

Genetic Control of Downy Mildew Resistance in Cucumber - A Review

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The oomycete pathogen *Pseudoperonospora cubensis* (Berk. and Curt.) Rostow. is a major foliar disease of cucumber (*Cucumis sativus* L.), especially in humid regions of the world (Palti and Cohen, 1980). Downy mildew was first described on cucumber by Berkeley and Curtis in 1868 and investigation into the genetic basis of resistance began in the early 20th century. Understanding the inheritance of downy mildew is fundamental to successful cucumber breeding programs. The objective of this article was to review the current knowledge of the inheritance of downy mildew resistance in cucumber.

Literature review

Early disease screening efforts at the Puerto Rico Agriculture Experiment Station focused on transferring resistance identified in a Chinese cultigen (Roque, 1937) into adapted varieties. The new Chinese cultivar introduced in 1933 was highly resistant, but had long and curved fruit that were not commercially usable. This cultivar was crossed with elite cultivars to combine the resistance with good horticultural traits. This eventually led to seven highly resistant lines having good characteristics. Of these, Puerto Rico selections 37, 39, and 40 were found to have resistance and good fruit quality, as well as yield superior to commercial cultivars used as checks.

Cochran (1937) used the Indian cultivar 'Bangalore' as a source of downy mildew resistance for crosses with popular slicing and pickling cultivars of the time. Cochran (1937) had some success with crosses to 'Bangalore' but did not determine the inheritance of resistance.

Jenkins (1946) used P.R. 37 as a resistant parent in studies of downy mildew in Minnesota as part of his Ph.D. dissertation research. He did not attempt to describe the inheritance of downy mildew resistance except to say that it was probably due to a number of factors. Part of his research involved a study of the correlation between physical traits and disease resistance. Of the traits observed (spine color, fruit color, fruit netting, spine texture, and growth habit) only growth habit appeared to have any relation to resistance. Jenkins suggested that determinate plants were more susceptible to

downy mildew than indeterminate plants. Barnes and Epps (1950) observed that even resistant plants became more susceptible to infection when fruit began to approach maturity. Determinate plants have concentrated fruit set, so that may explain their increased susceptibility to downy mildew.

'Palmetto', a cross between P.R. 40 and 'Cubit', was released in 1948 as a highly resistant slicing cucumber (Barnes, 1948). Resistance was attributed to two primary factors; high resistance to initial infection, exhibited by very few lesions, and limited sporulation resulting in decreased secondary infection. These resistance mechanisms were thought to be controlled by several genes. Limited acreage of 'Palmetto' was planted in 1948 and 1949. In those years, downy mildew was only found on 'Palmetto' when it was planted adjacent to susceptible cultivars, such as 'Marketer'. In 1950 and 1951, all 'Palmetto' fields inspected showed infection, regardless of proximity to 'Marketer' or other susceptible cultivars. In those years, the lesions were large and sporulated heavily, typical of lesions of susceptible cultivars (Epps and Barnes, 1952). Because it is unlikely that the change from 1948 to 1950 was due to a change in resistance, the change likely was in the pathogen population, either through mutation, selection in a mixed population, or migration of a race from a different region.

A new type of resistance was described by Barnes and Epps (1954) that was found in *Cucumis sativus* PI 197087 from India. PI 197087 was previously reported as having some resistance to downy mildew, as well as immunity to anthracnose (Barnes and Epps, 1952). The reaction of PI 197087 to downy mildew infection was characterized by small irregularly shaped, brown lesions, with a slight water-soaked appearance, becoming necrotic with sparse sporulation (Barnes and Epps, 1954), resembling a classic hypersensitive response (HR). Resistance from PI 197087 was used in the development of new cultivars, and the resistance in many current cultivars traces to PI 197087 (Wehner and Shetty, 1997). That resistance was effective for growers to produce cucumbers in warm humid regions of the U.S. without fungicides for over 40 years, but has not been as effective since a major epidemic in 2004.

Several studies have dealt with the inheritance of

downy mildew resistance in cucumber. Shimizu et al. (1963) reported that resistance in 'Aojihai' was controlled by three recessive genes (proposed s_1 , s_2 and s_3). Pershin et al. (1988), using cultivar 'Sadao Rischu', determined resistance to be controlled by at least three major genes exhibiting partial dominance that were linked to at least three genes for powdery mildew resistance.

Van Vliet and Meysing (1974) concluded that downy mildew resistance from 'Poinsett', probably originating from PI 197087, was controlled by a single recessive gene that they named p . In addition, they proposed that the downy mildew gene was linked with the genes for powdery mildew resistance and for dull green fruit color (D). In a following study, Van Vliet and Meysing (1977) confirmed that the gene for hypocotyl resistance to powdery mildew was linked with or identical to the gene for resistance to downy mildew. They also concluded that the resistance found in 'Poinsett', 'Ashley', 'Taipei', 'Natsufushinari', PI 179676, and PI 234517 was controlled by the same gene. However, they stated that downy mildew resistance in 'Ashley' resulted from PI 197087, whereas resistance actually traced back to P.R. 40 (Barnes and Epps, 1956). This would explain why 'Poinsett' was reported to be more resistant than 'Ashley' and suggests that its resistance is due to a different gene.

Fanourakis and Simon (1987) reported agreement with Van Vliet and Meysing (1974) confirming that downy mildew resistance is controlled by a single recessive gene. They also reported loose linkage with powdery mildew resistance (pm) and compact plant (cp) genes. They reported a discrepancy in their results for one F_2 and one backcross family which did not fit the single-gene hypothesis. They attributed this to difficulty in rating resistance based on phenotypic expression at the cotyledon stage. No linkage with dull green fruit (D) was found, but there was deviation from expected results in one F_2 family.

El-Hafaz et al. (1990) report that the cultivars 'Palmetto' and 'Yomaki' were resistant in Egypt. They concluded that resistance was the result of an epistatic interaction between a dominant susceptible gene and a recessive resistance gene. Badr and Mohamed (1998) also determined that resistance was controlled by a pair of dominant and recessive interacting genes. Angelov (1994) reported that resistance in PI 197088 was due to two recessive genes and that 'Poinsett' resistance was inherited as a single recessive gene. PI 197088 was collected from the same region and at the same time as PI 197087.

Doruchowski and Lakowska-Ryk (1992) had evidence that downy mildew resistance was controlled by three recessive genes ($dm-1$, $dm-2$ and $dm-3$), where $dm-3$ and either $dm-1$ or $dm-2$ had to be homozygous recessive

for maximum resistance. However, there was discrepancy in the F_2 results, which did not agree with their model. They argue that this resulted from testing too narrow a population. The three genes were included in the previous cucumber gene list (Pierce and Wehner, 1990), but should probably be removed as none of the genes were identified and no type lines are available to use in studies of separate genes. Petrov et al. (2000) claimed that the resistance in J-13, which was derived from Wisconsin 2843 (resistance originally from PI 197087 according to Peterson et al., 1985) was not inherited in a clear manner, but suggested it was due to one or two incompletely dominant genes.

PI 197088 was recently described as highly resistant to downy mildew in a large germplasm screening study and a multiple year re-evaluation of the most resistant and susceptible cultigens conducted at North Carolina State University (Criswell, 2008; Call, 2010). It appears that there are at least three genes for resistance to downy mildew in cucumber: one from the Chinese cultivar used in developing the PR lines, one from PI 197087, and one from PI 197088 (assuming that PI 197087 and PI 197088 share one resistance gene, $dm-1$). Inheritance studies are currently being conducted to determine if these genes are allelic.

Discussion

There are several proposed inheritance patterns for resistance to downy mildew. They range from three recessive genes (Doruchowski and Lakowska-Ryk, 1992; Shimizu et al., 1963) to three partially dominant genes (Pershin et al., 1988) to an interaction between dominant susceptible and recessive resistance genes (Badr and Mohamed, 1998; El-Hafaz et al., 1990) to one or two incompletely dominant genes (Petrov et al., 2000) to a single recessive gene (Angelov, 1994; Fanourakis and Simon, 1987; Van Vliet and Meysing, 1974; 1976). Conflicting results regarding the inheritance of downy mildew resistance in cucumber is likely due to four main factors.

First, the pathogen is highly variable and populations have not been well studied for the factors causing virulence (Lebeda and Urban, 2004). Multiple pathotypes and races have been identified (Lebeda and Widrechner, 2003). In some cases, more than one pathotype in a geographical region has been identified (Lebeda and Urban, 2004). Different races have been reported (Angelov et al., 2000; Epps and Barnes, 1952; Hughes and Van Haltern, 1952; Shetty et al., 2002) and there may be different genes involved in resistance to different races. Call and Wehner (2010) noted a change in rank of resistant and moderate cultigens from screening studies before

and after a change in the pathogen population. Cultigens highly resistant in 1988 and 1989 were only moderately resistant in studies conducted from 2005 to 2009. Those cultigens identified as highly resistant in the most recent studies were only moderately resistant in 1988 and 1999.

Second, differences in the environment, including temperature, humidity, rainfall and inoculum movement by wind all influence the severity of downy mildew infection (Cohen, 1977). Interactions between pathogen, host and environment are complex and not easily determined. Greenhouse tests are important for reducing environmental variability and should be conducted in addition to field tests. High variability in pathogen-host interactions due to environment can cause simply inherited traits to appear polygenic. This may be misleading and continuous variation with no clear segregation, even in homozygous inbred lines, can also indicate low heritability (Shaner, 1991). Horejsi et al. (2000) measured a low broad-sense heritability for downy mildew resistance, and noted large plant-to-plant variability in their study.

Third, different mechanisms of resistance have been studied (Angelov and Krasteva, 2000; Baines, 1991; Barnes and Epps, 1950; 1954; Palti and Cohen, 1980; Tarakanov et al., 1988). The previously mentioned inheritance studies used different mechanisms of resistance when evaluating plant response. Doruchowski and Lakowska-Ryk (1992) used necrotic lesions; Van Vliet and Meysing (1974; 1977) and El Hafaz et al. (1990) used sporulation intensity; Fanourakis and Simon (1987) used incidence of chlorotic and necrotic lesions on cotyledons; and Petrov et al. (2000) used chlorotic lesions for rating resistance. Other studies did not specify how resistance was measured. Different mechanisms of resistance may have different inheritance patterns.

Fourth, the original source of resistance varies over downy mildew inheritance studies. Some studies evaluated resistance sources from PI 197087 (India) while other studies evaluated resistance from P.R. 40 (China) and other germplasm sources. There are at least three genes for resistance to downy mildew, coming from P.R. 40, PI 197087, and PI 197088. Although, P.R. 40 is not available in the germplasm collection, cultivars tracing resistance to P.R. 40 are. Those include 'Ashley' and Ames 4833. The combination of the two different sources should provide either better resistance or more durable resistance. This combination can be found in PI 234517 (SC-50), which does have slightly higher resistance to downy mildew than 'Ashley' or PI 197087. However, the difference between PI 234517 and cultivars having resistance from PI 197087 alone was not significant (Wehner and Shetty, 1997). This is not surprising as

this resistance was overcome in 1950 in the cultivar 'Palmetto'.

Conclusions

There is evidence that PI 197087 currently in the germplasm collection has lost resistance to downy mildew. Barnes and Epps (1954) described the phenotypic response of PI 197087 as having small necrotic lesions with no chlorosis. Yet, Van Vliet and Meysing (1974) reported resistance in 'Poinsett' as necrotic spots on cotyledons with small chlorotic spots on true leaves. Shetty et al. (2002) described resistance as small necrotic or chlorotic spots, Chipper and Poinsett 76 both exhibited these symptoms. Wehner and Shetty (1997) state that PI 197087 showed only intermediate resistance, with chlorotic and necrotic spots. On the other hand, 'Chipper', Gy 4, M 21, 'Poinsett 76', 'Pixie', and 'Polaris' were all more resistant than PI 197087, even though their resistance was derived from PI 197087. Angelov (1994) reported that 'Poinsett' had only moderate resistance, with chlorotic lesions present on the leaves. Petrov et al. (2000) reported that resistance in WI 2843 (from PI 197087) was expressed as small chlorotic lesions. However, in their study 'Poinsett' showed small necrotic lesions. The loss of the typical resistance response phenotype in PI 197087 may be due to a combination of downy mildew overcoming the hypersensitive response described by Barnes and Epps (1954) along with genetic drift in the PI collection.

There probably are multiple genes for downy mildew resistance, each with a distinct inheritance pattern. However, the cucumber gene list should be corrected by removal of *dm-1*, *dm-2* and *dm-3* genes, while keeping the *dm* gene from PI 197087. Additional genes, for example from P.R. 40, could be included when their effects have been isolated and type lines identified.

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