rice is correlated more closely with potassium that is absorbed slowly than with potassium that is easily available. This brings up an important point: in intensive growth conditions with high doses of nitrogen and phosphate, potassium levels are far too low for rice to grow properly.

Is this a general phenomenon? It could explain the harmful effects of excessive nitrogen, which are linked to appearances of deficiency caused by unbalanced NPK fertilisation. Although fertilisers high in N, P, and K lead in the short term to higher yields, they bring losses of nutritional elements that are not replaced. Several studies of deficiencies in the trace elements, Ca, Mg, and S confirm this ratio.

As Bussler (1982) notes: 'As well as deficiency-related diseases with visible symptoms, latent deficiencies are attracting more and more attention. With more intensive use of fertilisers, the balance of all the nutritional elements becomes more important, especially in light soils.'

Some symptoms of deficiencies in nutritive elements have in fact been observed, particularly in poorly drained soils. Analyses have shown that although these soils contained high levels of organic matter, they were poor in interchangeable K, as well as in available P and Zn.

This explains why such 'organic' soils can be transformed into productive rice fields by adding certain trace elements, for instance Cu, Mo, and Zn, to an NPK fertilisation. (Quidez, 1978)

C. The influence of trace elements on the resistance of rice to its parasites.

The preceding analysis dealt with different studies of resistance in rice, according to the type of fertilisation. It shows that the balance between nutritional elements plays a major role, related to the level of protein synthesis caused by this balance in the plant. If the trace elements, through their action on enzymes, are a determining factor in protein synthesis, we should also consider their balance with the major elements such as N, P, K, and Ca.

These considerations, related so far mostly to resistance to attacks by insect pests, seem to apply just as well to diseases. As Shigeyasu Akai (1962) noted in regard to rice *Helminthosporiosis*: 'Until now, studies of the relationship between potassium and plant diseases has not shed much light on the influence of potassium supplementation on the mechanism of diseases.'

However, the author's research concerning potassium, based on hydroponically raised plants, showed that the proportion of large *Helminthosporiosis* spots on the leaves was lowest when treated with 'excess potassium.' In contrast, the proportion was highest with the 'potassium deficient' and 'nitrogen deficient' treatments.

Moreover, the germination rate of conidia in fungal parasites was lowest after treatment with 'excess potassium,' while it was highest after the 'deficient in nitrogen' treatment. This correlates with the composition of the liquid exudate from the rice leaves. This liquid contains various amino acids such as glutamic acid, aspartic acid, and leucin. According to Shigeyasu, 'It seems that the germination rate of the spores is almost proportional to the quantity of free amino acids contained in the leaves, The more the level of free amino acids is raised, *the more the germination rate of the spores is raised*. As to the level of potassium in the leaves, this has little influence.'

This would confirm the indirect role of potassium, by its action on protein synthesis and by its mobility within the plant. The K/N ratio also plays a role here. According to some researchers, application of nitrogen exclusively in the form of ammonia has an antagonistic effect on the rice paddy's absorption of potassium. The present writer has also observed a lower potassium level in the leaves of rice plants in plots with excessive nitrogen.

A K/Mg antagonism has also been noted. As Shigeyasu (*op. cit.*) comments concerning this, 'The influence of Mg and P should probably be taken into account in plots with an unbalanced K/N ratio, for example with an excess of nitrogen.' The author eventually concludes that *the development of Helminthosporiosis marks on potassiumdeficient rice is caused by an inhibition of synthesis of proteins due to free amino acids*. However, he concludes from these results that potassium must be considered not on its own but in relationship with the other elements. The criterion for a high rate of resistance, he states, is a high level of protein synthesis and, conversely, a low level of free amino acids.

With regard to trace elements, Shigeyasu (*op. cit.*) notes that susceptibility to *Helminthosporiosis* decreases with the application of iodine, zinc, or manganese. This also has a favourable effect on vegetative development, which confirms the beneficial effects of these trace elements on the protein synthesis process, which occurs through their action on the enzymes.

Conversely, the same author states that susceptibility to the parasite increases when there is a lack of magnesium, an excess of potassium, or an addition of cobalt. The author concludes: 'The effect of potassium on the resistance of rice to *Helminthosporiosis* cannot be assessed purely from the angle of potassium.' This conclusion is clearly the same that we can draw from the studies of Primavesi *et al.* (1972) in relation to resistance to *Piricularia* in rice, to which we now turn.

To sum up briefly the important work of these researchers, we note that they refer first of all to certain fundamental laws of biology, primarily the following:

- An abundant NPK fertilisation promotes a plant's susceptibility to disease.
- A certain balance between the elements is more important for crop production than an over-abundant fertilisation.
- There is a very delicate balance between macro-elements and microelements, just as between bases and acids.
- The most delicate balance is the one between nitrogen and copper.

The basic reason why this last point seemed important to the authors is that the disease *Piricularia* in their view arises from a nutritional imbalance: an excess of nitrogen, which itself stems from a deficiency in copper.

The K/N ratio is also important here, as with other plants. Excessive levels of nitrogen lead to deficiencies in potassium. Primavesi *et al.* (*op. cit.*) point out how an excess of nitrogen causes a fundamental modification of the metabolism of rice, leading to enrichment of the tissues and exudates in amino acids. The result is susceptibility to 'blast', as well as to *Helminthosporiosis* A further result is the ineffectiveness of fungicides as stressed by various researchers. This is pointed out particularly by SallaberryRibeiro (1970), by Primavesi *et al.*, and by Sanchez Neira (1970), all of whom conclude by recommending wellbalanced liming as an effective preventive measure.

The results of the trials by Primavesi *et al.* (*op. cit.*) are as follows:

- There is a high, statistically significant correlation between yield and levels of Ca and Mg.
- pH has an impact on the severity of the disease: healthy rice grows in water with a pH between 5.4 and 5.8; rice suffering from *Piricularia* grows in water with a pH level varying between 6.8 and 7.9.
- Nitrogen-rich fertilisers increase the susceptibility of rice, while potassium lowers it.
- Maximum yield is obtained with an average level of potassium.
- Higher levels of potassium are recorded in healthy rice (0.891%) than in rice suffering from *Piricularia* (0.739%).

Relations between the elements vary greatly depending on whether the rice is healthy or suffering from the disease. We give a table showing some of the ratios between various nutritional elements in rice plants, according to their condition of resistance (Table 11).

Table 11 clearly shows that diseased rice suffers from a marked deficiency in Mg, K, Mn, and Cu. It is worth recalling that we found some deficiencies of this type produced symptoms similar to those caused in grapevines by viral disease. This led us to raise the question of a cause-and-effect relationship between deficiencies and viral disease. We will return to this subject in the next chapter.

Table 11: Ratios between various elements in rice, according to their state of resistance to *Piricularia*.

Ratios	Values in healthy rice	Values in diseased rice
KCa	7.6	2.0
Ca/Mg	1.5	4.0
Ca/Na	2.1	2.2
K/Na	19.1	6.4
P/S	6.4	2.2
N/Cu	35.0	54.7
P/Mn	35.6	118.4
Base/Acid	3.6	2.3
Macro- elements/Mn	231.0	656.0

In regard to rice, the authors point out that manganese decreases with submersion. They also stress the great importance of copper. Their conclusion is as follows:

> 'It is clear that the contamination of the soil, water, and seeds with 'blast' spores has no influence on the health of the plant when the plant's nutrition is wellbalanced. Even in susceptible varieties, the disease does not persist. We can accept that levels of 18 ppm of Mn and 2 ppm of copper are sufficient to keep the plants in good health in the soils studied.'

We have looked at the effects of different types of fertilisation on the resistance of the rice plant to its different parasites. The results show the great importance of the plant's nutrition in the stimulation of protein synthesis. Herbicides make rice susceptible to its parasites, and this is true of cereal plants in general. They do this by inhibiting protein synthesis, and through imperfect selectivity. Just the opposite happens with a well-balanced fertilisation.

Although this phrase, 'well-balanced fertilisation,' is a common-place, it is never properly defined. We define it as obtaining a maximum degree of protein synthesis, especially during the more sensitive periods of the plant's physiological cycle, such as flowering. To achieve this, we have to understand the needs of each individual plant and not poison it with one pesticide or another.

As to perfecting the use of fertilisers, this is a subtle question, given the state of our knowledge. It can only be achieved through the correction of deficiencies. This is, moreover, the reason why all preventive measures that adhere to such a method must be based on examining both the soil and the plant. We shall return to this in the course of the next chapter, in which we examine *nutritional treatments with leaf sprays*.

Notes

1 Some recent analyses have shown that an increase in nitrogen fertilisation, from 150 U/ha N to 250 U/ha N, leads to a decrease of various elements in colza crops 15% in S, 11% in B, 8% in Mo, 3% in Mn while N increases by 10%.

Chapter 9

Correction of Deficiencies and Stimulation of Protein Synthesis to Prevent and Control of Diseases and Parasites

I. Introduction

The nutritional requirements of insects, pathogenic fungi, bacteria, and viruses are not identical. Instead, they all draw from the available 'pool' of free amino acids and reducing sugars. It is the state of proteolysis which sensitises the plant to its parasites. In Chapter 2, we listed the various factors that are likely to influence protein synthesis. They can also be classified according to the degree of control that the farmer has over them. He or she is most often subject to the variety of the plant, sometimes the root stock, the soil, and the climate. The farmer has power over fertilisation, grafting, and chemical treatments.

Fertilisation of the soil and fertilisation of the foliage should work together to bring about optimum levels of protein synthesis. This assures both protection against parasites and the nutritional quality of the harvest.

In practice, this optimum nutrition can be seen from two angles:

- the major elements, notably such 'traditional' ones as N, P, and K, to which can be added Ca, Mg, and others which, owing to their properties, might be better grouped with the micronutrients.
- the micronutrients the best known of which are Cu, Fe, Zn, Mo, Mn, Li, B, Co, Ni, and I. We might also add Cr, F, Se, Sr, and Va.

Given the length of this list and how little we know about the role of the elements in the physiology of the plant, it is easy to feel discouraged in the search for the nutritional balance that allows us to ensure simultaneously the health of the plant and (more ambitiously, to be sure) that of the people and animals that consume it.

First we must discover how to avoid deficiencies and then how to correct them as far as possible. It seems that, without realising it, traditional anti-fungal measures have often done this.

This chapter includes:

- a study of the balance of the cationic elements, deficiencies, and nutrition of the plant,
- a study of the way various anticryptogams work,
- a special study of boron and its properties,
- an example of the relationship between deficiencies and viral diseases,
- the control of scab (current difficulties and new perspectives),
- various examples of curing diseases through 'nutritional' sprays.

II. Cationic balances and the nutrition of the plant

Various researchers have studied the balance between the cationic elements K, Ca, and Mg, although less research, it seems, has been done on sodium. These studies have been carried out using a number of methods: hydroponic cultivation, leaf analysis, effects on the scion, and the effects of fertilisers on the soil or of sprays on the foliage.

However, it is not only cationic balances that influence this balance. The micronutrients intervene just as much. The reason is simple: they constitute part of the coenzymes, and as such they intervene directly in protein synthesis.

Mn, Cl, and B are *activators of enzymes*, while Cu, Fe, Zn, and Mo are *components of enzymes*. This explains the importance of their relationship with K, Ca, and Mg. Finally , regarding the criterion of the plant's 'resistance,' this explains the relationships of Mn, Cl, and B with one another and with nitrogen, and the equilibria between the different forms. There are many mysteries regarding this subject still to be resolved, and this is confirmed by the various studies in this chapter.

1. K/Ca and K/Mg ratios

Potassium seems to be the element that, both in soil fertilisation and foliage nutrition, most often creates resistance in the plant toward parasitical insects and diseases. K activates enzymes and over forty different enzymes are interconnected with this element. Accordingly, a K deficiency leads to an increase of soluble substances in the tissues. These include reducing sugars and soluble nitrogenous compounds, especially the amino acids asparagine and glutamine, essential nutritional factors for parasites.

High doses of K lower the proportion of amino acids and stimulate resistance to various diseases.

This influence of the ion K (since K is a non-component part of organic compounds) explains how it can interact with a number of other elements, including major ones such as Ca, Mg, and N, and various micronutrients such as iron, zinc, molybdenum, and boron. The relationships with Ca and Mg are likely to be of most interest, since their balance with these cationic elements has the greatest effect on the metabolism of protein synthesis.

Calcium is equally important for plant resistance. Shear (1875) lists no fewer than thirty disorders or diseases, in the strict sense,arising from calcium deficiencies. Among these are a number of different necroses or 'black hearts' (an inhibition of the multiplication of the meristem cells).

We know in fact that Ca has an effect on some enzymes and on the circulation of carbohydrates, while certain proteins have some affinities with calcium. In this way calcium encourages the synthesis of amylase. It plays an important role in the SiCa balance and keeps the clay in a flocculated state. It also determines the pH value of the soil and thus the level of bacterial activity.

These various functions explain the close relationship that calcium has with various other elements. Thus, while it can prove beneficial in exercising a 'buffering' effect on certain micronutrients, it can also, in the form of calcareous soil improvers, block manganese in cereals, zinc in maize, or boron in beetroot. A relationship has also been noticed between calcium and molybdenum, aluminium, and strontium.

A boron deficiency arising from a calcium deficiency can lead to formation of 'proliferations' or 'witches' brooms,' causing growth failure in meristems, as we have seen above.

As a result of the importance of the two elements K and Ca for the physiology of the plant, it is interesting to study the influence of the K/Ca balance. We shall also consider the K/Mg balance as a criterion for the level of protein synthesis at a given stage of the cycle (eg: flowering or ripening).

The equilibrium between K and Ca has been studied by Crane and Steward (1962) in *Mentha piperita*, cultivated in a hydroponic solution. The effects on the synthesis of proteins and their amino acid composition were also studied in relation to the length of the day (in other words, the photoperiod) (Fig.2)

On long days there is protein synthesis in conditions of high glutamine content, with growth occurring in proportion to an elevated K/Ca ratio.

On short days, growth occurs according to a relatively low K/Ca balance, while levels of soluble nitrogen are higher and those of protein nitrogen are lower. Proteolysis dominates, with an accumulation of asparagine. Asparagine is an amino acid required for the growth of fungi (we shall return to this in relation to fruit scab). It accumulates in those plants that are specifically fertilised with ammonium nitrate fertilisers. This could explain the susceptibility to disease caused by this type of fertilisation (Huber and Watson, 1974) and at the same time the renewal of attacks in the late autumn.

Generally speaking, a Ca deficiency reduces the quantity of protein produced, with sudden cessation of growth. On short days K deficiency brings a drastic increase in *asparagine* and on long days, an increase in *glutamine*. Similar phenomena are also found in other plants. Although the effect of K on the metabolism of nitrogen is still very poorly understood, nevertheless analyses of plants deficient in K show increases in soluble nitrate compounds, in particular amino acids, while proteins are found to diminish. Such results highlight the relationship between the K/Ca balance and the plant's resistance to various parasites.

The K/Mg balance

Magnesium is, of course, part of the chlorophyll molecule. It therefore forms part of the chain of decomposition and of carbohydrate synthesis. The reaction of ADP+P to ATP cannot take place without Mg. This explains the importance of the relationship between P and Mg.

The fact that the metabolism of phosphorus is closely related to that of calcium explains the CaMg antagonism. This is why the relationship between K and Ca may be used as a first approximation to standardise the metabolism of the plant, especially with respect to protein synthesis. In some plants, however, it is magnesium that seems to be the most variable, depending on conditions. This is so, for example, in the case of the grapevine, where special studies have looked at the effect of the K/Mg relationship on the nutrition of the plant (Delmas, 1970) and on the practical use of leaf diagnosis for similar ends (Ryser, 1982).

The work of Delmas on Merlot cultivated in a nutrient solution aimed to assess the effects of the elements N, P, K, Mg, Fe, Mn, B, Mo, and Zn on the vegetative state of the grapevine, and especially the potential for deficiencies.

According to Delmas (op. cit.), magnesium deficiency occurs at levels below 0.20% of dry matter, and a proportion of K higher than 1.5%, and thus a deficiency when the K/Mg ratio is higher than 7. This proportion is similar to that estimated by other authors (Levy and Gouny, for example).

This ratio varies greatly depending on the nature of the rootstock, as we have seen in Chapter VII. Thus the K/Mg ratio in the leaf blades follows the scale below (Table 12), depending on the nature of the rootstock used (Duval-Raffin, 1971).

Table 12: Values for the K/Mg ratio in the leaf blades of Merlot, in relation to the rootstock (DuvalRaffin, 1971)

Root system	Mg: mg/kg Dry Matter	K/Mg ratio
Merlot ungrafted	2 110	5.39
Merlot/SO4	765	14.27
Merlot/3 309	1 009	11.28
Merlot/Riparia	912	17.09

As the author points out, K and Mg in the grapevine are profoundly modified by grafting, so we need to determine the K/Mg ratio. Soils poor in Mg cause Mg deficiencies which impact on nutrition and thus on the resistance of the plant to diseases and parasites. This happens in the case of grape bunch desiccation.

According to Ryser (1982), there is a 'latent' deficiency when the ratio is around 1012, as can happen in some vineyards in French speaking Switzerland. The standard ratio in viticulture is between 2 and 10. A net Mg deficiency occurs when there is a ratio of more than 12.

Such figures provide a basis for correcting deficiencies and thus, as we see it, for controlling parasites, especially by nutritional sprays.

However, the cationic elements K, Ca, and Mg are not the only ones that affect the metabolism of the plant. The micronutrients seem to play a very important role in this respect, due to their relationship with the enzymes, while much remains unknown about their relationship with K, Ca, and Mg. Phenomena such as blocking and stimulation may occur, although the rules that govern these are not yet clearly established.

2. The balances between micronutrients

In his experiments with the grapevine, Delmas (*op. cit.*) has brought to the fore the importance of certain micronutrient ratios at the level of diet, especially the Fe/Mn ratio. Optimum ratios would be on the order of 1 during the flowering period and 0.6 during ripening. A Mn deficiency causes ratios to exceed 5. To be more precise, such a Mn deficiency leads to serious metabolic problems, which are all the more important since

- the level of P rises;
- the feed level of *iron,* previously, was reduced or excessive.

Finally, we also note the existence of a K/Mn balance, with the Mn deficiency leading to an accumulation of K in the roots. At the same time, a K deficiency is the result of a manganese deficiency.

In branch tendrils, a Mn deficiency provokes a disturbance of the Fe/Mn and P/Mn ratios. The latter ratio could influence the ripening of grapes that are undernourished, small, and rich in P.

This interaction among the nutritional elements, especially the micronutrients, is related to the absorption of those elements. Primavesi *et al.* (1982) point out that in rice the absorption of Mn may rise to 8,000ppm in certain varieties without any side effects, by counterbalancing Mn levels with those of Fe. In conclusion, the Fe/Mn ratio could be a good choice as an indicator for plant metabolism.

Lubet *et al.* (1983), confirming the importance of the role of iron in the diet and thus in the plant's resistance, emphasise that as a means of anticipating zinc deficiency in maize, the Fe/Zn ratio appeared to be a much better indicator than the zinc content as such. We know that the latter is a constituent of numerous enzymes. This explains better the histological modifications caused by functional problems in the enzyme chains. Zn acts as an essential element for many metalloenzymes of the dehydrogenase type.

We recall the close relationship linking zinc to the metabolism of phosphorous in the grapevine. Zincbased treatments lead to an increase in the P content in the leaves - that is to say, inorganic phosphates, phospholids, and nucleoproteins (Dobrolyubskii and Fedorenko, 1969). Such results could explain the beneficial effects noted by Dufrenoy (1934) with zinc sulphate treatments against the manifestations of infectious degeneration in the grapevine.

Zinc deficiency, especially in the tomato, causes an increase in the level of amino acids, with a considerable accumulation in particular of asparagine, because it cannot be polymerised into proteins (Krisna, 1963). From this arise growth problems and susceptibility to disease.

Finally, we draw attention to the relationship between molybdenum and the other elements. Delmas (*op. cit.*) indicates that a Mo deficiency causes leaves to be deficient in K but enriched in Ca. The ratio K/Ca passes from 0.22 of dry matter to 0.12 during the ripening period and from 0.37 to 0.07 after harvesting. Delmas also noted that an Mo deficiency provokes an enrichment of the leaves in Mn.

According to Primavesi *et al.* (1972) a delicate balance exists between copper and nitrogen. Thus the N/Cu ratio is 35.0 in healthy rice and 54.7 in rice infected with *Piricularia*.

In *Mentha piperita* a copper deficiency encourages the accumulation of soluble compounds, especially glutamine. If copper treatments actually enrich the tissues with copper and have an effect on proteic metabolism, this would explain its effectiveness against various diseases. (See below, p. 226).

Boron is another element whose deficiency has often been linked to susceptibility to disease.

Table 13: Foliage analysis of tomatoes hydrosoluble elements in mg/kg

Elements ⟶	NH4	PU4	K	Ca	Mg	Fe	Cu	Mn	Zn	B	Co	Cd	Ti	Ni	Mo
Subjects															
Healthy tomatoes	1000	3125	4850	3375	1475	115.0	62.5	40	137.5	160	7.5	5	2.5	92.5	12.5
'Virused' tomatoes	1250	3125	4900	1925	1400	107.5	15.5	11.5	115	110	T	T	T	137.5	32.5

N.B. T = Traces

Table 14: Ratios which indicate deficiencies

Elements ⟶	K/Ca	Ca/Mg	P/Ca	K/M	Fe/Mn	P/Mn	N/Cu	N/P
Subjects								
Healthy tomatoes	1.4	2.28	0.92	3.28	2.87	78.12	16	0.32
'Virused' tomatoes	2.4	1.39	1.62	3.50	9.24	271.7	80.64	0.40

N. B.: Mn, Cu, and Ca deficiencies in tomatoes with virus.

III. Deficiencies and viral disease in the tomato

Several years ago an agricultural chemicals firm provided us with the results of an analysis of tomatoes with and without virus. The differences between them proved to be very significant. This particular firm added objectively that it seemed 'extremely difficult to know if these differences were due to the viruses or if they arose subsequent to feed deficiencies.'

We believe that it is the deficiency that creates in the plant a state of susceptibility to disease, even if, once the virus is established, both symptoms and deficiencies become more pronounced. As far as the precise details of how a viral disease is triggered are concerned, Dufrenoy (1934) noted in the case of 'mottle leaf': 'The pathological cases are linked to premature appearance, as well as to a general cellular proteolysis and pectic degeneration.'

This proteolysis in citrus tissues infected with 'mottle leaf' was satisfactorily halted by $ZnSO_4$ based sprays neutralised by lime, as in Bordeaux mixture.

Tables 13 and 14 show the results of the analysis in mg/kg of tomato leaves, with and without virus respectively. Some ratios provide more specific evidence of certain deficiencies, which apparently cause proteolysis.

Thus, Ca, Mn, and Cu deficiencies are all found simultaneously. Copper and manganese deficiencies seem to be the most pronounced, although we cannot say what precisely is responsible for the disease. However, on the hypothesis that copper treatments could prove to be effective to some extent against bacterioses this would merely be due to the beneficial effect of the copper on protein metabolism in the tomato. This conclusion leads us to study the anti-fungal action of various 'fungicides,' both 'traditional' and 'synthetic.'

IV. On the mode of action of various anti-fungal products

It is evident that we know very little about how anticryptogamic products work. We may arrive quickly at the point where we know that this or that formula is more or less effective, but we would progress more quickly if we knew why. This, then, is good reason to take into account the reaction of the plant in the analysis of the product's effectiveness.

1. Copperbased products

The famous Bordeaux mixture was discovered when it 'was noticed that the vine stocks at the end of each row of grapevines, when sprayed with vitriol (copper sulphate) in order to protect them against thieves, suffered fewer attacks of mildew. Following this, Millardet and Gayon became the 'inventors' of Bordeaux mixture,' in trying to explain how the process works.

Even with Bordeaux mixture there are sometimes setbacks. After mildew attacks in 1932, the value of copper treatments was called into question by advancing the old cliché, 'The mildew has become habituated to copper.' (At this stage the word 'resistance' was not yet in circulation. Some authors (such as Villedieu (1932)) said that an excess of nitrogen and phosphate fertilisers could have made the grapevine more susceptible. Even in that era, it had already been shown that these fertilisers encouraged the invasion and multiplication of fungal parasites of the grapevine (Schaffnit).

The copper question was tackled by Semichon (1916). The effectiveness of copper, even on the surface, does present some problems, as we saw above. Semichon (*op. cit.*) distinguished between the 'surface' copper that remains on the leaves and the 'absorbed' copper. He observed that the surface or 'reserve' copper can only protect the grapevine for a very short period. This is for the simple reason that 'the copper hydrates, copper hydrocarbonates, and tetracopper sulphate which remain in the green parts of the grapevine after application become less and less soluble due to the effect of atmospheric agents.'

It is interesting to note at this point that, according to Semichon (*op. cit.*), leaves which are burnt by copper are immunised against mildew. Yet the question remains: how does the copper work? Should it be considered as a sort of toxic wall, after it has participated in the metabolism of the grapevine? Or should one not, instead, look more closely at the relationship between copper, plant, and parasite?

Copper, mediated by its action on enzymes (the oxidases), acts on the nitrogen metabolism of the plant. Thus it participates in protein structure, in combination with the substrate (Malström, 1965). This explains how vine leaves treated with Bordeaux mixture can produce much lower levels of soluble nitrogen in comparison with other chemical products (Pinon, 1977).

Crane and Steward (1962) have established, regarding *Mentha piperita*, that copper deficiency in the nutrient solution causes proteolysis, with an accumulation of glutamine in the tissues. One can therefore imagine that application of copper through foliar treatments could have the opposite effect. Similarly, Agrawal and Pandey (1972), experimenting on wheat, were able to show that copper treatments, by allowing sugars to be better utilised, increase levels of protein nitrogen in cereals. A positive effect such as this on protein synthesis will, in our view, only serve to stimulate the resistance of the plant.

The work of Marchal (1902) on lettuce shows that copper applied at levels of 5 to 7/10,000 in the nutritional environment prevents the development of Meunier (*Brema lactucae*).

Conversely, copper deficiencies resulting from the use of products containing nitrogen (fertilisers in the soil or chemical pesticides) could make crops susceptible to various parasites. This is the question we have previously considered in regard to the current spread of bacterial diseases. In this respect one could perhaps refer to the N/Cu ratio suggested by Primavesi *et al.* (1972) in rice.

2. Sulphur

Priest (1963) mentioned that during more than 2000 years that sulphur has been used to protect crops, it has never been used as a fungicide. Now it is used as such, especially in the case of *Oidium*.

That sulphur cannot be used on its own as a fungicide is proven by the fact that sulphur particles cannot penetrate into fungus cells. However, some early writers observed that the effect of sulphur in controlling *Oidium* on the grapevine, which they called 'remarkable,' was principally due to 'the way sulphur affects the vegetation of the plant generally.' One of them gave the following explanation:

> 'Grapevines which are sulphured, especially with a very fine spray, feel the effect of this excitation when they are sick. Immediately the pale and shrivelled leaves start to become green, supple, and shiny again, and the vine branches start to grow. If the disease is not too far advanced, it is noticeable how the whitish efflorescence which has begun to cover the berries disappears in the same way that parasites dogging an animal begin to disappear when it is fed a plentiful and healthy diet (Martres, 1862).'

Such a pertinent observation about the beneficial influence of sulphur, by its indirect effect on the physiology of the plant, ties in with the question posed by some authors about the influence of the nutritive environment on the vitality of the spores. Recent observations effectively confirm such an indirect effect of elementary sulphur. An example of this is the treatment by sulphur in oaks against *Oidium*. Observers have found that sulphur, unlike other chemicals, benefits the oak foliage. They conclude, 'The beneficial effect of sulphur is not directly linked to its power as a fungicide.' (Maennlein and Boudier, 1978). How then does it work? In this case too, the answer may be found by learning what happens in the case of sulphur deficiencies.

Crane and Steward (*op. cit.*) have shown that in *Mentha piperita* sulphur deficiency in the nutrient solution leads to an 'important change in the balance of nitrogen, favouring the soluble part, chiefly glutamine and arginine, and over the course of the longer days of summer.' This suggests that sulphur is a 'plastic' element closely associated with protein synthesis. Moreover, there are numerous authors who have underlined the close links that exist between the metabolism of sulphur and that of nitrogen.

We know that plants that are rich in nitrogen and poor in sulphur contain large quantities of free amino nitrogen, nitrates, and carbohydrates. This phenomenon is linked to a decrease in the reduction of nitrates and to reduced protein synthesis, together with a higher level of proteolysis (Nightingale, 1932 and Eaton, 1941). It has been shown that the leaves are able to absorb this elementary sulphur, because it is later found in the plant's proteins (Turrel and Weber, 1955).

If sulphur plays such a role in *indirectly* controlling *Oidium*, then it might have similar beneficial effects on plant diseases. Thus Dufrenoy (1936), stressing the damaging effect of deficiencies, as shown by the beneficial effect of certain fertilisers, recalls that sulphur at the rate of 200 to 1200kg/ha was successful in Florida in combating the bacterial disease *Bacterium solanacearum* in potatoes.

So the correction of a deficiency of sulphur through a process of a nutritional kind, at the same time stimulates the resistance of the plant to its parasites. This action of sulphur, by its positive influence on protein synthesis, would also appear to come under the general heading of trophobiosis.

Because *Cruciferae* need high levels of sulphur, it is logical to assume, as Polyakov and Vladinurskaya (1975) have done, that a sulphur deficiency decreases the resistance of cabbage to its various parasites.

So are the chemicals which have always been accepted as 'fungicides' in actual fact 'remedies,' acting indirectly by a positive effect on the metabolism of the plant?

3. How does maneb work?

If we look, for example, at a very widely used new 'fungicide' such as maneb, we will see just how difficult it is to prove its effectiveness simply in terms of its toxicity.

This is the question posed by Viel and Chancogne (1966). Initially, they observed that if maneb is suspended in water, it absorbs oxygen and decomposition takes place: the manganese is made soluble. Perhaps there could be an indirect effect through the reaction of the plant to the solubility of the manganese. This is in fact what has already happened with captan.

4. Is captan itself a fungicide?

This is the question that can be posed in the light of the results of the experiments of Somers and Richmond (1962) to verify the possible systemic action of this chemical. We should point out that we are dealing here with a phthalimide, a derivative of sulphur, with the formula $C_9H_8Cl_3NO_2S$.

Some solutions of captan have been applied to beans through irrigation, during experiments to control *Botrytis fabae*. Analysis showed the presence of levels of captan in the leaves to be so low they could not explain any possible chemotherapy effect through systematic activity. The authors came to the conclusion that such effectiveness could only be interpreted as: 'interference in the metabolism of the host plant, which is responsible for the anti-fungal effect observed in the leaves after an application has been made to the roots.'

5. How fosetyl (phosethyl) Al works

The new fungicide fosetylAl seems to have opened up 'new avenues in the control of plant disease' (Bompeix, 1982). As he writes, 'Contrary to the majority of fungicides, systemic or not, fosetylAl (or aluminium trioethylphosphonate with the formula $C_6HAlO_9P_3$) is characterised by a weak direct effect on fungus. This effect is not sufficient to explain the very good results obtained in practice.'

One notes an accumulation of polyphenols that presumably have a fungitoxic effect on the hyphae found at this level - that is, at the level of the necrotic blocking zones. Might not this accumulation of polyphenols be accompanied by a decline in the nutritional substances required by the fungus? It will be noted that necrotic blocking zones also occur with copper.

Bompeix (1981) concludes: 'All these facts point to a mode of action situated at the host/parasite interface, not a direct action on the parasites themselves.' Research is still being carried out on this subject, which no doubt will detect whether this immunity of the plant organ caused by the chemical, is caused by a toxic *or even an abiotic mechanism*.

In summing up this section, devoted to the study of the effects of various traditional or chemical antifungal products, we can state that the process of effectiveness proceeds through the reaction of the plant to the pesticide. The inhibition of growth of pathogenic fungi could derive from an absence of nutritional factors.

V. Boron in plant physiology and resistance

1. The physiological role of boron: its balance with the other elements

Boron is the micronutrient most often viewed as the cause of plant resistance, because of its effects on the plant's physiology. Yet its mode of action, like that of other elements, still remains very mysterious. Most knowledge of the role of the various micronutrients comes from studying the physiological problems caused by the lack of them. While boron does not seem to be essential for animals, the opposite is true for all plants.

As we have previously noted, boron deficiency has been linked to numerous diseases, whether fungal, bacterial, or viral. However, boron deficiency also affects the resistance of the plant to certain insect pests, such as mites, for example.

Conversely, applications of boron to the soil or in the form of leaf sprays, or even by impregnating seeds, stimulate the plant's resistance to certain diseases.

The relationship between boron and calcium

Calcium deficiencies, whether spontaneous or due to a boron deficiency, often show up through a shortening of the internodes, giving rise to some types of 'witches' brooms'. This happens especially with the maritime pine that grows in soils deficient in calcium. Calcium is linked to a number of enzymatic activities. In the plant, it is displaced in an upwards direction. According to Polyakov (1971), calcium acts by increasing the total level of phosphorus and different groups of phosphorous compounds. The role of micronutrients such as B or Cu is to intensify Ca penetration into the cells.

This 'synergy', well known between certain micronutrients, also comes into play with boron. D. Bertrand emphasises the fact that boron on its own has no significant effect; *it is only in the presence of Mg, Mo, and Mn (the 3 Ms) that the properties of boron become apparent.*

Moreover, the well-known fact that there is a relationship between boron and the transport of sugars would explain the necessity of its presence for the process of resistance, thanks to its action in providing the carbon chains required for protein synthesis.

What are the factors that provide the plant with the boron it needs? What antagonistic factors would be opposed either to its very presence, or to its availability?

2. The availability of boron and the requirements of the plant

The principal factors capable of affecting the availability of boron in ensuring the healthy functioning of the plant's physiology can be put into three categories:

- the type of soil and its pH
- fertilisation
- pesticide intervention.

As regards the soil, Coppenet (1970) observed that many areas are deficient in boron, for example, in the French Moyenne-Garonne region and the region around Toulouse.

Boron deficiencies have been recorded in a wide range of crops: apple trees, cauliflowers, alfalfa, beetroot, and grapevines. Thus there could be a relationship between such deficiencies and the frequency of certain diseases. It is perhaps no coincidence that, for instance, flavescence dorée in grapevines, which in extreme cases results from the proliferation of a mycoplasma or a virus, is particularly widespread in the Armagnac region. A boron deficiency was later identified in the vineyards of Gers and LotetGaronne.

Boron reserves cannot be activated in neutral or alkaline conditions. Cl. Huguet (1970) noticed that boron is the micronutrient most often deficient in perennial plants, especially in apple trees and grapevines. As he stressed, moreover, 'Pebbly soils do not provide sufficient amounts of boron either to grapevines or to cherry trees.' One might ponder the reason for this - insufficient boron in the bedrock, or particularly poor conditions for the dissolving of the boron? In the latter case, should we not also have to consider a lack of organic material and the role of micro-organisms? We will return to this point later.

To what extent do inorganic and organic fertilisers affect the availability of boron and, in a broad way, that of micronutrients in general?

As has been noted, micronutrients constitute less than 1% of the dry weight of plants. So why is it that deficiencies in these elements are recorded so frequently? It seems that Coïc (1971) has answered this question, when he commented: 'In a general way, a factor which affects the plant's physiology may also affect its levels of micronutrients.' Among these factors, Coïc mentions attempts to increase yields that actually produce an increase of organic material and therefore a decrease in the concentration of micronutrients in the plants and in their yields in general.

The 'intensive' agriculture used to increase in yields is based on chemical fertilisers, especially nitrogenous fertilisers and pesticides practically all of them nitrogenous and chlorates . As we have seen in this work, the parasites then follow one after another in an inevitable cycle. This is brought about by the plant's *sensitisation*, which may itself be the direct result of a deficiency.

How do chemical fertilisers and pesticides intervene in this specific case of the creation of deficiencies, especially those of micronutrients and, more specifically, boron?

First, disregarding slag, which is fairly rich in manganese, the amount of micronutrients provided by chemical fertilisers is insignificant when compared with manure or compost.

Secondly, these synthetic fertilisers generally increase the pH of the soil, thus lowering the assimilation potential of the micronutrients Mn, Fe, and Zn. For example, because of the antagonism between PO_4 and Zn, phosphate fertilisers can engender Zn deficiencies in fruit trees, maize, and flax.

However, it is perhaps chemical nitrogen fertilisers, often used in high doses in an effort to force high yields, which bear most of the responsibility for blocking micronutrients. This is a phenomenon that has long been recognised for copper. It causes hypoglycaemia and lowered fertility in livestock, when animals are fed with fodder deficient in copper. Périgaud and Démarquilly (1975) note that, in Brittany, incidences of poor fertility in cattle occur in areas where the soil is poor in copper, especially where farmers make applications of nitrogen exceeding 100kg/ha.

As we mentioned in Chapter IV, in regard to bacterial diseases, such blockage is linked not only to copper but also to boron. As Cl. Huguet showed, application of nitrogen to the soil in increasing doses causes a drop in boron in the leaves of cherry trees (Table XV).

In a second orchard, where at first no boron deficiency was found, applications of nitrogen fertiliser to the soil caused 'a decrease in levels of boron that became more pronounced over a period of years, as the tree's nutrition was increased. It seems that we have arrived just at present at a level of boron which is no longer the optimum for cherry trees' (Table 15) (Cl. Huguet, 1982).

Table 15: The decline in boron levels in cherry trees caused by nitrate fertiliser

Nitrogen fertiliser/ha	Levels of boron in 1980 (ppm)	Average levels of, over 11 years (ppm)
Controls: 0	44	47
100 kgs/ha	40	40
200 kgs/ha	32	37

Further, Cl. Huguet (1982) studied the effects of applications of zinc and boron sprayed on the foliage of cherry trees over a period of five years.

To start with, the trees were suffering from boron deficiency, causing early blossom drop. These results are put in concrete form in Tables 15 and 16.

Table 16: The effects of treatments on the boron content of the leaves (ppm of dry matter)

Treatments	Boron content (ppm)	Nitrogen content %
Without boron	19	2.38
With boron	59	2.56

In this case, one also notes a relationship between boron and nitrogen, but in an opposite, beneficial sense. There are protein compounds that become more pronounced following a correction of the boron deficiency. So, if the NB relationship sometimes appears contradictory, it is because they spring from the start from different cases - for example, in the two orchards where Cl. Huguet conducted his experiments.

1) In the first one, the tree's nitrogen status ranged from high to very high. In other words, proteolysis predominated, with boron as the 'limiting factor.' This explains why, under these conditions, applications of too much N make boron deficiency more pronounced.

2) In the second case, the tree's nitrogen status was critically low, and the boron content corresponded to a deficiency. This is why: 'The establishment of a feed with the correct boron levels can influence the whole metabolism, including the nitrogen metabolism, which improves slightly in this case.' (Cl Huguet) What the author means by this is that protein synthesis is in this way improved. From this stem the positive effects on the plants' resistance to various parasites, as we have seen in the course of this book.

3. The boron requirements of plants: Critical stages and application periods - effectiveness of sprays and boron treatments

All this demonstrates the importance of the boron requirements of various plants, especially fruit trees and grapevines. It also shows where the deficiency threshold lies. According to Ryser (1982), whose research concerns the practical use of foliage diagnosis, the ratio N + P/ K can be used to assess the risk of boron deficiency. If the ratio increases, there is a tendency towards a state of 'predeficiency.' This is a phenomenon of a quite general kind, equally likely to be found in viticulture, arboriculture, chrysanthemums, roses, and market gardens.

To what extent could chemical pesticides (largely nitrogenous and chlorinated), through repeated annual application, and by their cumulative, extended effects from one year to the next, upset the normal balance of elements and cause a harmful decline of boron in the plant's tissues?

Some analyses may perhaps suffice to provide the key to the current development of bacterial and viral diseases in fruit trees and grapevines, which we are at present witnessing.

Boron deficiency levels

The norm is very similar for both grapevines and fruit trees, and can be disrupted even on a general level in the same way. The generally accepted norm put forward is 25ppm for the leaves and 30ppm for the fullygrown fruit.

Mme Huguet (1979) proposes as a normal level of boron among apples a range of 25 to 50ppm for the leaves. In the 'Golden' variety, the content varies from 17 to 70ppm, and deficiencies can occur at levels of 20 to 9ppm. These figures seem to agree well with the threshold of 15ppm, generally proposed as being able to produce serious deficiencies.

The best times for applications of boron in the form of leaf sprays are thought to be those periods when the boron appears to be most crucial for the metabolism of the specific plant. Boron is, besides, very mobile, and a net increase in the level of boron is observed only a short time after application. This new level can thus exceed the normal level tenfold after the leaves have been treated.

Nevertheless, since boron is transported throughout the general structure of the plant, levels often return to normal after springtime applications, by the end of the season, and often as early as the summer.

In a general way, in fact, the authors admit that boron produces its effect from the beginning of the flowering period, in both sunflowers and grapevines. In the case of sunflowers, according to Polyakov (1971), cobalt begins to act before the flowering period, while copper and manganese are active during all stages of development. Polyakov (*op. cit.*) also worked on *Sclerotinia*, obtaining excellent results by soaking sunflower seeds in solutions of boron and also of cobalt, manganese, and copper. Salts were used on the seeds at levels of 0.1%.

These salts have been tested separately but, given what is known about the properties of boron mentioned above, it would be interesting to try out 'complexes' by combining boron with, for example the '3 Ms', i. e. Mg, Mn, and Mo.

In regard to the boron requirements of perennial species, Cl. Huguet gives them as the following:

Cherry trees: 10 tonnes of fruit/ha require 290g boron and 230g zinc.
Grapevines: *Ugni blanc* grafted on to 4 l B requires 108g B, 75g Mn, and 565g Fe.

For maximum absorption, the best periods for pear trees are as follows:
boron: from March to the beginning of April,
manganese: around midApril.

For grapevines, maximum absorption:
Boron: around midAugust
Manganese: around midApril.

4. On the influence of different types of fertiliser on the availability of boron and other micronutrients for the plant

The data on this subject are very incomplete. We must again point out that chemical pesticides and fertilisers may block micronutrients.

Moyer (quoted by Vicario, 1972) emphasises the fact that pesticide treatments that kill a large number of soil microorganisms can lead to toxicity toward plants, which is attributed to a lack of availability of phosphorus. (This arises from a process that is not yet fully understood.)

Moreover, Moyer points out that some pesticides containing nitrogen are themselves cations and may displace other cations such as Ca, Mg, and Zn. This leads to both toxic effects and deficiencies.

Similar phenomena may occur with fertilisers for similar reasons. Bussler (1982) notes that 'fertilisation with high levels of N, P, and K, though it can lead to higher yields, also increases the exportation of nutritional elements that are not replaced. A number of reports on micronutrient deficiencies in Ca, Mg, and even S confirm this relationship.'

In fact, cases of deficiencies in *copper* are more and more frequent, especially in cereals. Why specifically in cereals? Perhaps because 'intensive' cultivation, as we have seen above, combines the harmful effects of both fertilisers, especially nitrogenous ones, and chemical pesticides, starting with herbicides. If the blockage remains as a result of these cultivation techniques, one can justifiably ask what use is the application of micronutrients to the soil?

However, organic fertilisers and applications of manure and silage matter improve the assimilation of micronutrients, especially boron. Coïc and Tendille (1971) emphasise strongly that copper, of which we have just spoken, is more easily assimilated thanks to the bacterial development level of the soil. This is the same for other elements, especially phosphorus and boron. The phenomena of micronutrients being blocked in the soil by chemical pesticides could be the result of the pesticides' harmful effects on microorganisms. On the other hand, these microorganisms are abundant in manure and compost.

It is possible to see bacteria as packages of enzymes. This explains their influence on the phenomena of assimilation capacity, balances, synergy, and exchange.

While waiting for new research to bring us fresh data on the role of K, Ca, Mg, and numerous micronutrients in protein synthesis, we must rely on what we know about the correction of deficiencies in order to stimulate protein synthesis with a view to achieving maximum immunity for the plant. We must rely, that is, on the optimum level of the various balances we have looked at (K/Ca, K/Mg, Fe/Mn, Fe/Zn, etc.) as well as the perhaps more immediate consequences (N/Cu, N/K etc).

Given how little we know about the process of protein synthesis, we certainly cannot deny that such an undertaking presents certain difficulties. However, it could in the end present a lesser risk than the blind application of pesticides (i.e. without paying attention to the effects either on the plant itself or on the life of the soil).

Besides, we have also seen that it is probably through such an indirect effect that copper, sulphur, or some new fungicide can act to 'dissuade' the pathogenic agent from attacking the specific plant.

VI. Is it possible to control fruit scab by nutritional treatments?

1. The 'resistance' of fruit scab to chemical anti-fungals

We shall now look at how it might be possible to control a common disease, namely fruit scab. The new fungicides are not only powerless against this disease, but also cause phenomena of so-called 'resistance' (or proliferation by provoking susceptibility in the plant).

Fruit scab (*Venturia inoequalis*), together with mildew, *Botrytis* and *Oidium* in the grapevine, manifest strong 'resistance' phenomena in reaction to certain chemical fungicides. According to Olivier and Martin (1979), for example, in a number of orchards: 'Difficulties in treating whole trees, linked to rain measurement and associated with an active parasite population, have led to the more frequent use of benzimidazoles (in particular curatives). The conditions have promoted the appearance of phenomena of resistance to benzimidazoles in these orchards.' These authors continue:

> 'After the observation of this initial damage, treatments were stepped up and sometimes the doses exceeded authorised levels. Even so, the progression of the disease was not effectively halted. Under these conditions, the frequency of resistant strains among infestations of fruit scab increased steadily.' And finally:

'Ceasing to use benzimidazoles does not automatically mean that resistant populations will decline.' 'Observations of apple trees in Australia and Germany, and of pear trees in Israel, showed instead that these strains are being preserved. We have also come across an identical situation in France in the apple orchards we are studying' (Olivier and Martin, 1979).

Observations of 'resistant' strains have been confirmed by various parties. The following quotations are from a popular article (Phytoma, May 1982):

> 'The resistance of apple and pear scab to benzimidazolebased fungicides is shown by the significant damage which occurs, in spite of numerous treatments.'
>
> 'Some resistant strains are in fact capable of surviving for at least four years or more, even when no use at all is made of fungicides of this chemical family.' And further: 'In some orchards, a single application of benzimidazoles allowed a very small number of resistant strains to develop undetected during the autumn.'

Leroux comments: 'In order for resistance to manifest itself in practice (by a decrease in effectiveness), it is necessary that resistant individuals are selected (to the detriment of the sensitive ones). In other words, the frequency of resistant individuals must increase from one in a million to around 2030%'(Leroux, 1982).

Leroux recognises that the mode of action behind this selection and the factors that control it are largely unknown. 'Many of the observations made in practice involve situations where the final stage has been reached. That is to say, 90 to 100 per cent of the populations are resistant individuals, and treatment is completely ineffective' (Leroux, 1982). So, as Phytoma acknowledges: 'Not all unexplained attacks of fruit scab can be systematically linked to the issue of resistance' (Phytoma, May 1982).

These observations suggest that there is much confusion and that, in the face of an unexplained phenomenon, the terms used in an attempt to clarify it are likely to be inadequate. In this case, the term 'resistance' is inadequate to define a process that is nearly immediate, as well as persistent.

So might this phenonmenon be the result of proliferation? Is this the case in the stimulation of the development of Botrytis in tomatoes (Cox and Hayslip, 1956), strawberries (Cox and Winfree, 1957) or grapevines (Chaboussou, 1968), by various dithiocarbamates (maneb, nabam, zineb) used against *Oidium*? (See above, Chapter I).

Unless comparisons are made with controls treated with pure water, one cannot dismiss the hypothesis that the disease is stimulated by an indirect effect of pesticides on the plant's physiology. If it is indeed a nutritional effect that leads to the appearance of this pseudo'resistance', which may be comparable to a proliferation, it would be logical to assume that, conversely, one might obtain anti-fungal results by using 'nutritional' treatments which are beneficial for the plant. This would especially be the case, as we have seen above, with traditional fungicides such as copper and sulphur. This is what we shall now study, in regard to control of fruit scab. We shall begin with an investigation of the mechanism of the sensitising of the plant we wish to protect.

2. The nutritional requirements of Venturia inoequalis and the mechanism behind the sensitisation of the plant by new chemical 'anticryptogamics'

Certain studies of how different sources of nitrogen promote the growth and sporulation of various isolates of *Venturia inoequalis* have shown, in particular, that:

- Lhistidine is an excellent source of nitrogen for sporulation with certain isolates,
- DL alanine, DL phenylalanine, DL asparagine, DL aminobutyric acid, Lproline and glycine prove to be equally good sources of nitrogen for sporulation (Ross, 1968).

In other words, the same nutritional effects that encourage some amino acids (found in the exudations or in the tissues themselves) also encourage the development of pathogenic fungi.

Might this state where proteolysis dominates be caused by the action of synthetic fungicides, and could this be the origin of what is designated 'resistance'? Pathologists have remarked on the fact that this 'resistance' is observed precisely with those products that inhibit protein, sterol, and carbohydrate syntheses. If one accepts that pesticides participate in the metabolism of the plant by penetrating its tissues, it is possible that, as a result of synthesis being inhibited, the plant may be predominantly in a state of proteolysis, and thus susceptible to parasites for nutritional reasons.

In other words, these 'resistance' phenomena which multiply in such a strange manner can perhaps be explained as processes of proliferation which themselves result from a sensitising of the plant. The plant is in a state in which proteolysis predominates, a state that may itself have been caused by one or more deficiencies.

3. The provocation of deficiencies by synthetic pesticides

A. Soluble nitrogen and susceptibility to disease.

Looking at the relationships between diseases and the amino acids, Van Andel (1966) emphasises:

'The fact that the nitrate nutrition of the plant may influence its sensitivity indicates that nitrogen metabolism plays a role of its own in the relationship between the plant and the pathogen.'

Phenolic compounds have raised hopes of finding toxic antagonists of the parasites (as we discussed in Chapter 2). To quote Van Andel:

> 'The effect of the phenolic compounds, which according to Flood and Kirkham have been found to be very important in the resistance of apple trees to *V. inoequalis*, was found to depend on the nitrogen concentration of the amino acids.'

In other words, as we saw in Chapter 2, the phenols, like the other phytoalexines proposed, do not seem to have any sort of toxic effect on pathogenic fungi. They act as balancing elements for the amino acids, which play an eminent and decisive nutritional role against the parasites. This would seem to confirm the findings of the same author. As he observes, 'Some amino acids such as glycine, asparagine, aspartic acid, and glutamic acid, which hold a key position in the metabolism of nitrogen in plants, have been found to increase susceptibility to cryptogamic diseases.'

The influence of soluble nitrogen on the susceptibility of apple trees to scab can be found at the level of varieties. As Williams and Boone (1963) observe: 'The Cortland variety, which is susceptible to all strains of *Venturia inoequalis*, has an asparagine content of 1.969 in the leaves, whilst the McIntosh variety is resistant with levels of only 0.756.'

The variety therefore affects the resistance process by means of the level of protein synthesis that it is able to create, though this varies in accordance with local conditions.

B. The effects of treatments with chemical pesticides

It is essential to remember that all synthetic fungicide treatments increase total nitrogen levels in the plant being treated. There are, however, great differences in the amino acids when analysed separately (Symposium on integrated pest control in orchards, Boulogne, 1972.) As we saw above, nitrogen works in balance with boron. What happens at the level of the soil might equally well be produced as a result of repeated treatments with chemical pesticides, which are almost all nitrogenous. So, one might ask whether these frequent boron deficiencies in grapevines and fruit trees arise as a result of various chemical pesticides.

It is also important to record that the nitrogen content of the leaves varies widely, according to the way the nitrogen is used. Soenen (1975) notes, in regard to the percentage of N in dry matter:

- 'intensive' use: 2.44% N
- 'random' use: 1.9% N

The author attributes these differences to the effect of the pesticides. This confirms what we suggested above. Lefter and Pascu (1970) established that the degree of the fruit scab attack is proportional to the ratio: N % / K_2O %. Such a relationship would seem to justify certain practices in the control of fruit scab, whose action can only be the result of nutritional effects.

4. Nutritional treatments against fruit scab

Potassium is frequently found in the formulations recommended to control fruit scab. Demestjeva and Sturna (1970) report good results from the use of potassium chloride as a leaf spray (six sprayings during the season).

Apart from sulphur and copper (which can be considered as nutritional chemicals and are officially confirmed to act against fruit scab certain popular products employ a mixture of sulphur and potassium carbonate, as generally used in Germany. Potassium permanganate, which is rich in manganese, is also used.

Of course, potassium plays a fundamental role in protein synthesis. This is shown by the fact that every K deficiency causes a growth of liquid exudate in the plant that occurs following protein hydrolysis, with an accumulation of amino acids, especially asparagine. This is an amino acid which is recognised, as we have seen above, to be the preferred food of *Venturia inoequalis* and of a number of other pathogenic fungi.

Optimum protein synthesis, as shown in research carried out on apple and pear trees, has certain requirements. It is essential to guard against K deficiency, but K must be present in balance with calcium, which is also indispensable. Crane and Stewart (1962), working on *Mentha piperita*, were able to establish effectively that a K/Ca ratio where Ca is dominant encourages a physiological state with dominant proteolysis, while the opposite is the case if potassium is the dominant element.

In the first case asparagine is present in greater quantities, which should in principle encourage attacks of fruit scab. In the second case, however, the opposite is true: the plant is predominantly in a state of protein synthesis with a high glutamine content (Fig. 2).

We do not have the figures to allow us to posit a relationship between the K/Ca ratio and the resistance of the plant to fruit scab. However, we have one indication. For example, according to Soenen (1976), the optimum K/Ca ratio in the apple tree is 1.20. (Soenen does not specify to what extent this ratio would lead to resistance to fruit scab.

As for boron, we do not have at our disposal, for the moment, a single reference to its role in relation to fruit scab. It is extremely likely, however, that it plays an important role, to the extent that (as we have seen) it is a direct factor in protein synthesis.

Certain good effects of zinc-based fungicides may quite likely be due to the enrichment in zinc of the tissues of the plants treated with them. It has been established that every deficiency in zinc leads conversely to an increase of amino acids in the tissues, especially of asparagine.

VII. The immunity of the plant achieved by nutritional treatments

First, it is important to define what we understand by the term 'immunity'. As we have emphasized above, certain factors affect protein synthesis, causing the plant to show resistance. Yet even if the plant appears to be 'genetically' resistant, this cannot be the case for the whole of its annual cycle. The important thing is that the plant is resistant at the stage of 'sensitive periods,' especially at the moment of flowering.

Moreover, we have seen that different factors governing the plant's environment may serve, as it were, to 'erase' resistance. A harmful influence may often be exerted by chemical pesticides. So it seems that such an inhibition of protein synthesis (to which we attribute the sensitisation of the plant to its parasites) may have a deficiency as its immediate cause. This deficiency is the result of an 'inhibition' induced by the pesticide, possibly through repeated applications to the leaves or to the soil. In both cases, the outcome is malnutrition in the plant.

Toxicity and malnutrition could be (according to Vago) the two factors at the origin of diseases, especially viral diseases, due to the metabolic problems that they engender (Chapters 5 and 6).

This is the reason why, in the third section of this chapter, we gave examples of the difference in levels of various elements found in the tissues of healthy tomatoes and those badly infected with a viral disease. It is significant that various deficiencies are recorded, of Ca, Mn, and Cu - deficiencies that are not the consequence but the *cause* of the disease.

If, as we believe we have demonstrated above, deficiency = metabolic problems = disease (that is to say, parasitism), it would be normal to register a remission of the disease to the extent that we are able to correct every possible deficiency. It is at this point that we can see the extent of our ignorance. We do not know, for example, how to correct micronutrient deficiencies, since we are still quite far from knowing all the properties of these elements, or the exact manner in which the deficiencies are manifested.

In order to proceed further, we have to await further progress in plant physiology. However, what we already know should enable us to avoid the worst mistakes and even to have a positive effect on the resistance of the plant. Often, in the use of chemicals , we aim at the parasite but it is the plant which is damaged.

Synergies in micronutrients

While copper and sulphur seem to work well, use of these elements in isolation may not be the best way to achieve nutrition for maximum resistance. Many other micronutrients may also play a part.

We learn from D. Bertrand, for example, that the full range of properties of boron is only manifested in the presence of magnesium, manganese, and molybdenum. In the same way we see how copper and manganese work together in the growth of wheat (Piland and Willis, 1937) or in the resistance of rice to *Piricularia* (Primavesi et al., 1972).

Another example of 'synergism' and its practical results, comes from Dufrenoy (1930). As he points out, 'With a solution containing 26 elements, Hoagland and Snyder obtain strawberries which are much more vigorous and more resistant to Oidium and red spider mites than with a solution containing only 12 elements.'

All this in the meantime justifies the logic of using micronutrient complexes, allied with the major elements. This forms the basis of our suggestions for controlling fruit scab.

The balance: KCaMg

We draw here mainly on the work of Crane and Steward (1962) concerning the K/Ca ratio in relation to the level of protein synthesis. For the rest, we rely on the results obtained for the decline of various parasites through better potassium nutrition, as we have seen above.

Calcium is also a fundamental element for the plant's nutrition. Shear (1975) lists no less than 30 afflictions or diseases that arise as a result of calcium deficiency. Calcium and magnesium are very closely related. For example, Shear (*op. cit.*) notes that if Mg levels are increased to a sufficient degree, the level of absorption of Ca also rises. This explains the good results obtained in tackling diseases involving 'necroses,' and 'black heart,' or 'black rot.' At the moment, 'black rot' in chicory and necrosis in the terminal buds of chard grown under glass can be controlled by treatments based on a combination of Ca, Mg, and B.

In the same way, 'proliferation' in apple trees, which is attributed to the multiplication of a mycoplasma, is only secondary and can be treated effectively by a special combination of Co, Cu, and Mn. This is quite logical, bearing in mind that calcium deficiencies, whether spontaneous or as a result of boron deficiency, are often manifested in shrinking internodes known as 'witches' brooms' on the trees or in crinkly leaves. K and Ca are closely linked: Ca deficiency causes liquefaction of the middle lamella of the cell wall, because of lack of potassium pectate. This is what makes the K/Ca ratio important.

Chiefly by correcting this ratio in favour of potassium, we were able to control scale in citrus trees, as Vaithilingam (1982) managed to eliminate parasitic insects from rice (p. 206).

Treatments with micronutrients

Following his studies of diseases of the citrus and regarding deficiencies and their pathological effects, Dufrenoy expanded on other diseases, experimenting with various micronutrients and especially with zinc. It was found that the number of enzymes containing zinc as a component was very high.

This explains the histological modifications brought about by the functional problems of enzyme chains. He drew attention to the use of zinc sulphate in grapevines against 'fanleaf' or 'infectious degeneration of grapes' (Dufrenoy, 1934).

He notes: 'From the point at which the buds burst, the vine developed branch tendrils with longer internodes than in the 'fanleaf' vines. Then, on 24 July, measurements confirmed that this elongation of the merithalles was accompanied by abundant fructification.' We know that zinc deficiency, especially in tomatoes, causes protein synthesis to be inhibited, and that this coincides with an increase in amino acids. Significantly there is a considerable accumulation of asparagine, a nitrate foodstuff which, as we have seen, is essential for pathogenic fungi (Krisna Chutima, 1963).

As for boron, we recall that applications of this element, whether to the soil or as a leaf treatment, proved to be effective against a number of diseases. Thus, by correcting boron deficiency in beet, it is possible to eliminate *Phoma betae*. In the same way, silver leaf disease can be controlled in grapevines.

In the case of peach blight ravaging trees that are shrivelling in an environment too humid for them, Dufrenoy and Bruneteau (1937) showed that those trees treated with a boron salt re-establish themselves very quickly.

Similarly, the trials of Branas and Bernon (1954) showed that grapevines attacked with shrivelling by silver leaf disease [*Stereum purpureum*] reestablish themselves when boron treatments are applied to both the leaves and the soil. This is not really surprising, given the importance of boron in the plant's physiology, as we have seen above.

These findings lend weight to the hypothesis that boron deficiency lies at the bottom of a large number of diseases, especially viral diseases. As we have emphasized, the symptoms of viral diseases resemble those arising from boron deficiency. This would seem to be further proved by the example of 'apricot virula.' As Pena and Ayuso observe, 'As a consequence of the symptoms, the development of the disease, and *the result of analyses of the leaves*, the boron deficiency hypothesis could be advanced' (Pena and Ayuso, 1970).

We must also probably place the 'declines' in the same category of ideas: in other words, the various types of withering seen in fruit trees, for example, pears and apples. Campbell (1970) was able to obtain good recovery rates in pear trees suffering from 'apple decline' thanks to copper and zincbased sprays, after he discovered a number of 'latent' viruses in the trees. This would seem to confirm our opinion that all diseases, including viral diseases, are linked to metabolic problems that are in turn often linked to deficiencies.

Finally, the 'complex' disease that attacks certain species of *Prunus* in the Molières region in Tarn, seems also to be the result of one or several deficiencies. One thing is certain: the existence of a pathogenic agent, a mycoplasma, has also been suspected here. The existence of this organism has been shown convincingly in the infected host plant. But, as researchers recognise (Bernhard et al., 1977), the pathogenicity of this mycoplasma is far from obvious. Yet again we are faced with the same question: cause or effect? As far as we are concerned, this question still calls for the same response: it is the deficient nutrition of the plant that leads to its susceptibility, due to inhibition of protein synthesis. (Research is currently in process to determine, as far as possible, the factors that are responsible: root stock, variety, feeding with major elements, micronutrients, etc. One might add that all these factors relate to the nutrition of the plant.)

Finally, we recall that numerous authors have deliberately tried using micronutrients against diseases, with an indirect effect on the nutrition of the plant they are trying to protect. Mudich (1967) showed that, unlike Cu, Zn, and Mn, molybdenum caused a decrease in the susceptibility of potatoes to mildew. However, this same author quotes Galilov, according to whom potato diseases can be controlled with some level of success by the use of various micronutrients such as Cu, Mn, B, and Zn. We think that the differences in these results may arise from differences, to start with, in the composition of the soil, and especially from any later deficiencies.

The research of Prusa (1965) into the effects of various micronutrients on leaf roll in hops also produced interesting results. The author established that the disease was viral in character, and emphasised the close relationship between the external manifestations of the disease in other words, the symptoms and the nutritional conditions.

In the field, the disease was controlled by applications of various salts based on micronutrients, especially those with a base of boron, magne-sium, manganese, nickel, iodine, and zinc.

This led to the disease being controlled in Germany by zincbased preparations, whether in the form of sulphate or zineb. Still, we should point out equally significant effects as those achieved with zinc may be obtained with the use of boron or magnesium. Analysis shows that diseased plants have relatively low levels of these two elements .

On the other hand, one fact underlines clearly the *indirect nature* of these effects: *copper-based products aggravate the disease rather than combating it.*

We should also pause a moment to look at another procedure of indirect pest control, by an action beneficial for the metabolism of the plant. This is an elegant procedure, which consists of soaking seeds in micronutrient solutions.

Polyakov (1971) tried the following: Cu, Mn, Co, and B against *Sclerotinia* or grey mould in sunflowers. His method was to soak the seeds before sowing them. In practice, seeds were soaked for 10 hours in salt solutions of 0.1%. Two litres of solution are sufficient to soak all the seeds needed to plant one hectare.

These experiments show a clear decline, according to the evidence, with different micronutrients (Table 17).

Table 17: Percentage of attacks on sunflowers by *Sclerotinia* according to the micronutrients used to soak the seeds (averaged over 4 years)

Controls	Manganese	Cobalt	Boron	Copper
16.3	4.4	4.9	6.4	7.7

As can be seen, cobalt and manganese have shown the best results. However, one wonders, after what we have already seen above regarding synergetic effects, whether certain 'complexes' of micronutrients might not have been still more effective. Interesting research could be undertaken in this direction.

Polyakov's experiments (which note in passing that 'microelements' improve resistance to numerous diseases) also included an analysis of the leaves. It was shown that through the action of the micronutrientsone may bring about a decline in reducing sugars.

The author observed that this phenomenon is the consequence of a reflux towards the reproductive organs, and of rapid transformation of simple sugars into reserve nutritional substances. As a result the development of the pathogenic fungus is inhibited, since it no longer has the soluble sugars of the plant cells at its disposal.

The outcome is an explication that coincides completely with our theory of trophobiosis. Polyakov writes, 'The treatment of plants with micronutrients intensifies the physiological processes and thus causes the pathogenic agent to be inhibited or destroyed, as happens with varieties which are naturally immune.'

General Conclusions

It is universally recognised that the health of our crops is declining. There is a serious proliferation of parasites and disease, in spite of the use of powerful chemical pesticides, In fact it is precisely these chemical pesticides that cause the problem. At long last it has been admitted that they have 'repercussions,' otherwise called 'side effects.' In other words, their use causes other problems to flare up, in the shape of rapid proliferation of pests or the development of diseases (fungal, bacterial, and viral) - not to mention the appearance of new organisms recently identified as mycoplasmas, spiroplasmas, or 'viroids.'

In human and veterinary medicine such phenomena have been labelled 'iatrogenic illnesses.' When it comes to the health of plants, we should recognise similar 'failures' with equal clarity. These are normally characterised, however, in different ways, depending on whether one is dealing with the rapid multiplication of an insect or mite, or with the development of a pathogenic agent from the plant kingdom. One speaks of 'proliferation' when a treatment with one pesticide or another is followed by an increase in populations of spider mites, aphids, or psyllids. However, when fungal diseases are raging, such as Oidium or botrytis in grapevines or fruit scab in apple trees, the rapid multiplication is explained as a 'resistance' of the pathogenic agent to the fungicide in question.

According to the traditional idea, if there is a rapid multiplication of spider mites or aphids after a treatment with parathion or captan, these chemicals are said to have destroyed their natural predators. However, such an explanation does not apply to all 'imbalances' of this sort - for instance, rapid proliferations following treatments of the soil. It is even less useful when looking at the development of cryptogamic, bacterial, or viral diseases. This is why, as mentioned above, a different explanation is advanced: a 'resistance' of the pathogenic fungus to the specific fungicide.

But to adopt such an interpretation is to turn a blind eye to a series of results obtained in both laboratory and field tests. As we ourselves showed in the case of spider mites, the pests actually increased their biotic potential when fed on leaves treated with the suspect chemical. There is an increase in daily rate of reproduction and life expectancy, modification of the gender ratio in favour of the female, and shortening of the evolutionary cycle. Such a process, also found in aphids, explains their proliferation, which is nutritional in nature.

Such phenomena are not, however, limited to the animal world. It has also been shown, for example, that dithiocarbamates cause the development of *Oidium* and *Botrytis* when used to treat grapevines for mildew. (Chaboussou, 1968, and Vanev and Celebiev, 1974). The mechanism is therefore the same in both cases, and fairly easy to prove as long as one has adequate controls.

We therefore believe we have established a fundamental fact: namely, that the relationships between plant and parasite are primarily nutritional in nature. We have given the name 'trophobiosis' to this theory.

We have shown that soluble substances such as amino acids and reducing sugars cause the stimulation of biotic potential in parasitic organisms (for instance, spider mites, aphids, bacteria, pathogenic fungi, and viruses) which is expressed as proliferation in the field. These substances, which are localised in the cellular vacuole and in the vessels, form the raw materials of the proteins, with which they are in constant balance. We referred to various authors, in particular to the works of the eminent French biologist Jean Dufrenoy.

In the last analysis, then, the resistance of the plant to its different parasites depends on the balance between protein synthesis and protein breakdown. In other words, *a predominance of protein breakdown increases the plant's susceptibility*. This happens with the use of 'poisons' (which is what chemical pesticides are for plants), especially in multiple applications.

Conversely, *predominance of protein synthesis increases the plant's resistance*, or its 'immunity.' This is what happens with some 'remedies,' whose action on a plant's metabolism render it resistant thanks to the biochemical state created (at least for a certain period). This resistance seems to correspond to a great extent to that found in varieties that are resistant 'by nature,' that is, genetically.

In this new development of bacterial and viral diseases, chemical pesticides, especially for aggravating the poor health of fruit trees. This is why we gave some space to the issue.

It seems to us particularly significant that a plant bacteriologist such as our colleague Ride (INRA), in his research on the growing importance of infections of bacterial origin, sees the same connection. As he notes, 'This spread seems to occur as a result of the increase in treatments making use of synthetic chemicals.'

It is obvious that we here encounter two factors at work. Both of them intervene in the same harmful way at the level of the plant's physiology:

- the abandoning of mineral products, such as copper or zinc products.
- the adoption of chemical pesticides, especially dithiocarbamates.

What might at first appear to be a simple substitution of one product for another actually has a doubly harmful impact on the plant. Minerals like copper and zinc generally have a beneficial effect on the plant's metabolism, and thus on its resistance. Chemical products, on the other hand, have the opposite effect, by inhibiting protein synthesis. The impact of these two effects on the metabolism of the plant make it susceptible to various parasites, especially bacteria.

Moreover the old question about the nature of the relationship between plant and disease has still not yet found an answer. In the words of Trocmé:

> 'We cannot exclude the possibility of relationships between deficiencies and certain diseases, especially bacterial and viral diseases. It may be that these diseases encourage the manifestation of deficiencies, or that the deficiencies encourage the manifestation of these diseases. For example, trees deficient in boron or zinc may be more susceptible to certain viral or bacterial diseases' (Trocmé, 1964).

We believe that our trophobiosis theory could fill this gap. As we see it, there is a sequence that has been clearly proven, namely:

Deficiency → inhibition of protein synthesis →accumulation of soluble substances →improved nutrition of parasites →rapid multiplication and virulence of bacteria and viruses, etc.

It also seems vital to try to confirm as soon as possible the relationship that seems to exist between the use of chemical pesticides and the triggering of deficiencies. Several works have raised this issue. As we have already mentioned, the overwhelming majority of chemical pesticides are nitrogenous and often chlorinated as well. Thus the traditional balance between nitrogen and micronutrients (especially boron and zinc) may be upset in favour of soluble nitrogen, because of a deficiency. This in turn creates a situation that is unfavourable for the plant's resistance.

The theory of trophobiosis also explains why symptoms caused by deficiencies coincide with those caused by diseases. Official plant protection, however, assiduously casts doubt on this convergence, claiming that any cause-and-effect relationship is an illusion.

We are still a long way from grasping the extent and the gravity of the effects of chemical pesticides. This applies especially to multiple treatments that some research suggests may have a cumulative effect.

For the same reasons, chemical fertilisers may cause deficiencies. These can have extremely serious consequences for the plant's metabolism. Dufrenoy, for example, has shown that boron deficiency in broad beans causes disintegration of the nucleus of the cell. In conditions such as this, it is hardly surprising that viral diseases and other diseases of the root can develop, quite possibly as a result of the cumulative use of herbicides.

Viral diseases appear to differ very little from other diseases in their reactions to environmental factors or to contamination of the plant. In other words, just as with other diseases, it is the biochemical state of the plant that generally determines how susceptible it will be.

In this connection we gave a brief summary of the work of Vago, in relation to the effects of the environment on the development of viral diseases in insects. *It seems that, in both animals and plants, susceptibility to disease is the result of metabolic problems.*

Furthermore, these problems themselves may appear as a result of malnutrition or toxic effects. Such a process seems to stem quite often from the effects of chemical pesticides. This may occur through direct action on the plant at the time of application (especially of herbicides), which are scattered so liberally with no concern for the life of soil microorganisms. Or it may occur through the impact of chemical fertilisers and the deficiencies they can cause, which in turn affect the assimilation and nutrition of the plant.

It is no accident that, in exploring these issues of resistance and protection of the plant, we find ourselves face-to-face with the phenomenon of nutrition. At this point it is perhaps relevant to recall the work of Bodenheimer (1955), who disputes the Darwinian theory about the regulation of animal populations by predators and parasites. Bodenheimer stresses that this never prevents proliferation in the environment. Instead, Bodenheimer emphasizes the importance for population levels of the quality of the diet as 'the source of animal energy'.

For example, fluctuations in vole populations do not necessarily conform to the level of predators, but can be explained, he says, by differences in levels of fertility. These differences may themselves be subject to an indirect seasonal influence on the quality of the diet. Bodenheimer also mentions the voles' ingestion of certain gonadotrophic substances produced in certain seasons and in varying quantities.

In addition, according to Maurice Rose and Jore d'Arces (1957), the nutrition of organisms, via deficiencies, has played a fundamental role in the process of evolution itself.

To conclude: a theory only acquires value through the results it provides. In this respect, we can already say that the results so far obtained concerning the protection of various plants from different diseases serve to confirm our ideas and encourage us to continue along this path. These results are based on achieving well-balanced fertilisation and stimulation of protein synthesis through the use of complexes of micronutrients.

First, we need to overcome the idea of 'the battle': that is, we must not try to annihilate the parasite with toxins that have been shown to have harmful effects on the plant, yielding the opposite effect to the one desired. We need, instead, to stimulate resistance by dissuading the parasite from attacking.

This implies a revolution in attitude, followed by a complete change in the nature of research. Work on plant physiology and its relationship to the resistance of the plant would be particularly important. In other words, we need a deeper understanding of the relationship between the 'conditioning of the plant' and proliferation of the parasite.

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THIS BOOK CALLS INTO QUESTION THE CENTRAL ARGUMENTS OF INDUSTRIAL AGRICULTURE, sets out the theoretical foundation for ecological agriculture and quietly calls for a revolution. Based on his own research and that of others, Francis Chaboussou argues that we should improve the health of our crops rather than using chemicals to eliminate pests and diseases, because healthy crops resist attack. Furthermore, chemical pesticides and fertilisers weaken plants, making them vulnerable to disease.

Chaboussou says: '... we need to overcome the idea of the 'the battle': that is, we must not try to annihiliate the parasite with toxins that have been shown to have harmful effects on the plant, yielding the opposite effect to the one desired. We need, instead, to stimulate resistance by dissuading the parasite from attacking. This implies a revolution in attitude, followed by a complete change in the nature of research. Work on plant physiology and its relationship to the resistance of the plant would be particularly important.'

As farming becomes more intensive and GM crops are prmoted as another narrow 'techno-fix', Chaboussou's book reminds us that there are genuine alternatives to the chemical treadmill. The questions he raises about the impact of chemical pesticides and fertilisers on the health of plants urgently need to be investigated with new research.

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'Almost all conventional chemical agricultural technologies create favourable conditions for the growth of pest and disease organisms: this book shows that much of the problem can be explained through increases in soluble nitrogen, amino-acid and sugar concentrations in the plant cells.'

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'Although the ways in which synthetic fertilisers and pesticides affect plant physiology are not yet fully understood, Chaboussou's Theory of Trophobiosis gives a clear approach for how to deal with such problems in practice. learning how to treat the sick and not the sickness is an effective tool for plant protection, substantially reducing problems with pests and diseases in the field, especially for those working in agriculture without chemicals.'

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