

# 10<sup>th</sup> International Plant Virus Epidemiology Symposium

## *Controlling Epidemics of Emerging and Established Plant Virus Diseases - The Way Forward*

15-19 October 2007, ICRISAT  
Patancheru 502324, AP, India

Program and Abstracts



Organized and Hosted by the International Crops Research Institute for the Semi-Arid Tropics



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Patancheru 502 324, Hyderabad, Andhra Pradesh, India

### **Program and Abstracts**

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**OP-34: Centennial of research on groundnut rosette disease: what is known and what still needs to be known to achieve effective control of this menace in Sub-Saharan Africa**

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Several economically important plant virus diseases involve synergistic interactions between causative viruses, wherein the presence of one virus facilitates the other virus deficits in some molecular function for survival and spread. Groundnut rosette disease (GRD), first reported in 1907 from Tanganyika (presently Tanzania), is the most fascinating example of such an interaction, wherein three agents are intricately dependent on each other for survival and spread. GRD has been recognized in the all groundnut growing countries on the African continent, including its offshore islands such as Madagascar, but not anywhere outside Africa. GRD is responsible for annual groundnut loss of worth US\$ 150 million. The disease occurs in two predominant symptom forms - chlorotic rosette and green rosette, infection of which at early growth stage results in up to 100% yield loss. GRD is caused by a complex of three agents: *Groundnut rosette assistor virus* (GRAV; Family, *Luteoviridae*), *Groundnut rosette virus* (GRV; Genus, *Umbravirus*) and its Satellite-RNA (satRNA, Sub-Group 2 Satellite RNA). GRD symptoms are associated with the presence of the GRV-satRNA complex, in which variants of the satRNA are responsible for different symptom types, such as chlorotic rosette, green rosette and mottle rosette. GRAV replicates autonomously in plants and is transmitted by an aphid, *Aphis craccivora* Koch (Homoptera: Aphididae). GRAV alone causes no obvious symptoms, but it was shown to cause substantial yield reduction in susceptible genotypes. The satRNA depends entirely on GRV for its replication, and GRV must be associated with its satRNA for its packaging in the GRAV coat protein and subsequent transmission by the aphid vector. Through the ability to utilize the coat protein of GRAV, GRV-satRNA gains epidemiologically by acquiring a persistent relationship with the aphid vector for survival and spread. Resistance sources to GRD have been found in cultivated as well as wild *Arachis* germplasm. Breeding for host plant resistance at ICRISAT have contributed to the development of several groundnut varieties with acceptable levels of field resistance. However, all the resistant genotypes identified apparently contain the same genes conferring resistance to GRV & satRNA, and several of them are long duration types. There is a need to broaden the genetic base of GRD resistance to avert any breakdown of resistance under severe disease pressure, and breed short-duration GRD resistance varieties preferred by the farmers in SSA. This paper illustrates the successful international collaborative research efforts since the first report of GRD 100 years ago in unraveling the etiology, molecular mechanism of interaction between the agents and its invertebrate vector, leading to the development of control strategies. The paper will further emphasize the need for diverse approaches required to understand the critical information pertaining to the off-season survival of the disease agents and aphid vector and the need for new GRD resistant cultivars, if effective control of this major plant virus disease of groundnut in SSA is to be achieved.