Viroids

Viroids are infectious pathogens that affect only plants, therefore are also called as the plant pathogens. Structurally, viroids are smaller than viruses and possess circular strands of ribonucleic acids (RNA's) with no protein coating. These entities hijack the cellular machinery present in plant cells to replicate new copies of itself. It primarily affects all forms of higher plants.

The first discoveries of viroids triggered the historically third major extension of the biosphere—to include smaller lifelike entities —after the discoveries, in 1675 by Antonie van Leeuwenhoek (of the "subvisible" microorganisms) and in 1892 by Dmitri Iosifovich Ivanovsky (of the "submicroscopic" viruses). The unique properties of viroids have been recognized by the International Committee on Taxonomy of Viruses, in creating a new order of subviral agents.

The first recognized viroid, the pathogenic agent of the potato spindle tuber disease, was discovered, initially molecularly characterized, and named by Theodor Otto Diener, plant pathologist at the U.S Department of Agriculture's Research Center in Beltsville, Maryland, in 1971. This viroid is now called Potato spindle tuber viroid, abbreviated PSTVd.

Although viroids are composed of nucleic acid, they do not code for any protein.The 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 that allow self-cleavage and ligation of unit-size genomes from larger replication intermediates.

With Diener's 1989 hypothesis 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.

Structure Of Viroids

Viroids differ from the virus in structure and form. These consists of solely short strands of circular, and single-stranded RNA without the protein coats.

The plants that are infected by viroids are responsible for the crop failures and also causes loss of millions of dollars in the agricultural revenue every year. Some of the plants that are affected by these pathogens are potatoes, tomatoes, cucumbers, chrysanthemums, coconut palms, avocados, etc.

Viroids were first discovered by T.O. Diener in the year 1971. It was first examined in the potato spindle tuber viroid that caused a huge loss to the potato industry.

Viroids are the plant parasites like transcriptional machinery of the **cell organelles** such as the nucleus or the chloroplast since they are known to be non-coding. These replicate by the process of RNA–RNA transcription. They mainly infect the epidermis of the hosts after causing mechanical damage to the cell wall of the plant.

Characteristic Features Of Viroids

Some of the characteristic features of viroids are given below-

- Viroids are formed only on the RNA.
- These are known to be smaller in size and infect only the plants.
- These are among the smallest known agents causing infectious disease.
- Viroids are the species of nucleic acid with relatively low molecular weight and a unique structure.
- They reproduce within the host cell which they affect in and cause variations in them causing death.
- Viroids are mainly classified into two families namely Pospiviroidae- nuclear viroids and Avsunviroidae- chloroplastic viroids.
- Viroids are said to move in an intracellular manner, cell to cell through the plasmodesmata, and a long-distance through the phloem.

Viroid Diseases

Some of the diseases that are caused by the infection of viroid in plants are citrus exocortis, cucumber pale fruit, chrysanthemum stunt. These **infectious diseases** are spread by the propagation of seeds in plants by cutting, tubers, etc and also by mishandling the contaminated implements. The only disease that is caused by viroids in humans is Hepatitis- D.

The symptoms that are caused by the infection of viroid in plants include stunting of growth, stem necrosis, deformation of the leaves and fruits, and at last causing the death of the plant.

Most of the viroids are said to infect the plants, including coconut and the apple trees. The (PSTV) potato spindle tuber viroid causes significant crop damage to the potato yields causing the tubers to elongate and then crack. The other common type of viroid infection symptoms includes stunting and leaf epinasty.

Transmission

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.

Viroid infections can be transmitted by aphids, by cross contamination following mechanical damage to plants as a result of horticultural or agricultural practices, or from plant to plant by leaf contact.

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.

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.[13] 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. 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.

RNA world hypothesis

Diener's 1989 hypothesis had proposed that the 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 theory, because their properties make them more plausible candidates than other RNAs to perform crucial steps in the evolution of life from inanimate matter (abiogenesis).

Diener's hypothesis was mostly forgotten until 2014, when it was resurrected in a review article by Flores et al. in which the authors summarized Diener's evidence supporting his hypothesis as:

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.

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.

Our current understanding is that the disease-causing viroids were transferred from wild plants used for breeding modern crops. The widespread prevalence of these agents can be traced to the use of genetically identical plants (monoculture), worldwide distribution of breeding lines, and mechanical transmission by contaminated farm machinery. As a consequence, these unusual pathogens now occupy niches around the planet that never before were available to them.

The origin of viroids remains an enigma, but it has been proposed that they are relics from the RNA world, which is thought to have been populated only by non-coding RNA molecules that catalysed their own synthesis. Viroids have properties that make them candidates for survivors of the RNA world: small genome size (to avoid error catastrophe caused by error-prone replication), high G+C content (for greater thermodynamic stability), circular genomes (to avoid the need for mechanisms to prevent loss of information at the ends of linear genomes), no protein content, and the presence of a ribozyme, a fingerprint of the RNA world. Today's viroids can no longer self-replicate, possibly having lost that function when they became parasites of plants. What began as a search for virus-like agents that cause disease in plants has lead to new insights into the evolution of life.