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The system management approach of biological weed control: Some theoretical considerations and aspects of application

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Abstract. Theoretical considerations behind the system management approach of biological weed control are presented. These include, a part describing and explaining the effects of parasitic fungi on crop – weed competition, a part describing and explaining the epidemic spread of parasitic fungi on weeds, and a part relating crop – weed competition at the population level to epidemics. The theoretical framework developed may also provide a basis for the use of other natural enemies, like insects, for biological weed control following the system management approach. Aspects of application are discussed using data of the interaction between the annual weed *Senecio vulgaris* and the rust fungus *Puccinia lagenophorae*.

Key words: biological weed control, epidemics, natural enemy, plant competition, *Puccinia lagenophorae*, *Senecio vulgaris*

Introduction

The aim of the study presented here is to review theory and application of the system management approach of biological weed control. This approach was recently proposed by Müller-Schärer and Frantzen (1996) and the philosophy of this approach can be understood best by contrasting it with the two other major approaches of biological weed control, the classical and bioherbicide approach.

The classical approach, often also called the inoculative approach, is based on the idea that plants may become weeds when released from the effects of their natural enemies. Such a situation is, for example, created by the invasion of a new area by a plant, which is not accompanied by the natural enemies that attack it in its area of origin. The invasion of parts of Australia by the apomictic plant *Chondrilla juncea* L. (Wells, 1971) may illustrate this. The plant is not a weed in the native range and this might be explained by the presence of natural enemies. A strain of the rust fungus *Puccinia*

chondrillina Bubak & Syd. was collected in Italy, within the native distribution of *C. juncea*, and tested for its pathogenicity to *C. juncea* in Australia (Hasan, 1972). Afterwards, the fungus was introduced into Australia using a few infected plants as the inoculum sources for an epidemic that spread throughout the weed infested area (Cullen et al., 1973). This example illustrates very well the philosophy behind the classical approach: limited material of a natural enemy is introduced and control is effected, or not, by the reproduction and spread of the natural enemy throughout the area where the target weed occurs. This is a relatively cheap approach, once the initial research (agent selection, host specificity testing etc.) has been done. However, despite specificity testing, one drawback of this approach is the risk of attack of non-target plants after the introduction of the biocontrol agent in a new area (Simberloff and Stiling, 1996; Thomas & Willis, 1998).

The bioherbicide approach to weed biocontrol relies on natural enemies present within the native range of the weed and which have the potential to cause sufficient damage to the weed to significantly reduce its negative impact on crop yield. In nature, this potential for damage may not be expressed due, for example, to a persistently low abundance of the natural enemy, or a low abundance at the particular time required to control of the weed in a specific agricultural or horticultural situation. The aim of the bioherbicide approach is to increase the abundance of a natural enemy by culturing it under controlled conditions and, subsequently, applying it in relatively large amounts onto the whole weed population like a herbicide. An example of successful use of the bioherbicide approach is the control of the annual species *Aeschynomene virginica* (L.) B.S.P., which is a weed indigenous to the United States, with the fungus *Colletotrichum gloeosporioides* (Penz.) Sacc. forma specialis *Aeschynomene*, which also is indigenous to the United States (Daniel et al., 1973; Templeton et al., 1984). The fungus is cultured on artificial media in large quantities and subsequently applied onto a whole *A. virginica* population. Whereas the classical approach is based on the innate capacity of natural enemies to reproduce, the bioherbicide approach is based on reproduction of natural enemies under controlled conditions and subsequent spread by man.

Indigenous fungi belonging to the category of facultative parasites have been used until now in the bioherbicide approach. Indigenous fungi have the benefit of minimising the risk of attack of non-target plants and this contrasts with the use of exotic organisms following the classical approach. Facultative parasites have been used because they are relatively easily to culture on artificial media and this contrasts, for example, with biotrophic fungi. However, facultative parasites can be relatively unstable with respect to host specificity (Leonard, 1982). Indeed, the host range of *C. gloeosporioides* was extended

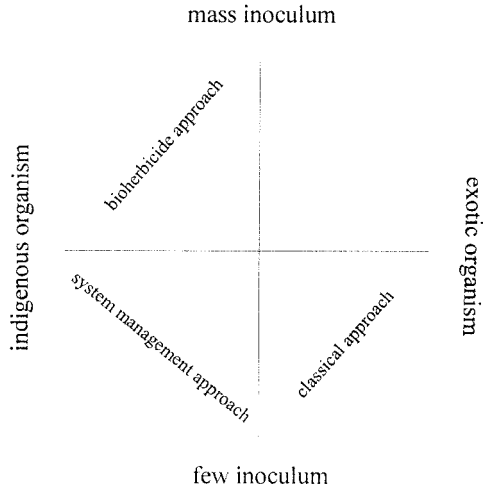

Figure 1. Three approaches to biological weed control contrasted by the amount of inoculum initially used to control a weed population and contrasted by the origin of the natural enemy. Notice that no approach is yet based on the use of mass inoculum of exotic organisms.

after its introduction to the market as a bioherbicide, and now includes some crop species (TeBeest, 1988; Cerkauskas, 1988).

The system management approach was proposed as complementary to the other two approaches to increase the palette of control strategies based on the use of natural enemies. The major differences between the three approaches are (1) the origin of the natural enemy, i.e., exotic versus indigenous, and (2) the amount of natural enemy initially used to control a weed (Figure 1). The system management approach uses indigenous organisms, like the bioherbicide approach, but relies on the innate capacity of the natural enemies to reproduce, which is similar to the classical approach. Following the system management approach enables the use of indigenous biotrophic fungi, which are relatively stable with respect to host specificity (Leonard, 1982), but which are not easily exploited as bioherbicides as they cannot be cultured on artificial media on a large scale presently.

Biodiversity is a major issue in ecology and natural conservation. Agriculture may also contribute to biodiversity by moving away from the clean crop concept, i.e., a crop growing completely free of weeds, towards a concept of tolerance of weeds providing they do not significantly reduce economic yield (Müller-Schärer and Frantzen, 1996). This may rely on the impact of weeds on crop yield being minimised by reducing the competitiveness of weeds without killing them. Such a modern view to weed control is taken into account developing the system management approach, and so the focus is on reduction of the competitiveness of weeds without killing

(Müller-Schärer and Frantzen, 1996; Frantzen and Hatcher, 1997; Frantzen and Müller-Schärer, 1998).

The preceding considerations result in three criteria to choose a biocontrol agent suitable to follow the system management approach:

(1) The agent should be an indigenous, or naturalised, natural enemy;
(2) The agent should be able to reduce the competitiveness of its host without killing;
(3) The agent should have an innate capacity to reproduce and to spread relatively easily.

The rust fungus *Puccinia lagenophorae* Cooke, infecting the annual weed *Senecio vulgaris* L., met these three criteria and we used it to develop the system management approach. Examination of points (2) and (3) makes it obvious that the derivation of a theoretical framework for the system management approach requires consideration of both competition and epidemics, which are caused by the reproduction and spread of pathogens like rust fungi. Hence, our aim here is to derive a theoretical framework from existing theories of both, plant competition and epidemics. Applying the theory, subsequently, to the pathosystem *S. vulgaris – P. lagenophorae* may contribute to validation of the theory.

Theory: can we link epidemics to plant competition at the population level?

Competition

Plant competition is a major issue in ecology, and biocontrol research may profit from the ecological knowledge about competition. Here, we review and extend existing literature to arrive at a method to model plant competition in such a way that it can be linked to epidemics. We shall proceed in three steps starting the analysis of intraspecific plant competition, subsequently interspecific competition, and finally the effects of pathogens on interspecific competition.

Initial analysis of intraspecific plant competition (Kira et al. 1953) used the equation,

$$w = K * N^{-a}, \tag{1}$$

where w is the mean dry weight of plants, K and a are constants, and N is the density. The values of K and a increase as plants grow. If a equals 1, Equation 1 simplifies to,

$$w = K/N \tag{2}$$

and this means the mean dry weight of plants reduces proportionally to the density, i.e., the total dry weight of a population is independent of density. This is the law of constant yield.

Equation 1 does not fit well to data collected at low densities. Therefore, other more complex equations were proposed. Watkinson (1980) proposed the equation,

$$w = w_m * (1 + \alpha * N)^{-b}, \tag{3}$$

where w_m is the maximum dry weight of a plant growing without competitors, α is a competition coefficient and b has a less clear biological function. The parameters w_m and α increase to a maximum as plants grow and assuming b equals 1 we have again the law of constant yield. The parameter α can be envisaged as the ground area needed by a plant to achieve the maximum weight. This area is determined not only by the need to take up resources sufficient for growth, but also by the dilution of inhibitory allelopathic substances produced by neighbours. In this sense, the term α integrates physical, nutritional, and chemical aspects of the plant environment. Since density N is often expressed as the number of plants per unit area ([N] / [L^2]), α is expressed in units of area per plant ([L^2] / [N]). Thus, the parameter α can be considered to be similar to parameter β in the spacing formula developed by De Wit (1960).

The parameter b of Equation 3 often has a value of 1 (Watkinson and Freckleton, 1997). Whether other values than 1 are statistical artefacts or expressing a biological phenomenon is still not clear (A.R. Watkinson, personal communication). Therefore, we adopt here a value 1 for b and write Equation 3 as:

$$w = w_m/(1 + \alpha * N) \tag{4}$$

and this equation describing intraspecific competition can be extended to describe also interspecific competition (Watkinson, 1981),

$$w_{cr} = w_{m,cr}/[1 + \alpha * (N_{cr} + \varepsilon * N_{we})], \tag{5}$$

where ε is the equivalence, or multiplication factor, of space (area) used by species *we*, e.g., a weed, compared to the target species *cr*, e.g., a crop. For example, if the weed requires twice as much area as the crop, ε is equal to 2. Thus, the area needed by a plant of species *we* is equal to $\alpha * \varepsilon$. Whereas the increase of the parameters $w_{m,cr}$ and α with time is known, the fate of ε in time has not been studied. Research directed to the relation between ε and growth of competing plants is clearly relevant to explaining crop losses due to weeds. We hypothesise here that ε increases with time if the growth rate

of the weed is higher than that of the crop, rather remains stable if the growth rates are similar, and decreases if the growth rate of the crop is higher.

The parameters $w_{m,cr}$, α, and ε are independent of N_{cr} and N_{we} and thus independent of total plant density ($N_{cr} + N_{we}$) as well as of the ratio between species (N_{cr} / N_{we}). Thus, we are looking at some intrinsic characteristics of the species *cr* and *we*. The maximum weight of any species is constrained by physiological processes, plant architecture, and the available amount of resources, e.g., a single individual of a vigorous perennial herb will be larger than a single plant of an annual grass and a plant growing on nutrient rich soil will be larger than one growing on nutrient poor soil. Similarly the parameter α will be species-specific and parameter ε depends on the specific characters of a given pair of species (a crop and a weed in the above example) given a specific environment.

In seeking to quantify the parameters of Equation 5 we should be aware of the difference between statistics and biology. Although the parameters are independent of N_{cr} and N_{we}, the estimates will not be precise if a rather small range of densities and proportions of species *cr* and *we* is used. The parameters are estimated using (non-)linear regression and the estimates will be more precise the broader the range of densities and proportions is. Thus, the optimum experimental design to determine the severity of plant competition should include a broad range of densities and proportions (Firbank and Watkinson, 1990; Cousens, 1991; Watkinson and Freckleton, 1997). Despite the advantages of this design, the so-called response surface analysis, it is not frequently used because of the relatively high input of labour. Here, we adapt Equation 5 with respect to both, time and pathogenic infection of species *we* and this results in,

$$w_{cr,t} = w_{m,cr,t}/[1 + \alpha_t * (N_{cr} + \varepsilon'_t * N_{we})] \tag{6}$$

and

$$\varepsilon'_t = \varepsilon_t * r_t, \tag{7}$$

where *t* is time of observation, ε' is the competition equivalence for infected plants of species *we*, and *r* expresses the impact of the pathogen on interspecific competition as expressed by ε. A likely range of values of *r* is zero to one. If *r* equals zero, the pathogen kills the plant and interspecific competition is eliminated. If *r* equals 1, the pathogen has no impact on interspecific competition. Values greater than 1 mean stimulation of interspecific competition and such values are not of interest with respect to biological weed control. No estimates of *r* are published yet. However, intuitively the negative impact of a pathogen on ε is expected to increase (and *r* expected to decrease)

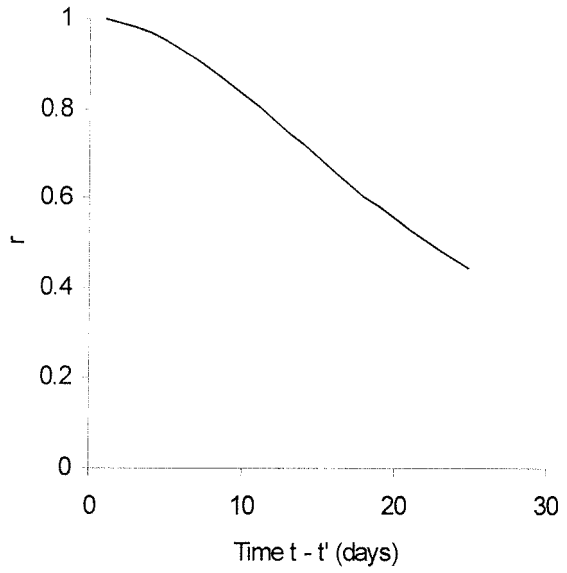

Figure 2. A hypothetical function describing the relation between the multiplication factor r of the competition equivalence ε (see text Equations 6 and 7) and the period that a weed is infected by a parasitic fungus. The parameters ε and r are estimated at time t and t' is the time at which the weed becomes infected.

with the duration of infection, i.e., ε will be higher in plants infected earlier and longer compared to those infected later (Figure 2). The hypothetical function of Figure 2 is based on two considerations: (1) hardly any effect of the pathogen is expected at the early stage of infection, i.e., during the latent period, and (2) the effect will not increase infinitely and, in the case of biotrophic fungi, r will not equal zero, which would imply kill of the host. Whereas we may generally expect a decline of r with increasing periods of infection, the specific relationship may differ between host – pathogen interactions and may vary among biotic and abiotic conditions.

Epidemics

The spread of a pathogen through a host population can often be treated as a so-called travelling wave, comparable to the waves expanding from the point where a pebble is thrown into a pond. Treating disease spread as a travelling wave allows quite simple mathematical modelling of epidemics. Except where other references are given, we shall consider such modelling using the two leading textbooks in botanical epidemiology (Zadoks and Schein, 1979; Campbell and Madden, 1990).

Reproduction and dispersal of pathogens such as the biotrophic fungi are closely linked with their hosts and environmental conditions that may or may not be suitable for reproduction and spread. Low temperature, low abundance of the host, and lack of water, are some common factors inhibiting reproduction and dispersal of pathogens. In Europe, winter is especially a period with unfavourable conditions and pathogens have developed strategies to cope with such conditions. Three major strategies of survival can be distinguished:

(1) survival of a pathogen within host plants without reproduction and spread;
(2) increase of reproduction to guarantee that at least one or a few dispersal units contact a new host plant under suitable conditions;
(3) survival outside the host as special survival units.

Although most pathogens use one or more of these strategies, the abundance of a pathogen typically diminishes over winter and, subsequently, only little material is present within or around a host population to function as inoculum sources for a new epidemic.

Thus, a new epidemic has to start from the few inoculum sources within or around a host population. Let us consider the case of some special survival units, like the teliospores of rust fungi, functioning as inoculum sources. The fungus emerges from the survival units and infects a host plant. Subsequently, fungal mycelium develops inside the host. After a certain time, the latent period p, spores are produced by the mycelium and liberated. Spores may be produced for only one day, or over a longer period, up to several weeks, and this period of spore production is called the infectious period i. Spores are dispersed and land either on the same host plant or plants in the neighbourhood. Spores germinate and infect a plant. In the case of spores infecting the same plant as they were produced we talk about auto-infection, infection of other plants is referred to as allo-infection. Successful penetration of a plant by one spore results in one lesion and plotting the number of new lesions on plants versus the distance from the plant liberating the spores results in the so-called contact distribution D. The average number of new lesions produced by one sporulating lesion on the spore liberating plant is called the net reproductive number R_0. The processes by which of spores are liberated, dispersed, land, infect, and produce new spores may be repeated various times during a season favourable for fungal reproduction and spread, i.e., a polycyclic epidemic.

Thus, a specific epidemic can be described using p, i, D, and R_0, and the effects of all kinds of biotic and abiotic factors on an epidemic are expressed by changes in these four parameters. Epidemiological research is, therefore,

directed to estimating the parameters under various conditions and linking the parameters together. The parameters also reflect the spatial and temporal components of an epidemic. Whereas D expresses the spatial component, the other three components express the temporal component.

Various density functions can be used to describe a contact distribution D, but there is an important division between so-called exponentially bound and exponentially non-bound functions. The common normal distribution is an example of an exponentially bound function, while the power law function is an example of an exponentially non-bound function. If exponentially bound functions accurately describe a contact distribution, then the epidemic will spread as a travelling wave at a constant velocity. However, epidemics best fitted using exponentially non-bound functions spread not as a travelling wave, but like a dispersive wave i.e., spreads with a continuously increasing velocity without a closed wave front (Ferrandino, 1993). There is an ongoing debate whether dispersive waves do indeed exist because they are difficult to demonstrate experimentally (Frantzen and Van den Bosch, 2000). We shall assume here that epidemics spread like travelling waves, and that D is described by exponentially bound functions.

The area occupied by an epidemic expanding as a travelling wave is a function of time t':

$$A(t') = \pi * R^2(t'), \tag{8}$$

where R is the radius of the area occupied by an epidemic. This function can be re-written as (Van den Bosch et al., 1988a),

$$A(t') = \pi * (e + c * t')^2, \tag{9}$$

where e is a factor correcting for the time required before an epidemic arrives at the constant velocity of expansion c. Frantzen and Müller-Schärer (1998) proposed to replace e by an unknown function $f(t_1)$,

$$A(t') = \pi * (f(t_1) + c * t')^2, \tag{10}$$

where t_1 is the time required to arrive at the constant velocity c. Such a function may include various elements relevant to the build-up phase of an epidemic: (i) the strength of the inoculum sources, (ii) the net reproductive number R_0 of the fungus, (iii) the contact distribution D, (iv) the latent period of the first generation of the fungus, i.e., the time required to develop lesions caused by the inoculum sources, and (v) the number of lesions per plant for which the epidemic is followed (adapted from Shigesada and Kawasaki, 1997). The constant velocity c can be calculated using the contact distribution D, the net reproductive number R_0, and the time kernel $i(t)$, which

is based on the latent (p) and infectious (i) period (Van den Bosch et al., 1988b, c). The importance of calculating velocity c is that D, p, i, and R_0 can be determined in relatively small-scale experiments under various conditions avoiding large-scale field experiments to monitor epidemics.

Competition and epidemics

Above we have considered relatively simple modelling approaches that (i) integrate the combined effects of intra- and interspecific competition with the effects of pathogenic infection for plant weight, reproduction, or any other relevant plant trait, and (ii) describe epidemics expanding through a host population like travelling waves. We need now to go a step further and link the two together. This is possible using the factor time (Frantzen and Müller-Schärer, 1998). Whereas that study was based on the concept of critical period with respect to plant competition, the link between competition and epidemics can be made more precisely using the Equations 6 and 7 developed here. The parameter r is a function of time (Figure 2) and r can be quantified for each time of observation (t) and related to the time of infection t'. Such quantification of t' is possible using an experiment in which the time of infection is controlled. Once t' is defined, we can use it in Equation 10 to describe the spread of an epidemic from an inoculum source. Knowing the function $f(t_1)$ and the velocity c of Equation 10 the area over which the competitiveness of the host species is reduced by infection from a single inoculum source is predictable. The effects of a single inoculum source can be scaled-up on the basis of the number and spatial distribution of inoculum sources within a host population. Thus, it is possible to predict the fraction of host population with reduced competitiveness.

The importance of a modelling approach is that all parameters required can be measured using relatively small-scale experiments. Most parameters can also be measured under controlled conditions enabling quantification of the effects of various biotic and abiotic factors on the parameters. Having estimates of the parameters for specific conditions, we can test the predicted effect of an epidemic on plant competition at the population level using a large-scale field experiment. If the prediction is correct, we have both, insight in the mechanisms behind the observed outcome and a framework to predict the outcome under a range of conditions.

Application: can we use *P. lagenophorae* epidemics to reduce the competitiveness of *S. vulgaris* populations?

Competition

The rust fungus *P. lagenophorae* reduces competitiveness of its host *S. vulgaris* with respect to non-infected con-specifics (Paul and Ayres, 1986a, 1987a), with respect to other wild plant species (Paul, 1989; Paul and Ayres, 1990) and with respect to the crop lettuce (Paul and Ayres, 1987b). All these studies were based on a relatively few densities and proportions of the competing species and the effect of time of infection on the reduction of competitiveness was not taken into account. These studies, therefore, demonstrated the negative impact of *P. lagenophorae* on *S. vulgaris* competitiveness, but do not allow us to obtain estimates of the parameters ε and r as functions of time. The parameters have now been estimated for various systems (Paul & Frantzen, unpublished).

Epidemics

We have not yet a precise description of function $f(t_1)$ of Equation 10, but enough information is available to make an initial approximation of its nature. The subsequent information is based on an inoculum source consisting of 1–4 moderately infected *S. vulgaris* plants. The net reproductive number R_0 was estimated at 383 and this relatively high R_0 results in a fast approximation of the velocity c, i.e., after one generation of *P. lagenophorae* (Frantzen and Van den Bosch, 2000). From the same study we have an estimate of the contact distribution D. The contact distribution could be described by a double exponential function with a standard deviation σ of 28 cm. This leads to the prediction that an area of about 0.25 m^2 is occupied by the first generation of *P. lagenophorae*. The period until appearance of the first generation lesions, the latent period p, is 10 days under controlled, optimum, conditions (Paul and Ayres, 1984) and this is the shortest latent period we know for *P. lagenophorae*. A latent period of 14 days was determined in a field experiment in spring (Frantzen and Müller-Schärer, 1998). We assume a latent period of about two weeks is common in Western Europe under the prevailing conditions in spring and early summer. The last point relevant to estimating the outcome of the function $f(t_1)$ is the build-up of a specific disease level. Field observations indicate that plants around an inoculum source become severely infected in the second generation of *P. lagenophorae*, i.e., after about twice a latent period, due to auto- and allo-infection. It is likely that the maximum effect of *P. lagenophorae* on competitiveness of

S. vulgaris results from this severe infection. On that basis, we estimate the build-up phase of a *P. lagenophorae* epidemic relevant to biocontrol of *S. vulgaris*, at about four weeks and an area of about 0.25 m^2 is occupied by severely infected *S. vulgaris* plants during this build-up phase.

Estimates are available to calculate the constant velocity of spread, parameter c of Equation 10. The net reproductive number and the contact distribution are already mentioned above. The time kernel $i(t)$ may consist of a latent period of 14 days (see above) and the infectious period may be described by a mean time of 10 days to produce a spore during the infectious period with a standard deviation of 4.7 days (Rossi, 1999). Using these estimates, the velocity c of *P. lagenophorae* spread is about 8 cm per day at a specific infection level. This is an underestimation of the velocity observed in the field (Frantzen and Van den Bosch, 2000). One interpretation of this underestimation might be that *P. lagenophorae* spreads as a dispersive wave and not as a travelling wave. At first sight, the greater velocity of *P. lagenophorae* expansion that results from spread as a dispersive wave would seem to be an advantage with respect to biological control. However, the spread of a dispersive wave in space is not uniform making predictions about time and place of infection less reliable. Thus, more rapid spread may also have some disadvantages with respect to providing biological control as a management tool that has predictable results for the farmer. Until better predictions of dispersive waves are possible, we will continue to follow a conservative strategy and treat epidemic expansion as a travelling wave.

We have now roughly an idea about the velocity of *P. lagenophorae* spread. An initial build-up phase of approximately four weeks is required to establish a severe infection level on an area of about 0.25 m^2 of the *S. vulgaris* population. Subsequently, the spread is relatively fast, e.g., the area occupied by severely infected *S. vulgaris* plants increases to about 20 m^2 in the next four weeks. A doubling of time results in a 20-fold increase of the area. We emphasise the key importance of the build-up phase with respect to biological weed control. This initial phase is the bottleneck to a fast control of *S. vulgaris* by *P. lagenophorae* epidemics (Frantzen, 2000), and is also a period when the epidemic may be relatively easily stopped for example by unsuitable environmental conditions.

More estimates of the epidemiological parameters expressing effects of factors like resistance of *S. vulgaris*, pathogenicity of *P. lagenophorae*, temperature, and so on, on epidemics will increase the precision of predicted velocity and may provide clues to stimulate the velocity. Studies are in progress to provide such estimates. However, we have to find a balance between precision and robustness of predictions and the estimated outcome

of $f(t_1)$ and the calculated c presented here may well be reasonable indication of the velocity of *P. lagenophorae* epidemics we may expect.

Competition and epidemics

Linking *P. lagenophorae* epidemics to competition between *S. vulgaris* and crops at the population level as proposed above is not yet possible because estimates of the competition parameters ε and r are missing. We can, however, argue that stimulation of *P. lagenophorae* epidemics by increasing the number of natural inoculum sources may result in control of *S. vulgaris* in spring crops at agricultural sites by looking at the dynamics of inoculum sources.

Plants of *S. vulgaris* with disease symptoms of *P. lagenophorae* are hard to find in spring (Paul and Ayres, 1986b). This general observation can be explained by the mechanism of winter survival of *P. lagenophorae*. The fungus survives as mycelium in *S. vulgaris* plants without visible disease symptoms of the host (Frantzen and Müller-Schärer, 1999). Infection of the plants in autumn reduces the probability of *S. vulgaris* survival and thus survival of *P. lagenophorae*. In extreme situations, no *S. vulgaris* plants of a population may survive due to *P. lagenophorae* infection in autumn and/or other environmental conditions. Whereas the host has the possibility to build up relatively quickly a new population by way of seeds in spring, the fungus does not have this possibility and inoculum has to come from outside the host population at a later date (Leiss and Müller-Schärer, unpublished). This situation is common at agricultural sites, but less so at ruderal sites. Thus, the lack of inoculum is probably the major reason why natural *P. lagenophorae* populations do not control *S. vulgaris*, and that the number of inoculum sources in *S. vulgaris* populations has to be increased at agricultural sites to control the weed in spring and early summer. The major question is then how many inoculum sources are required and, a secondary but vital practical issue, can such a number of sources be established? The answer to the first part of this question should come from calculations based on the theoretical link between *P. lagenophorae* epidemics and crop – *S. vulgaris* competition at the population level, and a subsequent test of the prediction in the field. Of course, the answer to the second part of the question is strongly related to the answer to the first one.

Outlook

The theoretical considerations presented here will hopefully serve three aims. The first aim is to generate predictions that can be used to design efficient field experiments. The second aim is to understand the impact of a control agent

like *P. lagenophorae* on a weed and this understanding may result in appropriate control strategies. The third aim is to have criteria to select a natural enemy as biocontrol agent. Clearly, in selecting *P. lagenophorae* using more general criteria our procedure was not according to this third aim. However, we can now declare more precise criteria to select a natural enemy for the system management approach, based on the model parameters developed above. The most important criterion is that the factor r is sufficiently low to minimise the competition equivalence ε according to the aim of a farmer. A relatively small-scale experiment may be sufficient to indicate likely values of ε and r. The next criterion is the period of infection required to achieve the desired value of r. It should be short enough to allow time for spread of the natural enemy. An estimate of the relationship between r and time can be provided by a function like that presented in Figure 2. To decide what is long or short depends, of course, also on the characteristics of spread of the natural enemy. The higher the net reproductive number R_0, the larger the contact distribution D, the shorter the latent period p, and the shorter the average time to produce dispersal units during the infectious period i, the faster the natural enemy spreads. Again, estimates of all these epidemiological parameters can be determined in relatively small-scale experiments.

We use here explicitly the term natural enemy instead of fungus to emphasise that we feel the system management approach can not only be followed using biotrophic fungi as control agents, but also using other natural enemies like insects. All the epidemiological terms used here can be replaced quite easily by appropriate terms of spread of other kinds of organisms. Doing so increases the palette of potential biocontrol agents and this may result in a higher probability of successful weed control following the system management approach.

A general objection against biological control and, therefore, also against the system management approach is the specificity of the control, i.e., only one weed is controlled by one biological agent. Whereas this specificity is an advantage with respect to avoiding the risk on attack of non-target organisms, it may increase the costs of pest control. The attitude of weed biocontrollers until now was to focus on major weeds that are difficult to control by herbicides justifying the additional costs. The system management approach might, like the classical approach, change this attitude because of the relatively low costs involved in this approach. Again taking the example of the control of *S. vulgaris* the cheapest way for the farmer to control this weed would be to allow *P. lagenophorae* infected plants to survive in a protected place over winter and, subsequently, to transfer them as inoculum sources into a *S. vulgaris* population in spring. By removing the 'bottle-neck' in epidemic build-up that results from loss of inoculum over winter, disease

spread will be rapid and reach higher levels early in the life of the weed, and of the crops it infests. This may require a certain amount of labour, but otherwise the costs to the farmer are negligible. This low-input approach may appear unattractively 'low-tech', but such simple procedures founded on a solid understanding of the biology of the weed and pathogen can, we argue, make a genuine contribution to future weed control strategies.

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