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Plant virus-ecology and epidemiology

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Abstract
Viral diseases rank second in terms of monetary losses after fungal diseases and provide a major challenge to agriculture worldwide. Climate change, rapid population increase and food insecurity are driving rapid alterations in agricultural system that favour destructive viral disease outbreaks. Viruses alter physiology of infected plants, results in symptom development that ultimately lead to huge yield loss, estimated to be more than 30 billion dollars annually (Jones, 2021). Emerging *Geminiviruses* cause economic losses estimated as: 1300–2300 million dollars in cassava (Africa), 300 million dollars for grain legumes (India), 140 million dollars in tomato (Florida) and 5 billion dollars in cotton (Pakistan) (Varma and Malathi, 2003). The economic losses due to viral diseases complicate food security and may pose a significant threat to mankind. Keeping all this in sight there is a robust got to investigate and understand virus ecology and epidemiology for developing any management programme for any viral disease. Ecology mainly focuses on virus behaviour in its habitat while as epidemiology focuses on viral diseases within host populations (Wilson, 2014).Discovery of cause of potato leaf roll disease in Netherland, importance of insect vectors, understanding of virus vector transmission (Harrison, 1980) and effectiveness of predictive modals (Jones *et al.*, 2014) were major mile stones in the advancement and development of plant virus ecology and epidemiology field. Both the areas are multi-layered with many variables having influence on host plants, viruses and the vector behaviour mostly having damaging effect but some may have beneficial effect as Cucumber mosaic virus offers cold resistance in sugar beet (Xu *et al.*, 2008). Vectors have a major role in virus transmission (Gallet and blanc, 2018). Viral diseases are due to specified molecular interactions via protein among virus and vector, understanding such interactions can support approaches for shielding plants from contamination by meddling virus take-up and transmission (Dietzgen *et al.*, 2016). Climatic changes have diminished effectiveness of cultural management practices and virus epidemics have become less predictable (Jones, 2014). Due to climatic change previously unknown viruses and vectors emerged, their introduction threatens both cultivated as well as newly introduced crops (Jones and Barbetti, 2012). The utilization of new molecular techniques have significantly speed up the pace of progress in understanding all the factors underlying this discipline. Numerous advantages that are provided by studying molecular epidemiology and ecology include improving the recognisability of plant virus populations, providing information about virus epidemic patterns not possible by conventional field examinations, knowledge regarding the virus resistance breakdown and testing the strength of virus or vector specific control measures (Jones, 2014). Rapidly increasing technological innovation is giving a significant means to address the problems that plant virus epidemics pose to future food security and the diversity of the earth's remaining vegetation and ensures that the subject has a very exciting nature.

Keywords: Climatic change, emerging virus, epidemiology, ecology, molecular, transmission

Introduction
Viral diseases rank second in terms of monetary losses only after fungal diseases. Rapidly accelerating population, expected to reach 10 billion by 2050 and climate change are the major challenges faced by twenty first century results in increased prevalence of viral diseases (Godfray *et al*., 2010) [21]. Amongst emerging plant diseases around 47 percent have virus as a causal organism (Yogita *et al.*, 2019) [87]. Plant viruses cause an estimated loss of US $ 60 billion in crop yield per annum worldwide (Roossinck., 2011) [66]. Viruses like *Apple mosaic virus* cause 30-50 (%) reduction in fruit yield, 50 per cent reduction in growth and about 20 per cent reduction in trunk diameter (Sutic., *et al.* 1999) [73]. Plant viral epidemics pose threat to world food security when there is climatic change and world population increasing (Jones and Barbetti, 2012) [32]. Basically economic losses on account of viral diseases add one more layer of intricacy and vulnerability in accomplishing food security. Keeping all above mentioned in sight there is a robust to investigate and understand plant virus ecology and epidemiology for developing any management program for any viral disease.

The current and future food crops will be protected from viral diseases by simply improving our understanding of plant virus epidemiology which includes interaction between virus, host and vector. Management should be knowledge based for understanding the role of ecological and epidemiological factors in the development and emergence of viral epidemics, likewise help in obtaining more information on the genetic diversity and evolution of plant virus population. Plant virus ecology and epidemiology are firmly interrelated but quite different concepts, first one focusing on the virus population within a given environment and other one on the complex relationship between the plant virus, host and the factors influencing the viral epidemics within host population (Wilson, 2014). Hull (2002) [80, 26] defined the virus ecology as the investigation of factors influencing the behaviour of a viral epidemic during a given circumstances and virus epidemiology as the investigation of determinants, factors and conveyance of plant virus diseases in host populations. The influence of ecological elements on the viral population behaviour within their habitats indicate the connections between viruses, hosts and vectors and also include host range, tissue tropism, symptom expression, transmission modes and mechanisms symptom expression, virus ingenuity within the environment and a specific scope of cultural practices and human activities having an impression in these communications (Aranda and Freitas -Astúa, 2017) [41]. Both ecology and epidemiology include numerous factors influencing virus, host and vector behaviour mostly having damaging effect on host but some may have beneficial effects as *Cucumber mosaic virus* offers cold resistance in sugar beet (Xu *et al*., 2008). Smith (1955) [84, 72] allude the period before the 1930s as the dark age of plant virus research after which a more scientific approach was initiated. In 1916, the infectious nature of *Potato leaf roll virus* disease was discovered by researchers from Netherland. This gave a final knockout to the idea of senile-decay and enhancing understanding that the degeneration of potatoes was on account of spread of virus that the degeneration of potatoes was on account of spread of virus infection within the crop. By 1939, The significance of insects as virus vectors turned out to be completely recognised by 1939 and also the relationship between insect borne virus and their vectors as persistent or non-persistent on the basis of the time taken by the insects to hold infectivity without having access to a virus source. The natural selection is responsible for the development of resistance in plants but compensated by selection of highly virulent variant from viruses suggested by Homes (1955). Swarming of plants under cultivation builds the need for high degrees of infection obstruction plants under cultivation builds the need for high degrees of infection obstruction. He provided samples of strain specific resistance to *Tomato spotted wilt virus* (TSWV; genus *Tospovirus*) in tomato. The progress in understanding vector-virus transmission mechanisms, ecological systems and epidemiology and advances in control methods was accented by Harrison (1980). Harrison & Robinson (2005) [23, 24] emphasised that tremendous application of latest molecular, cell biological and genetical techniques had occurred during the period of 1979–2004. Thresh (1976) [75] published a very influential review entitled as 'Gradients of plant virus diseases'. Gradients refers to change in virus incidence with distance from a virus infection source. Varma & Malathi (2003) [79] published a review also entitled as 'Emerging *Geminivirus* problems: a serious threat to crop production'. *Geminiviruses* had arisen as wrecking pathogens, particularly in the tropics and subtropics, their epidemics causing colossal financial losses and seriously threatening crop production before their publication that is before the two decades. *Geminiviruses* included the genera *Begomovirus*, *Curtovirus*, *Mastrevirus* and *Topocuvirus*, but *Begomoviruses* led to premier difficult issues in cassava, cotton, grain legumes and vegetables. Monetary losses because of *Geminiviruses* were assessed to be: 1300–2300 million dollars in cassava in Africa, billion dollars in cotton in Pakistan, 300 dollars million for grain legumes in India and 140 million dollars in tomato in Florida. The evolution of new latest virus variants, appearance of the 'B' biotype of the vector *B. tabaci*, and expansion in its population were major contributory factors responsible for the emergence and spread of latest *Geminiviruses*. Virus diversification occurred due to genomic recombination in *Geminiviruses*, not only among variants of an equivalent virus, but also between species and even genera. Modelling provides a helpful device to broaden comprehension of the complex interactions between vectors, viruses, host plants, and the environment. However, the worth of an epidemiological model's predictions relies on the quality of the data from which the model is developed and the legitimacy of the assumptions made (Frazer, 1977) [14].

Origin and Evolution of plant viruses:

Familiarity with the individual virus history can provide an important new insights for understanding their ecology and epidemiology. Pagan and Holmes, (2010) [57] estimate the origin up to 3500 years ago for the family *Lutheoviripae* and the two major genera of this family (*Luteovirus* and *Polerovirus*) to have approximately about 1000-2000 years ago. The radiation of the species in the two genera (*Luteo virus* and *Polerovirus*) of family *Luteoviridae* has occurred over the past 500 hundred years. Same results were obtained by Wu *et al*., (2011) [82] for the *Barley yellow dwarf virus* (BYDV, *Luteovirus*). Similarly, Yasaka *et al*., (2017) [19] marked the origin of TuMV 3500 years ago, with the current baselines issued over the past 5000 years. Yasaka *et al*., (2014) [86] also marked the origin of the modern diversity of *Cauliflower Mosaic virus* (CaMV) about 5000 years ago and Salva *et al*., (2012) [70] estimated that *Citrus tristeza virus* (CTV) originated about 1500 years ago by Fargette *et al*., (2008b). Gibbs *et al*., (2008b) [12, 18] traced the origin of the *Potyvirus* genus about 6600 years ago, and accordingly Gibbs *et al*., (2017) [19] marked the origin of PVY (Genus *Potyvirus*) upto 6500 years ago. Fuller *et al*., (2014) [15] reported that all the estimates of origin and radiation times for virus taxa find time points near to the origin or the intensification of agriculture, which has led to formulate the hypothesis that agriculture is basic ecological driver for the radiation of pathogenic plant viruses. The phylogenetic analyses have revealed that certain RNA plant viruses could be much more ancient, despite the general agreement on plant virus evolutionary time scales. This is explained by the case of the species in the genus *Tobamovirus*, whose origin has been traced back to 110 million years ago. This has suggested that a proto-tobamo virus infecting ancient angiosperms would have been the origin of *Tobamovirus* species through their co-divergence with solanaceous hosts (Gibbs *et al*., 2015) [20]. If long-term survival of *Tobamovirus* would strongly depend upon the adaptation to their present particular family of plants, co-divergence would be explained. Similarly,

diversification of *Geminiviridae* species through analyses of virus-related sequences integrated within the host genome has been traced back to 80 million years ago (Gibbs *et al*., 2010; Lefeuvre *et al*., 2011) [17, 39]. Besides these estimates of long-term evolutionary rates, coalescent Bayesian phylogenies are also helpful to analyze the short-term evolutionary time scales of plant viruses. As an example the origin of the EACMV epidemic in Central African Republic within the in the last 34-175 years supported sequences of the CP gene of this virus and was estimated by Duffy and Holmes, 2009 [10]. Using an equivalent gene, Rodelo-Urrego *et al*., 2013 [62] dated the origin of the PepGMV and PHYVV epidemics in wild pepper or chiltepin (*Capsicum annuum* var. *glabriusculum*) plants to about 30-50 years ago in Mexico. Almeida *et al*., (2009) [2] found that BBTV spread across Hawaiian Islands only in 17 years. Benítez-Galeano *et al*., (2017) [4] reported the origin of a plague caused by an isolate of CTV in 1977. Finally, the origin of the TYLCV epidemic in tomato plants was estimated in China about 80 years ago by Xu *et al*., (2017) [83]. The above estimates were in agreement with existing epidemiological evidence, indicating that phylogenetics can also provide relevant information both on disease evolution and epidemiology.

Plant virus ecology

The virus populations within a specific environment are studied under an in-dispensable discipline plant virus ecology. Plant virus ecology gives an insight of correlation and interactions of a complex of virus-host environment. This field is comprised of a number of aspects like plant virus biodiversity, sampling viruses from infected plants directly, from other ecosystems, how plant viruses invade the new emerging and invasive species, interactions between plant communities involving mixed and wild plant populations, insect vectors; persistent viruses showing epigenetic effects; soil born plant viruses and their surrounding soil ecosystems; molecular basis of viral genomics; ecological factors impacting upon plant viruses and modern technological innovations helping in research about the viruses (Islam, 2017) [29].

Plant virus biodiversity

According ICTV (master species list 2018) there are 5560 species of viruses, 1019 genera, 150 families and 19 orders. The non-host crops are being studied by the help of technological innovations. The biodiversity is reviewed by following result oriented approach, where samples from various environments are analysed against particular virus sequence but now plant virologists are adopting another way of individual plants sampling (Roossinck *et al*., 2010) [65]. In this way deeper ecological and evolutionary analyses possible as each sequence are often tracked towards its specific host (Roossinck, 2012) [31]. Regarding viral diversity, huge data is available about mixed virus infections or temporal viral infections in native plants. So it is very important to achieve more and more knowledge about viruses, their hosts and existing host and virus combinations which will cause diseases to the next level and will be very much damaging in future (Jonathan *et al*., 2012) [31].

Emerging plant viruses

International committee for Taxonomy of Viruses (ICTV) has enlisted approximately 900 species of plant viruses (King *et al*., 2012) [37]. Viruses have been found in a range of ecosystem a prevalent example is *Pepper mild mottle virus* found in waste water (Rosario *et al*., 2009) [67]. Studies showed fifty per cent of the newly emerging viruses belong to a category of DNA or RNA viruses (Anderson *et al*., 2004) [3]. RNA viruses perform host shifting mechanism easily than other type of viruses (Longdon *et al*., 2014) [40]. Viruses undergo evolutionary changes before invading new plant species, thus guaranteeing their survival within their new hosts (Longdon *et al*., 2014: Roossink *et al*., 2015) [40, 64].

Insect vector ecology

Plants become more attractive to insects as plants release some volatile compounds perceived by insects in presence of viruses, this may lead to the idea that insects feed more on infected plants (Mauck *et al*., 2012) [47]. Further studies showed that persistent viruses make plants more attractive than non -persistent ones (Mauck *et al*., 2012) [47]. Viruses also modify insect vector life cycles, fitness and behaviour either directly or indirectly. Direct changes occur in vector when the virus remain inside the insect for its whole life. For instance, TYLCV (*Tomato yellow leaf curl virus*) remained in the body of white fly thus influences vector settling, probing and feeding (Moreno-Delafuente *et al*., 2013) [50]. Further research revealed that the interaction is interdependent between virus and vector specifically for the biotype Q only (Pan *et al*., 2013).

Virus ecology in wild plant communities

While considering the ecology of viruses in wild plants, fitness is the important concept. When virus-infected wild plants grow in species mixtures in undisturbed natural plant communities, relative fitness of infected plants is defined as the survivorship arising from their ability to compete nicely with healthy plants of other species, reproduce sufficiently and produce subsequent generations of the seedling. Maskell *et al*. (1999) [45] investigated viruses infected wild cabbage (*Brassica oleracea*) in the UK, viruses significantly diminish both vegetative and reproductive performance of wild cabbage. This finding showed that viral infections have great potential to significantly damage natural plant communities.

Soil-borne virus ecology

The ecology of a fungus-transmitted virus is best studied with respect to *Potato moptop virus* (PMTV, genus *Pomovirus*) in potato. As PMTV is transmitted to roots and tubers of healthy potato by zoospores and spore balls of *Spongospora subterranea* (the powdery scab fungus). Spore balls carry virus internally. This virus have ability to survive inside *S. subterranea* resting spores for 12 years in soil after potatoes have been grown there. Roots of the common weed (*Solanum nigrum*) were often infected with PMTV when collected from infested fields so, this species could help maintain field infestations through crop rotation when potato is absent (Hull, 2014) [27].

Factors affecting plant virus ecology

Climate

According to NASA earth Observatory, in 21st century the global temperature could rise by 2-6 °C causing significant damage to the plant virus ecosystem. For example, whitefly population begin to accumulate at high temperature and high relative humidity and shows declines at low temperatures and

high rainfalls (Islam *et al*., 2017) [29]. Extreme weather events, including adverse rainfall, wind storms, heat wave patterns and dry spells, are now more predictable due to availability of improved technology now a day. These events divide the whole world into several ecologically sensitive regions. However, because plants in the geothermal soils of Yellowstone National park adapt to virus infected endophytes, plants and viruses readily adapt to extreme weather conditions (Márquez *et al*., 2007) [44].

Movement and feeding behaviour of insect vector

The temporal and spatial distribution of any disease depends on a positive correlation between the migration of insect of in vectors and population size. The movement of the vector must go through several stages in order to land properly on the host, after which it begins to feed (Fereres and Moreno, 2009) [13]. The Aphids may feel the plants less attractive for them after they fed upon them and got virus acquisition (Rajabaskar *et al*., 2014).

Host plant genotype and populations

The plant viruses can infect hosts with different taxonomic status (Woolhouse *et al*., 2001) [81]. The genetic diversity of virus is determined by their own genome structures, which evolve to increase resistance (Schneider and Roossinck, 2001) [69]. An outbreak of Pakistani *Cotton leaf curl virus* (CLCuV) resulted in a new cotton genotypes that was highly susceptible to local virus strains, resulting in wiping off cotton crop by CLCuV (Mansoor *et al*., 2006) [43].

The role of weed plants

Viruses can invade seeds and annual weeds and are clearly important in maintaining insect vector populations and growing host agro-ecosysyems (Norris and Kogan, 2005). Trebicki (2010) [55, 77] investigated several factors important for the epidemics of *Tobacco yellow dwarf virus* in Australia and found that the two weed species *Amaranthus retroflexus* and *Raphanus raphanistrum* were the viral ports through which *Orosius orientalis* (vector) transmits the virus to other crops.

Role of human beings

Today, the planet earth is experiencing rapid human activities day by day, that significantly affects plants, vectors and viruses results in instability to the virus vector-plant ecosystems (Patel and Fauquet, 2011) [59]. The rapid and aggressive human activities include introduction of more diverse, widespread and intensive agronomic practices such as: mono or dual cropping systems; better tillage, un-judicial chemical usage, untreated irrigation methods and similar cropping patterns. All of these methods help in virus dissemination and infectious disease break outs (Jones, 2009) [33]. Several examples of large scale agricultural activities and practices undertaken by citizenry inadvertently led to the emergence and outbreak of new diseases (Alexander *et al*., 2014) [1]. The simplest and best example of adaptations to the new host vegetation's is *Begomoviruses* (Navas-Castillo *et al*., 2011) [54].

Plant virus epidemiology

Robert (2001) defined epidemiology as the cyclical development of viral diseases in time and space within plant populations. Thus, epidemiology is related to how and why an epidemic spreads in an ecosystem.

- **Virus:** A viruses is a chain of nucleic acids (DNA or RNA) which lives in a host cell, uses parts of the cellular machinery of host to reproduce, and release the replicated nucleic acid chains to infect more cells.

Virus related factors

Physical stability of viruses and concentrations reached

The viruses which transmit by mechanical transmission need to be quite stable both inside and outside of the plant and should reach to a high concentration in the tissues for their survival and spread, as some viruses are highly unstable affecting their perennation. The survival and spread of certain viruses largely depend on high degree of stability and therefore the large amount of viruses are produced in the infected tissues. For example, TMV may survive for long periods in dead plant material in the soil, which will then ensure a source of infection for next crops (Johnson and Ogden, 1929) [30].

Rate of movement and distribution within host plants

Viruses or virus strains that migrate very slowly through the tissue of the plant from the point of infection are less likely to persist and spread efficiently than ones that move very quickly in the host. Viruses move fast in annual plants than long-lived shrubs or trees in which viruses move much more slowly through their hosts. Another crucial factor for aerial-borne viruses is the speed of movement within the host in relation to the life cycle or seasonal cycle of the vector. Viruses residing in seed have a crucial advantage in spread and survival (Hull, 2014) [27].

Severity of the disease

As a rapidly evolving systematic diseases, viruses that kill host plants are much less viable than viruses that cause mild to moderate disease that allow host to plants to survive, complete with surrounding plants, and reproduce efficiently. Synergistic interactions between viruses can lead to more severe diseases (Zhang *et al*., 2001) [89]

Mutability and strain selection

Molecular diversity in plant viruses is due to the mutation and recombination. The epidemiology of the virus is these variation within viral populations (Moury *et al*., 2006) [51]. Thus, the degree to which a virus have ability to mutate into strains that can cope effectively with changes within environment can affect survival and dispersal of the virus. When perennial weeds and wild hosts are the source of virus for an annual agricultural crop, successive crops may become infective with strains that have never been able to adapt well to the crop plant.

Host range

The interaction of multiple ecological and genetic factors result in plant viruses emergence a complex process ultimately leads to virus encountering a new host and adapting to it, also ensuring effective virus transmission to the new host population (Elena *et al*., 2014) [11]. Host range is defined as the number of host species used by a pathogen, is in theory a simple metric central to understanding pathogen epidemiology and pathogenicity. Some viruses for example affecting strawberries appear to be confined to the genus *Fragaria*, other viruses may be able to infect a wide range of

plants thus viruses vary greatly in the ability to infect a range of plant species, CMV has host range over 1200 host species in addition to 100 families (Hull, 2014) [27].

- **Host:** An organism in which virus replicate.

Host related factors

Genotype

Susceptible host lacking genes for resistance provide the perfect substrate for establishment and development of new infections resulting epidemics under favourable environmental conditions (Singh, 2001) [71].

Abundance and distribution of susceptible host

Epidemic develops in large population from relatively fewer infected individuals. Genetically uniform variety over large contiguous areas are conditions that favour the pathogen to extend the speed of multiplication to develop an epiphytotic (Singh, 2001) [71].

Type of host

Epidemics develop much more rapidly in the annual crops than perennial woody plants (Singh, 2001) [71].

Presence of weeds and alternate host

Trebicki *et al*., (2010) [77] in south-east Australia assessed factors important for the epidemiology of *Tobacco yellow dwarf virus* (genus Mastrevirus) in tobacco crop. The predominant leafhopper (*Orosius orientalis*) proved to be its vector. The virus was detected only in four plant species out of 40 sampled plant species, tobacco and customary bean (*Phaseolus vulgaris*), and the alternative weed host species *Amaranthus retroflexus* and *Raphanus raphanistrum*. The virus is transferrd by *O. orientalis* to these two cultivated species from the two weed hosts which acts as virus reservoirs. Prassada Rao *et al*. (2003) [60] studied a viral epidemic caused by *Tobacco streak virus* in peanut in India and the weed *Parthenium hysterophorus* played a serious role as an alternative host for virus, this virus is spread by its thrips vectors to peanut crops. Two other potential alternative hosts for the virus are sunflower and marigold, from which virus is spread to peanut vectored thrips. A comprehensive study of sweet-potato virus infection in wild species in Uganda, East Africa found as species of *Convolvulaceae* are known to be its alternative hosts. *Sweet potato feathery mottle virus* (SPFMV, genus *Potyvirus*), *Sweet potato mild mottle virus* (SPMMV, genus *Ipomovirus*) and *Sweet potato chlorotic stunt virus* (SPCSV, genus *Crinivirus*) all these viruses infect wild hosts in their natural habitats and wild hosts were potential virus reservoirs for spread to sweet potato crops.

- **Vectors**

Almost all plant viruses are transmitted by vectors which can be insects, nematodes, mites or fungi (Gallet and Blanc, 2018) [16]. *Caulimovirus, Tobravirus, Furovirus, Tungrovirus, Tritimovirus and Begomovirus* are transmitted by their respective vectors aphid, nematode, fungi, leaf hopper, Eriophyid mite and White fly. Viral diseases are due to specified molecular interactions via protein among virus and vector, understanding such interactions can support approaches for shielding plants from infection by interfering virus uptake and transmission (Dietzgen *et al*., 2016) [9].

- **Role of seeds, pollens, world trade and grafts in virus transmission**

In tomato a TMV strain is carried on the surface of tomato seeds externally and sometimes within their endosperm and testa, but never inside their embryo (Broadbent, 1965) [6]. Application of various treatments removed external seed contamination, but internally carried TMV was effectively removed by heating at 70°C for 3 days. Plants growing from TMV-contaminated seeds never became infected if left undisturbed after they are sown, but caught infection once they are transplanted. TMV then spread to the remainder of the crop. *Raspberry bushy dwarf virus* (RBDV; genus, *Idaeovirus*) is transmitted to raspberry seeds through the pollen, the embryo and also from infected pollen to plants (Murant *et al.*, 1974) [53]. Milne & Walter (2003) [48] in north-east Australia studied thrips-mediated pollen transmission of *Prunus necrotic ring spot virus* (PNSV; genus *Ilarvirus*) in stone-fruit orchards. They suggested that there is mounting circumstantial evidence that stone fruit flowers are oftenly infected by PNRSV via an interaction with virus-bearing pollen and this transmission mechanism is the best explanation of new infections in the field. In 1974, PepMV was isolated from a pepino (*Solanum muricatum*) crop that was growing in coastal Peru in the Andean region of South America. Symptomatology suggests yellow mosaic in young leaves of pepino virus is having stable particles, readily contact transmissible, has ability to infect a wide range of crops in the family *Solanaceae* including potato and tomato on sap inoculation. In 1999 it infected tomatoes in the Netherland (van der Vlugt *et al.*, 2000) [78] and then spread rapidly to several other European countries, China and North America. It is most economically important virus as it changes the look of tomato fruits and also cause surface contamination of its seed. It's presence in other continents was attributed to activities of international seed companies using South America to propagate tomato seed crops purchasable elsewhere and therefore the increased speed and volume of international trade in tomato seeds and fruits (Mumford & Jones, 2005) [52]. It spreads from plant-to-plant by contact and seed transmission. *Apple mosaic virus* (graft transmission) (Thokchom *et al*., 2009) [74].

- **Environment related factors**

Rainfall

Altered rainfall patterns cause huge viral epidemics transmitted by fungi or nematodes. Epidemics due to fungus-transmitted viruses are likely to become prominent in temperate regions (Jones, 2009) [33]. This is often due to enhanced activity and movement of vector zoospores resulting from enhanced temperature and soil moisture, which might increase the epidemics of *Furoviruses* and *Bymoviruses* of field crops like cereals, sugar beet and potatoes. The incidence of nematode-transmitted viruses in rain-fed crops in mid-latitudes is projected to diminish when there is decline in soil moisture (Jones, 2016). PMTV has its highest incidence in areas with very best rainfall which is correlated with increased incidence of the fungal vector (*Spongospora subterranea*) in wetter soils in Scotland (Cooper and Harrison, 1973) [8].

Wind

Wind may play a crucial role in determining the predominant

direction of spread both over long and short distance in addition to assisting or inhibiting spread of viruses by airborne vectors and pollen. Winged aphids are unable to fly when wind speed is very high, although their direction of flight is often influenced by the prevailing wind. In 1977, there an unexpected and big epidemic of MDMV in the corn crop of the northern US state of Minnesota, as the virus had been usually confined to southern states. Zeyen *et al.* (1987) [88] proposed that low-level jet winds rapidly carried infective aphid vectors from drought-stricken southern areas to Minnesota. Wind also leads to abrasive contact between plant leaves and helps in the spread of RYMV (Sarra *et al*., 2004) [68].

Air Temperature and carbon dioxide

The speed of multiplication and movement of airborne virus vectors is markedly affected by air temperature. For example, when conditions are reasonably warm, only then winged aphids tend to fly. However, very high temperature is effective in reducing certain aphid populations. Temperature have adverse effect on virus proliferation as it causes change in gene silencing (Chellappan *et al*., 2005) [7]. When plant hopper, vector of MRDV is exposed to 36°C it prevents virus replication in the vector and suppresses its transmission (Klein and Harpaz, 1970) [38]. It has been observed that elevated carbon dioxide concentration increases resistance in tobacco plants against PVY infection Matros (2006) [46]. But elevated CO2 can also have the reverse effect if it increases the biomass of virus-infected plants, thereby increasing the virus reservoir for spread to healthy plants by insect vectors, e.g. in oat crops elevated carbon dioxide concentration increases biomass of BYDV-infected oat plants which in turn increases the virus reservoir for spread to healthy plants by its aphid vector (Malmstrom and Field, 1997) [42].

Soil condition

The incidence of virus diseases is influenced by conditions of the soil in diverse ways. Highly fertile soils tend to have high incidence of viral diseases. For example, animal manure and a number of other inorganic fertilizers increase the incidence of *Leaf roll* and *rugose mosaic* disease in potato crops (Broadbent *et al*., 1952) [5]. Soil conditions also influence the survival of TMV in plant debris. Moist well-aerated soils inactivate the virus more, compared than dry, compacted or waterlogged soils (Johnson and Ogden, 1929) [30].

Over All effect of climate

The effectiveness of control measures such as some cultural measures and temperature sensitive single gene resistance is predicted to diminished by climate change and viral epidemics are projected to become less predictable, increasing difficulties in suppressing them successfully using latest management technologies. In many instances, losses in cultivated plants and damage to natural vegetation is likely to increase serious threat to world food security and plant biodiversity resulting from viral diseases (Jones, 2014) [35]. The extraordinary changes in ecosystem due to viral diseases can significantly bring changes in the range of cultivated plants and their cultivated area, results in the introduction of new kind of weeds and increase the activity of insect vectors promoting the disease spread (Harrington *et al*., 2001) [22].

Molecular epidemiology and ecology

The simple-ness of plant virus genome makes them ideal subjects for molecular epidemiological and ecological studies. These provide detailed information about viruses which were difficult to study, when biological assays or serological techniques were the only options. The genome sequences of virus isolates retain signatures that reflect their histories, provide information on epidemic patterns and gave an enhanced understanding of the ecological and evolutionary processes are made understood through molecular epidemiology and ecological studies. It provides current information, such as understanding of distant and rare events not provided by traditional field approaches. However, the patterns it finds can only be understood if factors such as natural host range and method of transmission in the field are familiar (Traore *et al*., 2009) [76]. For example, in tomato plants TYLCV epidemic in China occurred about 80 years (Xu *et al*., 2017) [83]. Molecular ecology uses genetic 'finger print' techniques for traceability of virus populations. Precise identification of virus strains and assessment of their spatial and temporal pattern of spread, and relative distributions within populations of cultivated plants, native plants and alternative weed hosts. Olarte-Castillo *et al*., (2011) [56] studied the molecular epidemiology of *Papaya ringspot virus* (PRSV; genus *Potyvirus*, family *Potyviridae*) isolates using phylogeography and molecular clock approaches from many parts of the world. The data generated suggests that PRSV originated about 2500 years ago in the Indian subcontinent and then dispersed first to Thailand and after then to the remainder of East Asia about 600 years ago. Transmission from India to other continents probably occurred within the last 300 years. The fine details of virus genetic variation present within epidemics that were impossible to obtain previously is now easily obtained by using currently powerful molecular tools (Jones., 2013) [34].

Case study:

Mirik *et al*, (2012) [49] performed remote monitoring of Wheat Streak Mosaic progression for site specific disease management in winter wheat by using sub-pixel classification of Landsat 5 TM Imaginery. The results of this case study indicate that there is great potential in using moderate resolution multi-temporal imagery with 30m pixel size for identifying and quantifying WSM within wheat fields. This can provide producers and administrators and quick and repeatable way for WSM management anytime and anywhere. When applied to specific management unit or farm, the geo referenced disease spots can then be used to facilitate more efficient treatment of WSM with Geographic Information System-based precision farming spray equipment.

Conclusion

With increase in the world population there is a need to control viruses and to mitigate crop losses. A good understanding of the virus ecology and epidemiology is required for developing the best management program for a crop viral disease. Molecular ecology and epidemiology are paving the way for diagnosing epidemic risk of viral diseases and enhancing sustainable response to economically important diseases. Viral diseases are due to specified molecular interactions via protein among virus and vector,

understanding such interactions can support approaches for protecting plants from infection by inhibiting virus uptake and transmission. Virus acquisition is promoted by attraction of vector to infected plants while their attraction to healthy plants promote virus transmission. Climatic change leads to virus epidemic both in cultivated as well as in natural vegetation due to emergence of previously unknown viruses, thus poses a serious threat to humanity and other species.

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