“Host-directed evolution of viroids”

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Within the constellation of viruses and virus-like pathogens, viroids are an oddity among oddities. These non-encapsidated non-coding, circular single-stranded RNA parasites are only known to infect plants. Unlike viruses, these non-cellular agents are parasites of the host transcriptional machinery that localize to the nucleus or plastid in a family-dependent manner. Without the ability to code for proteins, the mechanism through which viroid-infection induces disease has been hypothesized to be due to off-targeting effects of gene silencing. Most viroids have narrow host ranges with a few select species displaying an ability to infect genetically disparate species. One such viroid is Hop stunt viroid (HSVd), which can cause disease in both herbaceous and woody plants. The sub-viral agent can infect over 20 known species including apple, apricot, citrus, cucumber, grapevine, plum, and of course, hop.

Interestingly, this viroid appears to be a symptomless symbiont in cultivated grapevines. However, inoculation of hops with the grapevine-variant of HSVd induces economically significant yield losses. The symptoms include stunting and reduced branching – which prevents the bine from climbing. More importantly, HSVd infection reduces cone production, cone weight, and alpha-acids. The increase in virulence appears to be a reversal of the serial-passage paradigm of viral pathogens normally applied to attenuate virus virulence. Repeated passage of a virus in a non-host cell-line usually results in attenuation of virulence when inoculated into host cells. This reduced virulence has been seen Vaccinia virus, Poliovirus, and Dengue Virus after serial passage through non-host cells resulting in increased viral fitness in the new host but reduced virulence in the original host. Though one may suspect this is a normal part of HSVd pathogenesis in a new host, the observations made by Kawaguchi-Ito et al. (2009) raise important questions about the origins of HSVd and the stimulation of virulence.

For the study, HSVd variants isolated from hop, plum, citrus, and grapevine were used to infect hop plants. Data regarding cone yield and growth were taken over the course of 10 years to see the effect of viroid infection. Their longitudinal study analyzed the mutations in the four HSVd populations over 15 years – specific nucleotide mutations at predicted sites 25, 26, 54, 193, and 281 were seen in the grapevine-inoculum that mirror the wild-type variant infecting hops. The convergence of mutations from the grapevine-variant towards the hop-variant coincides with increased virulence in the hop host. This convergence was not seen in the plum- and citrus-variants, which clustered separately along their own evolutionary trajectories. This raises the question of whether viroids are taking advantage of a susceptible host or whether the host is selecting for a more virulent viroid.