Fitness and evolution in clonal plants: the impact of clonal growth[☆]

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Abstract. Seeds have often been emphasized in estimates of plant fitness because they are the units that carry genes to the next generation, disperse, and found new populations. We contend that clonal growth also needs to be considered when estimating fitness in clonal plants, regardless of whether fitness is measured from a genet or ramet perspective. Clonal growth affects genet fitness through both genet persistence and seed production. It affects ramet fitness through new ramet production, because both seeds and clonal propagants are considered offspring. The differential production of clonal propagants will contribute to fitness differences among individuals which may result in population-level changes in allele frequencies (i.e. microevolution). We describe a form of selection unique to clonal organisms, genotypic selection, that can result in evolution. Genotypic selection occurs when genotypically based traits are associated with differences in the rate of ramet production. It can lead to evolutionary change in quantitative trait means both directly and indirectly. It leads directly to change in the ramet population by increasing the proportion of ramets with more advantageous trait values. From the genet perspective, it leads indirectly to evolution within and among populations whenever significant portions of the genetic effect on a trait are inherited through seed. We argue that under most conditions, clonal growth will play a major role in the microevolution of clonal plants.

Key words: clonal growth, clonal selection, evolution, fitness, genet, genotypic selection, ramet

Introduction

Fitness is often estimated as lifetime reproductive success (Antonovics and Ellstrand, 1984), or lifetime seed production for plants (Primack and Kang, 1989; Strauss, 1997). However, this estimate has been difficult to apply to clonal plants because they are long-lived and can propagate both clonally and sexually. Unlike non-clonal organisms, the physical and genetic individuals are decoupled in clonal plants so that many physical individuals (ramets) can have the same genotype (Primack and Kang, 1989). Genets consist of all products

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derived from one zygote and are genetically identical (barring somatic mutations). Ramets are potentially or actually physiologically independent units of a genet and have all functions common to the physical individual of non-clonal organisms, including survivorship and reproduction. Genets have traditionally been viewed as the appropriate units of selection (Janzen, 1977; Cook, 1979; Harper, 1985; Tuomi and Vuorisalo, 1989a; Eriksson and Jerling, 1990), and, thus, for measuring fitness. But determining lifetime seed production for genets is problematic because they are composed of many ramets and are potentially immortal (Eriksson and Jerling, 1990; Fagerström, 1992).

In addition to seed production, clonal plants also produce propagants through clonal growth (see Klimeš *et al.*, 1997, for modes of clonal growth). Though clonal growth is often measured in comparative studies in clonal plants (53 out of 84 studies; Appendix 1), it has frequently been ignored as a component of fitness. Often only seeds are measured as an estimate of fitness (Primack and Kang, 1989). When we have used clonal growth as a measure of fitness, questions about seed production often arise (J.J. Pan, personal observation at the *Clone 2000: Current Progress and Development in Clonal Plant Research* conference; J.S. Price, personal observation). The reason for this could be that the relationship between clonal growth and seed production is difficult to determine. It is generally accepted that only seeds affect dynamics among populations and that clonal offspring only influence their own population (Silander, 1985b; Eckert, 1999; but see Grace, 1993). However, we believe that clonal growth is an important component of fitness and can have effects beyond the local population.

It is crucial that we move toward an approach that combines clonal growth and seed production in an integrated measure of fitness. Ecological studies require a measurement of fitness in order to compare alternative strategies or phenotypes in a range of environments. Microevolutionary studies require a measurement of fitness for assessment of selection and evolutionary constraints. Both kinds of studies are not possible without a reasonable estimate of lifetime reproductive success, which, for clonal plants, must include both clonal propagation and seed production.

In order to understand how clonal growth affects microevolution in clonal plant populations, it is critical to first understand its effects on fitness. Therefore, we have separated our paper into two sections. We first show the importance of clonal growth from both genet and ramet fitness perspectives. While most researchers seem to have an intermediate perspective, the ramet and genet perspectives are the extremes of a continuum and we use these two extremes to emphasize the range of implications of clonal growth for fitness. We then consider the evolutionary implications of selection for a trait that is favored through increased clonal growth (i.e. ramet production). The goal of this paper is to present an alterative to the notion that clonal growth is only a

component of ecological success and does not affect fitness or evolution in clonal plants.

Fitness in clonal plants

The genet perspective

Fitness in clonal plants has been difficult to assess, partly because of the difficulty of measuring genet fitness. The first component of genet fitness is survivorship or genet persistence (Eriksson and Jerling, 1990; Fig. 1a). Clonal growth influences genet persistence through exploration of the environment for limiting nutrients (Cook, 1985; de Kroon and Hutchings, 1995; Oborny and Cain, 1997), physiological integration (Pitelka and Ashmun, 1985; Jónsdóttir and Watson, 1997), and survivorship by spreading the risk of mortality among ramets (Cook, 1979; de Steven, 1989). Genets consist of populations of ramets that arise via clonal growth and are considered part of genet growth (Barnes, 1966; Fenner, 1985; Mogie, 1992). Similar to genets, ramet populations have their own birth and death rates (Kays and Harper, 1974; Harper, 1985). Genet mortality depends on the probability of all ramets of a genet dying (Cook,

(a)   **(b)**

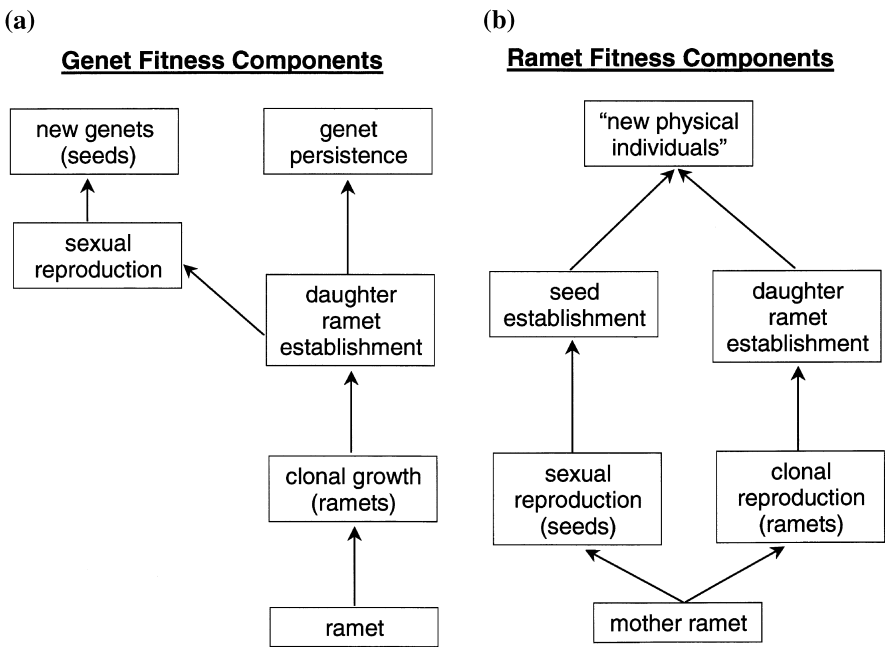**Genet Fitness Components**   **Ramet Fitness Components**

Figure 1. Contributions of clonal growth and seed production to (a) genet and (b) ramet fitness components, (a) is modeled after Figure 2 (Eriksson and Jerling, 1990).

1979). If mortality is not correlated among ramets (e.g., Schellner *et al.*, 1982), then genet mortality is the product of individual ramet mortalities (Sackville-Hamilton *et al.*, 1987). Thus, the probability of genet mortality decreases with greater numbers of ramets (or independent ramet fragments), because an agent of mortality will be more likely to 'miss' part of the genet (Schmid, 1990).

The second component of genet fitness is fecundity or new genet production, which is estimated through seed production. Seeds are important for colonization of new areas (Harper, 1977; Fenner, 1985; Cain *et al.*, 1998; but see Grace, 1993; Cain and Damman, 1997), as sources of new genotypes from sexual reproduction, and are the units by which genotypes can influence traits and genetic structure of other populations (Piquot *et al.*, 1998). Ramets are the units that undergo sexual reproduction in clonal plants, which means that seed production will be strongly influenced by clonal growth (Eriksson & Jerling, 1990; Hartnett, 1990; Maillette, 1992; Fig. 1a). More ramets will lead to greater seed production on average, especially if all ramets have the potential to reproduce (Harper and White, 1974; Augspurger, 1985; de Steven, 1989). For instance, *Puya dasylirioides* ramets are monocarpic, but the genet is polycarpic because it produces multiple ramets. Reproductive output of *P. dasylirioides* genets depends on the number of ramets produced and number of fruits produced by each ramet (Augspurger, 1985).

The influence of clonal growth on lifetime seed production can be further illustrated with a simple thought experiment that compares seed production of two genets with different strategies of allocation to sexual reproduction (number of seeds) and clonal growth (number of daughter ramets). Ramets of genet S (for seed production) produce two new ramets per year and 1000 seeds per ramet. Ramets of genet C (for clonal growth) produce four new ramets per year and 500 seeds per ramet. These allocation patterns reflect our assumption that there is a ramet-level trade-off between sexual reproduction and clonal growth (e.g. Westley, 1993). There is also evidence for trade-offs at the genet level (Abrahamson, 1980; Sutherland and Vickery, 1988; Verburg and Grava, 1998; but see Zamora *et al.*, 1998). However, the level at which the trade-off occurs should not affect the outcome of this thought experiment, except that the effects of clonal growth will take longer to manifest when there is a genet-level trade-off. For simplicity, the life history of our genets is pseudoannual, where ramets live for only one year. We also assume that seedling recruitment into the population is negligible, as is the case for many clonal plant species (Cook, 1985; Eriksson, 1993).

Greater clonal growth can lead to greater cumulative seed production very quickly, even when fewer seeds are produced per ramet (genet C; Fig. 2). Under conditions of density-independent population growth, the genet with greater clonal growth (C) has greater cumulative seed production after just three years (Fig. 2b). In year 4, genet S has eight ramets, produces 8000 seeds that year, and
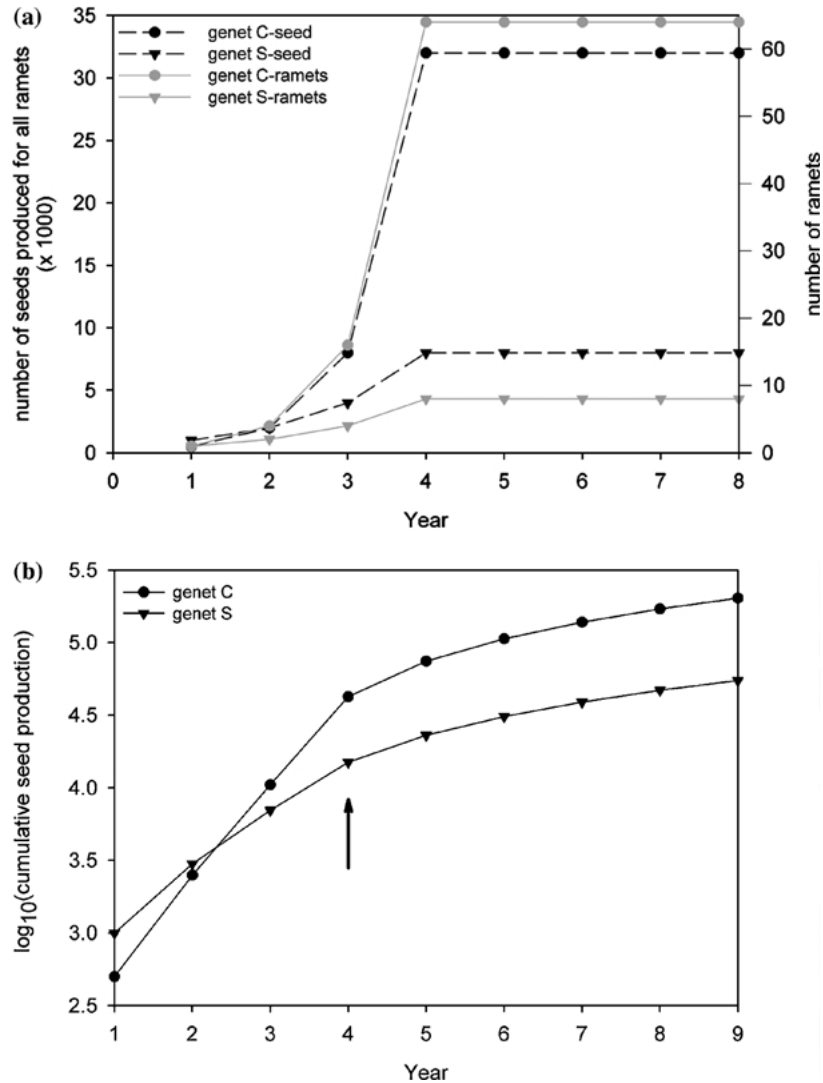
Figure 2. Comparison of genets with different growth strategies, one with greater clonal growth (C) and the other with greater seed production (S). (a) Seed production for all ramets (gray lines: circles for genet C and triangles for genet S) and number of ramets for each genet (black lines: circles for genet C and triangles for genet S) in a given year. (b) Cumulative seed production through time for each genet (circles for genet C and triangles for S). Genet C has greater fitness after just two years. Over a 4-year period of density-independent population growth, genet C produces three times as many seeds as genet S (42,500 and 15,000 seeds, respectively; denoted by the arrows). Even if density dependence occurs in the fifth year, genet C will have greater cumulative seed production than genet S (see text for details).

has a cumulative seed production of 15,000 seeds, while genet C has 64 ramets, 32,000 seeds that year, and a cumulative seed production of 42,500. Thus, by year 4, genet C has produced almost three times the number of seeds as genet S. In well-established populations, density dependence commonly occurs and can lead to a fixed number of ramets being produced for genets (Sarukhán, 1974; Fig. 2a, year 5 and beyond). Genet C will still have greater cumulative seed production than genet S in this situation, e.g. nearly four times as many seeds by year 9 (Fig. 2b). Even if ramet establishment under density dependence is not equal for these two genets, it is likely that genet C would be superior because future clonal growth should be proportional to current clonal growth (de Kroon *et al.*, 1992). Moreover, the probability of mortality of genet C should be lower than genet S because of risk-spreading. The longer the period of exponential growth or genet persistence in the population, the greater the effect of clonal growth on lifetime seed production. This thought experiment is currently being developed into a model (Price and Pan, in preparation).

Similar to interest earned on a bank account balance, compounding through clonal growth, i.e. daughter ramet establishment, leads to more consistent and greater lifetime seed production (Fig. 2). Genet seed production will be more consistent with more ramets because there will be a higher likelihood that at least a few of these ramets will be reproductive each year. Moreover, since clonal plants are usually perennials, yearly seedling establishment may be less critical. Thus, with more consistent and greater overall seed production, it will be more likely that seeds will be present when favorable environmental conditions occur.

The ramet perspective

Recently, a switch to a ramet- or meristem-based perspective of fitness has been advocated as a more tractable alternative to a genet-based perspective (Sackville Hamilton *et al.*, 1987; Fagerström, 1992; Wikberg, 1995; Winkler and Fischer, 1999), though it is far from being widely accepted. However, clonal propagation has a large effect on ramet fitness because daughter ramets are also measured for lifetime reproductive success (Fig. 1b). Ramets have two types of offspring, seeds that arise through sexual reproduction and daughter ramets that arise through clonal propagation (Sackville-Hamilton *et al.*, 1987). Unlike for genets, clonal propagation does not lead to risk-spreading of mortality for the mother ramet because clonal offspring are considered new individuals. Therefore, mother ramet survivorship depends only on one ramet, the mother ramet itself. Thus, clonal propagation affects ramet fitness only through fecundity.

Both seed and daughter ramet production need to be measured for ramet fitness, although the relative importance of each as a component of lifetime reproductive success is difficult to determine. For genets, where lifetime re-

productive success is measured as seed number, it may be reasonable to assume that genets that make more seeds will leave more offspring in the next generation. For ramets, estimating lifetime reproductive success is not as simple as counting the number of offspring (seed plus clonal offspring) because seed and clonal offspring do not have the same probability of establishing (Cook, 1985). That is, the proportion of seeds successfully establishing will not be the same as the proportion of ramets successfully establishing, so offspring number will not be as informative. For instance, daughter ramets generally have higher establishment rates than seed offspring (Grace and Wetzel, 1982; Shumway, 1995). These establishment differences coupled with variation in seed and daughter ramet production by mother ramets makes ramet fitness difficult to predict. Continuing with our previous example using genets C and S, we find that ramets with greater clonal propagation (genet C) can have greater fitness when seed offspring establishment is low (Fig. 3). On the other hand, when seed offspring establishment is moderate, then ramets with greater seed production (genet S) may have greater fitness.
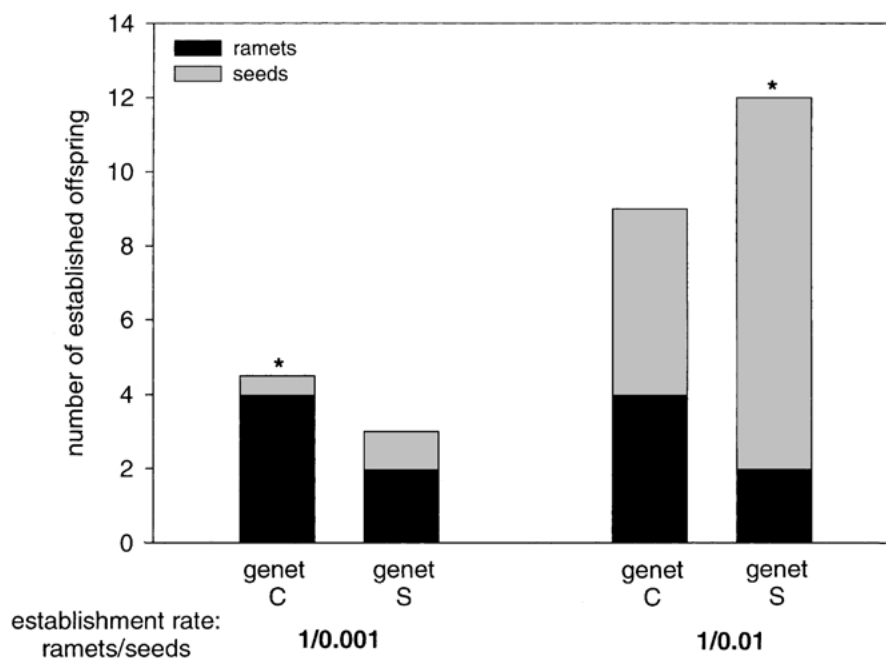
Figure 3. The influence of relative establishment rates on the number of established offspring. See text for details on genets C and S. When seed offspring establishment is very low (0.001) relative to ramet establishment (1), then genet; C will have more established offspring than genet S. The reverse is true when seed establishment is slightly higher (0.01 instead of 0.001). (*) denotes genet with greater offspring establishment. Black portion of the bar is for ramet offspring and gray portions of the bar are for seeds offspring.

Further considerations for measuring ramet fitness include genetic related-ness of the different offspring types and relative probabilities for establishment within and beyond the local population. Since clonal offspring are vegetatively derived, they have the whole genetic complement of the mother ramet (Abrahamson, 1980; Silander, 1985b). In contrast, seed offspring receive half of their genes from the mother ramet, unless they are the result of selfing (see next section). The influence of mother ramet-offspring genetic relatedness on ramet fitness will also depend on offspring establishment rates within and beyond the population. Clonal offspring will usually have a higher probability of establishment within the local population than seed offspring (Cook, 1985). This situation is likely to be reversed beyond the local population (Meyer and Schmid, 1999; but see Grace, 1993). Because the probability of offspring establishment outside the local population is very difficult to assess, measuring ramet fitness will not be simple.

Effects of clonal growth on evolution of clonal plant populations

Estimates of 'individual' fitness in clonal plants are not uncommon (84 studies in our survey, Appendix 1). However, empirical studies that address whether fitness differences are likely to lead to microevolution in clonal plant populations through selection of specific traits are extremely rare (three out of 84 studies; Ennos, 1985; Silander, 1985a; Totland, 1999). The rarity of empirical studies of evolution does not seem to indicate a lack of interest: 25% of the studies that reported estimates of fitness included speculation as to the potential effects of past, current, or future selection based on their results. More direct study of the potential for evolution of specific traits is needed in order to determine whether this speculation is warranted.

In the previous section, we argued that clonal growth is a major determinant of fitness from both a genet and ramet perspective. It has generally been accepted that clonal growth can change the relative representation of genotypes within populations (Silander, 1985b; Starfinger and Stöcklin, 1996; Pyšek, 1997). However, its evolutionary importance has been questioned (Harper, 1980; Eckert, 1999). The remainder of our paper addresses ways in which selection mediated by differences in clonal growth (i.e. ramet production) can lead to evolutionary changes that can have within and among population effects.

Phenotypic selection, genotypic selection and evolution

Phenotypic selection occurs whenever among-individual variation in a trait is associated with variation in fitness and results in a within-generation change in

the trait distribution of a cohort (Endler, 1986). There will be an evolutionary response to phenotypic selection whenever genetic relatedness causes offspring to resemble their parent(s) with respect to a trait (i.e. whenever the trait is heritable, e.g. Lande and Arnold, 1983). The resulting change in allele frequencies among generations is often referred to as microevolution (Lerner, 1958; Silander, 1985b). We will focus on this short-term, quantitative genetic evolution of population means (e.g. Via and Shaw, 1996; Pfrender and Lynch, 2000).

The term genotypic selection was introduced by Haldane (1957, cited in Lerner, 1958) to distinguish differential reproduction of genotypes (which will lead to changes in the genetic composition of the following generation) from differential reproduction of phenotypes (which may or may not lead to changes in the genetic composition of the following generation). Although Haldane envisioned genotypic selection as describing any case of differential reproduction of genotypes (i.e. similar to the modern day definition of natural selection, see Endler, 1986), we more specifically define genotypic selection as occurring when variation in a genotypically based trait is associated with differential rates of ramet production[1].

Genotypic selection is similar to the term clonal selection as used in the clonal animal literature (Via and Shaw, 1996; Pfrender and Lynch, 2000). However, in the agricultural literature, clonal selection usually refers to the process of identifying clones with agriculturally valuable traits (Brown and Glaz, 2001; Tsarouhas et al., 2001). Moreover, in immunology and oncology, clonal selection includes the production of genetic variants (Rajewsky, 1993, 1996; Sigal and Ron, 1994; Paul, 1999). We have chosen to use the term genotypic selection to emphasize that it results from the proliferation of individuals with identical genotypes and to avoid confusion with other usages of the term clonal selection.

Genotypic selection will occur when genet populations exhibit genotypically based variation in disease resistance and genets with lower resistance produce fewer new ramets. Under these conditions, genotypic selection for resistance is occurring because higher resistance is associated with greater ramet production. Resistance to herbivory and disease have been found to be related to variation in clonal propagation (Goldberg, 1988; Cain et al., 1991; Meyer and Root, 1993), though evidence of a genotypic basis for these traits was not established. Even though we focus on disease resistance in this paper, any trait that is associated with differential clonal propagation will be subject to genotypic selection. Furthermore, although a causal relationship between trait variation and differential clonal propagation may be common, heritable traits that are not themselves responsible for the variation in clonal propagation can also be favored by genotypic selection.

It is important to explore the effect of genotypic selection on both clonal and seed offspring because of their differential probabilities of establishment within

and beyond the population. Differences in ramet production among genotypes will directly change allele frequencies in the ramet population because clonal offspring are genetically identical to their parent. Regardless of whether this change in allele frequency is considered to be evolution, it will result in increased mating within or among genets with more advantageous values of the trait. Under most circumstances, this non-random mating among genets will cause a change in allele frequencies in the seed pool, allowing the changes in the ramet population to be passed on to future genet generations.

Direct effects of genotypic selection on the ramet population

Genotypic selection leads directly to change in the ramet population by increasing the proportion of ramets with more advantageous values of specific traits. In our example, genotypic selection for resistance results from the greater clonal propagation exhibited by ramets with higher levels of genotypically based resistance. Whether this increased ramet production results directly in evolutionary change depends on the fitness perspective employed. As genotypic selection proceeds, ramets with greater resistance will make up an increasing proportion of the population (assuming that disease acts as a relatively strong or consistent selective pressure), resulting in an increase in the mean level of resistance in the ramet population. Genotypic selection can be said to have caused evolution of resistance level (i.e. an inter-generational change in allele frequencies), as long as ramets are considered to be offspring that represent the next generation. However, if one retains a strict genet perspective, allele frequency changes in the ramet population will not qualify as evolution because these changes will often occur without changing allele frequencies in the corresponding genet population.

This difficulty of characterizing changes in allele frequency due to the separation of the genetic (genet) and physical (ramet) individual is not unique to clonal plant systems (Andrews, 1991). In other clonal systems, a compromise between the genet and ramet perspective has been adopted (e.g. bryozoans, Hughes, 1992; aphids, Service and Lenski, 1982; *Daphnia*, Baillieul *et al.*, 1996; *E. coli*, Kibota and Lynch, 1996; and even RNA viruses, Holland *et al.*, 1991). Although research in these systems usually focuses on clones, a ramet-like perspective has been adopted with regard to clonal propagants: they are treated as offspring.

Indirect evolutionary effects of genotypic selection on genet populations

Regardless of whether one accepts that changes in allele frequency in the ramet population are evolutionary, it is important to recognize that they can lead indirectly to genet-level evolutionary change. Genotypic selection leads indi-

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rectly to evolutionary change within and among genet populations whenever significant portions of the genotypic effect on a trait are inherited by seed offspring. Although the nature of the genetic components of trait variance, breeding system, and population structure all affect the degree to which genotypic selection can cause trait evolution through the seed pool, conditions that prevent it from having any evolutionary effect are likely to be rare.

The genetic effect on the value of a quantitative trait can be represented as the sum of additive and non-additive genetic effects (see Falconer and Mackay, 1996). Estimates of the additive effect on a trait quantify the proportion of its variance that is due to individual alleles independent of the effects of other alleles at the same locus or other loci. The non-additive effect is due to interactions among alleles. It includes dominance effects, which derive from interactions between alleles at the same locus, and epistatic effects, which depend on combinations of alleles at multiple loci. Clonally propagated individuals inherit intact genotypes and thus both non-additive and additive genetic components from their parent. On the other hand, sexual progeny inherit genes and not genotypes such that only the additive portion of the genetic effect can be consistently passed on through seed in obligately outcrossing, random-mating (panmictic) populations. This is because mixis and recombination will break up allele combinations, making the non-additive genetic effect of progeny genes unpredictable.

Furthermore, the degree to which genotypic selection will result in trait evolution through seed will depend strongly on the size of the additive genetic effect. When the genetic basis of resistance is comprised largely of an additive effect (i.e. resistance has high narrow-sense heritability), genotypic selection will lead to an increase in the mean level of resistance in the seed pool. As genotypic selection increases the number of ramets representing those genets that have greater resistance, a greater proportion of the seed pool results from matings among genets that have high additively based resistance. Since a parent's additive component is inherited by its seed offspring, genotypic selection will have a major impact on allele frequencies in the seed pool (i.e. evolution). These effects of clonal growth on the seed pool can cause it to have evolutionary effects that span genet generations and affect other populations.

Although the importance of genotypic selection for evolution through seed production is likely to be greater when the additive genetic effect on a trait is large, it can also lead to seed-based evolution when the non-additive effect makes up the majority of a trait's genetic basis. Under these conditions, however, predicting the impact of genotypic selection on seed-based evolution is complicated by the interaction of breeding system and population structure. These factors are important because they play major roles in determining the consequences of recombination for the inheritance of the non-additive effect.

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In self-compatible species, mating among ramets of the same genet will cause a significant portion of the non-additive effect on a trait to be passed on to seed offspring because it results in reduced breakup of allelic combinations (Baudouin *et al.*, 1995; Pollack and Sabran, 1999). The inherently spatial nature of clonal growth may cause ramets of a resistant genet to be clumped within a population, which is likely to increase the frequency of matings among ramets of the same genet (Silander, 1985b, Charpantier, pp. 583–600). This increase in selfing strongly limits the number of alleles available for substitution by recombination. When selfing leads to limited breakup of favorable gene combinations during seed production, trait values based largely on a non-additive genetic effect that have spread through a population by clonal propagation can also spread to other populations.

Inheritance of a non-additive effect is also possible in obligately outcrossing species because population substructure can limit the influence of recombination. In cases where the genetic makeup of sub-populations varies and gene flow between sub-populations is limited, recombination will be limited to a shuffling of the alleles that are by chance present in that sub-population. In sub-populations with multiple resistant genets, genotypic selection will lead to an over-representation of resistant genets, and mating among them will be common. If the gene complexes responsible for resistance are similar among resistant genets, their seed offspring are likely to inherit a portion of their non-additive genetic effect on resistance. Thus, it is possible for genotypic selection to cause evolution through outcrossed seed even without a substantial additive genetic effect.

In summary, we have presented a number of quantitative genetic scenarios where within-population spread of a genotypically based trait is likely to indirectly change the mean value of the trait in new genets (seeds). This suggests that differential clonal growth (i.e. ramet production) will affect genetic structure within and beyond the focal population.

The relationship between genotypic and hierarchical selection

Although genotypic selection may appear to be a type of hierarchical selection, it is not hierarchical in nature. Hierarchical selection theory posits that phenotypic selection operates through interaction among groups based on a higher level of organization (e.g. among genets) through its influence on the fitness of the reproductive units which comprise them (e.g. ramets; Tuomi and Vuorisalo, 1989b; Pederson and Tuomi 1995). Thus it requires that group-level traits (e.g. spatial dispersion or density of ramets within genets, see Vuorisalo *et al.*, 1997) modify fitness at the level of the reproductive units (Tuomi and Vuorisalo 1989b). In contrast, genotypic selection operates exclusively at the level of the reproductive units (i.e. among ramets), acting completely independently

from group-level traits. It is true that genotypic selection relies on variation among genotypes in number of clonal propagants, but genotype can hardly be considered an emergent group-level trait since it can clearly be assigned to individual ramets.

Furthermore, since hierarchical selection focuses on phenotypic selection, it examines interactions that might lead to variation in fitness without any information about the heritability of this variation (Vuorisalo *et al.*, 1997). Because of this, the evolutionary implications of hierarchical selection are difficult to assess. Genotypic selection, on the other hand, depends on inheritance of the entire genotype by daughter ramets. As we have seen, genotypic selection will favor certain alleles and allele complexes, which often results in evolutionary change in average trait values.

We have noted that an influence of genet-level traits is not required for genotypic selection. However, it may alter the likelihood that genotypic selection will lead to trait evolution through genetic changes in the seed pool. Variation in group-level characters, such as the spatial dispersion or density of ramets within genets, is likely to affect patterns of mating within or among genets, which could in turn affect ramet fitness (and therefore result in hierarchical selection; see Heisler and Damuth (1987) for a method to identify multilevel selection). As discussed above, non-random mating can allow allele combinations which may be favored through genotypic selection, to be passed on through seed. In this way, hierarchical selection may change the likelihood that genotypic selection will influence the traits and genetic structure of other populations.

Conclusions

In this paper, we have discussed the impact of clonal growth on fitness and evolution in clonal plant populations. We have shown that clonal growth affects genet fitness through survivorship and future seed production and ramet fitness through daughter ramet production. The production and establishment of daughter ramets will also affect trait distributions: directly through increases in the number of ramets with favorable values of the trait or indirectly by affecting future seed pools. Changes in trait distributions through genotypic selection can lead to evolution of clonal plant populations from either fitness perspective.

Having multiple types of individuals has long been problematic in the study of clonal plants. Janzen (1977) noted that 'the study of dandelion ecology and evolution suffers from confusion of the layman's "individual" with the "individual" of evolutionary biology'. Similarly, Harper (1980) said that 'a demography at the module level is almost irrelevant,…(an evolutionist) needs to

know about births and deaths of genets – of genetic individuals'. Twenty years later, the study of clonal plant ecology and evolution seems to be suffering from a polarization that has resulted from the distinction between ramets and genets. From the genet perspective of fitness ramets are not considered as offspring, and from the ramet perspective, the dynamics of genetic individuals are often ignored. Because both of these perspectives have substantial limitations, progress in the empirical study of fitness and evolution of clonal plants has been impeded.

Clonal plant research has been criticized for its limited attention to evolution (Eckert, 1999). We believe that the shortage of evolutionary studies results from a lack of understanding of the effects of clonal growth on fitness and evolution. This could be due to the perception of ecological and evolutionary fitness as distinctly different concepts. However, the separation of ecological and evolutionary fitness may not be valid because of the long lifespan of clonal plants (genets) and lasting effects of genetically identical offspring (ramets) on future seed pools. As we have argued in this paper, clonal growth clearly affects persistence of genes or gene combinations and seed production in both of these cases. For clonal plants, these two types of fitness are not distinct because there can be substantial overlap between ecological and evolutionary time.

We recommend that fitness be assessed with an integrated measure of clonal growth and seed reproduction. The current lack of integration has impeded progress in clonal plant evolution research. We have emphasized that an association between specific traits and increased clonal propagation will often lead to evolution via changes in the genetic makeup of the seed pool. It is our hope that by emphasizing the causal connection between these two forms of 'reproduction' and its evolutionary implications, we have provided some insight into the important variables which must be included in an integrated measure of clonal plant fitness.

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Note

[1] A trait can be said to have a genotypic basis when a genet effect can be detected in an experimental population of clonally propagated fragments of multiple genets (e.g. Silander and Antonovics, 1979; Grace and Wetzel, 1981; Cheplick, 1995; Price, in preparation).

Appendix 1

We searched for empirical papers on clonal plant research published between 1981 and 2000 in *Ecology, Evolution, Journal of Ecology, Oecologia, Oikos*, and *Plant Ecology* (formerly *Vegetatio*), using the following strategy. First, we identified all records in the Biological Abstracts (BiosisTM) database that contained data on clonal plant species with an emphasis on genera that were common in van Groenendael and de Kroon (1997):

((plant*) and (clonal or genet or ramet* or module* or rhizome* or stolon*)) or (*Carex* or *Fragaria* or *Glechoma* or *Lolium* or *Potentilla* or *Prunella* or *Ranunculus* or *Rubus* or *Rumex* or *Solidago* or *Spartina* or *Trientalis* or *Trifolium* or *Viola*)

Then, we narrowed this first set of papers to those that were likely to report fitness components (reproduction or fecundity or fitness or selection or evolution) in the title or abstract field.

We retrieved 334 papers of which only 84 were relevant. Two hundred and fifty papers were eliminated either because they did not address fitness components, were on non-clonal or caespitose species, model or theory papers, or did not have measures or comparisons of individual sexual or clonal propagation. For the remaining 84 papers, data on were recorded for five criteria: subject area, years of data, fitness components measured, individual of interest, and perspective on selection. A summary of our literature survey and its references are available upon request.

References

Abrahamson, W.G. (1980) Demography and vegetative reproduction. In O.T. Solbrig (ed.) *Demography and Evolution in Plant Population*. University of California Press, Berkeley/Los Angeles, pp. 89–104.
Andrews, J.H. (1991) *Comparative Ecology of Microorganisms and Macroorganisms*. Springer-Verlag, New York.
Antonovics, J. and Ellstrand, N.C. (1984) Experimental studies of the evolutionary significance of sexual reproduction. 1. A test of the frequency-dependent selection hypothesis. *Evolution* **38**, 103–115.
Augspurger, C.K. (1985) Demography and life history variation of *Puya dasylirioides*, a long-lived rosette in tropical sub-alpine bogs. *Oikos* **45**, 341–352.

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Baillieul, M., Selens, M. and Blust, R. (1996) Scope for growth and fitness in *Daphnia magna* in salinity-stressed conditions. *Funct. Ecol.* **10**, 227–233.

Barnes, B.V. (1966) The clonal growth habit of American aspens. *Ecology* **47**, 439–447.

Baudouin, L., Cao, T.V. and Gallais, A. (1995) Analysis of the genetic-effects for several traits in oil palm (*Elaeis guineensis* Jacq.) populations. 1. Population means. *Theor. Appl. Genet.* **90**, 561–570.

Brown, J.S. and Glaz, B. (2001) Analysis of resource allocation in final stage sugarcane clonal selection. *Crop Sci.* **41**, 57–62.

Cain, M.L., Carson, W.P. and Root, R.B. (1991) Long-term suppression of insect herbivores increases the production and growth of *Solidago altissima* rhizomes. *Oecologia* **88**, 251–257.

Cain, M.L. and Damman, H. (1997) Clonal growth and ramet performance in the woodland herb, *Asarum canadense*. *J. Ecol.* **85**, 883–897.

Cain, M.L., Damman, H. and Muir, A. (1998) Seed dispersal and the Holocene migration of woodland herbs. *Ecol. Monographs* **68**, 325–347.

Cheplick, G.P. (1995) Genotypic variation and plasticity of clonal growth in relation to nutrient availability in *Amphibromus scabrivalvis*. *J. Ecol.* **83**, 459–468.

Cook, R.E. (1979) 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, L.W. Buss and R.E. Cook (eds) *Population Biology and Evolution of Clonal Organisms*. Yale University Press, New Haven, CT, pp. 259–296.

de Kroon, H., Hara, T. and Kwant, R. (1992) Size hierarchies of shoots and clones in clonal herb monocultures: do clonal and non-clonal plants compete differently? *Oikos* **63**, 410–419.

de Kroon and Hutchings, M.J. (1995) Morphological plasticity in clonal plants: the foraging concept reconsidered. *J. Ecol.* **83**, 143–152.

de Steven, D. (1989) Genet and ramet demography of *Oenocarpus mapora* ssp. *mapora*, a clonal palm of Panamanian tropical moist forest. *J. Ecol.* **77**, 579–596.

Eckert, C.G. (1999) Clonal plant research: proliferation, integration, but not much evolution. *Am. J. Bot.* **86**, 1649–1654.

Endler, J.A. (1986) *Natural Selection in the Wild.* Princeton University Press, Princeton, NJ.

Ennos, R.A. (1985) The significance of genetic variation for root growth within a natural population of white clover (*Trifolium repens*). *J. Ecol.* **73**, 615–624.

Eriksson, O. (1993) Dynamics of genets in clonal plants. *Trends Ecol. Evol.* **8**, 313–316.

Erickson, 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, The Netherlands, pp. 79–94.

Fagerström, T. (1992) The meristem–meristem cycle as a basis for defining fitness in clonal plants. *Oikos* **63**, 449–453.

Falconer, D.S. and Mackay, T.F.C. (1996) *Introduction to Quantitative Genetics.* 4th edn. Longmans, London, UK.

Fenner, M. (1985) *Seed Ecology.* Chapman and Hall, London.

Goldberg, D.E. (1988) Influence of plant phenology on the insect herbivore-bittercress interaction. *Oecologia* **77**, 357–364.

Grace, J.B. (1993) The adaptive significance of clonal reproduction in angiosperms: an aquatic perspective. *Aquat. Bot.* **44**, 159–180.

Grace, J.B. and Wetzel, R.G. (1981) Phenotypic and genotypic components of growth and reproduction in *Typha latifolia*: Experimental studies in marshes of differing successional maturity. *Ecology* **62**, 789–801.

Grace, J.B. and Wetzel, R.G. (1982) Niche differentiation between two rhizomatous plant species: *Typha latifolia* and *Typha angustifolia*. *Can. J. Bot.* **60**, 46–57.

Harper, J.L. (1977) *Population Biology of Plants.* Academic Press, London.

Harper, J.L. (1980) Plant demography and ecological theory. *Oikos* **35**, 244–253.

Harper, J.L. (1985) Modules, branches, and the capture of resources, In J.B.C. Jackson, L.W. Buss and R.E. Cook (eds) *Population Biology and Evolution of Clonal Organisms*. Yale University Press, New Haven, CT, pp. 1–33.

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Harper, J.L. and White, J. (1974) The demography of plants. *Ann. Rev. Ecol. Syst.* **5**, 419–463.

Hartnett, D.C. (1990) Size-dependent allocation to sexual and vegetative reproduction in four clonal composites. *Oecologia* **84**, 254–259.

Heisler, I.L. and Damuth, J. (1987) A method for analyzing selection in hierarchically structured populations. *Am. Nat.* **130**, 582–602.

Holland, J.J., de la Torre, J.C., Clarke, D.K. and Duarte, E. (1991) Quantification of relative fitness and great adaptability of clonal populations of RNA viruses. *J. Virol.* **65**, 2960–2967.

Hughes, D.J. (1992) Genotype-environment interactions and relative clonal fitness in marine bryozoans. *J. Animal Ecol.* **61**, 291–306.

Janzen, D.H. (1977) What are dandelions and aphids? *Am. Nat.* **111**, 586–589.

Jónsdóttir, I.S. and Watson, M.A. (1997) Extensive physiological integration: an adaptive trait in resource-poor environments? In H. de Kroon and J. van Groendael (eds) *The Ecology and Evolution of Clonal Plants*. Backhuys Publishers, Leiden, pp. 109–136.

Kays, S. and Harper, J.L. (1974) The regulation of plant and tiller density in a grass sward. *J. Ecol.* **62**, 97–105.

Kibota, T.T. and Lynch, M. (1996) Estimate of the genomic mutation rate deleterious to overall fitness in *E. coli*. *Nature* **381**, 694–696.

Klimeš, L., Klimešová, J., Hendriks, R. and van Groenendael, J. (1997) Clonal plant architecture: a comparative analysis of form and function. In H. de Kroon and J. van Groenendael (eds). *The Ecology and Evolution of Clonal Plants*. Backhuys Publishers, Leiden, pp. 1–29.

Lande, R. and Arnold, S.J. (1983) The measurement of selection on correlated traits. *Evolution* **37**, 1210–1226.

Lerner, I.M. (1958) *The Genetic Basis of Selection*. John Wiley and Sons. Inc., New York.

Maillette, L. (1992) Plasticity of modular reiteration in *Potentilla anserina*. *J. Ecol.* **80**, 231–239.

Meyer, A.H. and Schmid, B. (1999) Seed dynamics and seedling establishment in the invading perennial *Solidago altissima* under experimental treatments. *J. Ecol.* **87**, 28–41.

Meyer, G.A. and Root, R.B. (1993) Effects of herbivorous insects and soil fertility on reproduction of goldenrod. *Ecology* **74**, 1117–1128.

Mogie, M. (1992) *The Evolution of Asexual Reproduction in Plants*. Chapman and Hall, London.

Oborny, B. and Cain, M.L. (1997) Models of spatial spread and foraging in clonal plants. In H. de Kroon and J. van Groenendael (eds) *The Ecology and Evolution of Clonal Plants*. Backhuys Publishers, Leiden, pp. 155–183.

Paul, W.E. (1999) *Fundamental Immunology*. 4th edn. (Chap. 25) Lippincott-Raven Publishers, Philadelphia.

Pedersen, B. and Tuomi, J. (1995) Hierarchical selection and fitness in modular and clonal organisms. *Oikos* **73**, 167–180.

Pfrender, M.E. and Lynch, M. (2000) Quantitative genetic variation in *Daphnia*: temporal changes in genetic architecture. *Evolution* **54**, 1502–1509.

Pollak, E., and Sabran, M. (1999) On the theory of partially inbreeding finite populations. VI. The survival probability of a two-locus allele combination when there is partial selfing. *Math. Biosci.* **159**, 97–112.

Piquot, Y., Pratt, D., Valero, M., Cuguen, J., da Laguerie, P. and Vernet, P. (1998) Variation in sexual and asexual reproduction among young and old populations of the perennial macrophyte *Sparganium erectum*. *Oikos* **82**, 139–148.

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

Primack, R.B. and Kang, H. (1989) Measuring fitness and natural selection in wild plant populations. *Ann. Rev. Ecol. Syst.* **20**, 367–396.

Pyšek, P. (1997) Clonality and plant invasions: can a trait make a difference? In H. de Kroon and J. van Groenendael (eds) *Ecology and Evolution of Clonal Plants*. Backhuys Publishers, Leiden, pp. 405–427.

Rajewsky, K. (1993) The power of clonal selection. *Nature* **363**, 208.

Rajewsky, K. (1996) Clonal selection and learning in the antibody system. *Nature* **381**, 751–758.

Sackville Hamilton, N.R., Schmid, B. and Harper, J.L. (1987) Life-history concepts and the population biology of clonal organisms. *Proc. Roy. Soc. Lond. B* **232**, 35–57.

Sarukhán, J. (1974) Studies on plant demography: *Ranunculus repens* L., *R. bulbosus*., and *R. acris* L. II. Reproductive strategies and seed population dynamics. *J. Ecol.* **62**, 151–177.

Schellner, R.A., Newell, S.J. and Solbrig, O.T. (1982) Studies on the population biology of the genus *Viola*. IV. Spatial pattern of ramets and seedlings in three stoloniferous species. *J. Ecol.* **70**, 273–290.

Schmid, B. (1990) Some ecological and evolutionary consequences of modular organization and clonal growth in plants. *Evol. Trend. Plant.* **4**, 25–34.

Service, P.M. and Lenski, R.E. (1982) Aphid genotypes, plant phenotypes, and genetic diversity: A demographic analysis of experimental data. *Evolution* **36**, 1276–1282.

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.

Sigal, L.H. and Ron, Y. (1994) *Immunology and Inflammation: Basic Mechanisms and Clincal Consequences*. McGraw-Hill, Inc., New York.

Silander, J.A. (1985a) The genetic basis of the ecological amplitude of *Spartina patens*. II. Variance and correlation analysis. *Evolution* **39**, 1034–1052.

Silander, J.A. (1985b) Microevolution in clonal plants. In J.B.C. Jackson, L.W. Buss and R.E. Cook (eds) *Population Biology and Evolution of Clonal Organisms*. Yale University Press, New Haven, CT, pp. 107–153.

Silander, J.A. and Antonovics, J. (1979) The genetic basis of the ecological amplitude of *Spartina patens*. I. Morphometric and physiological traits. *Evolution* **33**, 1114–1127.

Starfinger, U. and Stöcklin, J. (1996) I. Seed, pollen, and clonal dispersal and their role in structuring plant populations. *Prog. Bot.* **57**, 336–355.

Strauss, S.Y. (1997) Floral characters link herbivores, pollinators, and plant fitness. *Ecology* **78**, 1640–1645.

Sutherland, S. and Vickery, R.K., Jr. (1988) Trade-offs between sexual and asexual reproduction in the genus *Mimulus*. *Oecologia* **76**, 330–335.

Totland, O. (1999) Effects of temperature on performance and phenotypic selection on plant traits in alpine *Ranunculus acris*. *Oecologia* **120**, 242–251.

Tsarouhas, V., Kenney, W.A. and Zsuffa, L. (2001) Variation in freezing resistance during different phenological stages in some *Populus* and *Salix* clones: implications for clonal selection. *Silvae Genetica* **50**, 54–63.

Tuomi, J. and Vuorisalo, T. (1989a) What are the units of selection in modular organisms? *Oikos* **54**, 227–233.

Tuomi, J. and Vuorisalo, T. (1989b) Hierarchical selection in modular organisms. *Trends Ecol. Evol.* **4**, 209–213.

Via, S. and Shaw, A.J. (1996) Short-term evolution in the size and shape of pea aphids. *Evolution*, **50**, 163–173.

Verburg, R. and Grava, D. (1998) Differences in allocation patterns in clonal and sexual offspring in a woodland pseudo-annual. *Oecologia* **115**, 472–477.

Vuorisalo, T., Tuomi, J., Pedersen, B. and Käär, P. (1997) Hierarchical selection in clonal plants. In H. de Kroon and J. van Groenendael (eds) *The Ecology and Evolution of Clonal Plants*. Backhuys Publishers, Leiden, pp. 243–261.

Westley, L.C. (1993) The effect of inflorescence bud removal on tuber production in *Helianthus tuberosus* L. (Asteraceae). *Ecology* **74**, 2136–2144.

Wikberg, S. (1995) Fitness in clonal plants. *Oikos* **72**, 293–297.

Winkler, E. and Fischer, M. (1999) Two fitness measures for clonal plants and the importance of spatial aspects. *Plant Ecol.* **141**, 191–199.

Zamora, R., Gomez, J.M. and Hodar, J.A. (1998) Fitness responses of a carnivorous plant in contrasting ecological scenarios. *Ecology* **79**, 1630–1644.