Evolutionary Ecology (2004) 18: 647–667
DOI: 10.1007/s10682-004-5148-2

Research article

Clonal integration beyond resource sharing: implications for defence signalling and disease transmission in clonal plant networks☆

JOSEF F. STUEFER*, SARA GÓMEZ and TAMARA VAN MÖLKEN
*Department of Ecology, Radboud University Nijmegen, Toernooiveld 1, 6525ED Nijmegen, The Netherlands, (*author for correspondence, e-mail: j.stuefer@science.ru.nl)*

Co-ordinating editor: J. Tuomi

Abstract. Resource sharing between ramets of clonal plants is a well-known phenomenon, which allows stoloniferous and rhizomatous species to internally translocate water, mineral nutrients and carbohydrates from sites of high supply to sites of high demand. The mechanisms and implications of resource integration in clonal plants have extensively been studied in the past. Vascular ramet connections are likely to provide an excellent means to share substances other than resources, such as systemic defence signals and pathogens. The aim of this paper is to propose the idea that physical ramet connections of clonal plants can be used (1) to transmit signals, which enable members of clonal plant networks to share information about their biotic and abiotic environments, and (2) to facilitate the internal distribution of systemic pathogens in clonal plant networks and populations. We will focus on possible mechanisms as well as on potential ecological and evolutionary implications of clonal integration beyond resource sharing. More specifically, we will explore the role of physiological integration in clonal plant networks for the systemic transmission of direct and indirect defence signals after localized herbivore attack. We propose that sharing defence induction signals among ramets may be the basis for an efficient early warning system, and it may allow for effective indirect defence signalling to herbivore enemies through a systemic release of volatiles from entire clonal fragments. In addition, we will examine the role of clonal integration for the internal spread of systemic pathogens and pathogen defence signals within clonal plants. Clonal plants may use developmental mechanisms such as increased flowering and clone fragmentation, but also specific biochemical defence strategies to fight pathogens. We propose that clonal plant networks can act as stores and vectors of diseases in plant populations and communities and that clonal life histories favour the evolution of pathogens with a low virulence.

Introduction

Many clonal plant species form horizontal stems (rhizomes or stolons), which expand laterally from the parent plant and grow roots and new shoots at each of their nodes. This type of clonal growth leads to the formation of networks of physically interconnected, genetically identical, and functionally autonomous offspring individuals, called ramets. Physical connections between ramets allow for the internal transport of substances such as water, carbohydrates and mineral nutrients between different parts of the clonal network.

☆All authors have contributed equally to the ideas and preparation of this manuscript.

This phenomenon, termed physiological integration or resource sharing, has received rather broad attention in the last few decades, resulting in an extensive body of literature dealing with the physiological mechanisms as well as the ecological implications of resource integration in clonal plants. Since the pioneering work of Qureshi and Spanner (1971, 1973), and the seminal review by Pitelka and Ashmun (1985), clonal integration has attracted considerable attention from (eco)-physiologists (e.g. Chapman *et al*., 1991, 1992; Kemball and Marshall, 1995), ecologists (e.g. Alpert, 1991, 1996; Evans, 1991, 1992; Stuefer *et al*., 1994, 1996; Shumway, 1995) and theoretical biologists (Caraco and Kelly, 1991; Oborny *et al*., 2000, 2001; Magori *et al.,* 2003). These and numerous other studies have helped to elucidate the mechanisms, preconditions and constraints of carbohydrate, water and mineral nutrient sharing in clonal plants. They have also provided clear evidence for the ecological importance of clonal integration for enabling plant species to provide their (clonal) offspring with post-natal care, for avoiding the vulnerable life-cycle processes of seed germination and seedling establishment, and for allowing an efficient resource extraction from heterogeneous environments and the provisioning of internal support to damaged or stressed ramets (Pitelka and Ashmun, 1985; Marshall, 1990; Marshall and Price, 1997).

While it is clear that physical connections between ramets can be used to transport resources within clonal plants, much less is known about the possible function of stolon and rhizome connections for the internal distribution of non-resource substances and agents, such as plant hormones, defence signals, toxins, pathogens, and others. In spite of early suggestions that clonal plant networks may be used for sharing substances other than water, carbohydrates and mineral nutrients (Cook, 1978; Pitelka and Ashmun, 1985), the mechanisms, dynamics and implications of clonal integration beyond resource sharing has never been considered in a comprehensive way. Nonetheless, it seems likely that vascular ramet connections are an efficient and suitable means to distribute information among interlinked members of a clonal network, and that clonal fragments bear an inherent risk of intrusion and possible rapid internal spread of systemic diseases. Since any form of clonal integration (i.e. irrespective of what substance or agent is shared among connected ramets) makes use of the vascular system for long-distance transport within clonal plant networks, non-resource integration is likely to follow the same (or very similar) principles as ordinary resource integration, including known constraints and limits on clone-internal movement of substances along vascular pathways. Irrespective of this similarity, however, resource and non-resource sharing are likely to have very different implications for plant functioning, and for plant responses to the biotic and abiotic environment.

The existing knowledge and broad experimental experience with resource sharing in clonal plants can serve as an excellent basis for studying and pre-

dicting aspects of information sharing and disease spread in clonal plants. Plant traits and environmental features which are known to be of major importance for the dynamics and the extent of resource integration can also be expected to play a prominent role in non-resource sharing. Architectural and physiological features, such as the branching structure, the average distance between connected ramets, vascular sectoriality, source–sink dynamics, plant-internal water potential gradients, as well as life-history traits, such as the longevity of ramets and ramet connections, are all likely to affect processes and phenomena that rely on resource as well as non-resource integration. Sectoriality for instance, can prevent resources and anything else transported in the vascular system from reaching all parts of a plant (Stuefer, 1996; Vuorisalo and Hutchings, 1996; Watson and Casper, 1984). In clonal plants sectoriality can effectively isolate ramets or larger clone parts in terms of physiological integration (Hay and Sackville-Hamilton, 1996; Price *et al*., 1996).

In addition, aspects of the (biotic and abiotic) environment can promote and constrain physiological integration in clonal plants. Spatial habitat heterogeneity has often been shown to promote or constrain the exchange of resources between interconnected parts of clonal plants (Evans, 1991, 1992; Stuefer and Hutchings, 1994; Shumway, 1995). The longevity and transport capacity of connecting stolon or rhizome internodes is an example for plant-internal features that strongly affect the spatio-temporal degree and the quantitative amount of physiological integration and related processes in clonal plants (Stuefer *et al.,* 1998; Oborny *et al.,* 2001). These and other plant-internal and external features can thus be expected to critically affect non-resource integration and its implications in clonal plants.

The aims of the following sections of this paper are (1) to explore processes and phenomena which rely on the transmission of signals and other non-resource agents and substances through physical connections between ramets of a clonal plant, (2) to draw attention to possible ecological implications of non-resource integration, and (3) to put forward expectations, predictions and hypotheses which can be tested in future research initiatives. Due to the scarcity of specific background information, most of our predictions will be speculative in nature. We will focus on two main topics, namely the role of clonal integration (a) for distributing defence induction signals in response to herbivory and (b) on mechanisms and implications of the systemic spread of pathogens and pathogen defence signals in clonal plant networks.

Plant defences against herbivores

Plants have developed a broad array of mechanisms to cope with herbivores. Plant defence traits can be constitutive or inducible (i.e. plastic), and defence

strategies may be direct or indirect. Constitutive defence traits are always expressed, even at times and in environments where they are not needed (Wittstock and Gershenzon, 2002). Plastic defence traits are only expressed after initial damage or they may be induced by external signals such as volatiles (Bruin *et al*., 1991; Karban and Baldwin, 1997; Dicke and Hilker, 2003). Defence mechanisms can have direct or indirect effects on herbivores. Direct defences consist of inducible changes in tissue quality, plant palatability and toxicity, or in plastic alterations of anatomical and morphological traits that reduce the herbivores' preference for, or performance on, the host plant (Karban and Baldwin, 1997). Many plants can also make use of indirect defences against herbivores (e.g. damaged plants may release specific infochemicals to attract the natural enemies of the herbivore; Takabayashi and Dicke, 1996; Karban and Baldwin, 1997).

Inducible defences can either be exclusively expressed at the site of damage or they can also be activated in other, undamaged parts of the plant. The latter phenomenon, usually termed induced systemic resistance (ISR) is a common defence mechanism of plants against herbivores (Agrawal *et al*., 1999; Tollrian and Harvell, 1999). The complex signalling cascade leading to the local induction, systemic spread of the signal and non-local activation of defence traits is not yet fully understood (Roda and Baldwin, 2003). Nevertheless it seems clear that the jasmonic acid pathway and phloem-transmitted signals play a key role in the systemic induction of defence genes after herbivore damage (e.g. proteinase inhibitor genes in *Solanaceae*; Stastwick and Lehman, 1999; Thaler *et al*., 2002a, b; Stratmann, 2003). ISR, though mechanistically not fully understood, has been described for numerous plant–herbivore systems (Karban and Baldwin, 1997; Agrawal *et al*. 1999; Tollrian and Harvell, 1999).

Early warning system?

Herbivory triggers defensive responses in host plants showing systemic inducible resistance. Herbivore feeding (and in some cases also mechanical leaf damage) elicits a specific response, which leads to the production of a defence induction signal at the site of attack (Karban and Baldwin, 1997). This alerting signal then travels through vascular pathways from the site of damage to other parts of the plant. As a consequence undamaged plant parts will also activate their defensive machinery. Though never tested explicitly, stolon and rhizome connections between ramets of clonal plants are most likely to act as pathways for systemic defence induction signals (see Haukioja *et al*., 1990; Haukioja, 1991).

If herbivores attack one or a few ramets of a clonal plant, a warning signal may be produced at the site of damage and sent to other uninfested ramets

through physiological integration. This would allow undamaged ramets to receive a defence induction signal even if they are rather distant from the site of the first herbivore attack, and to activate direct or indirect defence mechanisms before the herbivores arrive. ISR in clonal plants can be seen as a preemptive defence strategy of connected, uninfested ramets against impending herbivore attacks. Such a spatial alerting strategy is beneficial whenever the induction signal spreads faster than the herbivore, and if initial herbivore attack is a reliable cue for future damage in connected ramets of a clonal plant (Karban *et al.*, 1999). Due to the potentially large size of clonal plant networks, they may constitute an ideal system to study the complex space–time relationships between benefits and costs of ISR on one hand, and cue reliability and the spatio-temporal dynamics of herbivore spread in relation to the speed and extent of systemic signal transduction, on the other hand. The rate and spatial dimensions of internal signal transmission can be expected to depend crucially on architectural plant characteristics such as average ramet distance, vascular sectoriality, source–sink relationships, and on the transport capacity of connecting internodes between ramets.

In most cases herbivore-caused damage of a given ramet is likely to entail a considerable risk of attack for adjacent ramets. However, the risk of attack by an herbivore present on a connected sibling ramet should decrease with increasing distance from the point of initial damage. In other words, the information content of the systemic warning signal is very likely to decrease with increasing distance between sender and receiver ramets in a clonal plant network. The rate of decrease (i.e. the exact decay profile of spatial autocorrelation in attack risk) depends on the behaviour, mobility and population size of the herbivore, the size and architecture (e.g. average ramet distances) of the clonal plant under attack, and on general canopy characteristics (e.g. presence of alternative host plants). To be beneficial in the long run, the physiological and ecological costs of inducible defences should be equalled or outweighed by benefits in terms of enhanced plant performance and fitness (Karban and Baldwin, 1997; Heil, 2001, 2002; Cipollini *et al.*, 2003). This implies that uninfested ramets of a clonal plant should only respond to the systemic signal and switch on their inducible defences if the danger of being attacked is high enough to justify the costs of inducing and temporarily expressing defence traits (Karban *et al.*, 1999). Any mismatch in time or space between systemic defence induction and herbivore attack is likely to lead to a costly misallocation of resources. Examples for such mismatches include cases in which herbivores disperse faster than the induction signal and cases in which the herbivore does not spread to adjacent ramets.

Intermediate levels of spatio-temporal spread of the induction signal may be the most appropriate response of many clonal plant networks to localized herbivore attack. If the spatial scale of the systemic defence induction is too

small (e.g. induction occurs only in nearest neighbour ramets), or if the induction does not spread fast enough within connected ramets, the herbivores may be able to disperse to surrounding ramets before systemic induction has occurred there. If, however, the scale of systemic induction is too large, distant ramets of a clonal fragment may be induced even though the risk of being reached by the herbivore is low. A graded response of ramets according to their distance to the site of attack, possibly mediated by a decrease in induction over space and time, may allow for the optimization of the cost-benefit balance of systemically induced defence mechanisms in response to local herbivore damage. No data are currently available to confirm or to reject this and other predictions about the mechanisms and implications of ISR in clonal plant networks.

Sharing ISR elicitors between integrated ramets of clonal plants could have important population-level consequences for plant performance, plant–plant and plant–herbivore interactions. The systemic induction of direct defence traits in clonal ramet populations could act as an effective spatial information and early warning system for spatially scattered network members in case of local herbivore attack. Such an early warning system could be particularly beneficial for clonal plants by conferring integrated ramets faster and better protection from herbivore damage than their surrounding, uninduced competitors. This effect is likely to be enforced by the selectivity of herbivores which may be discouraged from feeding on the induced parts of a clonal fragment and move preferentially to neighbouring host plants. In herbivore-prone environments the ability of clonal fragments to share ISR signals over considerable distances may critically affect competitive relations between clonal and non-clonal plants.

Physical inter-ramet connections may not only reduce, but can also increase the risk and intensity of future herbivore damage. In many clonal plant species, ramets are connected by aboveground structures (i.e. stolon internodes), which can be used as bridges by foraging herbivores, guiding them to uninfested ramets. This negative effect of physical integration should be strongest if clonal plants with a sparse growth habit (relatively long inter-ramet distances) are attacked by specialist herbivores with limited mobility. Except for enhancing protection through ISR, clonal connections between ramets might thus also increase the chance of being located by herbivores in specific cases.

Resource flows and transport pathways within clonal plant networks allow for, but also constrain the distribution of ISR signals among interconnected ramets. Resource flows in the xylem and in the phloem are largely governed by plant-internal source–sink relationships and water potential gradients, which are a function of environmental factors and of the developmental relations between connected ramets and branches (Marshall, 1990). Transport of resources and signals can be severely constrained by the physical construction

of the vascular system and/or by predominantly unidirectional flows of organic resources in several clonal species (Marshall and Price, 1997). In addition, herbivore attacks themselves, may change the source–sink relationships and resource movement patterns within interconnected groups of ramets (Haukioja, 1991; Honkanen and Haukioja, 1998; Honkanen *et al.,* 1999), thereby affecting the direction and/or speed of information sharing within the network.

The effects of induced systemic resistance in clonal plants should be strongest in plants with large-scale phloem integration among connected ramets (e.g. *Trifolium repens*; Chapman *et al.,* 1992; Stuefer *et al*., 1996; Marshall and Price, 1997) and little directional constraints on carbohydrate movement (e.g. *Potentilla* spp.; Stuefer *et al.,* 1994; van Kleunen and Stuefer, 1999; *Hydrocotyle bonariensis*; Evans, 1991, 1992). Strongly sectorial species, such as *Glechoma hederacea* (Price *et al*., 1992a, b, 1996) are likely to be constrained in the internal transmission of signals. Sectoriality, however, may not strongly affect ISR in clonal plants, if herbivore damage and defence induction occurs not on a single but on several adjacent ramets. Due to the basic differences in vasculature, monocotyledonous species are less likely to be constrained by sectoriality than dicots (Stuefer, 1996).

We can expect that young ramets and growing parts of the clonal network will be most likely to receive induction signals shortly after an attack has happened, because they usually represent strong sinks for carbohydrates (Marshall, 1990). Environmental heterogeneity, such as partial shading of a clonal plant network, may enhance the transmission rate and distance of ISR signals by promoting the transport of phloem based resources to stressed clone parts. Environmental heterogeneity imposing source–sink gradients is also likely to block or constrain the dispersal of defence signals from ramets that act as strong resource sinks. Whether herbivores can make use of this fact (e.g. by preferentially feeding on shaded ramets in habitats with small-scale heterogeneity in light conditions) is unknown to date.

Massive perfume attraction

Many plant species emit a specific blend of gaseous substances (volatiles) when they are damaged by herbivores. The volatile mixture and concentration profile of constituent compounds can be species-specific and may differ for different herbivores. In various systems damage-induced volatiles attract natural enemies of the herbivores (such as carnivores or parasitoids) that are causing the damage through feeding (Dicke *et al.,* 1993a; Pichersky and Gershenzon, 2002). Strong evidence from the scientific literature and the successful application of such triangular interactions for the biological control of herbivores (Karban and Baldwin, 1997, and studies quoted there) confirm

the effectiveness of this indirect defence mechanism in several plant–animal systems. Experimental studies have shown that herbivores or parasitoid recruiting volatiles may not only be emitted from the site of damage, but systemic induction of volatile emission can lead to the release of gaseous info-chemicals from undamaged parts of a plant (Dicke *et al.,* 1993b; Takabayashi and Dicke, 1997; Dicke and Dijkman, 2001). In the case of clonal plant networks a systemic emission of volatiles could hence lead to a significant amplification (in terms of air volume containing the info-chemical) of the indirect defence signal, and a highly increased chance of attracting natural enemies of the herbivore that caused the defence induction (see Fig. 1).

The release of volatiles from numerous ramets after systemic induction would facilitate the attraction of predators and parasitoids. However, the spatial de-coupling of herbivore position and volatile release due to the systemic transmission of induction signals among scattered ramets of a clonal network may reduce the information content of the volatile signal, thereby potentially jeopardizing the ability of predators and parasitoids to locate their prey. This situation occurs if infested and uninfested ramets emit volatiles in similar concentrations after an herbivore attack. According to Dicke (1994/1995), however, the emission of volatiles from uninfested plant parts may be weaker than the emission from the site of wounding. If so, the strength of the volatile signal produced by interconnected ramets decreases with increasing distance from the attacked ramet, thereby creating a concentration gradient which can facilitate the predator or parasitoids to locate the herbivores among infested and uninfested ramets of a clonal plant. Experimental studies are needed to clarify the existence and action of indirect defence signalling from clonal plant networks to enemies of their herbivores.

The clonal architecture can also affect the total volume and concentration of emitted volatiles. One can expect that species with a spatially scattered distribution of ramets ('guerrilla' – type of clonal growth) produce bigger and less concentrated volatile emissions than species with an aggregated ramet distribution ('phalanx' – type). In both cases, however, clonal plants are likely to release a bigger amount of volatiles than infested individual of comparable non-clonal plants or non-integrated ramets of clonal species.

Indirect defences of this type could provide clonal plants with an increased protection, which might result in an enhanced competitive strength of clonal versus non-clonal plants in herbaceous canopies. This prediction can be tested in experimental studies by comparing volatile emissions and carnivore attraction between integrated and non-integrated ramets of clonal plants, or by competition experiments between clonal and non-clonal plants grown in the presence of herbivores and a natural enemy that can perceive volatile signals. Such studies would also allow for the measurement of volatile concentration

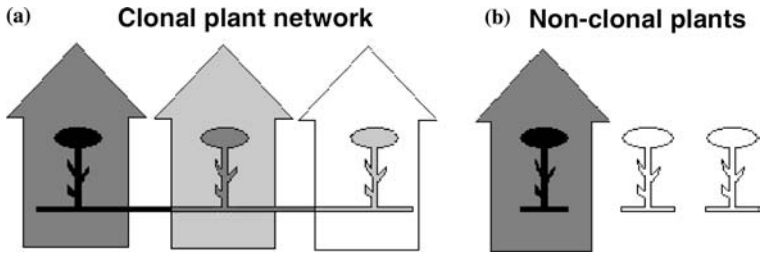

Figure 1. Emission of volatiles after initial herbivory in one ramet (black). (a) The attacked ramet sends a signal that induces volatile emission in adjacent ramets to attract the natural enemies of the herbivores. To help predators locate their prey, there should be a concentration gradient in relation to the distance from the attack point (fading coloured arrows). (b) In non-clonal plants, only the attacked individual can produce volatiles. Only very few and partly controversial studies (Dicke and Bruin, 2001 and studies mentioned therein) have reported volatile emission of neighboring plants after contact with volatiles from infested plants.

gradients around attacked ramets, and the assessment of the foraging precision of attracted enemies.

The beneficial effects of a massive emission of volatiles from interconnected ramets of clonal plants also depends on whether or not neighbouring plants are able to perceive and respond to these info-chemicals (Bruin and Dicke, 2001). Benefits to neighbours can arise from the direct perception of info-chemicals, or they may stem from an increased protection from herbivory by attracted carnivores or parasitoids. The net effect of information sharing with unconnected (and possibly unrelated) neighbours can be positive (through increased protection of a larger area) or negative (through increased performance and competitive strength of the neighbours). In other words eavesdropping (in the sense of an activation of defensive phenotypes after being exposed to info-chemicals produced by other damaged plants; Karban and Baldwin, 1997; Dolch and Tscharntke, 2000; Karban *et al.,* 2000) as well as mechanisms of group selection could favour or disfavour the emission of defence signals from clonal plant networks, depending on the competitive relationship and spatial arrangement of clonal fragments, genets and other species in natural populations and communities. These unresolved questions call for specific studies into the proximate mechanisms and ultimate implications of multi-trophic interactions between clonal plants, herbivores and their enemies.

It should be noted, that the information content, reliability and specificity of gaseous info-chemicals released to the air by damaged plants or ramets is lower than that of internally transmitted direct defence signals. The effectiveness of an alerting system based on volatile emissions is hence likely to be lower than the early warning system based on the systemic induction of direct defence traits in interconnected ramets of clonal fragments. In most of the studies reporting plant–plant communication, the activation of the response is thought

to be mediated by airborne info-chemicals (Karban and Baldwin, 1997 and studies therein, Dicke and Bruin, 2001 and studies therein, Karban *et al*., 2003) and in some cases by root-exudates (Chamberlain *et al*., 2001; Dicke and Dijkman, 2001; Guerrieri *et al*., 2002). However, in these cases of '*external warning'* the distance between infested and uninfested plants should be small in order to perceive the warning signal (both via air and soil) and to activate their defensive phenotype. In these studies the distance between infested and control plants is normally about 15 cm or less (e.g. 15 cm for volatiles perception in Karban *et al.,* 2000, less than 15 cm for root exudates perception in Chamberlain *et al.,* 2001 and Guerrieri *et al*., 2002).

Systemic pathogens: The dark side of network integration?

In spite of all obvious advantages, resource and information sharing has its risks. Modern, man-made information networks are well known for their vulnerability to *viruses* and *worms*. In direct analogy, populations of interconnected ramets of clonal plants may be especially susceptible to infections by systemic diseases, as physical links between clone members can be (ab-)used as internal dispersal highways, enabling pathogens to spread among connected ramets and to disperse rapidly within clonal ramet populations (Cook, 1985; Eriksson and Jerling, 1990). This risk might create selection pressures against communication and resource integration in clonal plants, and/or it may prompt a co-evolutionary arms race between networks and intruders (such as seen in electronic information networks). Although interconnected ramets of clonal plants can be functionally independent, they may not have independent risks of pathogen infection. After an initial infection, pathogens can trace uninfected ramets by following vascular connections through the use of the plant internal transport system for water and carbohydrates. Specialist pathogens may especially benefit from internal spread, because they can successfully locate and infect genetically identical hosts without the help of external vectors. From this perspective, clonal plants seem ideal hosts for systemic pathogens. In non-clonal plants, infections by internal disease transmission are restricted to one individual (Fig. 2).

Pathogen effects on host plants are extremely diverse, ranging from lethal or severely damaging effects, to symptom-less infections, and positive impacts on plant growth and performance. A wide range of qualitatively and quantitatively different pathogen actions is described in the literature: many pathogens cause a decrease in photosynthetic activity (Chia and He, 1999; Funayama and Terashima, 1999; Sampol *et al.,* 2003), while others affect hormone levels of their hosts (Pan and Clay, 2002) or alter resource allocation patterns to different

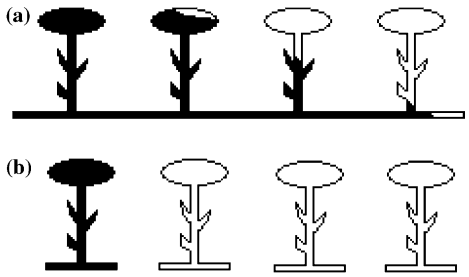

Figure 2. Systemic spread of a pathogen in clonal and non-clonal plants. In clonal plants (a) the pathogen can spread through many individuals. The spread of a pathogen in a non-clonal plant (b) is restricted to one individual. Black filling indicates the infected individuals.

organs and functions (García-Guzmán and Burdon, 1997). Pathogen infection may lead to a deformation of leaves (Gibbs and Harrison, 1976), reduced growth rates (Jones, 1992; Potter, 1993; Piqueras, 1999) and changes in the growth form of plants (Wennström and Ericson, 1992; Piqueras, 1999). In some cases, pathogens can effectively castrate (impede sexual reproduction) the host plant (García-Guzmán and Burdon, 1997; Groppe *et al.,* 1999; Pan and Clay, 2002). In rather rare cases, host plants may benefit from pathogen infections in terms of increased biomass production (Groppe *et al.,* 1999; Pan and Clay, 2002) or enhanced levels of allelopathy (Mattner and Parbery, 2001).

However, plants are by no means defenceless against pathogens. On the contrary, they have evolved an impressive array of mechanisms and strategies to tolerate, avoid or fight pathogens. In the following sections, we will focus on systemic pathogen spread and various defence mechanisms that (clonal) host plants may exhibit in response to disease infection and pathogen spread. We will concentrate on possible ecological implications of clonality in relation to systemic pathogens.

Race against time: Systemic spread of pathogens and defence signals

Pathogens can either be systemic or non-systemic. Systemic pathogens are able to move away from the initial site of infection and can contaminate other parts of the plant. In most cases they live perennially in the host plant. In contrast, non-systemic pathogens are restricted to the initial site of infection. They are often annuals that re-infect their host plants every year (Wennström, 1999). In the following sections we will focus exclusively on systemic pathogens, which can spread through the vascular system of their host plants. Fungi, for example, may grow along vascular vessels or sporulate directly into the xylem. Viruses can be transported in the phloem. They can directly be entered into the vascular system by feeding aphids or other animal vectors, or they can use

plasmodesmata to enter and exit the phloem. Once inside the phloem, viruses usually follow plant-internal source–sink flows (Leisner and Turgeon, 1993; Thompson and Schulz, 1999; Cheng *et al.,* 2000), thereby predominantly ending up at sites with high sink strengths (e.g. young, developing plant parts, resource deficient and damaged plant parts or ramets). In analogy, pathogens and pathogen propagules present in the xylem sap of plants are likely to move along water potential gradients from sites of water uptake to sites of high water loss through transpiration (Marshall, 1990; Stuefer, 1996).

Pathogen defence mechanisms can act on morphological, developmental and biochemical levels of the plant. An infected host plant may, for instance, escape the pathogen by a (partial or full) developmental switch from vegetative growth to flowering. Korves and Bergelson (2003) have recently shown that the time to flowering can be significantly shortened by pathogen infection in *Arabidopsis*. Several studies have shown that clonal plants may be able to escape their systemic pathogens by fast vegetative growth (Wennström and Ericson, 1992; Frantzen, 1994; García-Guzmán and Burdon, 1997) and by clone fragmentation (McCrea and Abrahamson, 1985; Kelly, 1995).

It is still unknown however, whether the timing and the extent of ramet isolation through clone fragmentation is a pathogen-inducible trait in clonal plant species. It is known for several groups of clonal plants (e.g. pseudo-annuals, Jerling, 1988; tussock-forming grasses, Wilhalm, 1995) that the connections between ramets are short-lived and that clones spontaneously fragment into individual ramets or small ramet groups as part of their regular development. Their ramets do not stay interconnected for extended periods of time, but become physiologically and physically independent after a short offspring production and establishment phase (Eriksson and Jerling, 1990; Piqueras and Klimes, 1998; Verburg and During, 1998; Piqueras, 1999). This seems counterintuitive because of the apparently low costs of maintaining connections and the broad evidence for positive effects of clonal integration on plant performance and fitness. It has been suggested that genet splitters may give up physical ramet connections to spread the risk of mortality (e.g. generated for instance by pathogens; Eriksson and Jerling, 1990) among independent ramets. According to this hypothesis, genet splitters could have lost their ability for prolonged integration due to past and/or current selection pressures created by systemic pathogens. This idea proved difficult to verify as most clonal species are either obligate splitters or integrators, and genetic variation for clone fragmentation is usually small or absent in most species. Direct experimentation with pathogens, intact and artificially severed clonal fragments may provide more insight into this topic.

Plants can defend themselves against viral pathogens by a mechanism known as post-transcriptional gene silencing (PTGS). PTGS is an effective defence mechanism targeted specifically at viruses, which protects plant cells by

degrading the nucleic acid of RNA viruses (Waterhouse *et al.,* 2001). PTGS can spread through the plant by an unknown signal that is capable of travelling both between cells (through plasmodesmata) and through the phloem. Infected plant cells can use this system to send a warning message to uninfected parts of the plant. These parts can then prepare their virus degradation machinery, in order to stop the infection (Waterhouse *et al.,* 2001, and references therein). This mechanism is induced whenever a pathogen carrying an avirulence (Avr) gene challenges a host plant with the matching resistance (R) gene. A so-called hypersensitive response is usually induced after infection. This process is mediated by salicylic acid (SA) and is commonly referred to as systemic acquired resistance (SAR; Gozzo, 2003). Through this mechanism, the host plant may temporarily exhibit a stronger resistance to following challenges by the same or in some cases also by other pathogens. SAR is active against viruses, bacteria and fungi (Conrath *et al.,* 2002; Gozzo, 2003; Maleck and Dietrich, 2003).

PTGS and SAR can systemically protect plants against invading pathogens. Both defence mechanisms may be of considerable importance in clonal plant networks, because they can internally spread to many (or all) functional individuals on a clonal fragment. In analogy to the early warning system against herbivores (see above) PTGS and SAR may be effective means to save connected sibling ramets from getting infected. The costs-benefit balance of PTGS and SAR will most likely depend on the effectiveness of protection against further pathogen damage, which is in turn a function of the relative speed with which pathogens and pathogen defence signals can travel within clonal plants.

The spatio-temporal extent and the dynamics of internal spread of systemic pathogens and appropriate defence mechanisms (SAR, PTGS, fragmentation) in clonal plants are largely unknown. Both the speed and the spatial extent of this spread may be system-specific and depend strongly on environmental conditions and on source–sink relationships between connected ramets. The outcome of the race between pathogens and specific defence signals should vary according to circumstances at the time and place of infection. In the absence of experimental data, any prediction as to whether systemic pathogens or systemic defence signals may win this race against time seems futile and excessively speculative. However, if pathogens could on average spread faster than the defence mechanism, clonality would be a major disadvantage for plants, and systemic pathogens should exert selection pressures against the prolonged maintenance of physical inter-ramet connections. If, on the other hand, the defence mechanisms were usually effective, clonal growth and physiological integration can be a great benefit for plants, because it allows for an effective protection of spatially scattered, yet functionally independent individuals. In non-clonal plants systemic defence mechanisms are restricted to a single individual (Fig. 3).

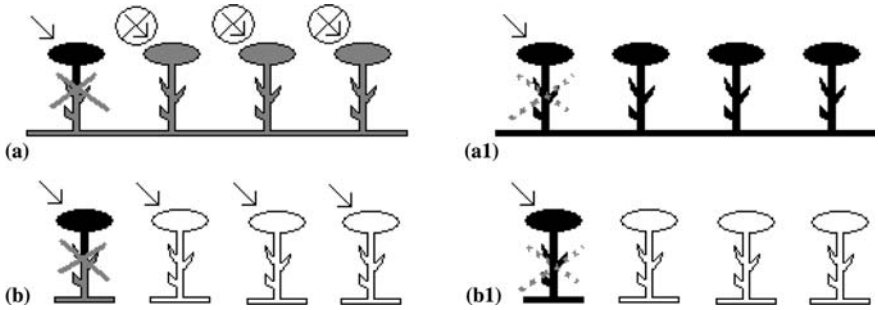

Figure 3. Race between the pathogen and the defence signals, in clonal and non-clonal plants. After infection by a pathogen (indicated with the left black arrows), PTGS and/ or SAR are induced. If these defence mechanisms (indicated by the grey cross) can be established faster than the infection, the defence signals will spread through many potential individuals in clonal plants (a), thereby protecting them from further infections. Whereas in non-clonal plants (b) only one individual is protected by these defence mechanisms and other individuals are still susceptible to infection. On the other hand, if the pathogen wins, many potential individuals are infected in clonal plants (a1), whereas only one individual is infected in non-clonal plants (b1). Black filling indicates the infected plant parts; grey filling indicates the plant parts protected by SAR and/or PTGS. Arrows indicate (possible) infection sites.

Clonal plants: Stores and vectors for diseases?

The presence of systemic pathogens in clonal plants may have serious repercussions on the population and community level, because clonal plant networks could serve as vectors for diseases and provide ideal long-term storage space for pathogens. Clonal plants can persist as long as the rate of clonal proliferation by initiating new meristems is higher or equal than the rate at which old plant parts die off (Thomas, 2002). Clonal plants can circumvent senescence and avoid the developmentally programmed death of the genetic individual by repeated rejuvenation from newly activated meristems (i.e. by spontaneous self-cloning). Therefore, clonal genets can be extremely long-lived (Oinonen, 1967; Kemperman and Barnes, 1976; Cook, 1985; Steinger *et al.,* 1996). Systemic pathogens that can persist in plants during the whole lifetime of the host may be preserved in clonal plants for very long, potentially endless periods of time. Specialist pathogens, could especially benefit from storage in clonal plants, since the need to dispense to new, maybe, hosts diminishes. In general, the availability of suitable rare/hosts can be expected to be higher for generalist pathogens, therefore storage in clonal plants may be less necessary for these pathogens.

Clonal plants could function as spatial vectors for pathogens in natural plant populations and communities. The spread of systemic diseases within populations and communities may be facilitated by the presence of clonal plant networks, because they allow pathogens to move between plants in the absence

of suitable external vectors, and without the production of specialized dispersal units. A pathogen that can persist in a clonal plant can use its host as a long-term base and spatial vector to spread to other plants within the system. From this perspective, clonal plant networks may represent spatio-temporal stepping-stones facilitating the spread of systemic diseases within populations and communities. Specialist as well as generalist pathogens are likely to use clonal plants as vectors and storage places. However, generalist pathogens probably use clonal plants in particular as spatial vectors. Because they can spread to many host species, clonal plants may provide a suitable starting-point for generalist pathogens to (re-) infect surrounding plants. The presence of generalist pathogens in long-lived, spatially extensive clonal networks may pose a threat to neighbouring plants. Specialist pathogens may benefit predominantly from clonal plants as temporal vectors. Clonal plants could give specialist pathogens the time to 'wait' for suitable host species that may not be present at all times. This notion of clonal plants as possible stores and vectors of diseases would predict that, in the long run and under comparable environmental conditions, populations with a high frequency of clonal plants might accumulate more resident pathogens and therefore suffer from higher disease loads than populations with a lower presence of clonal species. Specific data to test this prediction are not currently available.

High levels of virulence are likely to preclude systemic pathogens from using clonal host plants as long-term storage space and spatio-temporal vectors. Highly virulent pathogens are likely to kill or seriously damage entire clonal networks. To be ecologically and evolutionarily feasible, however, high levels of virulence must be coupled to very fast and efficient between-plant dispersal (Lively, 2001; Day, 2003). To date we do not have any compelling evidence for the existence or common occurrence of highly virulent *killer-pathogens* in clonal plants. We suggest that clonal plant life histories selectively favour pathogens with a low virulence, because the benefits conferred to the pathogen by a potentially unrestricted lifetime of clonal host plants might strongly select against high levels of pathogen virulence. This expectation is in concordance with general dispersion-virulence models that predict a positive relation between transmission rates and pathogen virulence (Lipsitch and Moxon, 1997; Lively, 2001; Day, 2003).

In specific cases clonal plants can actually benefit from pathogen infections. Groppe *et al.,* (1999) have shown that the internal concentration of the endophytic fungus *Epichloë bromicola* is positively correlated with the vegetative vigour of the clonal host plant *Bromus erectus*. Although infected plants showed a significant increase in vegetative growth and performance, endophyte infection also had strongly negative impacts on sexual reproduction. Seed output of the host plant was negatively correlated with fungal concentration. Pan and Clay (2002) reported a similar pathogen-mediated trade-off between

vegetative growth and sexual reproduction in their system. *Epichloë glyceria* infections enhance stolon production and accumulation of biomass in the clonal host plant *Glyceria striata*, and at the same time *E. glyceria* effectively castrates its host. By doing so, the fungal endophyte blocks the possible escape route for the host to dispose of the pathogen by flowering and sexual reproduction. In terms of vegetative growth and competitive ability of the host plant these fungi-plant associations can be considered mutualistic: the fungal endophyte enhances host performance and the host plant provides a suitable environment for the pathogen. In terms of life-history evolution of these pathogens, the lack of sexual reproduction of the host in combination with the virtual absence of genet senescence in clonal plants removes the necessity to disperse after successfully infecting a host. A very low pressure to disperse might eventually lead to the evolution of low virulence in these specific clonal plant pathogens, possibly generating a basis for the evolution of (partially) mutualistic plant–pathogen systems (Clay, 1990).

Conclusion

Based on the information and arguments provided above, we conclude that sharing substances and agents other than resources between ramets of clonal plants may have far-reaching consequences for the functioning of plant individuals, populations and communities, as well as for interactions between clonal plants on one hand, and pathogens, herbivores and the natural enemies of herbivores on the other hand. We are currently only at the beginning of research activities that will hopefully elucidate the various ecological roles, proximate mechanisms and ultimate implications of clonal integration beyond resource sharing. Future studies may shed light on these complex yet fascinating interactions.

Acknowledgements

We thank Hans de Kroon for comments on a previous version of the manuscript.

References

Agrawal, A.A., Tuzun, S. and Bent, E. (1999) *Induced Plant Defenses Against Pathogens and Herbivores: Biochemistry, Ecology and Agriculture*. APS Press.

Alpert, P. (1991) Nitrogen Sharing among ramets increases clonal growth in *Fragaria chiloensis, Ecology* **72**, 69–80.

Alpert, P. (1996) Nutrient sharing in natural clonal fragments of *Fragaria chiloensis*. *J. Ecol.* **84**, 395–406.

Bruin, J. and Dicke, M. (2001) Chemical information transfer between wounded and unwounded plants: backing up the future. *Biochem. Syst. Ecol.* **29**, 1103–1113.

Bruin, J., Sabelis, M.W. and Dicke, M. (1991). Mite herbivory induces airborne information transfer between plants. In F. Dusbàbek and Y. Bukva (eds.) Modern Acarology. Academia, Prague and SPB Academic Publishing, The Hague, pp. 599–605.

Caraco, T. and Kelly, C.K. (1991) On the adaptive value of physiological integration in clonal plants. *Ecology* **72**, 81–93.

Chamberlain, K., Guerrieri, E., Pennacchio, F., Pettersson, J., Pickett, J.A., Poppy, G.M., Powell, W., Wadhams, L.J. and Woodcock, C.M. (2001) Can aphid-induced plant signals be transmitted aerially and through the rhizosphere? *Biochem. Syst. Ecol.* **29**, 1063–1074.

Chapman, D.F., Robson, M.J. and Snaydon, R.W. (1991) The influence of leaf position and defoliation on the assimilation and translocation of carbon in white clover (*Trifolium repens* L.). 2. Quantitative carbon movement. *Ann. Bot.* **67**, 303–308.

Chapman, D.F., Robson, M.J. and Snaydon, R.W. (1992) Physiological integration in the perennial herb *Trifolium repens* L. *Oecologia* **89**, 338–347.

Cheng, N.-H., Su, C.-L., Carter, S.A. and Nelson, R.S. (2000) Vascular invasion routes and systemic accumulation patterns of tobacco mosaic virus in *Nicotiana tabacum*. *Plant J.* **23**, 349–362.

Chia, T.-F. and He, J. (1999) Photosynthetic capacity in *Oncidium* (Orchidaceae) plants after virus eradication. *Environ. Exp. Bot.* **42**, 11–16.

Cipollini, D., Purrington, C.B. and Bergelson, J. (2003) Costs of induced responses in plants. *Basic Appl. Ecol.* **4**, 79–89.

Clay, K. (1990) Fungal endophytes of grasses. *Annu. Rev. Ecol. Syst.* **21**, 275–297.

Conrath, U., Pieterse, C.M.J. and Mauch-Mani, B. (2002) Priming in plant–pathogen interactions. *Trends Pl S* **7**, 210–216.

Cook, R.E. (1978) Asexual reproduction: a further consideration. *Am. Nat.* **113**, 769–772.

Cook, R.E. (1985) Growth and development in clonal plant populations In J.B.C. Jackson (ed.) *Population Biology and Evolution of Clonal Organisms*. Yale University Press, New Haven, pp. 259–296.

Day, T. (2003) Virulence evolution and the timing of disease life- history events. *Trends. Ecol. Evol.* **18**, 113–118.

Dicke, M. (1994/1995) Why do plants talk? *Chemoecology* 5/6, 159–165.

Dicke, M. and Bruin, J. (2001) Chemical information transfer between plants: back to the future. *Biochem. Syst. Ecol.* **29**, 981–994.

Dicke, M. and Dijkman, H. (2001) Within-plant circulation of systemic elicitor of induced defence and release from roots of elicitor that affects neighbouring plants. *Biochem. Syst. Ecol.* **29**, 1075–1087.

Dicke, M., Bruin, J. and Sabelis, M.W. (1993a) Herbivore-induced plant volatiles mediate plant–carnivore, plant-herbivore and plant-plant interactions: talking plants revisited. In J.C. Schultz and I. Raskin (eds.) *Plant Signals in Interactions with Other Organisms*. Current Topics in Plant Physiology, American Society of Plant Physiologists Series, pp. 182–196.

Dicke, M., Vanbaarlen, P., Wessels, R. and Dijkman, H. (1993b) Herbivory induces systemic production of plant volatiles that attract predators of the herbivore – extraction of endogenous elicitor. *J. Chem. Ecol.* **19**, 581–599.

Dicke, M. and Hilker, M. (2003) Induced plant defences: from molecular biology to evolutionary ecology. *Basic Appl. Ecol.* **4**, 3–14.

Dolch, R. and Tscharntke, T. (2000) Defoliation of alders (Alnus glutinosa) affects herbivory by leaf beetles on undamaged neighbours. *Oecologia* **125**, 504–511.

Eriksson, O. and Jerling, L. (1990) Hierarchical selection and risk spreading in clonal plants. In J. van Groenendael and H. de Kroon (eds.) *Clonal Growth in Plants: Regulation and Function*. SPB Academic publishing, The Hague, pp. 79–94.

Evans, J.P. (1991) The effect of resource integration on fitness related traits in a clonal dune perennial, *Hydrocotyle bonariensis*. *Oecologia* **86**, 268–275.

Evans, J.P. (1992) The effect of local resource availability and clonal integration on ramet functional morphology in *Hydrocotyle bonariensis*. *Oecologia* **89**, 265–276.

Frantzen, J. (1994) The role of clonal growth in the pathosystem *Cirsium arvense – Puccinia punctiforis*. *Can. J. Bot*. **72**, 832–836.

Funayama, S. and Terashima, I. (1999) Effects of geminivirus infection and growth irradiance on the vegetative growth and photosynthetic production of *Eupatorium makinoi*. *New Phytol*. **142**, 483–494.

García-Guzmán, G. and Burdon, J.J. (1997) Impact of the flower smut *Ustilago cynodontis* (Ustilaginaceae) on the performance of the clonal grass *Cynodon dactylon* (Gramineae). *Am. J. Bot*. **84**, 1565–1571.

Gibbs, A. and Harrison, B. (1976) Effects of viruses on plants. In *Plant Virology*. *The Principles*. Edward Arnold, London, pp 19–32.

Gozzo, F. (2003) Systemic acquired resistance in crop protection: from nature to chemical approach. *Agric. Food Chem*. **51**, 4487–4503.

Groppe, K., Steinger, T., Sanders, I., Schmid, B., Wiemken, A. and Boller, T. (1999) Interaction between the endophytic fungus *Epichloë bromicola* and the grass *Bromus erectus*: effects of endophyte infection, fungal concentration and environment on grass growth and flowering. *Mol. Ecol*. **8**, 1827–1835.

Guerrieri, E., Poppy, G.M., Powell, W., Rao, R. and Pennacchio, F. (2002) Plant-to-plant communication mediating in-flight orientation of *Aphidius ervi*. *J. Chem. Ecol*. **28**, 1703–1715.

Haukioja, E., Ruohomaki, K., Senn, J., Suomela, J. and Walls, M. (1990) Consequences of Herbivory in the Mountain Birch (Betula-Pubescens Ssp Tortuosa) – importance of the functional-organization of the tree. *Oecologia* **82**, 238–247.

Haukioja, E. (1991) The influence of grazing on the evolution, morphology and physiology of plants as modular organisms. *Philos. Trans. Roy. Soc. Lond. B Biol. Sci*. **333**, 241–247.

Hay, M.J.M. and Sackville Hamilton, R. (1996) Influence of xylem vascular architecture on the translocation of phosphorus from nodal roots in a genotype of *Trifolium repens* during undisturbed growth. *New Phytol*. **132**, 575–582.

Heil, M. (2001) The ecological concept of costs of induced systemic resistance (ISR). *Eur. J. Plant Pathol*. **107**, 137–146.

Heil, M. (2002) Ecological costs of induced resistance. *Curr. Opin. Plant Biol*. **5**, 345–350.

Honkanen, T. and Haukioja, E. (1998) Intra-plant regulation of growth and plant–herbivore interactions. *Ecoscience* **5**, 470–479.

Honkanen, T., Haukioja, E. and Kitunen, V. (1999) Responses of Pinus sylvestris branches to simulated herbivory are modified by tree sink/source dynamics and by external resources. *Funct. Ecol*. **13**, 126–140.

Jerling, L. (1988) Clone dynamics, population dynamics and vegetation pattern of *Glaux maritima* on a Baltic sea shore meadow. *Vegetatio* **74**, 171–185.

Jones, R.A.C. (1992) Further studies on losses in productivity caused by infection of annual pasture legumes with three viruses. *Aust. J. Agric. Res*. **43**, 1229–1241.

Karban, R. and Baldwin, I.T. (1997) *Induced responses to herbivory*. The University of Chicago Press, Chicago.

Karban, R., Agrawal, A.A., Thaler, J.S. and Adler, L.S. (1999) Induced plant responses and information content about risk of herbivory. *Trends Ecol. Evol*. **14**, 443–447.

Karban, R., Baldwin, I.T., Baxter, K.J., Laue, G. and Felton, G.W. (2000) Communication between plants: induced resistance in wild tobacco plants following clipping of neighboring sagebrush. *Oecologia* **125**, 66–71.

Karban, R., Maron, J., Felton, G.W., Ervin, G. and Eichenseer, H. (2003) Herbivore damage to sagebrush induces resistance in wild tobacco: evidence for eavesdropping between plants. *Oikos* **100,** 325–332.

Kelly, C.K. (1995) Thoughts on clonal integration – facing the evolutionary context. *Evol. Ecol*. **9**, 575–585.

Kemball, W.D. and Marshall, C. (1995) Clonal integration between parent and branch stolon in white clover: a developmental study. *New Phytol.* **129**, 513–521.

Kemperman, J.A. and Barnes, B.V. (1976) Clone size in American aspens. *Can. J. Bot.* **54**, 2603–2607.

Korves, T.M. and Bergelson, J. (2003) A developmental response to pathogen infection in *Arabidopsis*. *Plant Physiol.* **133**, 339–347.

Leisner, S.M. and Turgeon, R. (1993) Movement of virus and photoassimilate in the phloem: a comparative analysis. *BioEssays* **15**, 741–748.

Lipsitch, M. and Moxon, E.R. (1997) Virulence and transmissibility of pathogens: What is the relationship?. *Trends Micr.* **5**, 31–37.

Lively, C.M. (2001) Propagule interactions and the evolution of virulence. *J. Evol. Biol.* **14**, 317–324.

Magori, K., Oborny, B., Dieckmann, U. and Meszena, G. (2003) Cooperation and competition in heterogeneous environments: the evolution of resource sharing in clonal plants. *Evol. Ecol. Res.* **5**, 787–817.

Maleck, K. and Dietrich, R.A. (2003) Defense on multiple fronts: how do plants cope with diverse enemies? *Trends Pl S* **4**, 215–219.

Marshall, C. (1990) Source–sink relations of interconnected ramets. In J. van Groenendael and H. de Kroon (eds.) *Clonal Growth in Plants: Regulation and Function*. SPB Academic Publishing, The Hague, pp. 23–41.

Marshall, C. and Price, E.A.C. (1997) Sectoriality and its implications for physiological integration. In H. de Kroon and J. van Groenendael (eds.) *The Ecology and Evolution of Clonal Plants*. Backhuys publishers, Leiden, pp. 79–107.

Mattner, S.W. and Parbery, D.G. (2001) Rust enhanced allelopathy of perennial ryegrass against white clover. *Agron. J.* **93**, 54–59.

McCrea, K.D. and Abrahamson, W.G. (1985) Evolutionary impacts of the goldenrod ball gall-maker on *Solidago altisima* clones. *Oecologia* **68**, 20–22.

Oborny, B., Czaran, T. and Kun, A. (2001) Exploration and exploitation of resource patches by clonal growth: a spatial model on the effect of transport between modules. *Ecol. Model* **141**, 151–169.

Oborny, B., Kun, A., Czaran, T. and Bokros, S. (2000) The effect of clonal integration on plant competition for mosaic habitat space. *Ecology* **81**, 3291–3304.

Oinonen, E. (1967) The correlation between the size of Finish bracken (*Pteridium aquiliunum* (L.) Kuhn.) clones and certain periods of history. *Acto Forestal Fennica* **83**, 1–51.

Pan, J.J. and Clay, K. (2002) Infection by the systemic fungus *Epichloë glyceriae* and clonal growth of its host grass *Glyceria striata*. *Oikos* **98**, 37–46.

Pichersky, E. and Gershenzon, J. (2002) The formation and function of plant volatiles: perfumes for pollinator attraction and defense. *Curr. Opin. Plant Biol.* **5**, 237–243.

Piqueras, J. (1999) Infection of *Trientalis europaea* by the systemic smut fungus *Urocystis trientalis*: disease incidence, transmission and effects on performance of host ramets. *J. Ecol.* **87**, 995–1004.

Piqueras, J. and Klimes, L. (1998) Demography and modelling of clonal fragments in the pseudoannual plant *Trientalis europaea L. Plant Ecol.* **136**, 213–227.

Pitelka, L.F. and Ashmun, J.W. (1985) Physiology and integration of ramets in clonal plants. In J.B.C. Jackson (eds.) *Population Biology and Evolution of Clonal Organisms*. Yale University Press, New Haven, pp. 399–435.

Potter, L.R. (1993) The effects of white clover mosaic virus on vegetative growth and yield of clones of S.100 white clover. *Plant Pathol.* **42**, 797–805.

Price, E.A., Hutchings, M.J. and Marshall, C. (1996) Causes and consequences of sectoriality in the clonal herb *Glechoma hederacea*. *Vegetatio* **127**, 41–54.

Price, E.A.C., Marshall, C. and Hutchings, M.J. (1992a) Studies of growth in the clonal herb *Glechoma-hederacea* .1. Patterns of physiological integration. *J. Ecol.* **80**, 25–38.

Price, E.A.C. and Hutchings, M.J. (1992b) Studies of growth in the clonal herb *Glechoma-hederacea* .2. The effects of selective defoliation. *J. Ecol.* **80**, 39–47.

Qureshi, F.A. and Spanner, D.C. (1971) Unidirectional movement of tracers along the stolon of *Saxifraga sarmentosa*. *Planta* **101**, 133–146.
Qureshi, F.A. and Spanner, D.C. (1973) Movement of [14C] sucrose along the stolon of *Saxifraga sarmentosa*. *Planta* **110**, 145–152.
Roda, A.L. and Baldwin, I.T. (2003) Molecular technology reveals how the induced direct defenses of plants work. *Basic Appl. Ecol.* **4**, 15–26.
Sampol, B., Bota, J., Riera, D., Medrano, H. and Flexas, J. (2003) Analysis of te virus- induced inhibition of photosynthesis in malmsey grapevines. *New Phytol.* **160**, 403–412.
Shumway, S.W. (1995) Physiological integration among clonal ramets during invasion of disturbance patches in a New England salt marsh. *Ann. Bot.* **76**, 225–233.
Steinger, T., Körner, C. and Schmid, B. (1996) Long-term persistence in a changing climate: DNA analysis suggests very old ages of clones of alpine *Carex curvula*. *Oecologia* **105**, 94–99.
Stastwick, P.E. and Lehman, C.C. (1999) Jasmonic acid-signaled responses in plants. In: Agrawal, A.A., Tuzun, S. and Bent, E. (eds.): *Induced plant defenses against pathogens and herbivores: biochemistry, ecology and agriculture*. APS Press, pp. 117–136.
Stratmann, J.W. (2003) Long distance run in the wound response – jasmonic acid is pulling ahead. *Trends Pl S* **8**, 247–250.
Stuefer, J.F. (1996) Potential and limitations of current concepts regarding the response of clonal plants to environmental heterogeneity. *Vegetatio* **127**, 55–70.
Stuefer, J.F., de Kroon, H. and During, H.J. (1996) Exploitation of environmental heterogeneity by spatial division of labour in a clonal plant. *Funct. Ecol.* **10**, 328–334.
Stuefer, J.F., During, H.J. and de Kroon, H. (1994) High benefits of clonal integration in two stoloniferous species, in response to heterogeneous light environments. *J. Ecol.* **82** , 511–518.
Stuefer, J.F., During, H.J. and Schieving, F. (1998) A model on optimal root-shoot allocation and water transport in clonal plants. *Ecol. Model* **111**, 171–186.
Stuefer, J.F. and Hutchings, M.J. (1994) Environmental heterogeneity and clonal growth: a study of the capacity for reciprocal translocation in *Glechoma hederacea* L. *Oecologia* **100**, 302–308.
Takabayashi, J. and Dicke, M. (1996) Plant–carnivore mutualism through herbivore-induced carnivore attractants. *Trends Pl S* **1**, 109–113.
Takabayashi, J., Dicke, M. (1997). Herbivore-induced plant volatiles with multifunctional effects in ecosystems: a complex pattern of biotic interactions. In T. Abe, S.A. Levin and M. Higashi (ed.) *Biodiversity, an Ecological Perspective*. Springer, pp. 131–145.
Thaler, J.S., Karban, R., Ullman, D.E., Boege, K. and Bostock, R.M. (2002a) Cross-talk between jasmonate and salicylate plant defense pathways: effects on several plant parasites. *Oecologia* **131**, 227–235.
Thaler, J.S., Farag, M.A., Pare, P.W. and Dicke, M. (2002b) Jasmonate-deficient plants have reduced direct and indirect defences against herbivores. *Ecol. Lett.* **5**, 764–774.
Thomas, H. (2002) Ageing in plants. *Mech. Ageing Dev.* **123**, 747–753.
Thompson, G.A. and Schulz, A. (1999) Macromolecular trafficking in the phloem. *Trends Pl S* **4**, 354–360.
Tollrian R. and Harvell C.D. (1999) *The Ecology and Evolution of Inducible Defenses*. Princeton University Press, Princeton.
van Kleunen, M. and Stuefer, J.F. (1999) Quantifying the effects of reciprocal assimilate and water translocation in a clonal plant by the use of steam-girdling. *Oikos* **85**, 135–145.
Verburg, R.W. and During, H.J. (1998) Vegetative propagation and sexual reproduction in the woodland understorey pseudo-annual *Circaea lutetiana* L. *Plant Ecol.* **134**, 211–224.
Vuorisalo, T. and Hutchings, M.J. (1996) On plant sectoriality, or how to combine the benefits of autonomy and integration. *Vegetatio* **127**, 3–8.
Waterhouse, P.M., Wang, M.-B. and Lough, T. (2001) Gene silencing as an adaptive defence against viruses. *Nature* **411**, 834–842.
Watson, M.A. and Casper, B.B. (1984) Morphogenetic constraints on patterns of carbon distribution in plants. *Annu. Rev. Ecol. Syst.* **15**, 233–258.
Wennstrom, A. (1999) The effect of systemic rusts and smuts on clonal plants in natural systems. *Plant Ecol.* **141**, 93–97.

Wennström, A. and Ericson, L. (1992) Environmental heterogeneity and disease transmission within clones of *Lactuca sibirica*. *J. Ecol.* **80**, 71–77.

Wilhalm, T. (1995) A comparative study of clonal fragmentation in tussock-forming grasses. *Abstracta Botanica* **19**, 51–60.

Wittstock, U. and Gershenzon, J. (2002) Constitutive plant toxins and their role in defense against herbivores and pathogens. *Curr. Opin. Plant Biol.* **5**, 300–307.