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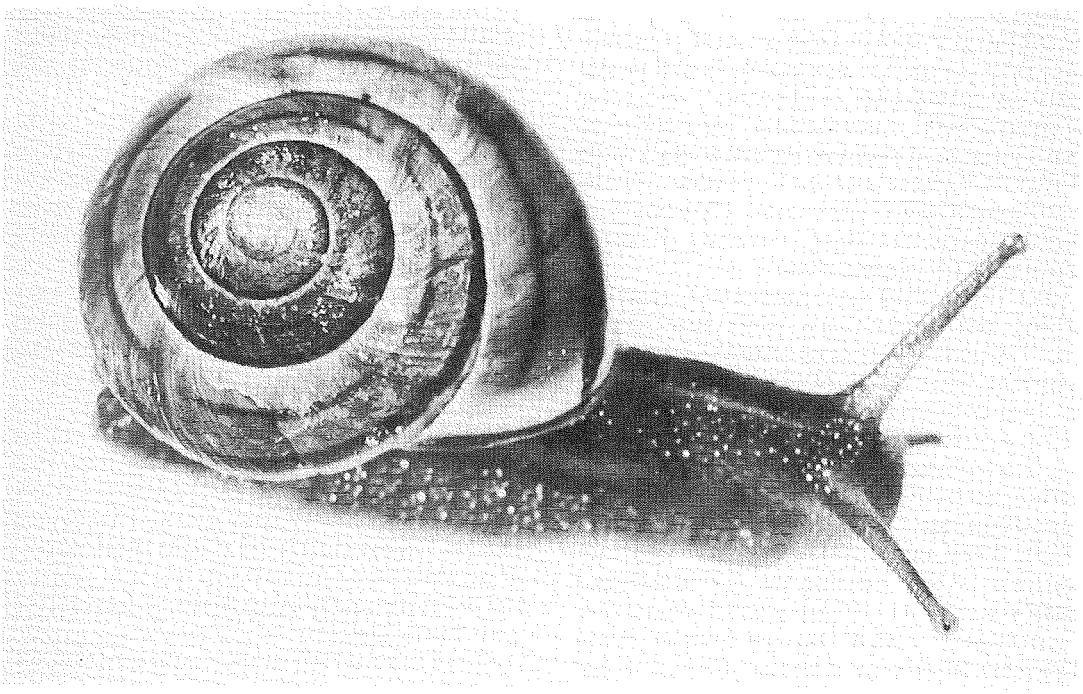

Tema: **Soil Pathosystem** Trädgårdssnäcka — *Cepaea hortensis.* Foto: Karl-Fredrik Berggren.

INNEHÅLLSFÖRTECKNING:

Soil pathosystems — a multidisciplinary subject

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The course ''Soil Pathosystems''

This issue of ''Växtskyddsnotiser'' covers some of the topics discussed at the 6th Scandinavian post-graduate course in Plant Pathology. The course was held at Garpenberg, Sweden, Nov. 21—28, 1985. Its title and leading theme was, as in this issue, ''Soil Pathosystems''. Thirthy six of the 42 students attending the course gave an oral presentation or a poster for discussion in the poster sessions. Summaries of students', and some of the teachers', contributions, were collected in a course compendium. Before attending the course at Garpenberg in November, all the participants had studied some 1200 pages of recommended literature, covering different aspects of the course theme.

Our main, and very apreciated, teachers during the Garpenberg week were Prof. J.L. Lockwood, Michigan State Univ., East Lansing, USA (part of his contribution is covered in a resent publication; Lockwood 1986), and Prof. E.I. Newman, Univ. of Bristol, England (some of his central topics were pathogens in natural ecosystems and rhizodeposition, see i.e. Newman 1978, Newman 1985). They interacted excellently and managed to avoid splitting up the broad and somewhat diffuse theme given into smaller ''sub-subjects''. Eight teachers from the Scandinavian countries gave one lecture each (some are published in the following pages), and further arranged demonstrations and evening discussions. Our ability to stand the very tight schedule and the intense scientific discussions at Garpenberg was greatly increased by socials and ''singing sessions'' arranged by Agronomist Paula Persson.

The theme ''Soil Pathosystems''

A pathosystem could be defined as a pathogen, its host and their environment, which typically could be seen as being in a kind of ''dynamic eqilibrium''. For soil pathosystems the environment is soil with all its variability in texture, organic content, water, nutrients, soil atmosphere etc. and a myriad of soil bacteria, fungi, protozoa, algae, arthropodes and nematodes. The host part encountered is most often the plant root with its specific physiology, root turn over, rhizodeposition, rhizosphere, a somewhat undefined surface and possibly infected with mycorrhiza and N-fixing bacteria. The soilborne pathogen itself is usually more dependent on the growing system (i.e. soil cultivation, plant debris, crop rotation) and more difficult to handle epidemiologically than pathogens borne by air, vectors or plant seeds.

Thus, in centering on the system as such, soil pathosystems — apart from being hidden in the soil — are complex, variable and demand broad knowledge from those studying them. Concepts from widely different areas are needed. The interaction of people from various disciplines in the course was also very rewarding. Many of us were again reminded that plant pathologists may have a great deal of learn from soil scientist, soil microbiologists, root physiologists, ecologists etc., but also that plant pathology may greatly enrich the thinking of these specialists. I believe that no one attending the course will ever forget that, apart from soil physical and chemical factors, other microorganisms and roots, there are a lot of nematodes, bugs, protozoa, rhizodeposition and fungistatis interacting with the soilborne pathogens we study. We further get a quite clear view of the enormous need for better knowledge about soil biology. Thus, the subject ''soil ecology'' seems to a great extent an unknown subject, not least concerning many details of interest for plant pathologists.

This, our lack of knowledge of what really happens in soil and along the plant roots, has sometimes given expectations that there may be many still unknown phenomena, of scientific but also of great practical interest, hidden in the soil biological system. Since a few years back, research on soil pathosystems has also been something of a ''hot area'' in the science of plant pathology. We have a renewed interest in things like biological control, soil amendment, crop rotation, disease decline effects, disease suppressive soils, allelopathic effects etc. for combatting our soilborne plant diseases or increasing crop yield. Partly this rise in interest could be seen as one side of the increasing interest in organic farming and other movements of this kind. Presently there are also great research efforts placed in areas like non-pathogenic rhizosphere microorganisms, minor pathogens, plant growth promoting rhizobacteria, deleterious rhizobacteria etc. Some recent research results in these areas are, from a practical point of view, quite ''thrilling'', as was also underlined in the course contributions. However, we also agreed in the course that it may take very long before we understand the background or are able to use such ''thrilling'' research results practically because of lack of background knowledge and basic principles. Here, our hope is that our young researches do not seek other, and perhaps easier research areas, because of the complexibility of the system, the great methodological difficulties and consequently, ususally slow results. One aim of the course was to encourage our younger Scandinavian researchers not to.

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Origin and evolution of fungal plant pathogens

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DE NEERGAARD, E. 1986. Origin and Evolution of Fungal Plant Pathogens. *Växtskyddsnotiser 50:* 6, 152—157.

Phylogenetic studies of the plant pathogenic fungi are often based on the assumption that parasitism is an advanced character compared with saprophytism. According to this, the highest developed plant pathogens will represent phylogenetic "dead ends". There are several theories of the origin of the groups of fungi which include plant pathogens. The ancestors of the Phycomycetes may be different Procaryotes. Ascomycetes and Basidiomycetes may be derived from Protozoa, Phycomycetes, Red Algae, or Chytridiomycetes, dependent on which theory is applied.

The fungal plant pathogens are characterized by their high geological age, their rate of evolution under certain conditions, and their high ability of diversification when new ecological opportunities arise. All these factors imply a high evolutionary potential. In certain cases parasitic forms seem to be ancestral to saprophytes. This statement contradicts the blind-alley-theory.

Short-time evolution is observed in agriculture where different crop systems influence several population-dynamic interrelationships in the host-parasite system. The concept of the pathosystem as a unit should be preferred when planning future strategies for crop protection.

Introduction

The ability of developing highly specialized forms is a remarkable and characteristic feature of plant pathogens as well as of other parasites. Specialization permits a relatively large number of species to utilize a given set of resources. As a consequence many species may co-exist under equilibrium conditions. Comparisons with predators show that they, in contrast to parasites, are relatively large and mobile and exploit a relatively fine-grained (uniform) environment. Populations of predators will tend to be monomorphic and unspecialized. Few species of these generalists can co-exist under equilibrium conditions. Parasites, on the other hand, represent the extreme in the exploitation of a coarse-grained (non-uniform) environment. Variations in resources (i.e. hosts) in time and space lead to development of geographic races and polymorphism (Price, 1980).

The evolution of parasites is often regarded as slow and their specialization as leading to phylogenetic "dead ends" (the blind-alley-theory). This is based on the opinion that generalized organisms persist as species through time for periods longer than specialized organisms. Furthermore, a common trend in evolution is a general increase in size of individuals, thus leading to increased complexity and greater control of the environment, and so, supposed, greater independence from the environment (Price, 1980). The idea on which this is based is expressed by Huxley (1953): "Progress is considered as constantly leading life into regions of new evolutionary opportunity."

This point of view is challenged by Price (1980) who prefers **the concept of evolutionary potential,** based on the fact that natural selection results in a series of adaptive reactions to ecological opportunities. He states that no group of organisms can surpass the parasites in their potential for continued adaptive radiation (this expression will be explained later).

Evolutionary potential

The phylogenetic age of parasites, their rate of evolution, and their extreme adaptive radiation (variability) are arguments leading to the opinion of parasites as organisms with a big evolutionary potential, this as a contrast to the "dead-end-theory". These three factors, on which estimates of the potential could be based, will be dealt with more detailed in the following text.

Phylogenetic age

The knowledge of the origin of fungi is based mainly on comparative studies of recent species. The fossil record is highly insufficient, only some 500 species of fungi are known.

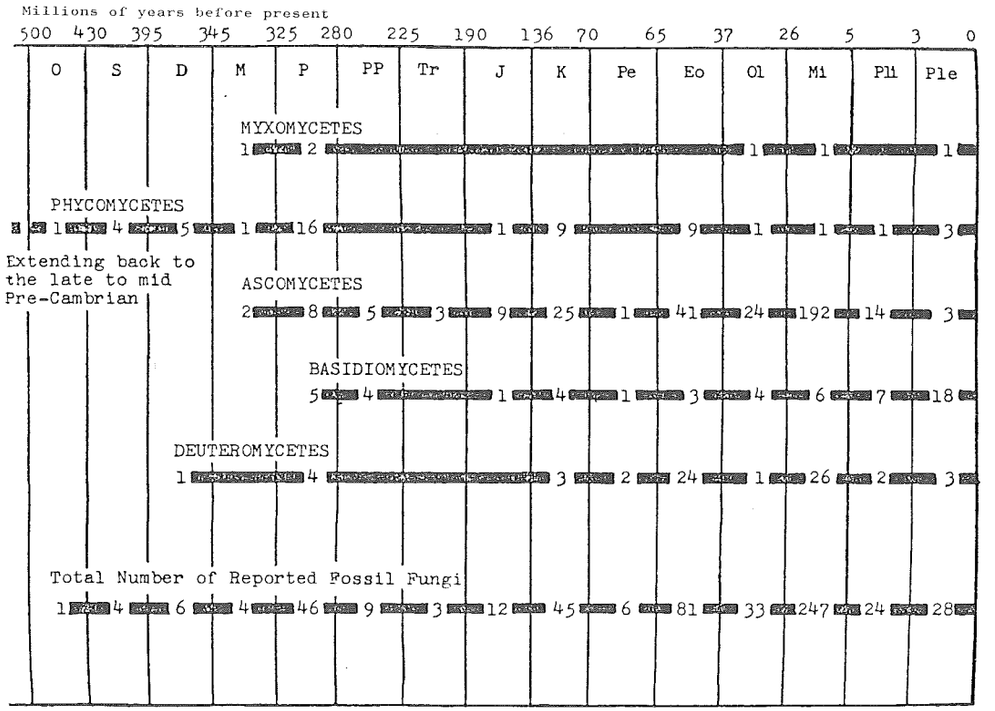

Figure 1. The Classes of Fungi in geological times (Tiffney & Barghoorn, 1974).

Their distribution in the fungal system reflects the frequencies of their substrates. Thus records on epiphyllous fungi from the Tertiary period (Eocene-Miocene) are abundant due to the fact that many leaves of angiosperms from this period have been found. In the same way most of the fungi known from the Carboniferous period are wood-inhabiting, caused by the commercial investigation of coal-layers. There are remarkably few fossil fungi known from the periods in between. The frequency of parasitic fungi among the fossils does not reflect a high ratio of parasitic/saprophytic species, but might be caused by the lower probability of preservation of saprophytic organisms.

Evolutionary schemes, based on studies of certain biochemical synthesis pathways (of for instance cell wall components, and biosynthesis of lysine and tryptophan) suggest an evolutionary line from ancestral Chytridiomycetes to the first Ascomycetes and Basidiomycetes (Bartnicki-Garcia, 1970), see figure 2. This investigation confirms the theories of relationship between some of the fungal classes based on comparative morphological and biological studies. It doesn't, however, fill the gape in our knowledge of the origin of the groups known today.

As ancestors for the group formerly named the Phycomycetes, which are older than the other groups, different classes have been pointed out: Algae and Protozoa. Non-septate hyphae-like filaments are dated back to 2000 mill. years ago. This is at the same time of the first Eucaryotes. This suggests a Procaryotic ancestor to this group, for instance the Blue-green Algae, or another more primitive Procaryote organism. Simple heterotrophs are necessary in any functioning ecosystem, thus explaining the role of fungi already in that period (Tiffney & Barghoorn, 1974).

The Myxomycetes, Ascomycetes, Basidiomycetes and Deuteromycetes are all known from the Devonian-Carboniferous period, that is 300—400 mill. years ago (Figur 1). These four groups are well distinguished at this time, and it is unlikely that they are derived from each other (Tiffney & Barghoorn, 1974), maybe except the Basidiomycetes which are a bit younger. According to different theories the Ascomycetes and the Basidiomycetes can be derived from (1) Phycomycetes, (2) Red

Algae. (Another theory points out the Protozoa as ancestor. In that case the Taphrinales will be the primitive part of the Ascomycetes.)

(1) Phycomycete ancestor. According to the theory of Savile (1955, 1968) the first Ascomycetes were the result of a selection pressure for airborne spore-dispersal in Phycomycetes which were **parasitic** in the early land plants. The first land plants appeared in the Silurian, 420 mill. years ago.

(2) Red Algae. Studies of the occurrence of the co-enzymes NADP and NAD show that Ascomycetes and Basidiomycetes represent a primitive form, close to the Procaryotes and primitive Eucaryotes. Other biochemical studies support the hypothesis that Red Algae might be the ancestor of Ascomycetes (Demoulin, 1974). Red Algae present a wide range of specific parasites on other algae. According to this theory, **parasitism** seems to be the first step in heterotrophic life, taken by Red Algae ancestral to fungi. Saprophytism could have evolved later from necrotrophic parasites. Parasitism in Red Algae seems to originate through adelphoparasitism (parasitism restricted to closely related species), so a complete sequence will be: Autotrophic life — Adelphoparasitism — Biotrophic alloparasitism — Necrotrophic alloparasistism — Saprophytism. Necrotrophic parasitism could have evolved as an answer to hypersensitivity, which acts as an effective reaction against biotrophic parasites in some hosts. By possibly killing their hosts necrotrophic parasites may not be as efficient as biotrophic ones, which may retain themselves alive for a longer time, but with the development of vascular plants they have found — in the decaying of their remains — an ecological niche that allowed an explosive evolution of saprophytic forms (Demoulin, 1974). The first Basidiomycetes would have been very close to the recent rust-species, but are supposed to have parasitized algae. Marine rusts may still exist (marine Ustilaginales were recently discovered). By comparison between the Red Algal trigenetic life-cycle and the long rust cycle it is possible to support the theory of relationship between the two groups (Demoulin, 1974). The emergence from the sea could have taken place in either of two ways: (1) Parasites following algae in their conquest of land. (2) Driftwood colonization preceeding the emergence from the sea. Closed and hypogeous fruiting bodies were certainly evolved as a response to desiccation. A parallel for this is the presentday

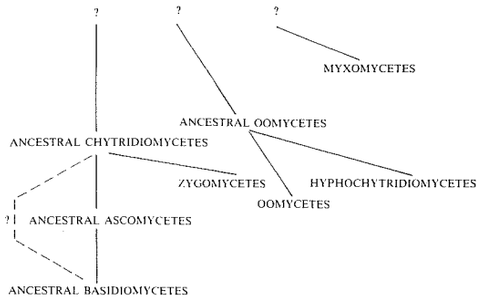

Figure 2. Evolutionary scheme based on the studies of Bartnicki-Garcia (1970) of biosynthesis pathways.

driftwood-inhabiting Pyrenomycetes which produce perithecia buried in the sand (Demoulin, 1974).

Saprophytism seems to have evolved again simultaneously or after the origin of the Basidiomycetes. Some dubious fossil species of Agaricales are as old as the first rust fungi 300 mill. years, certain agarics 150 mill. years younger (Tiffney & Barghoorn, 1974).

In these theories parasitism is a very old way of life. Of course saprophytism exists in all ecosystems, but it is likely to have evolved independently during the geological ages. Both theories describe an evolution in the direction from parasitism to saprophytism. In these instances saprophytism is an advanced character. Many soilborne fungi increase their ability of survival by having saprophytic stages. The theories create an argument against the "dead-end-theory". On the other hand, parasitism is a possible life strategy for a long period. An example is the rust fungi. Their phylogenetic age is considerable, thus indicating a high level of evolutionary potential.

Rate of evolution

The pressure from the environmental factors upon the parasitic habit promote the fractioning of gene pools and makes inbreeding and asexual reproduction advantageous. This leads to rapid divergence of populations, race formation and eventually speciation.

Short life-cycles, short generation time and high fertility will result in high reproductive rates, which permit dramatic changes in population size, and rapid differentiation of populations. The probability of evolution and

speciation is higher in populations which are predominantly homozygous or haploid, thus having all their genes expressed to selection in each generation (Price, 1977).

The evolutionary potential of parasite populations are positively influenced by these factors, and in addition to them, genetic drift and the founder principle will increase the probability of evolution and speciation compared with a normal randomly breeding population of the same size (Price, 1977). **(Genetic drift:** Random fluctuations of gene frequency. **Founder principle:** When a new population is established in isolation its gene pool is not identical with that of the parent population because of sampling error. These differences are often enhanced leading to increased divergence.)

Adaptive radiation

Adaptive radiation is defined as "evolutionary diversification, often over a relatively short period of time, of a group of organisms, presumably following their entry into a new adaptive zone" (Ehrlich *et al.,* 1974). The extreme adaption to special, discontinuous environments is one of the main characteristiscs of parasites. This type of evolution is favoured by two features which are developed among the parasitic fungi: The high spore-production, and the dispersal of units which are able to reproduce themselves vegetatively (Price, 1977). The adaptive radiation is extensive among the plant pathogens, and its degree of development depends on four factors:

1. The diversity of hosts.
2. The size of the host target.
3. The evolutionary time available for colonization of hosts.
4. The selection pressure for co-evolutionary modification.

The three first-mentioned points are self-explaining. The last item is exemplified best by the co-evolution of certain rust fungi and their hosts. An example, suitable for showing the typical features, is the Crown rust of oats *Puccinia coronata Cda. f.sp. avenae.* See figure 5 (Wahl *et al.,* 1984).

Rhamnus is beleived to be the primary host. The fungus radiated from this source to the secundary festucoideae hosts. In Israel a wide range of alternative graminicolous hosts of *P. coronata* exists. As many of the oats species have their center of origin and genetic diver-

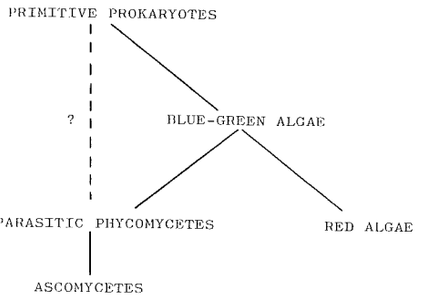

Figure 3. The relationship between the Ascomycetes and their ancestors according to Savile (1955, 1968).

sification in Israel it is beleived that the history of the fungus took place in this region. Another piece of evidence supporting this hypothesis is the existence of the microcyclic *Puccinia mesnieriana Thum.* on *Rhamnus.* The microform is beleived to descend from *P. coronata* by some sort of regressiv evolution. The semi-arid environment favours evolution of short-cycled rusts. So, aecidia of *P. coronata f.sp. avenae* and telia of *P. mesnieriana* are now existing on *Rhamnus,* occasionally even found on the same leaf.

The idea of co-evolution is supported by 3 groups of evidence: (1) Occurrance of primitive rusts on ancient plant forms. (2) Restriction of older rust genera to a narrow host range, while younger rusts inhabit a broader range of hosts that have originated in most recent geologic ages. (3) Evolution of correlated species (certain rusts of similar or different life cycles occurring on related hosts display close resemblance sufficient to indicate descent from a common ancestor) (Anikster & Wahl, 1979).

The evolution of plant pathogens in the agricultural system

Evolution of plant pathogens is affected by changes in the environment, caused by modern crop systems. The ecological basis for natural balance is influenced by a series of factors (Pimentel, 1977):

(1) Monocultures. Removal of the climax-vegetation prior to growth of crop plants is an alteration of the ecosystem. Remaining micro-organisms now utilize the crop plants as hosts. (2) Introduction of crops into new biotic communities. A new crop plant may attract a new parasite species. An example is

the introduction of the potato from Latin-America to USA, where the Colorado-potato-beetle had been living with another, wild Solanum-species and co-evolved with it. (3) Introduction of pest species. Example: Dutch Elm Disease in the USA was the result of the introduction of the pathogen and its vector. (4) Movement of crops to different climatic regions. Some plants escape severe attack by the ability of existing in a climatic region where the pathogens have difficulty surviving. (5) Crop systems. Crop breeding, continuous culture of annuals, and planting time may influence the host-pathogen balance. (6) Alteration of soil contents of nutrients will influence the parasite population. (7) Pesticides may alter the crop physiology. 2,4-D has been shown to increase the susceptibility of maize plants to *Ustilago maydis* and *Drechslera maydis*.

The dynamics of plant diseases

Disease in natural plant communities seldom reaches epidemic proportions. This is because a dynamic equilibrium exists between the host and its parasites. The co-evolution leads to lower aggressivity of the pathogen, and the host does not become so resistant that the parasite if destroyed.

Of importance in the agricultural system is the breeder's effort to stabilize disease by introducing resistance factors from gene centers of cultivated crop-plants. This often fails as a long-term control of endemic diseases. Continued resistance breeding takes place by assembling land-race-populations and by selecting pure lines from them. This leads to loss of genetic material (a sort of genetic erosion). Worse is the case where modern sorts of cereals replace populations in centers of diversity, which often means losses of land-race populations. These types could be maintained in genebanks: Collections of races expecially from centers of origin (Shattock, 1977).

Recent studies of populations of powdery mildew in barley mixtures in Denmark have shown that the virulence genes, corresponding to the resistance genes of the host, were favoured, and their frequency increased, due to selection for virulence. — In the wintertime on a susceptible winterbarley variety a selection pressure in the opposite direction is observed, the "unnecessary" virulence genes now show decreased frequency. This mechanism is less clear, and it is supposed to be influenced by a number of other fitness-affecting factors (lower fitness of complex genotypes?) (Munk, 1984).

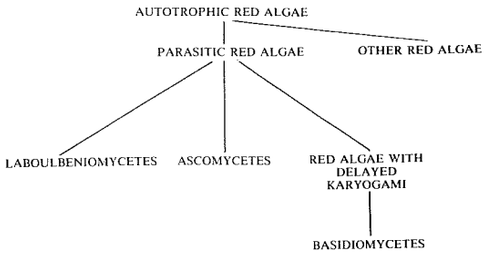

Figure 4. Evolutionary scheme based on the theory of Demoulin (1974).

In order to obtain a better understanding of the ecological aspects of plant resistance, Robinson (1976, 1980) has introduced some new concepts. He regards the pathosystem as a unit, a sub-system of the entire ecosystem, by describing the behavior of the host population and the pathogen population as one system. Any level in this system will be defined in terms of both host and parasite. The pathosystem is divided in two sub-systems, the vertical and the horisontal. The vertical subsystem (corresponds to the race-specific resistance mechanisms) is able to control only the allo-infection (the exodemic). Auto-infection (the esodemic) will be controlled by the horisontal sub-system only. (Some pathosystems lack the vertical system, and it seems that the horisontal system is able to control the exodemic too.) Robinson states that the horisontal system represents the original and fundamental control of the pathosystem, while the vertical system is relatively new in evolutionary terms. It occurs only in some species of hosts, and against only some of its pathogens. The best possible **agricultural stable strategy** should be based on a simulation of the wild pathosystem, where the resistance in the host is neither too low, nor too high (because an unnecessary survival value will decline under negative selection pressure), so the positive and negative selection pressures are equal and in balance, both for horisontal resistance in the host, and for horisontal parasitic ability in the parasite (Robinson, 1981).

Conclusions

If we look at the fitness, or evolutionary potential, of the parasitic form of life, we can state that:

(1) even though parasites are not complex organisms themselves **their reaction to environmental changes is quite complex.**

(2) **Their age as species or genera is in certain cases considerable.**

(3) Many groups have evolved saprophytism to improve their ability of surviving under certain conditions, thus proving that **parasitism is not necessarily a blind alley in phylogeny.**

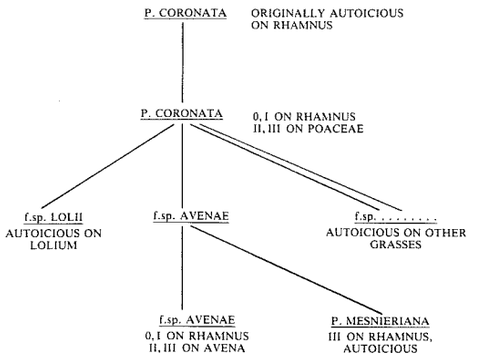

Figure 5. Coevolution of *P. coronata* and its hosts according to Wahl *et al.* (1984).

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Summary; see page 160.

Minor and nonparasitic plant pathogens

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Introduction

It is very difficult to devide all pathogens into clearly defined groups. Some pathogens are very aggressive and do great damage on crop plants every year. The diseases they produce therefore get much attention. If these diseases are economically important, much money will be put into their research and control efforts. The diseases will be recognized as major diseases and the pathogens as major pathogens. On the other hand: If a pathogen is not thought to cause considerable damage, or does so only under unusual environmental conditions, the we would call it a minor pathogen.

This is not at all clear definition, and therefore some scientists have tried to make clear definitions based on histopathological activity of the pathogens.

Salt (1979) has defined a minor pathogen as "saprophytes or parasites damaging only meristematic and cortical cells and surviving in soil as saprophyte, as resting spores or as sclerotia". This definition includes the socalled exopathogens or nonparasitic plant pathogens which damage plants without entering their tissues. Probably their pathogenic activity depends on production of phytotoxic substances in most cases.

The second group consists of weak parasites that only attack cortex or meristematic tissues.

In the third group are obligate parasites of the outermost cell layers of the root.

Predisposing factors

Soil condition is very important for most minor pathogens. Water in abundance is necessary for swimming of zoospores of Phycomycetes and the holocarpic zoosporic fungi. Temperature is the factor deciding the growthrate of the microorganism and the respiration rate, and the factors together control the amount of free oxygen in the soil. Low oxygen concentration can weaken the roots unless pathogens like Pythium are affected, and create more diseases. As many minor pathogens are primary saprophytes fresh organic material in soil will speed up their growth and increase their inoculum potential. The indirect effect of all these factors through the influence on other organisms can often be more important.

Foliar damages like frequent cutting, heavy grazing or diseases can weaken the root system and make the plants more vulnerable to root pathogens.

Estimation of damage

Most root diseases show very indifferent above ground symptoms and this applies especially to the minor pathogens. In case of parasitic fungi, infected root segments can be counted, but this is very time consuming. In the case of exopathogens the search for the pathogen is much more complicated. Wolzt (1978) has suggested ten steps in exopathogenic research.

1. Make sure that the disease is not caused by parasitic plant pathogen och physiogenic causes.
2. Name, discribe and list known microbial associates of the disease.
3. Search for a potential toxin in the litterature.
4. Test potential compounds for symptom production and refer to litterature for potential toxins and listings of microorganisms that produce them.
5. Test microorganisms from the disease site and vicinity in pure culture, for toxin production. Bioassey.
6. Obtain pure cultures of potential exopathogens from additional sources, culture and test them for toxin production.
7. Employ gnotobiotic and in vivo infestation and inoculation procedures.
8. From the above information reconstruct the potential disease biological situation, and produce the disease in vivo.
9. Identify the most favorable anvironmental conditions for the disease.
10. Develop management recommendations.

The above procedure applies to those pathogens that produce toxins.

Examples of minor pathogens

Because of difficult diagnosis and the fact that minor pathogens are often found with major pathogens in mixed infections, rather few such diseases are well known.

Bacteria

Bacteria are frequently suspected for causing minor diseases. Deleterious rhizobacteria (Suslow & Schroth, 1982) are now thought to be common in most soils. They have been found in many genera, for example *Enterobacter, Klebsiella, Citrobacter, Flavobacterium, Achromobachter, Arthrobacter* and *Pseudomonas*. Probably their deleterious effect depends on phytotoxin production. Suslow and Schroth (1982) found that about 25% of rhizobacteria on sugarbeet in California were detrimental while only 2% were growth promoting. They weighed the tops of sugarbeet plants grown in field soil and found that 14 isolates reduced the growth from 21.6% to 47.2%.

It is possible that bacteria which are not rhizobacteria are toxin producers too. It is almost certain that *Bacillus cereus* produces toxin which causes frenching of tobacco under certain environmental conditions.

Nonparasitic fungi

There are several examples of fungi that produce diseases without parasiting the plants. Several fungi which normally are seed borne or saprophytic in soil can produce toxins which cause disease in plants. Many aspergilli produce toxins, for example *Aspergillus wentii,* which causes yellow strapleaf of chrysanthemum and *A. flavus* and *A. tamarii* which have been shown to produce toxins that cause chlorotic seedlings of tobacco. *Alternaria alternata (A. tenuis)* causes similar symptoms on citrus seedlings and other plants. Known parasites like *Phytophtora cryptogea* which causes crown rot on tomato, apparently causes nonparasitic stunting of tobacco, and most isolates of *P. megasperma* seem to produce the same toxin(s).

Slightly parasitic fungi

As said before, fungi that only parasitize cortical or meristematic tissue are defined as minor pathogens. Most of these fungi belong to genera which are members of the normal soil microflora. The most common are *Fusarium, Pythium, Phytophthora,* sterile fungi and *Cylindrocarpon*. Many fungi in this group produce phytotoxins and can therefore also act as exopathogens. Others seem to be able to invade cortical tissue without producing any symptoms. Thus *Pythium* for example has been isolated from 68% of apparently healthy root segments of barley.

Although many of the species of the named genera are minor pathogens, this does not at all apply for all of them. *Fusarium oxysporum* is for example a major pathogen on many plant species and very destructive species of *Phythium* and *Phytophthora*are well known. But it has been shown that even with major pathogens there can be considerable infection (up to 20% of the roots) unless the growth of the plants is affected (Salt, 1979). This could at least partly be explained by the fact that cortical cells are often very short lived, and the fungi are mostly colonizing tissue that no longer is physiologically active. Thus Deacon and Henry (1978) found that 50% of the root cortex of healthy wheat plants in the top 6 cm of seminal roots were dead after 5 weeks.

Histopathological and physiological studies have revealed that many pathogens do not affect plant growth as long as they are restricted to cortical tissue and assimilates are translocated to the roots. If these pathogens do affect plant growth it has in some cases been shown that they are able to produce toxins in vitro and might also do so in the plant. Fitt and Hornby (1978) examined roots of wheat which had been grown in sand infested with different pathogens (see fig. 1). The pathogens were *Aureobasidium bolleyi, Cochliobolus sativus, Fusarium culmorum, Gaeumannomyces graminis, Phialophora radicicola, Pythium scleroteichum* and *Wojnowicia graminis.* The last three fungi did not penetrate the stele in 5 weeks (see fig. 1) nor did they affect shoot growth. *G. graminis* and *A. bolleyi* did not affect shoot growth until they disrupted the stele, while *C. sativus* and *F. culmorum* affected shoot growth before disrupting root steles, possibly by toxin production.

The holocarpic zoosporic fungi

These fungi are common on roots of cereals and many other plants. Some of them have a vide host range. In England the most common genera and species are *Rhizophydium graminis, Olpidium* spp., *Lignieria* spp., *Po-*

lymyxa graminis and *Lagenocystis (Lagena) radicicola* (Salt, 1979). Most of these fungi are restricted to superficial rhizodermal cells and root hairs and cause no apparent disease. *Lagenocystic* is however suspected for causing stunted roots of cereals. *Olpidium brassicae* has also been found to reduce uptake of iron. *Polymyxa betae* is suspected for causing root malformation on beets and is probably a virus vector (Beet Necrotic Yellow Vein Virus). *P. graminis* is a virus vector and so is *Olpidium brassicae.*

Conclusion

The minor pathogens are a very heterogenous group. The separation between major and minor pathogens is also somewhat unclear but it is useful to have Salts (1979) definition as a guideline. As many of the major diseases are now under fairly good control, many scientists are becoming more interested in the videspread and complex minor diseases. The difficulties in the research on minor pathogens are very great and probably it is necessary that specialists in many fields in plant and soil science work together to solve these complex problems. If we shall be able to reduce crop losses caused by minor pathogens, we will probably be able to do so by altering the cultural methods rather than by use of pesticides. More resistant plant varieties will undoubtly also contribute much to the solution of many of these problems.

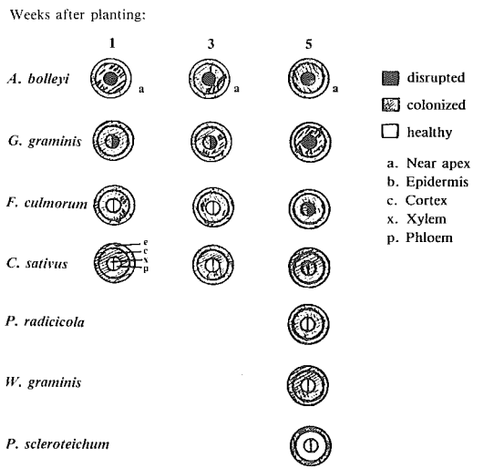

Fig. 1. The extent of invasions and tissue disruption of seminal wheat roots grown in sand infested by root-infecting fungi. (After Fitt & Hornby, 1978).

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Continued from page 157.

DE NEERGAARD, E. 1986. Origin and evolution of Fungal Plant Pathogens. *Växtskyddsnotiser 50:* 6, 152—157.

Fylogenetiske studier af de plantepatogene svampe baseres oftest på antagelsen af at parasitisme er en avanceret egenskab sammenlignet med saprophytisme. Dette medfører at de højest udviklede plantepatogener kommer til at fremstå som blindveje i udviklingen. Der er flere teorier for oprindelsen af de grupper af svampe hvori plantepatogener optrædet. Phycomyceternes forfædre kan være forskellige prokaryote grupper. Ascomyceter og Basidiomyceter afledes af Protozoer, Phycomyceter, Rødalger eller Chytridiomyceter, afhængig af, hvilken teori man anvender.

De plantepatogene svampe karakteriseres ved deres høje geologiske alder, deres store udviklingshastighed under særlige forhold, og deres betydelige evne til at udspalte sig til mange former når nye livsmuligheder optræder, hvilket alt sammen peger på et højt evolutionært potentiale. I visse tilfælde synes parasitiske former at være ophav til saprofyter. Dette som et argument imod blind-gyde-teorien.

Evolutionen i kortere tidsrum iagttages i jordbruget, hvor forskellige dyrkningsforanstaltninger påvirker en række populationsdynamiske sammenhænge i vært-parasit systemet. Opfattelse af patosystemet som en helhed bør foretrækkes ved planlægning af fremtidige strategier for sygdomsbekæmpelse.

Patogen-undertrykkjande jord

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SUNDHEIM, L. 1986. Patogen-undertrykkande jord. *Växtskyddsnotiser* 50: 6, 161—164.

I patogen-undertrykkjande jord er det biologiske faktorar som er forklaringa på den auka bufferverknaden mot plantepatogen. Det er eksempel på at langvarig planteproduksjon har bygd opp ein populasjon av antagonistar som held sjukdomsframkallande organismar i sjakk. Monokultur gjennom nokre år kan gje tilbakegang av rotdrepar i korn. Antagonistiske bakteriar synes å vere den mest sannsynlege forklaringar på rotdrepartilbakegang. Det at patogen-undertrykking kan induserast eksperimentelt ved intens dyrking, opnar nye vyer for å utnytte dette fenomenet. Mest nærliggjande er det å isolere dei biologiske faktorane som ligg bak og så bruke dei til frøbeising eller dypping av plantematriale i ein biologisk bekjemping.

I mange kulturar er plantevernet sterkt fokusert på overjordiske plantedeler. Helsetilstanden til rotsystemet på plantene er ikkje så lett synbar. Cook (1982) hevdar at skader på planterøtene er viktigaste barrieren for auka avling i mange kulturar i dag. Betre rothelse vil truleg gje både større og meir stabile avlingar.

Patogen-undertrykkjande jord (Suppressive soils på engelsk) var definert av Baker og Cook (1974) som jordarter der sjukdom blir undertrykt sjølv om patogenet blir tilført ein mottakeleg vert i eit gunstig miljø.

Walker og Snyder (1934) fann at *Fusarium*-visnesjuke på ert var vanleg i lette jordarter sør i delstaten Wisconsin i USA. Etter at jorda vart nedsmitta med *Fusarium oxysporium f.sp. pisi,* held soppen seg i jorda i lang tid. På tyngre jordarter i sentrale og nordlege deler av Wisconsin var det ikkje tilsvarande problem med visnesjuke sjølv etter intensiv ertedyrking. Walker og Snyder blanda ein del smitta jord med ni deler frisk jord av dei to jordtypene. Dei sådde erter i jorda i veksthusforsøk og noterte angrep i båe jordartene. Etter dyrking av erter i dei same pottene to gonger til var det klare skilnader i visnesjuke på erter dyrka i dei to jordartene.

Walker og Snyder konkluderte at dei to jordartene hadde ulik buffer-evne mot smitte av visnesjukesoppen.

Menzies (1959) var den første som nytta uttrykket "Soil suppressiveness". Han kartla utbreiinga av flatskurv *(Streptomyces scabies)* i delstaten Washington i USA. Menzies fann ingen sammanheng mellom kjemisk og mekanisk samansetning av jorda og utbreiinga av flatskurv. Storparten av poteten i delstaten var dyrka på lett jord med lågt innhald av organisk materiale og nøytral til svak alkalisk reaksjon. Menzies blanda flatskurv-undertrykkjande jord i ulike blandingstilhøve med jord som disponerte for flatskurv og fann at evna til å undertrykkje flatskurv kunne overførast fra ei jordart til ei anna. Damping av jorda øydele evna til å undertrykkje flatskurv og Menzies konkluderte fra det at ein biologisk faktor var årsaken til patogen-undertrykkjing.

Tabell 1. Visnesjuke i tredje kultur av erter dyrka i ei blanding av ni deler frisk med ein del smitta jord — *Wilts in 3rd pea culture grown in a 1:9 mixture of infected and uninfected soil* (Walker & Snyder, 1934)

	Procent visnesjuke — *Per cent wilts*
Lett jord fra sør i Wisconsin — *Light soil from southern Wisconsin*	61%
Leirjord fra nord i Wisconsin — *Clay soil from the north of Wisconsin*	8%

Rotdepartilbakegang

Monokultur induserer patogen-undertrykkjing i mange kulturar. Det er to ulike mønster for sjukdomsutvikling ved dyrking av same kultur år etter år. Sjukdomsangrepa kan auke og stabilisere seg på eit visst nivå. Men det fins og døme på at angrepa minkar etter nokre år med monokultur. Rotdrepar *(Gaeumannomyces graminis)* på korn er det best kjende døme på det siste mønsteret. Dei første 4—5 åra med einsidig korndyrking er det vanleg at rotdrepar-åtaka aukar, men så kan angrepa minke i intensitet slik at avlingsnivåer stabili-

Tabell 2. Rotdreparangrep og byggavling etter ulike forkulturar — *Take-all disease and barley yields after different previous crops* (Hansen, 1966)

Ledd *Treatment*	Forkultur — *Previous crop*					1956 Korn t/ha *Cereals t/ha*	1956 Angrep *Disease index*	1957 (Bygg) — *(Barley)* Korn t/ha *Cereals t/ha*	1957 (Bygg) — *(Barley)* Angrep *Disease index*
1	B	B	B	O/H	H	30,1	0	32,6	41
2	B	B	B	O/H	B/K	26,7	78	25,2	81
3	B	B	B	B/K	B/K	12,8	100	30,4	58

B = bygg — *barley* H = havre — *oats* K = kveite — *wheat* O = oljevekster — *rape/turnip rape*

Tabell 3. Spesifisiteten i patogenundertrykkjande jord i Dijon — *Specificity in suppressive soils in Dijon* (Alabouvette et al., 1980)

Verknad mot — *Effect against*	Ingen verknad mot — *No effect*
Fusarium oxysporum f. sp. melonis	*Fusarium solani*
Fusarium oxysporum f. sp. lycopersici	*Pythium spp.*
Fusarium oxysporum f. sp. dianthi	*Phytophthora spp.*
Fusarium oxysporum f. sp. raphani	*Verticillium dahliae*
Fusarium oxysporum f. sp. cucumerinum	*Sclerotinia sclerotiorum*
Fusarium oxysporum f. sp. lini	*Pyrenochaeta lycopersici*
Fusarium oxysporum f. sp. cyclaminis	*Phomopsis sclerotioides*

serar seg på eit nivå som ligg noko over avlinga i åra med sterkast angrep. Denne sokalla "rotdrepartilbakegangen" vart først registret av Zogg (1951) i Sveits. Gerlagh (1968) observerte at i dei nydyrka polderane i Nederland kom det raskt sterke rotdreparangrep på kveite. Men etter ei tid med monokultur av kveite vart det rotdrepar-tilbakegang slik at avlingane auka noko.

I feltforsøk i femtiåra fann Hansen klare indikasjonar på at ein rotdrepartilbakegang var under oppbygging i Norge (Hansen, 1966). Resultata fra ein fleiråreig forsøksserie med ulike forkulturar for bygg samanlikna med monokultur av bygg eller kveite er stilt saman i tabell 2.

Forsøksledd 3 i denne forsøksserien hadde i 1957 bygg etter fem år med bygg eller kveite. Rotdreparangrepet var mindre og avlingsnivået var betre i 1957 enn i 1956. Det tyder på at ein viss rotdrepartilbaktegang var etablert på desse rutene. Men avlingstala fra 1956 fortel at det kosta mykje å kome dit. Ledd 2 hadde i 1955 ein kultur som ikkje var mottakeleg for rotdrepar og det ga gov avling i 1956, men i 1957 var det sterkast angrep på dette forsøksleddet.

Naturleg patogen-undertrykkjande jord

Franske forskarar i Dijon fann at på visse alluviale jordarter i Rhône-dalføret kan melon dyrkast år etter år utan at det blir skade av visnesjueksoppen *Fusarium oxysporum f. sp. melonis.* Jorda har vore nytta til grønnsakdyrking i mange hundre år. På liknande jordarter berre 5 km unna er visnesjuke eit stort problem i melon og andre agurkvekstar. Men etter damping eller kjemisk jorddesinfeksjon mista jorda si patogen-undertrykkjande evne. Reinokulering med soppar isolerte fra jorda bygde oppatt den undertrykkjande evna i jorda (Alabouvette et. al., 1979). Feltforsøk på naturleg patogen-undertrykkjande jord i Rhônedalføret viste at jorda hadde undertrykkjande verknad mot fleire former av visnesjukesoppen *F. oxysporum.* Tomat vart beskytta mot *F. oxysporum f. sp. melonis* og nellik vart ikkje angrepen av *F. oxysporum f. sp. dianthii.* Men det var ingen verknad mot visnesjuke- eller rotråtesoppar i slektene *Pythium* eller *Phytophthora.* Såleis er den undertrykkjande evna til jorda verksam berre mot ei spesiell gruppe av jordbuande plantepatogen (tabell 3).

Spesifisiteten i den patogen-undertrykkjande evna til dei alluviale jordartene i Dijon gjer det vanskeleg å tenkja seg nokon praktisk utnytting av dette fenomenet. Forsøk med blanding av ulike jordarter har vist at den patogen-undertrykkjande evna kan overførast, men det skal til så store volum at det er lite aktuelt å utnytte det i praksis.

Tilsetting av glukose til patogen-undertrykkjande jord auka respirasjonen 3 gonger så mykje som etter tilsetting av same mengde glukose til normal jord. Alabouvette et al. (1985) hevdar at det indikerar tre gonger større biomasse i den patogen-undertrykkjande jorda enn i normal jord. Mikrobiell aktivitet aukar raskare i undertrykkjande jord. Den store totale biomassen i undertrykkjande jord fører til sterk konkurranse om energi og det kan vere noko av forklaringa på dei spesielle eigenskapane til visse jordarter i Dijon-området. Men det forklarar ikkje spesifiteten i patogenundertrykkjinga.

Nyleg fann Garibaldi (1984) liknande patogen-undertrykkjande jord i Nord-Italia. Visnesjukesoppen *Fusarium oxysporum f. sp. dianthi* vart sterkt hemma på visse jordarter og nellik kunne dyrkast utan visnesjukeproblem. Jorddesinfeksjon øydela den undertrykkjande evna til jorda.

Innblanding av relativt små mengder patogenundertrykkjande jord auka såleis buffarevna mot visnesjuesoppen i smitta jord (tabell 4).

Indusert patogen-undertrykkjing

Patogenet må vere tilstade for at jord skal bli undertrykkjande. Kan gjentatte plantingar i smitta jord indusere evna til å hemme eit visst patogen? Henis et al. (1978) sammanlikna fire jordarter fra Colorado i smitteforsøk med *Rhizoctonia solani* og fann at to vart temmeleg undertrykkjande, ei jord vart middels undertrykkjande og ei jord vart ikkje endra.

Vi har undersøkt om norske jordarter kan induserast til å bli undertrykkjande for *Rhizoctonia solani* (Davik & Sundheim, 1984). Etter smitting med to ulike mengder av *R. solani,* anastomose gruppe AG 2, sådde vi blomkål i seks ulike jordarter i karforsøk. Spiringa vart bestemt etter 10 dagar. Etter fjerning av spirte og uspirte frø, sådde vi nytt frø i same jorda og talde opp spiringa 10 dagar seinare. Dette vart gjentatt fem gonger slik at det vart tilsaman seks blåmkålsåingar i jordprøvene.

Tabell 4. *Fusarium*-visnesjuke i nellik etter innblanding av ulike volum patogen-undertrykkjande jord til normal jord i smitteforsøk med *Fusarium oxysporum f. sp. dianthi* — Fusarium-*wilts in carnations after admixture of different volume proportions of suppressive soil in normal soil. Infection experiment with* Fusarium oxysporum f.sp. dianthi (Garibaldi, 1984)

Volym % tilsett — *Volume % added*	Visnesjukeindeks — *Disease index* (0—100) Planting — *Planting* 1	2	3
0	100	98	97
2	73	63	38
10	64	58	34

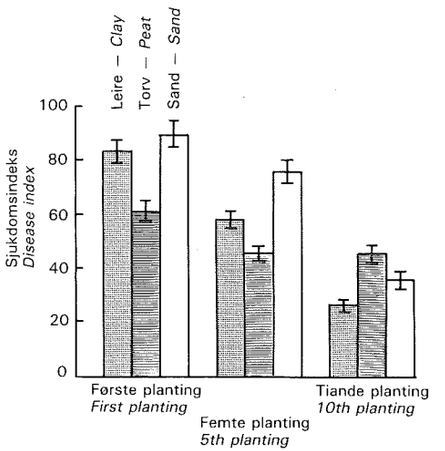

Fig. 1. Sjukdomsindeks ved første, femte og tiande planting i leire, torv og sandjord. — *Disease index at the first, 5th and 10th planting in clay, peat and sandy soil.*

Ved første såing var det ingen oppspiring i smitta potter. Ved sjette såing var det ein betydeleg grad av undertrykking av patogenet i jord 2, 3, og 4, medan det var liten eller ingen verknad av monokultur i dei tre andre jordartene (tabell 5).

I tilsvarande forsøk planta vi to veker gamle blomkålplanter i smitta jord og noterte sjukdomsutviklinga dagleg. Etter fjorten dagar vart alle planterestar fjerna og nye blomkålplanter planta i jordprøvene. Dette gjentok vi for å få i alt 10 plantingar i same jorda. Resultata fra ei sandjord, ei leirjord og ei myrjord er framstilt i fig 1.

Sjukdomsindeksen avtok sterkt i sandjorda og i leirjorda fra første til femte og tiande planting. Monokultur hadde mindre verknad på sjukdomsindeksen i myrjorda. Sjukdomsutvikling etter planting er framstilt i fig 2.

Tabell 5. Prosent drepte spirer ved første og sjette såing av blomkål i seks ulike jordarter — *Cauliflower seedlings killed in 1st and 6th planting in 6 different soil types* (Davik & Sundheim, 1984)

Jord — *Soil*	Inokulum % *Inoculum %*	Første såing — *First sowing*			Sjette såing — *Sixth sowing*		
		0	0,1	1,0	0	0,1	1,0
1		29	100	100	45	100	100
2		73	100	100	33	24	42
3		30	100	100	47	64	33
4		70	100	100	90	25	35
5		51	100	100	75	99	100
6		37	100	100	31	68	93

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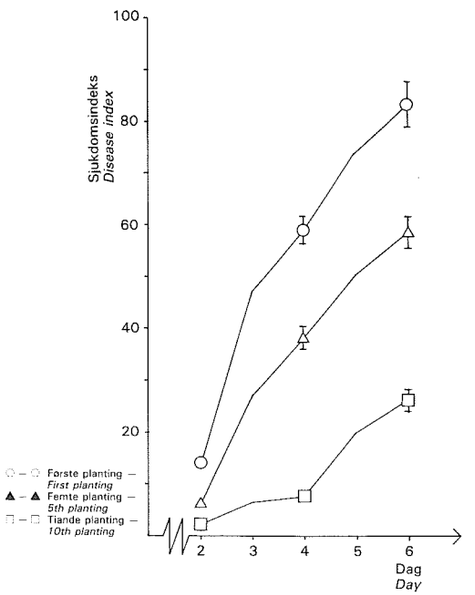

Fig. 2. Gjennomsnittleg sjukdomsindeks i ei leirjord smitta med *Rhizoctonia solani.* I jorda vart det plante blomkål 10 gonger. — *Average disease index in a clay soil infected with* Rhizoctonia solani, *in which cauliflower had been grown 10 times.*

Summary; see page 170.

Organism interactions in disease

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MAGNUSSON, C. 1986. Organism interactions in disease. *Växtskyddsnotiser 50:* 6, 165—167.

Both soil arthropods and plant parasitic nematodes may promote plant diseases. Due to their deep interference with root metabolism and root growth regulation, plant parasitic nematodes show the highest interactive capacity. As environmental factors also affect interactions, high-order interaction experiments are needed to resolve complex disease conditions.

Interactions in plant disease

The lack of an early recognition of multiple-pathogen etiology relates to the dominance of the doctrine of specific etiology, in which disease is related to a specific cause (Powell, 1979). Reality is more complex and demands a more dynamic disease concept (Wallace, 1978; Powell, 1979). This paper considers mainly the implications of soil animals in promotion of plant disease. Although, the term "interaction" contains a multitude of ecological relations, it is often practical, with regard to plant disease, to use the term in a statistical sense, indicating a condition where the combined effect of studied factors are not additive (Wallace, 1983). If promotion of disease is considered, this implies synergistic interactions.

Root conditions

The root system is the major contact surface between the plant and its environment, and exhibits a fundamental diversity in structure and function (Scott, 1978; Street et al., 1978; Drew, 1979). In soil, physical conditions, structure, metabolism, and growth regulation of roots are functionally linked in an interactive framework (Fig. 1), within which a multitude of responses may occur due to soil animal activity. Plant roots are well provided with defense mechanisms (Horsfall & Cowling, 1980). The optimal integration of defense mechanisms may, however, depend upon a proper function of this interactive framework.

Herbivory and disease

Below ground herbivory is a neglected field of study. This especially pertains to micro- and macroarthropods (diplopods and insect larvae). Plant parasitic nematodes have been studied more intensively. In vegetables, damaging secondary invasions of microorganisms may occur due to damage by root feeding larvae of sciaridae, cabbage root flies, and carrot fly (Nilsson et al., 1985). In agricultural crops, symphyllids, diplopods, and collembolans have been recognized as a "soilpest" complex with a possible contribution of fungi like *Aphanomyces, Fusarium, Phoma* and *Pythium* to sugar beet mortallity (Brown, 1985). Also larvae of the clover root curculio, *Sitona* sp. and fungi have been implicated in decline of various legumes and in bacterial wilt of alfalfa (Beute & Benson, 1979). Evidence is accumulating also for an etiological role for microarthropods in plant disease (Beute & Benson, 1979).

While arthropods mainly cause mechanical damage to plants, plant parasitic nematodes induce more far-reaching alternation (Magnusson & Lagerlöf, 1983). Plant parasitic nematodes have long been recognized as interacting synergistically with microorganisms in disease complexes (Powell, 1971; Taylor, 1979). Such interactions have also been reported on crops of nordic interest: eg. *Pratylenchus* and *Meloidogyne* with several fungi and some bacteria on pea, alfalfa, red clover, potato, oil seed rape and some horticultural crops, *Heterodera schachtii* with *Verticillium* on sugar beet and oil seed rape, *H. avenuae* with *Rhizoctonia* on wheat, and *Globodera rostochiensis* with Verticillium on potatoes (Harrison, 1971; Meagher & Chambers, 1971, Oyekan & Mitchell, 1971; McKinley & Talboys, 1979; Müller, 1980; Welty et al., 1980; Bookbinder et al., 1982; Mauza & Webster, 1982; Insunza & Eriksson, 1983; Rowe et al, 1985). Observed

synergistic effects include, increased colonization by the pathogen, earlier symptom expression, increased disease insidence, increased disease severity, decreased growth of plants, decreased yield, and resistance breaking. Synergistic effects have also been noted when nematodes attack parts of the root systems isolated from the fungus, indicating long-distance effects (Faulkner et al., 1970).

Interactive capacity of soil animals

Clearly, soil animals have different capacities to promote disease, and their interactive potential may be related to their ability to interfere with essential components of the interactive framework (Fig. 1). Although much information is missing for micro- and macroarthropods, their interactive capacity seems inferior compared to plant parasitic nematodes. The deep interference of nematodes with vital functions in root metabolism and growth regulation (Fig. 1) is manifested in far-reaching structural and physiological alterations in roots of cultivated plants (Endo, 1975; Jones, 1981; Glazer et al., 1985). Most certainly, the reported higher involvement of plant parasitic nematodes in disease complexes is related to these fundamental aspects rather than to the suitability of nematodes for experimentation and the devotion of nematologists!

The effects of interactions depend much on factors of the environment. Heat stress is important in the *Pratylenchus-Verticillium dahliae* induced "early-dying" syndrome of potato (Rowe et al., 1985) and the concentration and form of inorganic nutrients affected a *Fusarium*-wilt complex in muskmelon (Spiegel & Netzer, 1984). There are reasons to suspect a role also for mycorrhizae in disease complexes, although this has hardly been studied. "Cross-protection" between pathogens (McIntyre, 1980) could be one explanation for observed reductions in synergistic effects in high-order interactions (Wallace, 1983).

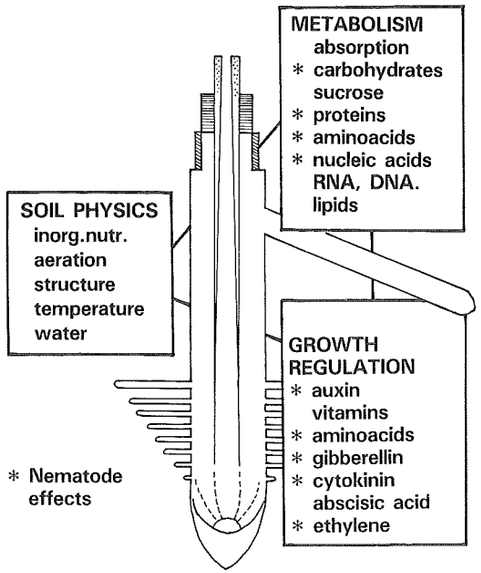

Fig. 1. Interactive framework in soil.

Complexity and research

Problems encountered in studying disease complexes are overwhelming. The complex reality still demands high-order interaction experiments. Factorial designs and response surface techniques allow testing of complex hypotheses embracing also different levels of causation. This implies more effort per experiment, but fewer experiments are needed to reach a given level of insight (Hilborn & Stearns, 1982). This is, however, contradicted by common strategies of grant application and approval which imply small scale efforts to precede more elaborated.

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MAGNUSSON, C. 1986. Organisminteraktioner i växtsjukdomar. *Växtskyddsnotiser 50:* 6, 165—167.

Såväl markartropoder som växtparasitära nematoder har förmågan att förstärka växtsjukdomar. Till följd av de växtparasitära nematodernas djupgående störningar i rotmetabolismen och rötternas tillväxtregulation besitter dessa den största interaktiva kapaciteten. Eftersom också omgivningsfaktorer påverkar interaktioner, krävs fler-faktor försök för att klarlägga komplexa sjukdomstillstånd.

Root diffusates and plant parasitic nematodes

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MAGNUSSON, M. L. 1986. Root diffusates and plant parasitic nematodes. *Växtskyddsnotiser 50:* 6, 168—170.

The effects of root diffusates on plant parasitic nematodes are discussed. Factors in root diffusates can stimulate or inhibit the hatching of the juveniles, affect their molting, or attract the nematodes to their hosts.

Root diffusates can affect different stages of plant parasitic nematodes by stimulating or inhibiting the hatching, activating the juveniles to molt, and attracting the nematodes to the host. Although much work have been done several questions still remain to be answered. The nature of different nematode-attractants in diffusates are poorly known as well as the mode of action, and the response of the nematodes (Marrian *et al.,* 1949; Green, 1971; Shepherd & Clarke, 1971; Lee & Atkinson, 1976; Perry & Beane, 1983; Tefft & Bone, 1985). Most of the papers published so far concern the hatching factors of the cyst forming nematodes *(Heteroderidae),* with some notes on root-gall nematodes (*Meloidogyne* spp), and a few other genera.

Diffusates

The hatching factor in root diffusates has been extensively studied for the potato cyst nematodes *(Globodera rostochiensis* and *G. pallida)* and for the soybean cyst nematode *(Heterodera glycines).* The hatching factor for the potato cyst nematode has not, so far, been isolated. The active chemical seems to be rather unstable in soil. It is apparently acidic in nature, stable at pH 1—7, water soluble, non-volatile, containing a lactone group, and one or two hydroxyl groups. It is believed to have an empirical formula of approximately $C_{18}H_{24}O_8$ (Marrian *et al.,* 1949). A hatching agent for the soybean cyst nematode, glycinoeclepin A, which is a diabasic acid with a molecular weight of 446, has recently been isolated from the roots of *Phaseolus vulgaris* (Masamune *et al.,* 1982). The hatching agent in the soybean roots remains, however, to be isolated.

Hatching

Most nematode species hatch without delay once the juvenile has reached a certain stage of development in suitable environmental conditions (Lee & Atkinson, 1976). Several cyst nematode species, however, need a stimulus released into soil by host plants before hatching is activated. Some *Heterodera*-species, such as *H. glycines* and *H. goettingiana* hatch in root diffusates from host plants, while species like *H. avenae* and *H. schachtii* are able to hatch both in host-root diffusates and in water (Green, 1971; Shepherd & Clarke, 1971; William & Beane, 1979; Steele *et al.,* 1982; Perry & Beane, 1983; Tefft & Bone, 1985).

In the genus *Globodera* most species have a narrow host-range, and their hatching usually depends on exposure to host-root diffusate. The most important species in this genus are the potato cyst nematodes with only very few hosts in the family *Solanaceae.* Only about 2% of the juveniles hatch in water (Sheperd & Clarke, 1971; Lee & Atkinson, 1976) the majority of the juveniles beeing activated only after exposure to root diffusates from potato, tomato, and a few other hosts (Triffitt, 1930, 1934; Sheperd & Clarke, 1971; Lee & Atkinson, 1976; Forrest & Farrer, 1983). The spontaneous hatching of the potato cyst nematodes frequently reported in fallow conditions is assumed to be caused by substances produced by other soil organisms (Ellenby & Smith, 1967). Some nonhosts, such as some grasses and cereals have been observed to stimulate the hatching of the potato cyst nematodes (Triffitt, 1934; Mägi, 1974; Magnusson unpubl.), although the hatching seems to be slower in non-host root diffusates as compared to the potato root diffusate (fig. 1) (Magnusson unpubl.). Indications of synergistic effect of root diffusates on hatching of the potato cyst nematode have also been reported. More juveniles hatched *in vitro* when potato and nonhost root diffusates were present together as compared with potato root diffusate alone (Mägi, 1974).

Nonhosts can also have an inhibitory effect on cyst nematode hatching. The hatching of the potato cyst nematodes is inhibited by root diffusates from mustard and some *Umbelliferae* (Triffitt, 1930; Mägi, 1974; Forrest & Farrer, 1983). Root diffusates of *Tagetes* spp., however, did not inhibit hatching of the potato cyst nematode despite the naturally occurring nematicide in the roots (Omidvar, 1961).

The mode of action of the hatching factor(s) in the root diffusates is not completely understood. There are observations of an increase in oxygen consumption of unhatched potato cyst nematode juveniles within 24 hours after exposure to potato root diffusate, and changes in adenylate energy charge of the cyst contents, which led to altered metabolism within the cysts changing presumably the permeability of egg shells (Atkinson & Ballantyne, 1977a, b). The hatching factors in potato root diffusate also activate the unhatched juveniles to move vigorously, and finally to cut a straight slit across the egg with the stylet (Green, 1971; Sheperd & Clarke, 1971; Lee & Atkinson, 1976).

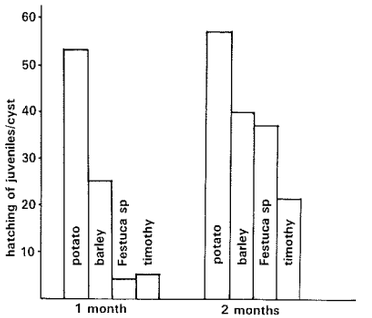

Fig. 1. Total numbers of 2nd stage juveniles of *G. rostochiensis* hatching *in vitro* in root diffusates from potato and some non-hosts after 1 and 2 months respectively. Means of four replicates.

Molting

The effect of root diffusates on molting of plant parasitic nematodes has only been reported of the ectoparasitic genus *Paratylenchus. P. projectus* and *P. dianthus* juveniles survive for long periods in soil without hosts. Very few larvae molt in water, while the majority of them molt in carnation root diffusate (Rhoades & Linford, 1959). Similar behaviour has been reported of *P. nanus* (Fisher, 1966).

Host findning

The movement of plant parasitic nematodes to their hosts frequently involves a random component, but chemoreceptors are probably important at least when the nematode is close to the host roots (Lee & Atkinson, 1976). Gradients of CO_2 concentrations seem often to be a main attractant to some species, eg. *Pratylenchus penetrans* and *Meloidogyne hapla,* but some species, for instance *H. schachtii* and *P. scribneri* fail to show a marked aggregation around roots of nonhosts, and they may be influenced by specific stimuli present near the host roots (Green, 1971; Lee & Atkinson, 1976). The response of the nematodes to the attractants from the roots is greatly influenced by environmental factors such as moisture and aeration, but also by the developmental stage of the nematode (Green, 1971).

Synthesis

The stimulating factors in root diffusates play an important role for many plant parasitic nematodes, especially for the species/genera highly specialized to live as plant parasites. The majority of the studies consider cyst nematodes, not only because of the noted efficiency of root diffusates, but also because of the economic significance of the cyst nematodes. Isolating of at hatching factor for any economically important cyst nematode species may prove of great value if this were to result in the synthesis of a cheap analogue chemical useful for control.

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MAGNUSSON, M. L. 1986. Rotdiffusat och växtnematoder. *Växtskyddsnotiser 50:* 6, 168—170.

Rotdiffusatens betydelse för växtnematoder diskuteras. Faktorer i rotdiffusat kan stimulera eller inhibera kläckningen av juveniler, påverka deras hudömsning, eller attrahera nematoderna till värdväxten.

Continued from page 164.

SUNDHEIM, L. 1986. Suppressive soils. *Växtskyddsnotiser* 50: 6, 161—164.

Research on disease suppresive soils are reviewed. Nine different Norwegian soils were inoculated with *Rhizoctonia solani* anastomosis group AG 2. Cauliflower seeds were sown with ten days intervals in six different soils. After six repeated sowings germination percentages were at the same level as in the non-inoculated control in three of six soils tested. In one soil pre-emergence damping-off was reduced from 100% in the first sowing to 33% in the sixth sowing.

In three different soils, two weeks old cauliflower plants were planted ten times with two weeks intervals. Repeated plantings reduced disease severity in all three soils. In a clay soil and in a sandy soil the disease index was reduced more than in a peat soil.

Examensarbeten från Institutionen för växt- och skogsskydd, SLU

SJÖBERG, A. 1986. Studier av insektparasitära nematoder i Mälar/Hjälmar-bygden.
(Handledare: Universitetslektor Martin Burman, professor Bengt Eriksson), *Sveriges lantbruksuniversitet, Institutionen för växt- och skogsskydd; Examensarbeten 1986:*4.

Arbetet utgör ett led i studierna av förutsättningarna för att använda insektsparasitära nematoder i bekämpningen av skadeinsekter i vårt land. Förekomsten av ifrågavarande nematoder i några olika mellansvenska biotoptyper analyserades mot bakgrund av årstid, vegetation, jordart, markfuktighet samt utbredning i markprofilen.

Provområdena var Ekenäs gård (nära Flen) i Södermanland, Lövsta hage norr om Uppsala samt Hosjön i Knutby-trakten (Uppland). Provtagningar gjordes under sommaren 1984, dels under försommaren (maj), dels under sensommaren (slutet av augusti). Nematodförekomst påvisades med s.k. *Galleria*-fällor.

Nematoderna i fråga förekom i samtliga de studerade biotoperna, blandskog, lövskog, löväng, hagmark, betesvall, åkermark och våtmark. Av försommarens 88 jordprover var 44% positiva *för nematodförekomst,* att jämföra med ca 20% av sensommarens 87 prover. Flertalet positiva fynd var från lättare jordarter, inklusive lättlera och mulljord. *Såväl försommar som sensommar* påvisades nematodförekomst ner till ett djup av 80 cm i flera jordprofiler.

Undersökningarna bekräftar de senaste årens erfarenheter att insektparasitära nematoder, bl.a. *Neoaplectana* spp., är ganska allmänt förekommande i svenska jordar och biotyper.

LERENIUS, C. 1986. Några miljöfaktorers inverkan på inträngningsresistens mot potatisbladmögel *(Phytophthora infestans).*
(Handledare: forskningsassistent Barbro Berggren och professor Vilhelm Umaerus), *Sveriges lantbruksuniversitet, Institutionen för växt- och skogsskydd; Examensarbeten 1986:*1.

Ospecifik resistens kan delas upp i olika komponenter beroende på var i utvecklingscykeln svampen hejdas. En av resistenskomponenterna är inträngningsresistensen som påverkar svampens groning på och penetrering av bladytan.

Syftet med examensarbetet var att undersöka hur inträngningsresistensen mot potatisbladmögel *(Phytophthora infestans* (Mont.) de Bary) påverkas av miljöfaktorer som tillgång på växtnäring och dagslängd. Dessutom gjordes en litteraturöversikt som belyser dessa miljöfaktorers inflytande på resistensen mot bladmögel.

Två potatissorter med stor skillnad i inträngningsresistens odlades utomhus sommaren 1984. I växtnäringsförsöket sattes sticklingar i kärl med brist på kalium, kväve eller magnesium samt kontrollkärl. I ett dagslängdsförsök odlades potatis i lådor med normal, lång dagslängd (cirka 18 h) respektive kort dagslängd (10 h).

Svampens groningsbeteende studerades i båda försöken. Blad inokulerades med zoosporer, och andel grodda sporer, andel sporer med appressorium och groddslangslängd noterades. I dagslängdsförsöket undersöktes även infektionsfrekvensen genom sprayinokulering varvid antalet lesioner per kvadratmeter i förhållande till antal deponerade, grobara zoosporer bestämdes.

Vid kort dagslängd ökade infektionsfrekvensen jämfört med lång dag. Svampens groningsarbete tycktes däremot inte påverkas av dagslängden, men denna undersökning var av liten omfattning och resultaten därför osäkra.

Växtnäringsbristerna påverkade kraftigt plantornas utveckling. Plantorna i led med magnesiumbrist utvecklades och åldrades tidigare än andra led och samtidigt fanns en tendens att svampen hellre grodde och bildade appressorier i detta led. Av undersökningen framgår inte huruvida effekter på svampen berodde på magnesiumbristen eller på plantans utvecklingsstadium. Övriga näringsämnen hade inga tydliga effekter på svampens groningsbeteende.

Inträngningsresistensens natur måste undersökas ytterligare. Miljöfaktorernas effekter på andra delar av infektionsförloppet, t ex sporangiernas kläckning och svampens penetrering av bladytan, bör studeras. Resultaten från detta arbete tyder på att inträngningsresistensen påverkar andra faktorer än svampens groning.

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