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Evolution of plant pathogens in the race for survival

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Abstract
Evolution can be termed as the process by which different kinds of living organisms are believed to have developed from earlier forms during the history of the Earth. In an interacting and dynamic environment, evolution is a constant process which helps the organisms to survive the changing environment and compete with other organisms for food and space. In the context of plant host and their pathogens, they are always entangled in an evolutionary arms-race to gain advantage over one another. Darwin was one of the first pioneers who studied evolution and put up the theory of evolution which includes the survival of the fittest. In a host-pathogen interaction, the host will always try to defend itself from the pathogen whereas the pathogen will find ways to penetrate the defense of the host. When a pathogen is introduced into a new area, limited resistance in the host and excessive aggressiveness in the pathogen can lead to explosive outbreak of the disease due to lack of prior co-evolution of the host and the pathogen. A study in the genome evolution of some plant pathogens provides useful insights how pathogens adapt to infect specific hosts. In today's world, climate change is a major issue. Similarly, climate change has a major role to play in the evolution of plant pathogens and how they interact with their host and the changing environment. Agriculture today, which is highly mechanized with high resource inputs, particularly in developed countries result in new disease occurrence due to loss of genetic variation and evolution of plant pathogens. Practices like monoculture over vast areas of land reduce the genetic variation of the host resulting in strong directional selection. One of the most common and most important examples of evolution is the evolution of the Ug99 race of wheat stem rust fungus which has evolved to overcome the resistance of major wheat varieties.

Keywords: Evolution, interaction, Ug99

Introduction
Evolution in simple words can be defined as the process by which different kinds of organisms are believed to have developed from their earlier forms during the history of the Earth. It is the change in the heritable characteristics of biological population over successive generation. Evolutionary processes give rise to biodiversity at every level of biological organisation, including levels of species, individual organisms and molecules. Evolution is a means of survival of an organism in an ever-changing dynamic environment. If an organism does not evolve new mechanisms to adapt to new environmental challenges then another form of more evolved and sophisticated organism may take-over the space of the less evolved organism.

When we talk about an interacting dynamic environment, then in this situation evolution can never be an isolated case. Evolution of one species is always accompanied by the co-evolution of another species. Similar is the case of plant-pathogen interaction, the evolution of the plant is always associated with the co-evolution of its pathogen and vice-versa. If the plant or the pathogen does not co-evolve with each other then they will be taken over by better evolved species respectively. The plant pathogen and their host are always entangled in an evolutionary arms-race to gain advantage over one another. The pathogen always tries to breach the defence of the plant whereas the plant always tries to defend itself from the attack of the pathogen. The plant may alter their genome to resist the attack of the pathogen or may as well change their phenotype for the same.

Darwin was the first person who studied evolution of species in depth when he noticed the different beak pattern of finches in the Galapagos Islands which were evolved according to their feeding habits. He proposed that all species have variation within them and that some variations are inherited and some are random.

Climate change is also a factor which may bring about evolution of an organism and may change the host pathogen interaction pattern. It may lead to phenotypic plasticity or the

organism to migrate to new areas or to evolve new attributes to survive the change in the climate.

Several plant disease epidemics have been reported which have been caused due to evolution of a pathogen against a certain host for which the host was not resistant enough. The spread of the Ug99 strain of wheat rust pathogen is a classic example of it which was first reported in Uganda in 1999 and has spread to almost all major wheat growing areas of the world.

Mechanism of evolution

There are four basic mechanisms of evolutionary changes:

1. **Mutation:** In this case, one of the genes of an organism in the population may mutate and lead to the emergence of an organism with a different phenotype and result in an evolutionary change.
2. **Migration:** In this case, an organism from one population may migrate to a different population of the same species with different characters and breed among them to produce offspring which may be different from the parent population.
3. **Genetic drift:** In this case, certain incidents may lead to the emergence of a dominant new species. For example, if in a mixed population of green and brown beetles, by accident a large number of green beetles are killed due to an accident, then the population will be composed mainly of brown beetles and slowly the green beetles will be outnumbered.
4. **Natural selection:** In this case, the appearance of the organism and its selection for survival decides the evolution of the organism. For example, on the bark of the tree green beetles are more visible than the brown beetles and are thus more targets for birds which feed on them. Slowly the population of the green beetles will decrease and will be overtaken by brown beetles.

Theories of Evolution

Darwin's theory of evolution

Darwin studied the theory of evolution on the Galapagos Islands on the beak pattern of the bird, finches. He observed that the beak pattern of the bird differed with the feeding pattern of the bird. He also observed that the beak pattern of the bird evolved with their evolution of their feeding habit. Thus, from this he came to some conclusions which have been summarised below:

1. All species of organisms arise and develop through the natural selection of small, inherited variations that increase the individual's ability to compete, survive and reproduce.
2. All species have variation within them.
3. Some variation inherited; some random.
4. Organisms produce more offspring than which actually survive. Survivors therefore have some characteristics which contribute to survival.

Red Queen Hypothesis

"Here, you see, it takes all the running you can do, to keep in the same place"

The Red Queen hypothesis proposes that organisms must constantly adapt, evolve and proliferate not merely to gain reproductive advantage but also simply to survive while pitted against ever-evolving opposing organisms in an ever-changing environment. In simple words it means that if an organism stops evolving according to the needs of the environment, its place in the dynamic environment will be taken over by another more evolutionarily advanced organism. It means that there is always an evolutionary arms race going on between an organism and its surrounding. Plants and their pathogens have, since time immemorial, been engaged in an evolutionary arms race.

Court-Jester Hypothesis

This hypothesis somewhat contrasts the Red Queen hypothesis. It states that abiotic forces rather than biotic competition between species function as a major driving force behind the process of evolution which produce speciation. It implies that events random in respect to the biota occasionally change the rules on the biotic playing fields. As a result of this it leads to accelerated biotic response from the organism. Court-Jester hypothesis basically puts forward the idea that changes in the physical environment rather biotic interactions themselves are the initiator of major changes in organisms and ecosystems leading to evolution of the organism according to the need.

Scenario of evolution

Disease pressure will favour changes in host proteins that are target for pathogen attack to neutralize the effect of virulence factors from the pathogen. The pathogen on the other hand will be pressurized to keep updating its virulence factors to be able to continue to attack and cause disease in the changing targets. Some of the proteins that are produced by a pathogen during infection can be directly or indirectly recognised by the products of R genes resulting in disease resistance. This is the basis of the zig-zag model of resistance.

Zig-Zag model of resistance

According to this model, when a pathogen attacks a host, it leads to the signalling of Pathogen-associated-molecular-patterns (PAMPs) in the host. Then the host, to counter the attack of the pathogen activates PTI i.e. PAMP-triggered-immunity. Again, the pathogen releases certain effectors to suppress the effect of PTI which is known as Effector-Triggered-Susceptibility (ETS). The plant host recognise these effector proteins and in turn activates the Effector-Triggered –Immunity (ETI) which suppress the effector proteins of the pathogen. This arms race in pathogenicity of the pathogen and the resistance capacity of the host takes place until a threshold for hypersensitive reaction or Programmed cell death is reached. There is always a direct or indirect recognition of the effector proteins in the plant host system.

Whenever two interacting organism co-evolve, there will always be environments which may be favourable for one organism and unfavourable for the other organism. In case of plants and their pathogen co-evolving, there are certain factors which may be favourable for the plant and unfavourable for the pathogen and vice-versa.

Co-evolutionary advantage of plants

1. Sexual recombination might provide effective evolutionary compensation by generating novel combination of resistance factors (Red Queen hypothesis).
2. In short-term, sexual reproduction provides host with a mechanism for the production of genetically unique progeny in each generation by shuffling existing defense related alleles.

3. Sexual reproduction can also increase defense polymorphism in host population by generating new allele through intragenic recombination.
4. In the long run, sexual reproduction facilitates the formation of novel mutant allele and allelic combination favourable for host defense and enhances the removal of deleterious mutations that otherwise could accumulate in plants during asexual reproduction.
5. Plant population in nature are highly patchy with many diverse genotypes of the same species as well as phylogenetically closely and more distantly related species distributed in the community.
6. Population level resistance as well as other genetic and ecological traits may vary greatly from year to year. This spatiotemporal feature of the genetic structure of the plant population can have negative effects on the evolution of corresponding pathogen.
7. High genetic variation and environmental heterogeneity among host population mean that a large percentage of pathogen propagules may not lead to infection because they cannot find the right host at the right time and place.

Co-evolutionary advantage of plant pathogens

1. Evolutionary rates in species are negatively correlated with generation time. Species with shorter generation time tend to have higher evolutionary rates possibly because their genomes are copied more frequently, generating more DNA replication errors per unit time.
2. In a host-pathogen interaction, pathogens are generally at an evolutionary advantage because of shorter generation time compared to their host.
3. Over a few pathogen generations, advantageous mutants can come to dominate a pathogen population, thereby rendering host defense systems ineffective. Clearly, this advantage is magnified in cropping systems in which host defense are relatively static and genetically homogeneous over a range of spatiotemporal scales.
4. Plant pathogens are also at an evolutionary advantage owing to their typically very large population. Single disease lesion may generate large numbers of pathogen propagules, each of which can serve as independent unit of reproduction.
5. Population size contributes positively to the generation of new mutants and negatively to the loss of existing mutants.
6. Natural population with large effective size tends to have greater genetic variation as more mutants are expected and fewer are lost due random genetic drift.

Evolution via inter-specific hybridisation

Plant disease epidemics resulting from introduction of exotic pathogens are a well-known phenomenon. Limited resistance in the host and excessive aggressiveness in the pathogen reflect their lack of prior co-evolution and can result in explosive outbreak of disease. Introductory events also present a window of evolutionary opportunity for the pathogen. When introduced into new environment, the pathogen will be subject to novel or episodic selection, reflecting sudden exposure to new biotic and abiotic influences such as new host population, new vectors, new competitors or a different climate. These influences provide the potential for rapid evolution. Contact that can occur between closely related but previously geographically isolated pathogens can present an opportunity for rapid emergence of new or modified pathogens via inter-specific gene flow. In its endemic location, a plant pathogen tends to be subjected to routine selection constraints, favouring maintenance of a relatively stable population structure over time. Falling within the category of episodic selection is the sudden contact that can occur between closely related but previously geographically isolated pathogens as a result of introductions. The potential outcome when closely related but previously geographically isolated pathogen come in contact depends on many factors including the frequency of niche contact, the nature of any genetic barrier to hybridisation, the degree to which the genomes of the two species can recombine and the ability of any resulting hybrid to compete with the 'parent' species. The opportunity for hybridization between previously alopatric pathogens is likely to increase as world trade in plants intensifies and more plants and their associated pathogens are introduced into new bio-geographic environments. Increasing stress level on plants due to global environment disturbance effects such as climate change and pollution, together with planting of more host species geographically off-site, are likely to increase the availability of hosts suitable for colonization by novel genotypes.

Trade-offs and fitness penalties

Trade-offs and fitness penalties can impede the emergence of super-infective pathogens. By gaining or accumulating infectivity, pathogens are able to infect more hosts but at the same time reduce their competitiveness when the corresponding resistance is absent. Isolates of the rust pathogen *Melamspora lini* carrying more infectivity factors against *Linum marginale* tend to produce fewer spores per pustule than less infective strains. Higher aggressiveness resulting from greater host exploitation potentially increase plant-pathogen reproduction rates, it also shortens the time the pathogen is able to persist on an infected host and be transmitted. This trade-off between reproduction and transmission often selects for pathogens with inter-mediate aggressiveness, although optimum strategy depends on host and pathogen life history.

Classic example

The *Myxoma* virus was introduced into Australia to control European rabbit population in 1950. Initially the virus was extremely aggressiveness leading to high rabbit mortality. However, a sharp drop in aggressiveness was observed after a few pandemics as the highly aggressive strain was killing the rabbit host too quickly thereby reducing its chance of being transmitted to new rabbit population. Hence the virus remained confined to one location only and gradually lost its infectivity.

Genome evolution in plant pathogens

Food security is of global importance and crop disease caused by plant pathogens are a major constraint to agriculture worldwide. Many of these pathogens have a similar biotrophic life stage during which they contact host cells and secrete effector proteins that alter plant responses to infection. Biotrophic infection strategies have evolved independently in diverse lineages of plant pathogens. Pathogen structures secrete large number effector proteins that enter the host cells and manipulate defense response and cellular metabolism. Many effectors require the short amino acid motif RXLR for entry into plant cells independently of the other pathogen machinery. The study found that most pathogen genes and genomes regions to be highly conserved but genes involved in

host-pathogen interaction appear highly diversified especially in the predicted RXLR containing effectors.

For example: The *Hyaloperonospora arabidopsidis*, which is a pathogen of the model plant *Arabidopsis thaliana*, is exclusively biotropic and cannot be grwn in culture. This pathogen is believed to have evolved from *Phytophthora* like hemibiotrophic ancestors. It was found that its genome contains a unique set of diversified RXLR containing effectors but has lost many of the hydrolytic enzymes that Phytophthora species used to digest host cell walls, as well as many of the genes that induce host cell death. The reduction of these protein classes is inferred as resulting from selection for stealth allowing *H. arabidopsidis* to avoid trigger host defense mechanism during its extended biotrophic interaction. Likewise it was found that genomes of three species of fungal powdery mildew pathogen are deficient in several classes of conserved primary and secondary metabolism genes. The powdery mildew genomes encode a unique class of secreted proteins with a conserved acid motif YXC. These genes are highly diverse among the three species which infect very different host plants suggesting that most of these effectors are associated with host species specific adaptation.
A study was made comparing the genome of *Sporisorium reilianum* to that of the related fungi *Ustilago maydis*, both of which infect maize. Most of the predicted secreted effectors are common to both species but show much higher divergence than the rest of the genome. Thus, even within a host species, selection imposed by the host immune system or selection that targets different host process can lead to rapid diversification of pathogen effectors

Plant pathogen evolution and climate change
Plant pathogens have three major adaptive responses to climate change:

1. To exploit the phenotypic plasticity: It is the capacity of a genotype to exhibit variable phenotypes in different environments. It enables individual genotypes to adapt to changed environment without the need for novel mutations.

For example: *Passiflora suberosa* leaves show different phenotypic characteristics under different environment of varying sunlight.

2. To migrate to areas with more suitable climate: Organisms generally follow temperature optima. There is a growing concern that climate change may enable the establishment of alien species in hitherto unsuitable regions. Plants from new origins will bring along new and unknown invasive pathogens, possibly well adapted to the altered climatic condition in the country of destination. For example, *Phytophthora cinnamomi* is sensitive to frost and will benefit from warmer winters possibly expanding is geographic range.
3. To evolve new attributes: Climate change may act as strong evolutionary force on plant pathogens. Climate change altering plant metabolism and quality may impose additional indirect selective forces on pathogens and pests. Major pathogens have emerged through three mechanisms i.e. host jump, hybridisation and horizontal gene transfer.

For example: *Puccinia psidii* jumped from native Myrtaceae to introduced Eucalyptus trees in South America.

Phytophthora alni that infects alders in Europe is a hybrid of *Phythophthora cambivora* and *Phythophthora fragariae. Phythophthora* species had never been reported on alders before the rise of hybrids.
Ophiostoma novo-ulmi acquired a gene from *Ophiostoma ulmi* and evolved new attributes.

Disease occurrence and evolution of plant pathogens in modern agricultural practice
In modern society, agriculture has become highly mechanized, industrial-style systems with high resource inputs, intensification and reduced diversity. These shifts impose crucial changes on the patterns of host-pathogen dynamics and generally favour widespread epidemics and thus rapid evolution of pathogen infectivity.

Monoculture: Most modern agriculture is characterized by situations in which crop varieties with closely related genetic background are planted across large areas especially in developed countries. Growing crops with identical physiological characters and genetic make-up can result in greater yield per unit area because management procedures can be standardised and mechanized, competition from other species for nutrients can be more easily controlled and better use can be made of available light and space. Monocultures in agriculture are mainly composed of single varieties that have been bred for high yield and resistance to certain disease. In short term, the narrowed genetic base of crops in monoculture has made them more vulnerable to disease epidemics. Pathogen strain that can infect one individual in a crop can affect all and hence quickly spread over an entire region because of reduced environmental heterogeneity and genetic barriers do not exist to prevent the spread. Monoculture imposes strong directional selection on pathogens and their widespread use is considered to be responsible for rapid loss in the effectiveness of R genes and agrochemicals. Novel infectivity that emerges in pathogen population as a result of single point mutation can quickly increase in frequency through strong directional selection and spread through natural or human-mediated gene flow even if overall fitness of new pathogen variant is somewhat lower.

Production intensification
Minimizes the effect of trade-offs that restrict the emergence of super infectivity. Growing crops over wide regions and agricultural intensification tend to minimize spatio-temporal heterogeneity thereby potentially favouring increased pathogen infectivity and aggressiveness. Continuous tissue availability associated with agricultural intensification not only supports large pathogen population size, which in turn minimize the effect of drift with regard to reducing pathogen genetic variation but is also likely to diminish trade-offs between aggressiveness and transmission in pathogen population as continuous cropping provides new tissues to support further growth and reproduction of the pathogen in the next season.

Global connectivity: Facilitates disease invasion and the spread of novel pathotypes, which increase the evolutionary potential of pathogen population. Increased international trade in agricultural products, exchange of plant materials for research and teaching and hitchhiking of pathogens via various transportation modes have greatly increased the spread of many infectious plant diseases across major geographic boundaries. Vulnerability of many agricultural

crops to exotic pathogens often occurs because breeding has proceeded for many years without challenge by the relevant pathogen. As a consequence, through combination of genetic drift and negative selection associated with potential fitness costs, both major and minor gene resistance may be lost when diseases are absent. In addition to the invasion of new diseases, globalization can promote the spread of new infectivity and agrochemical resistance among existing pathogen population, for example the invasion of *Phytophthora infestans* mating type A2 into Europe in the 1980s. This caused the simultaneous breakdown of host R genes and rendering the agrochemicals ineffective across a large geographical area within a short time.

Use of Agro-chemicals: Continued application of agrochemical on a large geographic scale not only cause adverse environmental effects but also speed up changes in the pathogen population and increase their overall aggressiveness.

Breeding: Breeding for high yield and quality artificially removes undesired traits, which may be crucial for hosts to combat pathogens. Some traits that are important for the host are often targets for removal in human-directed breeding programme or may be screened out unintentionally by plant breeders due to negative co-relations between host resistance to pathogens and crop productivity when pathogens are absent.

Case study 1

The Emergence of Ug99 races of the stem rust fungus is a threat to world wheat production

Ug99 is a virulent strain of old crop disease black stem rust that has evolved to overcome the resistance prevalent in wheat varieties during the Green Revolution of the 1960s. Ug99 was first observed in Uganda in 1999. It has overcome many wheat resistant genes like Sr24, Sr25, Sr26, 27, 31 and 38. After Uganda, the spore of Ug99 spread to Kenya through wind current followed by Ethiopia, Yemen, Iran, southern Sudan thereby threatening regions of the near east, eastern Africa, central and southern Asia. It not only spreads rapidly but also generates new variants able to break the resistance of the wheat varieties grown in these regions. The deleterious effect of Ug99 is more pronounced with stem rust. About 80% yield loss were recently recorded in Kenya and Uganda, though fortunately neither of these countries rely completely on wheat as staple food.

The predicted pathway: The FAO has already alerted Afghanistan, India, Pakistan, Turkmenistan, Uzbekistan and Kazakhstan about the dangerous wheat fungus that may cause huge loss to wheat production by destroying entire fields. Stem rust outbreak caused by Ug99 depends on the following key factors: area and distribution of susceptible material; optimum temperature and moisture; amount of initial inoculum and air movement.

Ug99 can attack India following either of the two routes:

1. Route A: East Africa directly to southern Pakistan/western India (has no known precedent and of much lower probability)
2. Route B: East Africa-middle east-west Asia- South Asia (has higher probability). Conditions favourable for outbreaks of epidemics currently exist in the migration path of highest probability.

The ICAR in collaboration with the CIMMYT started a testing programme of wheat at Njoro in Kenya since 2005. The tests have shown that there are 13 Indian wheat varieties with resistance to Ug99 and its variants. This diverse resistance in wheat varieties will help them fight against the deadly pathogen and other future threats of this magnitude.

The emergence of Ug99 races of the stem rust fungus is a threat to world wheat production

Because of the susceptibility of 90% of the wheat varieties grown worldwide, the Ug99 group of races was recognised as a major threat to wheat production and food security. Demand for wheat in the developing countries is projected to increase 60% by 2050 at the same time, climate change induced temperature increases are expected to reduce wheat production in developing countries by 29%. The last major stem rust epidemic occurred in Ethiopia in1993 and 1994 when Enkoy, a popular wheat variety suffered major losses however the rest of the world has remained unhurt by stem rust for the last 3 decades. Severe stem rust infections were observed on CIMMYT wheat nurseries planted in Uganda in 1998. Because these materials were known to be resistant to stem rust, the susceptibility drew large attention. Uredinospores were collected from field nurseries for avirulence/virulence phenotyping in a greenhouse in South Africa in early 1999 on a set of wheat differentials carrying designated race-specific resistant genes. A new race with novel virulence on resistant gene Sr31 was detected and designated as Ug99. Race Ug99 is the first known race of *P. graminis tritici* that has virulence to resistant gene Sr31. Race Ug99 carries virulence to gene Sr31, but this unique virulence is present together with virulence to a majority of the genes of wheat origin as well as virulence to gene Sr38 introduced into wheat from *Triticum ventricosum*. This striking virulence combination in Ug99 explains the widespread susceptibility of wheat varieties and germplasm worldwide. Loss of key resistant genes notably Sr24 and Sr36 was detected in Kenya in 2007.The pathogen is changing rapidly; 7 variants are now recognised as being part of the Ug99 race lineage. All are closely related, having nearly identical DNA fingerprints but differ slightly in their avirulence/virulence profiles. Simple sequence repeats marker similarity of Ug99 races indicates their evolution from a common ancestor. The presence of Ug99 variants in southern Africa has potential implications for other wheat growing regions. The main concern is the regions' potential as source of onward movement to either Australia or the Americas although with very low probability. Watson and de sousa provide strong evidence for three wild borne transmission events of *P. graminis tritici* races from south Africa to Australia during the period 1925-1983.Within the framework of the Borlaug Global Rust initiative (BGRI) and focussed on the Ug99 race lineage, the Global Cereal Rust Monitoring System (GCRMS) has been initiated. In its current form GCRMS initiates and disseminates up-to-date information on stem rust incidence and severity as well as race. Over 200,000 wheat varieties, germplasm collections and advanced breeding materials were screened during 2005-2010 for resistance to Ug99 and its derivative races at Njoro, Kenya and parts of Ethiopia. Varieties exhibiting adequate resistance to Ug99 accounted for only 5%-10% of the total areas. The huge susceptibility areas in Pakistan and India and China result from the predominance of either mega-varieties such as PBW343 in India and Inqualab 91 and Seher 2006 in Pakistan or the susceptibility of most varieties in china. In contrast to bread wheat, a greater proportion of durum wheat

varieties and germplasm show resistance to races of the Ug99 lineage. This difference is largely attributed to resistant gene Sr13 present in high frequency in durum wheat germplasm. In most wheat growing regions of the world, existing environmental conditions will favour stem rust infection which could build to epidemic build-up. Plus, the fact that susceptible wheat varieties are grown over large areas and that a large proportion of current breeding materials are susceptible to Ug99 and other newly identified races, means that these pathogens have the potential to cause a wheat production disaster that would affect food security worldwide.

Case study 2

Dutch Elm epidemic in Europe

Dutch elm disease, a major ecological accident of the 20th century, is providing remarkable insights into rapid evolution of a plant pathogen outside its endemic environment. Elm trees are confined mainly to the temperate regions of the northern hemisphere. China and Japan have a total of about 25 elm species, while Eurasia, north America and the Himalayas each have about 5-6 species. Dutch elm disease is the elm's main enemy which is caused by ascomycete fungi of the genus *Ophiostoma*, that spreads within the tree's vascular system. It is transmitted by the elm bark beetles of the genus *Scotylus.*

Dutch elm disease was unknown in Europe and North America before 1900, but there have been two enormous destructive pandemics of the disease across the northern hemisphere in this century. These two endemics were caused by the spread of two different species of fungal pathogen, *Ophiostoma ulmi & O. novo-ulmi*. The geographic origins of the pathogens remain unknown but are believed to have come from Asia. The optimum temp for growth of *O.novo-ulmi* is approx 22^0C and *O.ulmi* is 28ºC suggesting that the former may be naturally adapted to a temperate and the latter to a sub-tropical environment. *O.ulmi* is moderate and *O.novo-ulmi* is highly aggressive pathogen of European elm. These two are genetically divergent based on molecular fingerprint study.

O. novo-ulmi is not a single entity but exists as two distinct forms called the Eurassian (EAN) or North American (NAN) races. EAN is slightly less pathogenic than NAN. They also differ in number of properties like colony morphology, perithecial sizes and molecular fingerprint. Crosses with both the species are highly fertile and breed true. In crosses between them, *O. novo-ulmi* rejects *O. ulmi* as fertiliizing (male) partner, although *O. ulmi* can be fertilized by *O. novo-ulmi.* The resulting ascospore progeny show a remarkable range of non- parental phenotype including female sterility. Many are of low vigour and fitness. Most are weaker pathogens even than the *O. ulmi* parent. This reproductive isolation has been interpreted as evidence that they were previously geographically separated as well as differently adapted species.

The first pandemic: The first pandemic of Dutch elm disease caused by *O. ulmi* began in Northwest Europe around 1910. Thereafter the disease spread rapidly eastward on a series of epidemic fronts across Europe and into southwest Asia.

The second pandemic: In the early 1970s, a severe new Dutch elm disease outbreak occured in Britain and neighbouring parts of Europe, caused by previously unknown *O. novo-ulmi.*

The discovery of *O.himal-ulmi:*

China was long considered the origin of the Dutch elm disease but a survey in 1986 revealed no evidence of the pathogen. However, a survey in the western Himalayas led to the discovery of an entirely new endemic species of Dutch elm disease pathogen now named as *O. himal-ulmi.* It is very aggressive to European elms. It shares many other physiological similarities to *O. novo-ulmi.* But it is apparently in natural balance with the native Himalayan elms and the elm bark beetles.

Replacement of *O. ulmi* by *O. novo-ulmi* and the potential for genetic exchange

Commonly when *O. novo-ulmi* arrives at a new location, *O. ulmi* is already present. *O. novo-ulmi* rapidly replaces *O. ulmi* and the latter declines at about 10% of the total pathogen population per annum. Classic example of survival of the fittest. Direct competitive antagonism of *O. ulmi* by *O. novo-ulmi* and one of them is more pathogenic than the other. During this replacement process the close proximity of these two in the bark around the beetle galleries provides physical opportunity for inter-specific genetic exchange. Moreover, they are not fully reproductively isolated as limited sexual hybridization is possible between them. New research has shown that rare *O. ulmi-O. novo-ulmi* hybrids do occur in nature but are transient, quickly disappearing in competition with the parent species. However, they could act as genetic bridges allowing unilateral gene flow from one species to another.

Introgression of *O. ulmi* DNA, including a pathogenicity gene

One clue that gene flow was occurring between *O. ulmi & O. novo-ulmi* came from RFLP based DNA fingerprinting studies. In a search for cloned DNA fragments that unambiguously discriminated *O. ulmi* isolates from *O. novo-ulmi* isolates when used as hybridization probes, some EAN *O. novo-ulmi* isolates were found to exhibit rare *O. ulmi* like DNA polymorphism, suggesting they had acquired *O. ulmi* gene via introgression.

Rapid changes in *O. novo-ulmi* population structure

Another clue to the existence of gene flow between *O. ulmi & O. novo-ulmi* has come from rapid changes in the genetic structure of local *O. novo* populations. These changes include in particular a sudden increase in the frequency of so-called vegetative compatibility (VC) types. If adjacent colonies of *O. novo-ulmi* are of different VC type, viruses cannot pass readily from one colony to another via hyphal fusions because the fusion cells die. However, when the adjacent colonies are of same VC type, viruses can spread rapidly between them because the fusion cells are functional. Each time *O. novo-ulmi* has arrived at a new location in Europe, it has usually spread as a clone of a single VC type. These clones are of uniform colony morphology and single mating type. Deleterious viruses tend to spread abundantly in the expanding VC colonies. However, within a few years, the clonal population diversifies into multiple new VC types. This change is accompanied by a sudden increase in diversity in colony patterns and other characteristics and by the appearance of other mating types. Furthermore, as the new VC types appear the frequency of deleterious viruses in the population falls rapidly. In New Zealand, an immigrant VC clone has continued to persist unchanged for almost a decade.

In this case *O. ulmi* was not present prior to the arrival of *O. novo-ulmi.*

Two inferences: *O. novo-ulmi* clones tend to diversify into new VC types only where *O. ulmi* was originally present as in Europe and North America. Second, only where the virus activity in the clones is very high as in Europe do the clones diversify both rapidly and extensively. These inferences suggest that the novel VC genes are acquired by *O. novo-ulmi* from *O. ulmi* and the selection pressure exerted by viruses favours the survival of the novel VC types over the original VC clones. Results of a molecular study to test the hypothesis that the novel VC genes come from *O. ulmi* are consistent with the hypothesis. Segments of *O. ulmi* DNA has been found flanking the novel vc genes in *O. novo-ulmi.*

Transfer of viruses from *O. ulmi* to *O. novo-ulmi*

There is a possibility that the deleterious viruses that spread in the *O. novo-ulmi* vc clones are also acquired from *O. ulmi.* A preliminary comparisiion of viruses in *O. ulmi* and *O. novo-ulmi* isolates obtained from the same epidemic front site in Europe indicates very close similarity in their RNA sequences.

Conclusion

As a conclusion, it can be stated that evolution is an ever-occurring phenomena in the dynamic environment that we live in. Evolutionary arms-race always takes place among organisms interacting in the environment for food and space for survival. In the host-pathogen interaction dynamics, evolution cannot be an isolated event. Co-evolution of two or more interacting species always occurs in an environment. Evolution is essential for the survival of interacting species in the environment. If a species does not keep on evolving with the changing environment and condition, its survival will become difficult as it will have to compete with other more evolved species for food and space. More evolved species will obviously be more efficient than the less evolved organism at gathering food and survival thus jeopardizing the existence of the less evolved species.

With the change in cultivation practices and changing climatic condition, more and more pathogens are evolving to attack and infect our crop plants. These pathogens are new and their epidemiology and mode of infection may not be known to us leading to epidemic disease development. It is essential that interaction among pathogens is studied in new environments with new organisms so that a track of their evolution can be maintained. This will be helpful if in case a highly evolved pathogen attacks our crop then we can go back to the origin of the evolution of the pathogen and make strategies for the effective management of the pathogen.

References

1. Braiser CM. Rapid evolution of introduced plant pathogens via interspecific hybridization. J Biosci. 2001; 51:123-133.
2. Burdon JJ, Thrall PH. Co-evolution of plants and their pathogens in natural habitats. Science. 2009; 324:755-756.
3. Coates ME, Beynon JL. *Hyaloperonospora arabidopsis* as a Pathogen Model. Annu. Rev. Phytopathol. 2010; 48: 329-345.
4. Dodds PN. Genome Evolution in Plant Pathogens. Science. 2010; 330:1486-1487.
5. Gilbert GS, Parker IM. Rapid evolution in a plant-pathogen interaction and the consequences for introduced host species. Blackwell Publishing Ltd. 2010; 3:144-156.
6. Lo lacono G, van den Bosch F, Paveley N. The evolution of plant pathogens in response to host resistance: Factors affecting the gain from deployment of qualitative and quantitative resistance. J Theor. Biol. 2012; 304:152-163.
7. Khavkin EE. Potato Late Blight as a Model of Pathogen-Host Plant coevolution. Russ J Plant Physl. 2015; 62:439-451.
8. Shang Y, Xiao G, Zheng P, Cen K, Zhan S, and Wang, C. Divergent and Convergent Evolution of Fungal Pathogenicity. Genome Biol. Evol. 2016; 8(5):1374-1387.
9. Singh RP, Hodson DP, Bhavani S, Njau P, Jin Y, Singh S, Singh PK, govindam V Herrera-Foessel S, Huerta-Espino J. The Emergence of Ug99 Races of the Stem Rust Fungus is a threat to World Wheat production. Annu. Rev. Phytopathol. 2011; 49:465-481.
10. Van der Does, HC Rep M. Virulence genes and the Evolution of Host specificity in Plant-Pathogenic Fungi. MPMI. 2007; 20: 1175-1182.
11. Zhan J, Thrall PH, Papaix J, Xie L, Burdon JJ. Playing on a pathogen's weakness: Using evolution to guide sustainable plant disease control strategies. Annu. Rev. Phytopathol. 2015; 53:19-43.