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MOLECULAR ECOLOGY

Trading defence for vigour

Plants adapt to changing environments by optimizing the fitness costs associated with key biological functions. A comparison of a laboratory strain with other wild Australian accessions of *Nicotiana benthamiana* reveals that trading viral defence for vigour confers an adaptive advantage in arid habitats.

Alberto Carbonell

The herbaceous species *Nicotiana benthamiana*, endemic to Australia, is extensively used in plant research due to its unusual and enigmatic hypersusceptibility to viruses (Fig. 1). Writing in *Nature Plants*, Bally *et al.*[1] analyse in 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 defence. Remarkably, the LAB strain, originally from the Northern Territory of Australia, and the accession SA, from South Australia, are the only wild accessions containing the *RDR1* insertion and the only accessions hypersusceptible to viruses. Both grow in similar desert areas of Central Australia, and have higher vigour and shorter life cycles than other insert-free accessions occupying less extreme habitats. This trade-off of defence for vigour provides an adaptive advantage for the extremophile accessions whose arid territory prioritizes vigour over pathogen resistance for survival.

The discovery and first collection of *N. benthamiana* in Australia (then known as New Holland) dates from approximately 1837–1843, on the third voyage of HMS *Beagle*[2]. However, records on how *N. benthamiana* accessions were collected 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* insertion[2]. Bally *et al.*[1] identified records from the libraries of the universities of Adelaide and California which describe an accession of *N. benthamiana* being collected by Cleland in Central Australia who subsequently sent it to Goodspeed in California in 1939, suggesting that this was 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?

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Figure 1 | The *N. benthamiana* strain used in laboratories has traded antiviral defence for early vigour to survive to the extreme habitat of Central Australia where it originates. A plant infected with a GFP-tagged virus (right) displays typical viral symptoms compared with a non-infected plant (left). Images were taken under visible (top) or UV (bottom) light.

Bally and colleagues' 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 original source of the insertion. 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 (ka). As the separation between SA and LAB dates from 710 ka, this implies that the insertion occurred between 880 and 710 ka.

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 of plant architecture, leaf shape, floral structure and seed size across different geographic populations. Insert-free accessions living in non-desert areas are less susceptible to virus but also have lower growth rates and produce larger flowers facilitating insect attraction and cross-pollination. These phenotypes are consistent with the fact that in non-desert areas there are abundant insect pollinators and viral vectors which favour the maintenance of *Rdr1* functionality conferring virus resistance; hosts under pathogen pressure allocate more resources to defence that might otherwise have been dedicated to growth and survival. In contrast, LAB and SA accessions have higher growth rates and floral structures favouring self-fertilization: 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 vigour, fast seed setting and self-fertilization, confer a survival advantage in arid Central Australia, where the limited rainfall not only reduces the number of insects to act as pollinators and viral vectors, but also demands that plants complete their life cycles 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[4]. 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-mediated antiviral silencing pathway[5]. This observation led to the proposal that the dysfunctional Nb*Rdr1* might have arisen as a consequence of strong selection pressure favouring RDR6-mediated antiviral defence due to *N. benthamiana*'s hypersensitivity to multiple viruses[5]. Now, Bally *et al.* unambiguously show that the disruptive

insertion in Nb*Rdr1* simultaneously increases virus susceptibility and accelerates growth rate and seed setting. Furthermore, they found the dysfunctional Nb*Rdr1* most likely results from a fortuitous DNA insertion maintained as an adaptive response conferring higher fitness in a particularly extreme and 'low-virus' habitat.

The work of Bally *et al.* provides a fascinating illustration of how, despite providing substantial fitness gains under pathogen pressure, the costs of defence can 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 which optimally balance the costs of defence and vigour to ensure both high and durable yields. ❐

Alberto Carbonell is in the Instituto de Biología Molecular y Celular de Plantas (CSIC-Universidad Politécnica de Valencia), Avenida de los Naranjos s/n, 46022 Valencia, Spain.
e-mail: acarbonell@ibmcp.upv.es

References

1. Bally, J. *et al. Nature Plants* **1,** 15165 (2015).
2. Goodin, M. M., Zaitlin, D., Naidu, R. A. & Lommel, S. A. *Mol. Plant Microbe Interact.* **21,** 1015–1026 (2008).
3. Nakasugi, K. *et al. PLoS ONE* **8,** e59534 (2013).
4. Yang, S. J., Carter, S. A., Cole, A. B., Cheng, N.-H. & Nelson, R. S. *Proc. Natl Acad. Sci. USA* **101,** 6297–6302 (2004).
5. Ying, X.-B. *et al. Plant Cell* **22,** 1358–1372 (2010).