Effects of genetic diversity in experimental stands of *Solidago altissima* – evidence for the potential role of pathogens as selective agents in plant populations

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**Summary**

1. Pathogens are assumed to maintain high genetic diversity and the need for sexual reproduction in plants by continually reducing the fitness of genotypes that become dominant in a population. If this were true, a mixed stand should perform better than the average pure stand of the same plant genotypes. To test this hypothesis I deliberately created thirty-two 16-shoot stands of different genetic diversity of *Solidago altissima*, a plant species which in the field can form mono- or polyclonal patches, and allowed natural infestations by the mildew *Erysiphe cichoracearum* to occur.

2. Preliminary experiments revealed significant genetic variation in the source population of *S. altissima* with regard to pathogen level. However, the pattern of this variation changed between years, indicating that different strains of *E. cichoracearum* were dominant at different times.

3. In the main experiment pathogen levels at the beginning tended to be higher in more diverse than in less diverse plant stands. As the growing season progressed, levels of *E. cichoracearum* became higher in the genetically less diverse than in the genetically more diverse stands.

4. Despite the effects on pathogen level, genetic diversity did not significantly influence mean plant performance per stand (height growth and biomass at harvest). However, individual plant performance and pathogen level were positively correlated with each other at low levels of *E. cichoracearum* and negatively correlated at higher levels. Small plants possibly were in a phenological condition of low susceptibility to pathogen attack. The proportion of above-ground biomass allocated to leaves steadily decreased with increasing pathogen level.

5. The present study suggests that small-scale genetic diversity in the perennial plant *Solidago altissima* can influence pathogen levels which in turn may affect plant performance and ultimately fitness. I suggest that selection pressures exerted by plant pathogens may play a major role not only in the maintenance of sexual reproduction but also in the maintenance of lateral clonal growth that leads to a mixing of genotypes in polyclonal patches within plant populations and communities.

**Keywords**: clonality, *Erysiphe cichoracearum*, genetic diversity, plant–pathogen interaction, *Solidago altissima*

**Introduction**

The potential consequences of widespread reduction in the genetic diversity of plants is a major concern to environmentalists. It is assumed that genetic diversity plays an important role for the maintenance of viable populations. One theory predicts that genetically uniform plant populations are susceptible to specialist pathogens which, once they encounter such a population, may lead to its extinction, whereas in genetically variable plant populations some resistant genotypes may already exist or will rapidly be produced by genetic recombination (Adams, Ellingboe & Rossman 1971; Levin 1975; Hamilton 1982;

The predictions are the following: (1) if the plant–pathogen hypothesis were correct, specific resistance and virulence interactions between plants and pathogens should occur; (2) reduced genetic diversity in experimental plant stands should lead to an increased pathogen level; and, (3) plant performance should decrease with increasing pathogen level. According to this sequence, I first assessed variation in pathogen level among genetic families. Next, I analysed the influence of genetic diversity on stand development and disease progress. Then, I determined the relationship between pathogen level and plant size as a performance measure.

Most of the experimental evidence so far available in support of the plant–pathogen hypothesis has come from studies in agroecosystems (Wolfe 1985; Burdon 1987; Thompson & Burdon 1992). However, while disease epidemics in crop plants generally seem to occur more frequently in genetically uniform than in mixed stands, it is still questionable whether this also applies to wild plants (Parker 1988; Alexander 1991).

To assess the ecological importance of genetic diversity in nature it would be best to use a plant species which can produce uniform patches but at the same time has a large genetic variation within populations. This is the typical situation for clonal plants with limited lateral growth, for example the goldenrod Solidago altissima (Weis, Hollenbach & Abrahamson 1987; Schmid et al. 1988; Maddox et al. 1989; Dolt 1991; Meyer 1992; Schmid & Weiner 1993). I used this species in the experiments and allowed pathogen infection, mainly by the mildew Erysiphe cichoracearum, to occur naturally as in the study of Alexander (1991) rather than to manipulating it deliberately (e.g. Jarosz & Levy 1988).

Materials and methods

The tall goldenrod Solidago altissima L. (syn. S. canadensis L. var. scabra Torr. & Gray) (Asteraceae) is a clonal perennial which was introduced from North America and has become naturalized widely in Europe (Vosser 1983; Weber & Schmid 1993). In North America and Europe it colonizes old-field sites and often becomes a dominant during secondary succession (Bazzaz 1968; Bornkamm 1984). In the populations studied no new genets established after the initial colonization of a site by wind-dispersed seeds and the population development exclusively reflected the death or continued clonal growth of existing genets (Hartnett & Bazzaz 1985; Meyer 1992). In the process of clonal growth, above-ground shoots are annually produced from persistent below-ground rhizomes. The spatial structure of shoot populations depends on genet density and lateral spread by rhizomes which range from c. 5–20 cm (Schmid et al. 1988). Often individual genets of 10–20 shoots form monoclonal patches during early population development but later these patches become diffuse as different genets grow more and more into each other (Maddox et al. 1989; Meyer 1992).

In North America S. altissima is attacked by a large number of specialist herbivores which are absent in Europe (Zwölfer 1976). However, in the study region near Basel (Switzerland) the plants are regularly attacked by the mildew Erysiphe cichoracearum D.C. Because this pathogen is a polycyclic fungus it can infect the leaves of the plants repeatedly during one growing season. To analyse the genetic basis of plant resistance to this pathogen I collected seeds and rhizomes from different genets in an invading population of S. altissima in autumn 1987. The field site was an old field abandoned from cultivation in 1983 and all sampled genets of S. altissima were 3 years old and had 3–9 shoots.

Experiments to assess the genetic basis of mildew resistance in the plant population

Rhizome cuttings and seeds of the different genets were propagated in the glasshouse of the Botanical Institute, University of Basel. All rhizome cuttings of each single genet probably represented a clone with a unique genetic identity (based on morphology and quantitative genetic characters; Schmid & Weber, unpublished data) and all seeds of each single genet represented approximately a maternal half-sib family (S. altissima is self-incompatible and shows multiple paternity; Schmid & Weiner 1993). In the following, the term 'parent' will be used to refer to a single genet collected from the field, the term 'clone' or 'clonal replicates' to refer to progeny derived from the rhizome cuttings of a parent, and the term 'half-sib family' to refer to progeny derived from the seeds of a parent (or derived from rhizome cuttings of these seed-derived progenies). When the plants had all reached the rosette stage they were transplanted to an experimental garden which was located 1600 m from the field site in the same river valley and which would have been naturally invaded by S. altissima, had it not been weeded. A total of 24 clones and 24 half-sib families were used in the experiments described here. Together they were derived from 29 parents: 19 parents provided enough of both types of offspring (referred to as 'common families' or 'families'), five parents only provided clones, and five other parents only provided half-sib families.

In a first experiment in 1988 the plants were grown in rectangular plots with 4 × 6 planting positions at 16-cm spacing. The plots contained either
pure sand or enriched garden soil as substrate. In each plot either every clone or every half-sib family was represented by a single-shoot individual. Eight plots (four replicates for each substrate) were used for the clones and 16 plots (eight replicates for each substrate) for the half-sib families (see Schmid & Weiner 1993). For the half-sib families the infestation by *E. cichoracearum* towards the end of the growing season was assessed using a 4-level visual score (1, low; 2, medium; 3, high; 4, very high).

When the plants from the first experiment were harvested, new rhizome cuttings were made from both clones and half-sib families and again propagated to the rosette stage in the glasshouse. In a second experiment in 1989, plants from the 24 clones (but not the half-sib families) were again grown at 16-cm spacing in the rectangular plots containing either pure sand or enriched garden soil. The clones occupied the sixteen plots (eight replicates for each substrate) which had been used for the half-sib families in the previous year; and this time the clones were scored for level of infestation by *E. cichoracearum*. For the 19 common families the clones scored in the 1989 experiment could be viewed as maternal parents of the half-sib families scored in the 1988 experiment. In both experiments all plants were also scored for a large number of morphological characters (Schmid & Weber, unpublished data).

**Experiment to assess the relationship between genetic diversity, pathogen level, and plant performance**

The third experiment, carried out in 1989, was the main experiment and was designed to test for effects of genetic diversity in plant disease resistance within plots on pathogen level and plant growth. Based on the observations in 1988 the four least mildew-resistant and the four most mildew-resistant parents of the 19 common families were identified. The rhizome cuttings from both clones and half-sib families were transplanted to small, quadratic plots in the experimental garden during the second half of May 1989. In each plot 16 plants were grown 16 cm apart in the normal garden soil. There were four levels of genetic diversity and three levels of average mildew resistance (considering a low + high mixture as intermediate level) provided by the following seven treatments:

1a 16 plants from a single clone of a low-resistance parent (4 replicates)
1b 16 plants from a single clone of a high-resistance parent (4 replicates)
2a 16 plants from a single half-sib family of a low-resistance parent (4 replicates)
2b 16 plants from a single half-sib family of a high-resistance parent (4 replicates)
3a 16 plants from half-sib families of four low-resistance parents (4 replicates)
3a 16 plants from half-sib families of four high-resistance parents (4 replicates)
4 16 plants from half-sib families of two low-resistance and two high-resistance parents (8 replicates).

The levels 1–4 of the factor genetic diversity are named ‘Pure clones’, ‘Pure half sibs’, ‘Mixed similar half sibs’, and ‘Mixed different half sibs’ in the figures. To ensure that the same genetic variation within half-sib families was represented at each of the diversity levels 2–4, three cuttings had been prepared from each of sixteen seed-derived plants per family and one cutting per plant was assigned to one plot per level.

The plots with the different treatments were arranged randomly over the free area of the entire experimental garden (Fig. 1). The distance between plots was at least 1 m. In the statistical analysis of the data two blocks were later formed to account for

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**Fig. 1** Map of the experimental garden in 1989. The areas marked with thin borders were used for several experiments with herbaceous plants. The 32 stands of *Solidago altissima* planted for the main experiment of this paper were spread over the remaining experimental area and are indicated by the square plots marked with thick borders. Their different genetic diversities are indicated by shading (see text). Although they had been randomized over the entire garden they were later grouped into two blocks when data were analysed. The identity of an exceptionally mildew-resistant stand is indicated (‘Pure clone from parent no. 8’).
potential differences in soil conditions between the twenty-five plots on the left and the seven plots on the right in Fig. 1.

The following measurements were made on all plants in the main experiment on 9 June, 4 July, 2 August, 11 September, and 9 October 1989: phenological status (rosette-bolting–inflorescence buds–beginning to flower–flowering–flowers wilted–fruiting), height, proportion of leaves with visible infection by *E. cichoracearum* (score from 0–5 corresponding to 0, 0–5, 5–25, 25–50, 50–75 and 75–100%), and average intensity of infection by *E. cichoracearum* on visibly infected leaves (score from 0–4 corresponding to absent [no visibly infected leaves present], low, medium, high, very high). An overall measure for the level of infestation by *E. cichoracearum* was obtained by multiplying the two scores (range 0–20). In addition to these measurements all plants were also investigated for the presence of other pathogens and of aphids on each date. Unidentified fungi were observed on leaves mainly early during the growing season, but no single aphid could be found in the more than 2000 cases where they had been sought. All plants were harvested between 12 and 27 October 1989 and separated into stem, leaves, and reproductive structures (branches with flower heads and subtending small leaves). The dry mass of these components was measured to the nearest 0.01 g.

**Statistical Analysis**

The statistical analyses were carried out with general linear modelling techniques (Neter & Wasserman 1974) using the *GENSTAT* (Payne *et al.* 1987) and JMP® (SAS 1989) computer programmes. Because plots of residuals indicated that the assumptions of homoscedasticity and normality were met, ordinary analysis of variance was used for untransformed characters including pathogen levels. For the nonmetric character ‘phenological status’ ordinal logistic regression was used together with analysis of deviance (McCullagh 1980; McCullagh & Nelder 1989). Plot means were analysed to assess treatment effects. Variation among individual plants was used to assess family effects, treatment-family interactions, and differences among plots. For characters which were measured several times during the growing season the univariate analyses were followed by repeated-measures analyses. In this case the degrees of freedom for the within-subjects mean squares were corrected with the Huynh-Feldt epsilon factor (Elashoff 1986). Throughout this paper analysis of variance is viewed as an exploratory tool and significance levels are given as a guide to interpretation of results rather than an absolute level of error probability.

**Results**

**The Genetic Basis of Mildey Resistance in the Plant Population**

In the first two experiments in the rectangular plots, levels of *E. cichoracearum* were higher on plants growing in soil than in sand (*P < 0.01*) and varied significantly among plots (*P < 0.001*). Differences in pathogen level within plots among half-sib families in 1988 (*P < 0.01*) and among clones in 1989 (*P < 0.001*) reflected broad-sense heritabilities (measured across the two soil environments) of 27.1% and 32.0%, respectively. However, because there was a strong family–year interaction (*P < 0.001*), the mean pathogen levels of the 19 common families were not significantly correlated between years, except that the half-sib family and the clonal replicates from parent 8 stood out by their high resistance (Fig. 2).

**Effects of Genetic Diversity on Stand Development and Pathogen Level**

As a consequence of the poor correlation between the mean resistance of families from year to year, the selection of families representing low and high resistance in 1988 was no longer valid in 1989. Therefore the effect of resistance level was never significant in the main experiment and was excluded as a treatment factor in all final analyses. The plot with the clonal replicates from parent 8 in the main (third) experiment was exceptional with regard to pathogen level;
Table 1 Repeated-measures analyses of variance for plot means of plant height in cm (a) and for plot means of level of *Erysiphe cichoracearum* (b) (see Fig. 3). The sum of squares for each line was calculated after accounting for the influence of all other lines (so-called Type-III sum of squares; SAS 1989)

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Degrees of freedom</th>
<th>Unadjusted sum of squares</th>
<th>Adjusted sum of squares</th>
<th>F</th>
<th>P†</th>
</tr>
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<tbody>
<tr>
<td>(a) Plant height</td>
<td></td>
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<tr>
<td>Block</td>
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<td></td>
<td></td>
<td></td>
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<td>Time</td>
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<td>115327</td>
<td>204.982</td>
<td>0.000</td>
</tr>
<tr>
<td>Block x time</td>
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<td>1.3</td>
<td>502</td>
<td>0.893</td>
<td>0.351</td>
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<tr>
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<td>1.256</td>
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<tr>
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<td>35.3</td>
<td>14909</td>
<td></td>
<td></td>
</tr>
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<td>(b) Level of <em>E. cichoracearum</em></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td>0.340</td>
<td>0.565</td>
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<td>Genetic diversity</td>
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<td>0.717</td>
<td>0.551</td>
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<td></td>
</tr>
<tr>
<td>Time</td>
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<td>3.0</td>
<td>1030.0</td>
<td>38.180</td>
<td>0.000</td>
</tr>
<tr>
<td>Block x time</td>
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<td>38.9</td>
<td>1.443</td>
<td>0.237</td>
</tr>
<tr>
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<td>194.6</td>
<td>2.405</td>
<td>0.022§</td>
</tr>
<tr>
<td>Plot x time residual†</td>
<td>106</td>
<td>79.0</td>
<td>714.8</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Error term for preceding lines.
† Using Huynh–Feldt degree-of-freedom adjustment for the lines ‘Time’, ‘Block x time’, and ‘Genetic diversity x time’ (see Materials and methods).
‡ Including the nonsignificant (P > 0.2) block–genetic-diversity–time interaction.
§ P = 0.003 if clonal replicates from parent 8 are omitted (see text).

it was almost never attacked by *E. cichoracearum*. Means and significance levels were therefore also calculated excluding this plot to assess the influence of the outlier.

In the main experiment there was considerable variation among plots within treatments for the measured plant characters (plot effect usually highly significant if tested against residual variation among individuals). Differences in genetic diversity did not significantly affect the phenological development and height growth of the stands (Table 1a, Fig. 3a). Pathogen levels initially tended to be higher in stands of greater diversity but later this trend was reversed (Fig. 3b). Because of this reversal the main effect of genetic diversity averaged over time was not significant (second line in Table 1b). Instead, there was a significant diversity–time interaction (seventh line in Table 1b). On the fifth observation date both the score for the proportion of leaves with visible infection by *E. cichoracearum* and the level of *E. cichoracearum* showed significant decreases with genetic diversity in univariate analyses if the plot with the clonal replicates from parent 8 was excluded (*P* < 0.01), but not if it was included (*P* > 0.1). When these decreases were tested by monotonic regression (calculated from one-way analyses of variance of plot means using the programme of Gaines & Rice 1990) with the order restriction 1 > 2 > 3 > 4 for the four levels of the factor genetic diversity, a significance level of 7% was reached for both measures even if the plot with the clonal replicates from parent 8 was included and a very high significance resulted if this plot was excluded (*P* < 0.001).

![Fig. 3](image-url) Plant height (a) and pathogen level (b) (calculated by multiplying score for proportion of leaves infected with score for average intensity of infection) of *Solidago altissima* in experimental stands of different genetic diversity (see text) during the main experiment carried out in 1989. The line for 'Pure clones' in (b) was calculated excluding the extreme stand with clonal replicates from parent 8; this stand is represented by the white squares along the x-axis.
Large variations in pathogen levels among plots within treatments \( P < 0.001 \) for plot effect and for plot–time interaction if tested against residual variation among individuals) were only in part due to environmental heterogeneity. They also included genetic variation among families because, for the two low-diversity treatments, ‘Plot’ obviously had to be confounded with ‘Family’ (differences among the eight plots that each contained a different clone or among the eight plots that each contained a different half-sib family). Soil conditions and the spatial arrangement of the plots over the experimental area might have been responsible for environmental heterogeneity among plots within treatments. During the early phase of the experiment (4 July) the level of *E. cichoracearum* was highest in the south-eastern part of the experimental garden (Fig. 4a). However, from 11 September to 9 October, when the infestation by *E. cichoracearum* reached a second peak (Fig. 3b), these plots were less affected than the plots in the other parts of the experimental garden (Fig. 4b). Pathogen levels then tended to be higher in the western, wind-exposed plots. Within plots, border plants from the western edge were consistently more attacked by *E. cichoracearum* than were plants from the eastern edge and centre plants were least attacked \( P < 0.05 \). This was also true for the rectangular plots in the first and second experiment.
The relationship between pathogen level and plant performance

On the second observation date in the main experiment, plants with a high level of *E. cichoracearum* were phenologically less advanced than plants with low levels of *E. cichoracearum* (*P* < 0.01). This relationship was reversed towards the end of the experiment when phenologically more advanced plants had a higher level of *E. cichoracearum* than phenologically less advanced plants (*P* < 0.001). Both plant height towards the end of the experiment and above-ground biomass were strongly positively correlated with the level of *E. cichoracearum* up to a score of 5 and weakly but significantly negatively correlated with the level of *E. cichoracearum* above this score (Table 2). Therefore, second-degree polynomial regressions were fitted for each family (Fig. 6). Significant ‘quadratic-term–family’ interactions indicated that the relationships between plant height and pathogen level (*P* < 0.05) and between above-ground

Genetic diversity of stand

Fig. 5 Pathogen-level ‘reaction norms’ of eight families of *Solidago altissima* over four levels of increasing genetic diversity of stands (1 = Pure clones, 2 = Pure half sibs, 3 = Mixed similar half sibs, 4 = Mixed different half sibs). (a) and (b) represent the results for an early and late observation date during the main experiment carried out in 1989 (pathogen level by mistake was not recorded for one pure clone on 9 October). Note that for the two low-diversity treatments the lines connect means obtained from 16 plants growing in a single plot each whereas for the two high-diversity treatments the lines connect means obtained from 16 plants growing in four plots each. The mean of the exceptionally mildew-resistant stand indicated 'Pure clone from parent no. 8' in (b) is connected to the remaining stands of this family by a broken line in both (a) and (b).

zontal rather than ascending reaction norms (*P* < 0.001 for diversity–family interaction, with or without plot with clonal replicates from parent 8). Towards the end of the experiment, with the exception of clonal replicates from parent 8, all families had higher levels of *E. cichoracearum* in pure stands than in mixtures, i.e. the reaction norms were more or less parallel (Fig. 5b; *P* < 0.001 or *P* > 0.1 for diversity–family interaction, respectively, with or without plot with clonal replicates from parent 8).
Table 2  Direction, size ($r^2$), and significance ($P$) of correlations between levels of *Erysiphe cichoracearum* and two measures of plant performance (cf. Fig. 6) for two ranges of pathogen levels

<table>
<thead>
<tr>
<th>Range of pathogen levels</th>
<th>Direction of correlation</th>
<th>$r^2$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low (score 0-5)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height on 9 October</td>
<td>positive</td>
<td>0.395</td>
<td>0.000</td>
</tr>
<tr>
<td>Above-ground biomass</td>
<td>positive</td>
<td>0.328</td>
<td>0.000</td>
</tr>
<tr>
<td>High (score 5-20)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height on 9 October</td>
<td>negative</td>
<td>0.017</td>
<td>0.025</td>
</tr>
<tr>
<td>Above-ground biomass</td>
<td>negative</td>
<td>0.031</td>
<td>0.004</td>
</tr>
</tbody>
</table>

biomass and pathogen level ($P < 0.01$) differed among families. For the first relationship, however, the significant interaction was only due to the clonal replicates from parent 8; these small plants were not attacked by *E. cichoracearum*. Leaf biomass at harvest did not increase as strongly as above-ground biomass with increasing low levels of *E. cichoracearum* and the proportion of above-ground biomass allocated to leaves, i.e. relative leaf biomass, was negatively related to level of *E. cichoracearum* over the entire range of scores ($P < 0.001$).

There were no indications in the first two experiments in rectangular plots of linear or nonlinear relationships between family means of plant height or biomass and family means of level of *E. cichoracearum* [genetic correlations in the sense of Via (1984) not significantly different from 0]. Similarly, no such relationships between family means were found in the main experiment.

**Discussion**

**GENETIC DIVERSITY AND PATHOGEN LEVEL**

A prerequisite for the plant–pathogen hypothesis is that there is genetic variation among plant families in resistance against particular pathogen strains and among pathogen strains in virulence against particular plant families (see prediction 1 in the Introduction). I found significant genetic variation in the study population of *S. altissima* with regard to level of *E. cichoracearum*. Because the pattern of this variation changed over time, i.e. resistance levels were not genetically correlated between years (Fig. 2), it is likely that different strains of *E. cichoracearum* were dominant at different times, indicating that the pathogen population was also genetically variable.

In contrast to the prediction that average pathogen levels should have been lower in the more diverse than in the less diverse stands of *S. altissima*, diverse stands initially tended to be more strongly attacked by *E. cichoracearum* and other, unidentified fungi in the experiments (Fig. 3b). This could mean that pathogens had already infected some genetic families before the start of the experiment and that these families then acted as sources for secondary infections (Burdon 1987). However, even if there were no infections to start with it is conceivable that it might sometimes be a disadvantage to be next to a susceptible plant and this is more likely in mixtures of genotypes than in genetically uniform stands.

As the growing season progressed, levels of *E. cichoracearum* became higher in the genetically less diverse than in the genetically more diverse stands. Near the end of the growing season plants of all genetic families except one (number 8) had lower pathogen levels if they were grown with a majority of plants from other families (Fig. 5b). This result is in agreement with the prediction from the plant–pathogen hypothesis (prediction 2 in the Introduction). Nevertheless, the result is remarkable, first because to my knowledge it has not been observed previously in experimental stands of wild plants (e.g. Alexander

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**Fig. 6** The influence of the level of *Erysiphe cichoracearum* on plant height (a) and above-ground dry mass (b) in the eight families of *Solidago altissima* at the end of the main experiment carried out in 1989. Curves are second-degree polynomials ($P < 0.001$ that linear and quadratic regression coefficients = 0 in overall model).
1991) and second because the spatial scale at which genetic diversity occurred was small (16 plants plot"). The dispersal of the pathogen, which could have been blown in from the west by wind, within the experimental area must have been very localized. Even within plots plants at the western edge were most and plants in the centre were least attacked by *E. cichoracearum*.

The result that all genetic families except one were less attacked in more diverse than in less diverse stands would not have been expected with a genetically uniform pathogen population unless the one genetic family (number 8) occurred in most mixed stands (which was not the case) and 'protected' the other families in these stands. Therefore this result can be viewed as a further indication that the pathogen population was genetically diverse. The exceptional family derived from parent 8 was special because in the main experiment its clonal replicates growing in a single plot were almost never attacked by *E. cichoracearum* (the half-sib offspring were more often attacked).

**GENETIC DIVERSITY, PATHOGEN LEVEL, AND PLANT PERFORMANCE**

Because pathogen levels were initially higher and later lower in the genetically diverse than in the genetically homogeneous stands it is perhaps not surprising that no effects of genetic diversity on mean final plant size per plot were observed when the experiments were terminated (cf. prediction 3 in the Introduction). However, the level of *E. cichoracearum* did show a significant relationship with the size of individual plants. Two biological processes may have been involved in this relationship: at low levels, plant size and level of *E. cichoracearum* were strongly positively correlated whereas, at higher levels, the correlation was significantly negative (Table 2). This indicated that except for small plants or low pathogen levels the pathogen did have a negative influence on plant performance. Further potential evidence for such a negative effect was that the relative biomass of leaves (proportion of above-ground biomass allocated to leaves) decreased over the entire range of pathogen levels, i.e. plants with high pathogen levels had a lower absolute leaf biomass than plants with low pathogen levels at the same above-ground biomass. Because the perennating below-ground parts of the plants were not harvested in the main experiment, nothing can be said about the potential longer-term influences of high pathogen levels (a slightly negative correlation between pathogen level and number of rhizomes produced in the 1988 experiment was not significant).

The positive correlation between plant size and the level of *E. cichoracearum* at small sizes or low levels could be interpreted in several ways. Smaller plants may have been later 'detected', unable to support large pathogen populations, or in a phenological condition of low susceptibility. Towards the end of the main experiment there was a strong relationship between phenological status and level of *E. cichoracearum*, suggesting that the pathogen might have grown better on older, senescing tissue than on younger tissue. Indeed, the plot with the exceptionally mildew-resistant clonal replicates of parent 8 stood out visually by its fresh-green appearance and low canopy height. If resistance were to be paid for by slow development and small size it would appear to be very costly and an imaginary scenario of directional selection in favour of the resistant clone unlikely. However the trend, indicated by the exceptional family, of a genetic trade-off between pathogen level and plant size was not significant when the means of all families were correlated [genetic correlations in the sense of Via (1984)]. The evidence in the literature for fitness costs of plant resistance to pathogens seems to be relatively weak (Burdon 1987; De Nooij & Van Damme 1988; Parker 1990; Ennos & Swales 1991; Marquis & Alexander 1992).

**CLONALITY VS. SEX AS MECHANISMS FOR PLANTS TO ESCAPE PATHOGENS**

The present study yields the general conclusion that small-scale genetic diversity in the perennial plant *Solidago altissima* can influence pathogen levels which in turn may affect plant performance and ultimately fitness. A coevolutionary scenario between the plant and its pathogens may therefore explain the existence of large genetic variation in this species.

How specific is the reciprocal adaptation between plant and pathogen? Would pathogens isolated from different host individuals grow better on their 'home' plant genotypes than on 'away' plant genotypes? Although I have not yet tested this, the results of the garden experiments indicate that much variation in pathogen level can be related to systematic or stochastic environmental effects (differences among plants grown in different soil types in the first two experiments or grown in different plots in all experiments). This, together with the large variation between seasons and years, suggests that reciprocal adaptation at the local scale is not tight and that only 'diffuse' coevolution between plant genetic families and pathogen strains might be going on in this system. It is conceivable that selection by heterogeneous environments was more important than frequency-dependent selection. The importance of stochastic factors in plant–pathogen interactions has recently been stressed by Burdon, Jarosz & Kirby (1989) and by Thompson & Burdon (1992).

Given that the genetic variation in the plant population is at least in part a response to a rapidly evolving or genetically variable pathogen population, the crucial question is what the mechanism of this response could be. Because the investigated plant
species *S. altissima* has the ability to form clones of some tens or hundreds of shoots (Werner, Bradbury & Gross 1980; Hartnett 1983), as the clones grow in size and age more of them may be detected by specific pathogens. If this were the case it might be argued, in analogy to a similar situation in ageing trees, that the only escape for the plants would be sexual reproduction (Hamilton 1980; Ladle 1992; Stearns 1992). However, in a clonal plant, it may be difficult for a nonsystemic pathogen to detect all the shoots of an individual clone, especially if they are laterally spread out, lose their physical connections (see Cook 1979), and intermingle with other clones to form locally diverse patches similar to those that have been simulated in the present study. In view of the spatial stochasticity mentioned above and results of new spatial models of interacting populations (Hassell, Comins & May 1991), this mechanism to escape pathogen detection by clonal growth seems to be a conceivable alternative to the mechanism using sexual reproduction (see Parker 1988; Schmid 1990). There exists indeed good evidence that intermingling among genets in *S. altissima* increases with time during old-field succession (Maddox et al. 1989) and that individual clones produce increasingly fewer but longer rhizomes as they age (Meyer 1992). It should be rewarding to theoretically explore whether selection pressures exerted from plant pathogens not only may play a major role in the maintenance of sexual reproduction but also in the maintenance of lateral clonal growth in plants.

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