Viroid

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A **viroid** is a small infectious pathogen composed of simply a short strand of circular, single-stranded RNA. All known viroids are pathogens exclusively of plants; some are of economic importance. Compared to viruses, viroids are extremely small in size, ranging from 246 to 467 nucleobases.[1] In comparison, the genome of the smallest known viruses capable of causing an infection by themselves are around 2,000 nucleobases in size.

Viroid

Scientific classification

(unranked): Subviral agents

(unranked): **Viroid**

Families

Pospiviroidae
Avsunviroidae

Viroids, the first known representatives of a new domain of "sub-viral pathogens", were discovered and named by Theodor Otto Diener, plant pathologist at the U.S Department of Agriculture's Research Center in Beltsville, Maryland, in 1971.[2][3] The first viroid to be identified was *Potato spindle tuber viroid* (PSTVd). Some 33 viroid species have been identified.

Although viroids are composed of nucleic acid, they do not code for any protein.[4][5] A viroid's replication mechanism uses RNA polymerase II, a host cell enzyme normally associated with synthesis of messenger RNA from DNA, which instead catalyzes "rolling circle" synthesis of new RNA using the viroid's RNA as a template. Some viroids are ribozymes, having catalytic properties which allow self-cleavage and ligation of unit-size genomes from larger replication intermediates.[6]

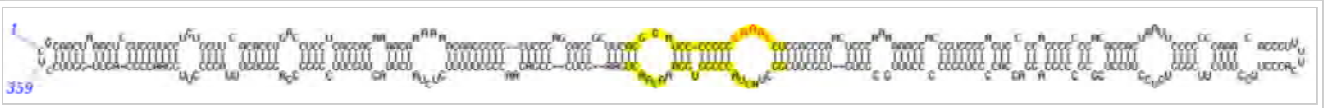
With Diener's 1989 hypothesis[7] that viroids may represent "living relics" from the widely assumed, ancient, and non-cellular RNA world—extant before the evolution of DNA or proteins—viroids have assumed significance beyond plant pathology to evolutionary science, by representing the most plausible RNAs capable of performing crucial steps in abiogenesis, the evolution of life from inanimate matter.

The human pathogen hepatitis D virus is a "defective" RNA virus similar to a viroid.[8]

Contents

Taxonomy

- Family Pospiviroidae
  - Genus *Pospiviroid*; type species: *Potato spindle tuber viroid* ; 356–361 nucleotides(nt) [9]
  - Genus *Pospiviroid*; type species: *Citrus exocortis* ; 368–467 nt [9]
  - Genus *Hostuviroid*; type species: *Hop stunt viroid* ; 294–303 nt [9]
  - Genus *Cocadviroid*; type species: *Coconut cadang-cadang viroid*; 246–247 nt[9]
  - Genus *Apscaviroid*; type species: *Apple scar skin viroid* ; 329–334 nt [9]
  - Genus *Coleviroid*; type species: *Coleus blumei viroid 1* ; 248–251 nt [9]

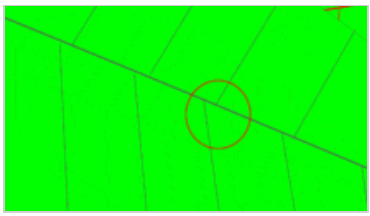


Putative secondary structure of the PSTVd viroid

- Family Avsunviroidae
  - Genus *Avsunviroid*; type species: *Avocado sunblotch viroid* ; 246–251 nt [9]
  - Genus *Pelamoviroid*; type species: *Peach latent mosaic viroid* ;335–351 nt [9]
  - Genus *Elaviroid*; type species: *Eggplant latent viroid* ; 332–335 nt [9]

Transmission

Viroid infections are transmitted by cross contamination following mechanical damage to plants as a result of horticultural or agricultural practices. Some are transmitted by aphids, and they can also be transferred from plant to plant by leaf contact.[9][10]


The reproduction mechanism of a typical viroid. Leaf contact transmits the viroid. The viroid enters the cell via its plasmodesmata. RNA polymerase II catalyzes rolling-circle synthesis of new viroids.

Replication

Viroids replicate in the nucleus (*Pospiviroidae*) or chloroplasts (*Avsunviroidae*) of plant cells in three steps through an RNA-based mechanism. They require RNA polymerase II, a host cell enzyme normally associated with synthesis of messenger RNA from DNA, which instead catalyzes "rolling circle" synthesis of new RNA using the viroid as template[11][6]

RNA silencing

There has long been uncertainty over how viroids induce symptoms in plants without encoding any protein products within their sequences. Evidence suggests that RNA silencing is involved in the process. First, changes to the viroid genome can dramatically alter its virulence.[12] This reflects the fact that any siRNAs produced would have less complementary base pairing with target messenger RNA. Secondly, siRNAs corresponding to sequences from viroid genomes have been isolated from infected plants. Finally, transgenic expression of the noninfectious hpRNA of potato spindle tuber viroid develops all the corresponding viroid-like symptoms.[13] This indicates that when viroids replicate via a double stranded intermediate RNA, they are targeted by a dicer enzyme and cleaved into siRNAs that are then loaded onto the RNA-induced silencing complex. The viroid siRNAs contain sequences capable of complementary base pairing with the plant's own messenger RNAs, and induction of degradation or inhibition of translation causes the classic viroid symptoms.[14]

Living relics of the RNA world

Diener's 1989 hypothesis[7] proposed that unique properties of viroids make them more plausible macromolecules than introns, or other RNAs considered in the past as possible "living relics" of a hypothetical, pre-cellular RNA world. If so, viroids have assumed significance beyond plant virology for evolutionary science, because their properties make them more plausible candidates than other RNAs to perform crucial steps in the evolution of life from inanimate matter (abiogenesis). These properties are:

1. viroids' small size, imposed by error-prone replication
2. their high guanine and cytosine content, which increases stability and replication fidelity
3. their circular structure, which assures complete replication without genomic tags
4. existence of structural periodicity, which permits modular assembly into enlarged genomes
5. their lack of protein-coding ability, consistent with a ribosome-free habitat
6. replication mediated in some by ribozymes—the fingerprint of the RNA world

Diener's hypothesis was mostly forgotten until 2014, when it was resurrected in a review article by Flores et al.,[15] in which the authors summarized Diener's evidence supporting his hypothesis (see above). In the same year, *New York Times* science writer Carl Zimmer published a popularized piece that mistakenly credited Flores et al. with the hypothesis' original conception.[16]

The presence, in extant cells, of RNAs with molecular properties predicted for RNAs of the RNA World constitutes another powerful argument supporting the RNA World hypothesis.

History

In the 1920s, symptoms of a previously unknown potato disease were noticed in New York and New Jersey fields. Because tubers on affected plants become elongated and misshapen, they named it the potato spindle tuber disease.[17]

The symptoms appeared on plants onto which pieces from affected plants had been budded—indicating that the disease was caused by a transmissible pathogenic agent. However, a fungus or bacterium could not be found consistently associated with symptom-bearing plants, and therefore, it was assumed the disease was caused by a virus. Despite numerous attempts over the years to isolate and purify the assumed virus, using increasingly sophisticated methods, these were unsuccessful when applied to extracts from potato spindle tuber disease-afflicted plants.[3]

In 1971 Theodor O. Diener showed that the agent was not a virus, but a totally unexpected novel type of pathogen, one-80th the size of typical viruses, for which he proposed the term "viroid".[2] Parallel to agriculture-directed studies, more basic scientific research elucidated many of viroids' physical, chemical, and macromolecular properties. Viroids were shown to consist of short stretches (a few hundred nucleobases) of single-stranded RNA and, unlike viruses, did not have a protein coat. Compared with other infectious plant pathogens, viroids are extremely small in size, ranging from 246 to 467 nucleobases; they thus consist of fewer than 10,000 atoms. In comparison, the genomes of the smallest known viruses capable of causing an infection by themselves are around 2,000 nucleobases long.[18]

In 1976, Sänger et al.[19] presented evidence that potato spindle tuber viroid is a "single-stranded, covalently closed, circular RNA molecule, existing as a highly base-paired rod-like structure"—believed to be the first such molecule described. Circular RNA, unlike linear RNA, forms a covalently closed continuous loop, in which the 3' and 5' ends present in linear RNA molecules have been joined together. Sänger et al. also provided evidence for the true circularity of viroids by finding that the RNA could not be phosphorylated at the 5' terminus. Then, in other tests, they failed to find even one free 3' end, which ruled out the possibility of the molecule having two 3’ ends. Viroids thus are true circular RNAs.

The single-strandedness and circularity of viroids was confirmed by electron microscopy,[20] and Gross et al. determined the complete nucleotide sequence of potato spindle tuber viroid in 1978.[21] PSTVd was the first pathogen of a eukaryotic organism for which the complete molecular structure has been established. Over thirty plant diseases have since been identified as viroid-, not virus-caused, as had been assumed.[18][22]

See also

- Circular RNA
- Microparasite
- Non-cellular life
- Plant pathology
- Plasmid
- RNA world hypothesis
- Satellite (biology)
- Virus
- Virus classification
- Virusoid

References

1. Lewin, Benjamin.; Krebs, Jocelyn E.; Kilpatrick, Stephen T.; Goldstein, Elliott S.; Lewin, Benjamin. Genes IX. (2011). *Lewin's genes*. Sudbury, Mass.: Jones and Bartlett. p. 23. ISBN 9780763766320.
2. Diener TO (August 1971). "Potato spindle tuber "virus". IV. A replicating, low molecular weight RNA". *Virology*. **45** (2): 411–28. doi:10.1016/0042-6822(71)90342-4. PMID 5095900.
3. "ARS Research Timeline – Tracking the Elusive Viroid". 2006-03-02. Retrieved 2007-07-18.

4. Tsagris EM, Martínez de Alba AE, Gozmanova M, Kalantidis K (September 2008). "Viroids". *Cell. Microbiol.* **10** (11): 2168–79. doi:10.1111/j.1462-5822.2008.01231.x. PMID 18764915.
5. Flores, Ricardo; Di Serio, Francesco; Hernández, Carmen (February 1997). "Viroids: The Noncoding Genomes". *Seminars in Virology*. **8** (1): 65–73. doi:10.1006/smvy.1997.0107.
6. Daròs JA, Elena SF, Flores R (2006). "Viroids: an Ariadne's thread into the RNA labyrinth". *EMBO Rep.* **7** (6): 593–8. doi:10.1038/sj.embor.7400706. PMC 1479586. PMID 16741503.
7. Diener TO (1989). "Circular RNAs: relics of precellular evolution?". *Proc. Natl. Acad. Sci. U.S.A.* **86** (23): 9370–4. Bibcode:1989PNAS...86.9370D. doi:10.1073/pnas.86.23.9370. PMC 298497. PMID 2480600.
8. Alves C, Branco C, Cunha C (2013). "Hepatitis delta virus: a peculiar virus". *Adv Virol*. **2013**: 560105. doi:10.1155/2013/560105. PMC 3807834. PMID 24198831.
9. Brian W. J. Mahy, Marc H. V. Van Regenmortel (ed.). *Desk Encyclopedia of Plant and Fungal Virology*. Academic Press. pp. 71–81. ISBN 978-0123751485.
10. De Bokx JA, Piron PG (1981). "Transmission of potato spindle tuber viroid by aphids.". *Netherlands Journal of Plant Pathology*. **87** (2): 31–34. doi:10.1007/bf01976653.
11. Flores R, Serra P, Minoia S, Di Serio F, Navarro B (2012). "Viroids: from genotype to phenotype just relying on RNA sequence and structural motifs". *Front Microbiol*. **3**: 217. doi:10.3389/fmicb.2012.00217. PMC 3376415. PMID 22719735.
12. Hammond RW (1992). "Analysis of the virulence modulating region of potato spindle tuber viroid (PSTVd) by site-directed mutagenesis". *Virology*. **187** (2): 654–62. doi:10.1016/0042-6822(92)90468-5. PMID 1546460.
13. Wang MB, Bian XY, Wu LM, Liu LX, Smith NA, Isenegger D, Wu RM, Masuta C, Vance VB, Watson JM, Rezaian A, Dennis ES, Waterhouse PM (2004). "On the role of RNA silencing in the pathogenicity and evolution of viroids and viral satellites". *Proc. Natl. Acad. Sci. U.S.A.* **101** (9): 3275–80. Bibcode:2004PNAS..101.3275W. doi:10.1073/pnas.0400104101. PMC 365780. PMID 14978267.
14. Pallas V, Martinez G, Gomez G (2012). "The interaction between plant viroid-induced symptoms and RNA silencing". *Methods Mol. Biol*. Methods in Molecular Biology. **894**: 323–43. doi:10.1007/978-1-61779-882-5_22. ISBN 978-1-61779-881-8. PMID 22678590.
15. Flores R, Gago-Zachert S, Serra P, Sanjuán R, Elena SF (June 18, 2014). "Viroids: survivors from the RNA world?". *Annu. Rev. Microbiol*. **68**: 395–414. doi:10.1146/annurev-micro-091313-103416. PMID 25002087.
16. Zimmer, C (September 25, 2014). "A Tiny Emissary From the Ancient Past". *New York Times*. Retrieved November 22, 2014.
17. Owens RA, Verhoeven JT (2009). "Potato Spindle Tuber". *Plant Health Instructor*. doi:10.1094/PHI-I-2009-0804-01.
18. Pommerville, Jeffrey C (2014). *Fundamentals of Microbiology*. Burlington, MA: Jones and Bartlett Learning. p. 482. ISBN 978-1-284-03968-9.
19. Sänger HL, Klotz G, Riesner D, Gross HJ, Kleinschmidt AK (1976). "Viroids are single-stranded covalently closed circular RNA molecules existing as highly base-paired rod-like structures". *Proc. Natl. Acad. Sci. U.S.A.* **73** (11): 3852–6. doi:10.1073/pnas.73.11.3852. PMC 431239. PMID 1069269.
20. Sogo JM, Koller T, Diener TO (1973). "Potato spindle tuber viroid. X. Visualization and size determination by electron microscopy". *Virology*. **55** (1): 70–80. doi:10.1016/s0042-6822(73)81009-8. PMID 4728831.
21. Gross HJ, Domdey H, Lossow C, Jank P, Raba M, Alberty H, Sänger HL (1978). "Nucleotide sequence and secondary structure of potato spindle tuber viroid". *Nature*. **273** (5659): 203–8. doi:10.1038/273203a0. PMID 643081.
22. Hammond RW, Owens RA (2006). "Viroids: New and Continuing Risks for Horticultural and Agricultural Crops.". *APSnet Feature Articles*. doi:10.1094/APSnetFeature-2006-1106.

External links

- Viroids/ATSU (http://www.atsu.edu/faculty/chamberlain/Website/Lects/Prions.htm)