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Molecular Ecology

Defense for vigor: a fitness trade-off for plant survival

Plants adapt to changing environments by optimizing the fitness costs associated with key biological functions such as pathogen defense or growth. A comparison of the laboratory strain and other Australian wild accessions of *Nicotiana benthamiana* reveals that trading-off viral defense for vigor confers an adaptive advantage for those individuals living in arid habitats.

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Nicotiana benthamiana, an herbaceous species endemic to Australia, is extensively used in plant research due to its singular and enigmatic hypersusceptibility to viruses (Fig. 1). Writing in Nature Plants Bally *et al.*[1] analyze in exquisite detail the origin, diversity, evolution and ecology of wild *N. benthamiana* accessions, which unequivocally associates the viral hypersensitivity of the laboratory (Lab) strain with a 72-bp DNA insertion in the *RNA-DEPENDENT RNA POLYMERASE1* (*RDR1*) gene involved in antiviral defense. Remarkably, Lab originally from the Northern Territory of Australia and SA from South Australia, the only two wild accessions containing the *RDR1* insertion and being hypersusceptible to viruses, grow in similar desert areas of Central Australia, and have higher vigor and shorter life cycles than other insert-free accessions occupying less extreme habitats. This particular defense for vigor trade-off provides an adaptive advantage for the extremophile accessions to survive in an arid territory where vigor rather than pathogen resistance is critical for survival.

It has been documented that the discovery and first collection of *N. benthamiana* in New Holland (Australia) dates approximately from 1837-1843, on the third voyage of the HMS *Beagle* ship[2]. However, records on how *N. benthamiana* accessions met the research community are rather scant. In a recent effort to determine the provenance of the most used laboratory strains of *N. benthamiana*, Gooding *et al*. concluded that all laboratory strains actually derive from a single accession containing the *RDR1* mutation[2]. Bally *et al.*[1] disclosed the records from the University Libraries of Adelaide and California which describe that an accession of *N. benthamiana* was collected by Prof. Cleland in Central Australia who subsequently sent it to Prof. Goodspeed in California in 1939, suggesting that this was probably the primary accession later dispersed around the laboratories all over the world.

But how and when did the widely used Lab isolate acquire the *RDR1* insertion? Bally *et al.´*s analysis of the *N. benthamiana* Lab genome[3] identified the 72-bp sequence not only in the *RDR1* gene but also in a 100 kb untranscribed non-coding region that seems to be the parental sequence. Phylogenetic and molecular

clock analyses of the *RDR1* sequences of different wild accessions collected throughout Australia estimate that the Lab and SA accessions form a sub-clade that diverged from the insert-free lineages about 880 thousand years ago (Kya). As the separation between SA and Lab dates from 710 Kya, this implies that the insertion occurred between 880 and 710 Kya.

Why was this insertion retained in the accessions from desert areas? Bally *et al.* provide an elegant answer to this intriguing question. As a widely distributed species, *N. benthamiana* exhibits extensive phenotypic variations on plant architecture, leaf shape, floral structure and seed size across different geographic populations. The insert-free accessions living in non-desert areas are less susceptible to virus. Moreover, they have lower growth rates and produce larger flowers facilitating insect attraction and cross-pollination. These phenotypes are well consistent with the fact that in non-desert areas there are abundant insect pollinators and viral vectors which favor the maintenance of *RDR1* functionality conferring virus resistance, demonstrating that hosts under pathogen pressure allocate more resources to defense which might have been dedicated to growth and survival. In contrast, Lab and SA accessions have higher growth rates and floral structures that favor self-fertilization, with small corolla tubes and with anthers and stigmas in close-proximity. This limits the chances of incorporation of functional *RDR1* through outcrossing with other insert-free accessions. Therefore, it appears that the *RDR1* insertion and its related phenotypes, such as enhanced vigor, fast seed setting and self-fertilization, confer a survival advantage in arid Central Australia, where the limited rainfall not only reduces insect pollinators and viral vectors but also demands that plants finish life cycle in a short time .

The suggestion that the 72-bp loss-of-function *RDR1* insertion causes the virus hypersensitivity in *N. benthamiana* was reported more than a decade ago. Also, it was shown that transgenic *N. benthamiana* expressing insert-free *RDR1* from *Medicago truncatula* exhibits enhanced resistance to several tobamoviruses [4]. More recently, it was found that expressing functional *RDR1* from *Nicotiana tabacum* in *N. benthamiana* enhanced viral infection by suppressing the RDR6-mediate antiviral silencing pathway[5]. This observation led the authors to propose that the dysfunctional *NbRDR1* might have arisen as a consequence of the strong selection pressure favoring RDR6-mediated antiviral defense due to *N. benthamiana´*s hypersensitivity to multiple viruses[5]. Now, using transgenic experiments Bally *et al*. unambiguously show that the disruptive insertion in *NbRDR1* simultaneously increases virus susceptibility and accelerates growth rate and seed setting. Furthermore, they found the dysfunctional *NbRDR1* most likely results from a fortuitous DNA insertion which was maintained as an adaptive response conferring higher fitness in a particularly extreme and “low-virus” habitat.

To conclude, Bally *et al.´*s work provides a fascinating illustration of how defense costs, despite providing substantial fitness under pathogen pressure, can actually be quite high in terms of reduced growth and seed setting. This conclusion constitutes a serious warning about the risk of breeding obsessively for crop yield, which can lead to the generation of top-performing cultivars with weakened immunity. In this context, a real agricultural challenge in the near future would be to find, for each crop, those cultivars presenting the optimal balance between defense and vigor costs to ensure both high and durable yields.

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Figure 1 The *Nicotiana benthamiana* strain used in laboratories has traded antiviral defense for early vigor to survive to the extreme habitat of central Australia, where it originates. Images of a plant infected with a virus tagged with the Green Fluorescent Protein (right) displaying typical viral symptoms compared to a non-infected plant (left). Images were taken under visible (up) or UV (down) light.