Spatial Patterns of Host-Pathogen Complex: A Geostatistical Insight

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A B S T R A C T

Present study discussed spatial pattern techniques with respect to plant pathogens. Study was framed under four segments related with (a) basics of spatial pattern analytical tools and ideal strategies for their detection, (b) overview of certain techniques largely being utilized for different host-pathogens, (c) multivariate analysis to identify patterns for use of various spatial indices and finally (d) identification of gaps relate with spatial pattern analysis of pathogens. Spatial pattern indices used in fifty nine host-pathogen studies were evaluated and there utilization frequencies were accessed through skweness, kurtosis, Kolmogorov-Smirnov and Chi-square. Agglomerative Hierarchical Cluster classified the studies into six different groups. Following gaps were identified (a) some indices still not worked out for pathogens spatial patterns detection (b) a generalized framework still requires for phylogenetic connection between hosts or with its family and with distribution patterns of pathogen, (c) inter and intra-steric (pathogen and host) factors controlling spatial patterns of pathogen rarely approached, (d) spatiotemporal aspects of pattern detection are also not fully explored and (e) very few studies have approached management practices based on spatial patterns of pathogens. Some open ended tasks are: what is the line of difference between specialized and non-specialized pathogen for their spatial pattern? Is the availability of high inoculums ensuring uniform pattern? (if all other conditions are conducive), factor that controls random and clumped patterns and information's on year to year variations of pathogen spatial patterns with similar crop/host.

Introduction

The horizontal organization of species can be effectively described by explaining its physical arrangement or distribution within the community which in turn can prove to be an utilitarian tool in relation to spatial patterns of a species. This physical arrangement refers to two-dimensional location of individual (e.g., latitude and longitude), analogous to the (X, Y) coordinates of the species. Knowledge of species spatial patterns might give a more noticeable command for distinguishing mechanism such as ecosystem functioning and stability, designing adequate management, recuperation, or restoration actions (Mathur 2014) and/or modification of micro-climate and biomass partitioning (Mathur and Sundaramoorthy, 2008).

With respect to the impact of spatial distribution of the host on the spatial distribution of the pathogen, model of McRoberts *et al.,* (1996) predicted that

aggregation of the host reduces the speed of disease development. Bolker (1999) showed that spatial discontinuities introduced by host aggregation causes the fluctuation in infection rate. Willocquet *et al.,* (2000) with rice-*Rhizoctonia solani* complex had studied disease level with host spatial pattern while, Gosem and Lucas (2009) simulated the disease development with reference to host aggregation magnitude. Association between plant density and disease distribution for Basal stem rot in oil palm, and for seven C4 grasses - *Pyrenophora tritici-repentis* and *Gaeumannomyces graminis* var. *tritici* (Ggt) were evaluated by Azahar *et al.,* (2011) and Cox *et al.,* (2013), respectively. Similarly, spatial pattern knowledge appertains to pathogen and plant disease can also enhance our understanding for epidemics dynamics and disease spread mechanism.

For example, random pattern of infected plants would suggest that the pathogen does not spread within the field or in other words, no secondary transmission occur and it can also enhance our understanding to solve disease dynamic issues like sources of inoculums, mechanisms of propagule dissemination, and reproductive strategies of the pathogen population (Wu and Subarea 2004). From geostatistical point of view in random pattern "the probability of plant or plant unit being diseased is independent of the disease status of other plants" however, for non-random pattern like uniform or clustered "the probability of plant or plant unit being diseased is not independent of the disease status of other plants". In general notion, for host-pathogen, uniform pattern indicate a regular arrangement of infected and healthy plants. While random means that all distinguishable arrangements of infected and healthy plants are equally possible, or that in every point in the crop there is the same probability of a plant being infected. In a clustered pattern, every plant in the field does

not have an equal probability of being infected so that a diseased plant increases the probability of nearby plants being infected (Trumper and Gorla, 1997). Host original distribution type and utilized mathematical approaches significantly affect such patterns.

Here a compressive effort has been made on various attributes of spatial pattern analysis of plant host-pathogens. This will provide detailed insight about tools and techniques utilized for pathogen distribution patterns. Present paper segmented into four, first one related with basics of spatial pattern with appropriate examples of different host-pathogens. Segment two dealt with ideal strategies for spatial pattern detection and overview of certain techniques largely being utilized for different host-pathogens, in third segment forty different studies were treated with multivariate approaches to get an insight about the trends of indices selection and fourth segment pertains to existing gaps and with their preliminary available work, if any.

Segment A

In general, a population will have of three basic spatial patterns: (1) regular pattern in which individuals within population are uniformly spaced; (2) random (or chance) pattern in which all individuals have an equal chance of living anywhere within an area; and (3) aggregated pattern in which individuals have a higher probability of being found in some area than in others (Mathur, 2014). These three basic spatial patterns commonly described by three probability distribution: (1) the regular pattern is quantified by the binomial distribution of which the variance (σ^2) is less than the mean (μ), i.e., $\sigma^2/\mu < 1$; (2) the random one is described by Poisson with the variance equal to the mean, i.e. $\sigma^2/\mu = 1$; and (3) the clumped one is modeled by the negative binomial distribution that has the mean $\mu = kp$ and the variance $\sigma^2 = kp(1+p)$,

and thus $\sigma^2/\mu = 1+p>1$ (Yin *et al.,* 2005). In this k is the degree of aggregation and $p = \mu/k$.

Quantitative evaluation of spatial dynamics of diseases may be related or linked with certain issues: is the disease pattern is aggregated?, how does the degree of aggregation fluctuate with time, is there any spatial dependence with progress of disease?, can such spatial dependence be quantified?, what is the rate of disease spread, disease gradient or spore dispersal gradient?, and what is the link between disease pattern and its management?.

For plant pathology and epidemiological studies, spatial pattern analysis includes *N* sampling units (groups), *n*, individuals per sampling units, and *X* diseased individuals per sampling unit. Sampling units could be groups of plants (e.g. quadrates), plants or plant units (e.g., branches), individual plants (e.g., *n* plants per sampling unit [quadrate]), leaves (e.g. *n* leaves per sampling unit [plant]). Some representative studies pertain to spatial patterns detection for various host-pathogen complexes at various geographical locations are depicted in Table 1 (and supplementary material 2) and variables utilized in these studies are described in subsequent part of the this article. Equations of different indices are presented in supplementary material 1.

Segment B

The initial step in pattern detection is often involves testing the hypothesis that stated that distribution of the number of individual per sampling unit is random. If this hypothesis is rejected, then the distribution may be in the direction of clumped or uniform (Mathur, 2014). Thus, patterns detection can be bifurcate into randomness and/or quantification of departure from randomness (Figure 1). For testing the randomness researchers test their hypothesis by using various approaches like Complete Spatial

Randomness (CSR), the Diggle test, quadrate counting testing, Kalmogorov-Sminrnov and through Monte-Carlo approaches. If the direction is toward a clumped dispersion, negative binomial and certain indices of dispersion may be tested (Mathur, 2014 and 2015).

Complete Spatial Randomness (CSR) asserts that the points are randomly and independently distributed in the region of interest. There are at least three reasons for beginning an analysis with CSR: 1. rejection of CSR is a minimal prerequisite for any serious attempt to model an observed pattern; 2. tests are used to explore a set of data to assist in the formulation of plausible alternatives to CSR; 3. CSR operates as a dividing hypothesis between regular and aggregated patterns. This tool applied for *Cocos nucifera-Phytoplasma, Citrus* black spot-*Guignardia citricarpa, Prunus persica-Plum pox* virus, *Medicago sativa-Hypera postica*, Citrus-*Citrus tristeza virus,* Wheat-*Gaeumannomyces graminis* var. *tritici*, Apple-*Monilinia fructigena,* Apple-Sooty blotch/flyspeck disease (SBFS) complex and bull's eye and bitter rots, *Phaseolus vulgaris-Sclerotinia scleotiorum*, *Lithocarpus densiflorus- Phytophthora ramorum, Ganoderma boninese* (Basal stem rot of oil palm), Strawberry plant (*Fragaria × ananassa*)- *Phomopsis obscurans* and Crown rot of wheat and barley-*Fusarium pseudograminearum* complexes (Supplementary material 2). Everhart *et al.,* (2011) have used these approaches for characterization of three-dimensional spatial aggregation and association patterns of brown rot symptoms within intensively mapped sour cherry trees.

The Kolomogorov-Smirnov is a nonparametric test for the equality of continuous, one-dimensional probability distributions that can be used to compare a

sample with a reference probability distribution (one-sample K–S test), or to compare two samples (two-sample K–S test). The null distribution of this statistic is calculated under the null hypothesis that the samples are drawn from the same distribution (in the two sample case) or that the sample is drawn from the reference distribution (in the one sample case). In plant pathology K-S test have been utilized by Lescure *et al.,* (1992), Lin *et al.,* (2004), Camarero *et al.,* (2005) Freita-Nunes and Rocha (2007), Carneiro (2009) and Everhart *et al.,* (2011). Everhart *et al.,* (2012) used this for spatio-temporal patterns of pre-harvest brown rot epidemics within individual peach tree canopies. This approach also used for host-pathogen like *Prunus persica-Monilinia fructicola, Prunus cerasus-Monilinia laxa* and *Triticum aestivum* 'WPB926- *Rhizoctonia oryzae*. However, this test can only be applied to continuous distributions.

Monte Carlo procedure can be used to evaluate the departure of a pattern from CSR. Kelly and Meentemeyer (2002) used this as a supportive tool for study the landscape dynamics of the spread of sudden oak death (*Lithocarpus densiflorus- Phytophthora ramorum*) in California. For Citrus black spot-*Guignardia citricarpa* and *Prunus persica-Monilinia fructicola* this procedure was utilized by Sposito *et al.,* (2008) and Everhart *et al.,* (2012), respectively. Gent *et al.,* (2007) used this technique to determine the probability of classifying mean disease incidence for *Humulus lupulus- Podosphaera macularis.*

On the other way departure from the randomness can also be quantified based on their sampling procedure that may be natural or arbitrary samplings. From natural sampling, distribution can be evaluated using Poisson distribution, negative binomial and through using different indexes for dispersion. While

for arbitrary sampling methods like quadrate variance methods, distance models, kernel estimation of intensity (k means) and nearest neighbor distribution (G, F and K functions) can be use (Mathur, 2014). Basically natural sampling units are appropriate for species fruiting on leaves, logs or cones, whereas arbitrary sampling unites are required for litter decomposers and for root fungi (Mueller *et al.,* 2004). In the subsequent part of the paper, these approaches will be explain and summarized with host-pathogen perspective.

Spatial paternity for natural sampling

Poisson probability distribution

For a randomly dispersed population, the Poisson model ($\sigma^2 = \mu$) gives probabilities for the number of individuals per sampling units, provided the following conditions hold (a) each natural sampling units has an equal probability of hosting an individual, (b) the occurrence of an individual in a sample unit does not influence its occupancy by another (c) each sampling unit is equally available and (d) the number of individual per sampling unit is low relative to the maximum possible that could occur in the sampling unit (Nicot *et al.,* 1984). Review of Karlis and Xekalaki (2005) on mixed poison distribution models provided the term oversispersion which indicate the higher variance value of mixture model then that of simple component model. However, such types of relationships still not work out for distribution patterns of plant pathogens.

Binomial and negative binomial distribution

Commonly used approaches to determine spatial patterns are related with to compare the observed frequency distribution with theoretical frequency distributions such as Poisson, binomial, negative binomial, Neyman type A, and beta-binomial distributions (BBD)

(Madden and Hughes 1995; Hughes and Madden 1998). Based on the best fit (maximum likelihood, Lloyd patchiness) of observed frequencies to these distributions, a spatial pattern is considered aggregated, random or uniform (Campbell and Noe, 1985; Campbell and Madden, 1990 and Madden and Hughes, 1999). It is generally accepted that for count data, a good fit to the Poisson distribution suggests random distribution (Madden and Hughes 1995) while a good fit to the negative binomial distribution indicating heterogeneity (here heterogeneity indicated the patchy/uniform, coarse/dense and aggregated/random (Mathur, 2014) Similarly, for a binary variable a good fit to the binomial distribution indicates homogeneity while a good fit to the beta-binomial distribution suggests heterogeneity (here the homogeneity and heterogeneity subjected to number of individuals).

Basically beta-binomial distribution provides a good description of populations of diseased individuals in many patho-systems (Hughes and Madden, 1993; Hughes *et al.,* 1996; Tanne *et al.,* 1996, Xiao *et al.,* 1997; Hughes and Maddan, 1998 and Jones *et al.,* 2011).

Beta-binomial and binomial distribution are useful tools to represents functional relationships between disease incidences at two levels in a spatial hierarchy. By using these functions, incidence of disease at the higher level can be predicted, without curve fitting, based solely on the incidence and degree of aggregation at the lower level.

A good fit to the binomial distribution would suggests a random spatial pattern of disease incidence, while a good fit to the beta-binomial would suggest an aggregated spatial pattern of disease incidence (Gosme *et al.,* 2007). The negative binomial distribution is mathematically related to the Poisson and logarithmic series (Yin *et al.,* 2005). Disease incidence is appraised of the probability (π) of a plant or other plant unit being diseased. If the probability of a plant being diseased is not constant, then the binomial is not appropriate for representing the distribution of diseased entities per sampling unit (such nonconstancy include variation in host, pathogen, or environmental conditions throughout a field; and the multiplication of the pathogen in sampling unit being proportional to the initial disease or inoculums in the field, Madden and Hughes, 1995). Some representative examples of beta-binomial distribution are *Cocos nucifera-Phytoplasma,* Wheat-*Gaeumannomyces graminis* var. *tritici, Triticum aestivum* L. 'WPB926- *Rhizoctonia oryzae,* Apple- *Monilinia fructigena*, Apple-*Erwinia amylovora,* Apple-Sooty blotch/flyspeck disease (SBFS) complex and bull's eye and bitter rots, Citrus-Citrus Tristeza Virus (CTV), *Phaseolus vulgaris-Sclerotinia scleotiorum, Humulus lupulus - Podosphaera macularis,* Strawberry plant (*Fragaria × ananassa*)- *Phomopsis obscurans*, *Grapes-Phomopsis viticola, Humulus lupulus - Pseudoperonospora humuli. Prunus armeniaca* and *Prunus persica* Sharka disease of stone fruits - pulm pox potyvirus (PPV), Cauliflower- *Verticillium dahliae* and *Glycine max-Pratylenchus scribneri* and -*Glycine max-Hoplolaimus galeatus.*

Dispersal Indices for Natural Sampling

After detecting the pattern of a species it is necessary to measure the degree of aggregation and in order to measure the degree of aggregation there are large number of indices of dispersions are available like Fisher's variance-to-mean ratio, David and Moore's index of clumping, the slope of the log (variance) to log (mean) line in the Taylor's empirical power law (Taylor, 1961), Morisita's index, (Iδ), Lloyd's mean crowding and indices of patchiness (Mathur, 2014). Some highly accessed indices are:

Index of dispersal (I$_D$)

This index represent observed to expected ration and for random distribution of observed ration the variance to mean ration (VM) is expected to be 1 (Equation 1). An I$_D$ value equals to 1.0 indicates a random distribution; zero (or <1) indicates uniform while, more than one indicates the clumped distribution. According to Ludwig and Reynolds (1988), for small sample size (N<30), Chi-square (χ^2) is a good approximation with N-1 degrees of freedoms. If the value for χ^2 falls between the χ^2 tabular values at the 0.975 and 0.025 probability levels (P>0.05), agreement with a random distribution is accepted (i.e., $s^2 = \bar{x}$). On the other hand, values for χ^2 less than the 0.975 probability level suggest a regular pattern (i.e., $S^2 < \bar{x}$), whereas χ^2 values greater than the 0.025 probability level suggest a clumped pattern (i.e. $S^2 > \bar{x}$) (Table 1 and Supplementary Material 2).

Intra-cluster correlation (ρ)

This measures the tendency of the plants within a sampling unit (quadrate) to have a similar disease status. Positive values of ρ indicate aggregation of disease and this can be calculated directly from the quadrate data (Equation 2). This index can also be quantify through Index of dispersion (*D*) by using ρ = (D-1)/ (n-1) and in addition, if the beta-binomial distribution fits the data, this index is related to θ as ρ = θ/ (1+θ). Gottwals *et al.,* (2007) quantified this index for spatial pattern analysis of citrus canker in Sao Paulo, Brazil.

Lloyd's mean crowding (M[*])

This index can be quantified by parameters like mean severity of the disease from selected plants and with sample variance (Equation 3). Values of LIP lower than, equal to or greater than 1.0 indicate regular, random or patchy spatial pattern, respectively and the

significance value can be checked with chi-square test (Michereff *et al.,* 2011). This index is independent of density and easy to calculate. Ideal sample size (n) can be estimate for each crop area based on the coefficient of variation of the mean (CV$_X$) and random pattern of diseased plants (Equation 4). This index used for *Citrus suhuiensis-Diaphorina citri, Medicago sativa-Hypera postica, Capsicum annuum-Cercospora capsici*, Apple- *Monilinia fructigena*, Cauliflower- *Verticillium dahliae, Lycopersicon esculentum, Cyamopsis tetragonoloba, Lycopersicon esculentum tetragonoloba* - *Meloidogyne javanica, Solanum melongena-Meloidogyne javanica* and *Lactuca sativa-Sclerotinia sclerotiorum.*

Morisita's index of dispersion

Morisita's index of dispersion (I$_\delta$) has been extensively used to evaluate the degree of dispersion/aggregation of spatial point patterns (Tsuji and Kasuya, 2001). This index is based on random or regular quadrate counts. Because Morisita's index can be calculated for different quadrate sizes, the scale of analysis is not inherent, and it can be used to investigate pattern over a range of densities and scales (Kristensen *et al.,* 2006). The value of this index equal to 1 indicates random, more than one indicates aggregated, and less than one uniform distribution. Schuh *et al.,* (1986) have used this approach for analysis of spatial patterns in sorghum downy mildew infected with Pernosclerospora sorghi, similarly, Thal and Campbell (1986) have used this for analysis of disease severity data for alfalfa leaf spot caused by Leptosphaerulina briosiana (Equation 5). Murolo *et al.,* (2014) have used this tool for bois noir grapevine disease caused by stolbur phytoplasma (Equation 6).

Further use and interpretation of this index can be found with following host pathogen systems *Lactuca sativa-Sclerotinia*

sclerotiorum, Sorghum bicolour-Peronosclerospora sorghi, Triticum aestivum-Sinapis arvensis, Glycine max-Pratylenchus scribneri and -*G. max-Hoplolaimus galeatus, Trichilia tuberculata-Scolecopeltidium maayteni, Solanum melongena-Meloidogyne javanica*, *Lycopersicon esculentum tetragonoloba -Meloidogyne javanica, Humulus lupulus - Pseudoperonospora humuli* (Supplementary material 2).

Taylor's power law

The Taylor's power law (Taylor 1984) relates the variance of the number of individuals of a species per unit area of site or habitat to the corresponding mean by a power law relationship ($S^2 = am^b$). The parameters a and b are population parameters. Parameters b is considered to be an intrinsic measure of population aggregation varying continuously from zero for regular distribution ($S^2 = a$ with a < 1) through 1 for random distribution ($S^2 = m$ with a =1) to ∞ for strongly contagious distribution (Siddiqui and Shaukat 2002). For disease incidence data, the binary form of Taylor's power law provides a simple model with only two parameters to detect and quantify aggregation at the scale of individual sampling units (Turechke and Maddan 1999; Turechek *et al.,* 2001; Xu and Madden, 2002 and Bannot *et al.,* 2010).

Binary form of power law relationship relate the observed variance (V_0) of disease incidence between quadrats to the variance expected (V_R) if a binomial distribution is assumed for the number of diseased plants per quadrate (Xu and Ridout, 2000 Equation 7 and 8)

Gosme *et al.,* (2007) have used power law analysis for quantification of size; shape and intensity of aggregation of take all disease during natural epidemics in second wheat crop caused by *Gaeumannomyces graminis* var.

tritici. They used binary form of Taylor's power law to relate heterogeneity of disease incidence by taking the natural logarithm of observed and theoretical variance, power function transformed into a linear function (Equation 9). Xu and Madden (2014) described the limits of binary power law and according to them, performance of this parameter affected whenever there is a positive correlation among neighbors on the probability of a plant becoming infected, or where disease development is not influenced by the neighbors.

For *Onion-Botrytis squamosa* Carisse *et al.,* (2008) have used this tool to describe the relationship between mean (*M*) and variance (*V*). Other host pathogen systems for which this utilized are *Citrus* black spot-*Guignardia citricarpai, Citrus suhuiensis-Diaphorina citri*, *Medicago sativa-Hypera postica,* Citrus-*Citrus tristeza virus* (CTV), *Citrus-Xanthomonas axonopodis* pv. *citri, Centella asiatica-Cercospora centella, Lycopersicon esculentum tetragonoloba -Meloidogyne javanica.*

Ordinal run test

For ordinary runs test we can use various symbols, consider the following pattern of 10 symbol representing a crop row with disease (+) and healthy plants (-) + - - - + + - - ++. Here, there are five runs, reading left to right plants, the expected number of runs (E ((U)) can be calculated through equations 10, 11 and 12.

The null hypothesis (H_0) of run test state that a number of observed runs are not significantly lower than the expected number of runs obtained from a randomly distributed population, and the alternative hypothesis (H_1) state that the observed number of runs is significantly lower than the expected number of runs obtained from a randomly distributed

population. Gent *et al.,* (2006) had performed the ordinary run analysis to characterize large scale patterns of diseased cones among plants in a transect and for this test they considered plant as diseased if at least one diseased cone was observed in the sampling unit. Examples of this tool with following host pathogen systems are *Prunus persica-Plum pox* virus, Citrus-*Citrus tristeza* virus (CTV), *Prunus armeniaca* and *Prunus persica* Sharka disease of stone fruits caused by the pulm pox potyvirus (PPV), *Cucumis melo* var. *reticulates-Mosaic* virus 2 (WMV 2), Apple-*Erwinia amylovora, Phaseolus vulgaris-Sclerotinia scleotiorum*, *Humulus lupulus Podosphaera macularis.* Jones *et al.,* (2011) through run analyses reported significant positive relationship between disease aggregation and disease incidence for *Phaseolus vulgaris-Sclerotinia scleotiorum*

Arbitrary sample analysis

A spatial point pattern can be defined as a set of locations, irregularly distributed within a region of interest, which have been generated by random mechanisms (Diggle, 1983). Spatial point patterns are based on the coordinates of events such as the locations of outbreak of a disease. It is also possible that they include attribute information such as the time of outbreak occurrence. The basic interest of a spatial point pattern analysis will be to detect whether it is distributed at random or represents a clustered or regular pattern. These methods can be classified into different categories based on various criteria. For example, Cressie (1993) classifies the methods into five broad categories: quadrate methods, distance methods, kernel estimators of intensity, nearest neighbor distribution functions and K -function analyses. Each method has its advantages and problems, and works well for particular situations or datasets. Detailed descriptions of these methods and the associated test statistics can be found in the

texts such as Ripley (1981), Cliff and Ord (1981), Diggle (1983) and Cressie (1993). I categorized the arbitrary sample analysis into quadrate variance method, distance methods, kernal estimation and nearest neighborhood method.

Quadrate-variance methods

Quadrate-variance method concerned with spatial pattern of individual of species that are found continuously across a community (e.g. hosts in a forest). In this method some type of arbitrary sampling unit chosen to obtain a sample and consequently results may be influenced by size and shape of sampling unit. To address this problem, methodologies have been developed that allows examining the effect that varying the size of the sampling unit has on the detection of some underlying pattern. Collectively these are called quadrate – variance methods. These methods are based upon examining the changes in the mean and variance of the number of individuals per sampling unit over a range of different sample sizes (Ludwig and Reynolds, 1998). Data can access through belt transects of contiguous quadrats (i.e. a joined or continuous series of quadrats) placed in a linear manner across the population of interest. The variance is calculated at different "block sizes". The block sizes are obtained by combining progressively the N quadrats (therefore increasing the theoretical sample unit size) in a prescribed manner. Two types of quadrate-variance methods are generally utilize. One is based on Blocked-Quadrat Variance (BQV) and the other on Paired-Quadrat Variances (PQV). The BQV methods utilize changes in quadrate size via the blocking or combining of adjacent or abutting quadrats to identify the pattern intensity. The PQV methods utilize changes in quadrate spacing. BQV includes some fundamental aspects summarized that BQV is the original method for analysis of spatial pattern by combining quadrats into

blocks. In this approach, the number of quadrate is included in only one block. In this method the variance is determined for the powers of 2 as well as limitations on the necessary rectangular shape of certain block sizes (*e.g.*, a block size of 8 must be 2 × 4) (Dale 1999). Two avoid this limitation; Hill (1973) developed another blocking scheme, the two-term local quadrate variance (TTLQV). Dale (1999) have further defined the properties of Three-term local quadrate variance (3TLQV); Four-term local quadrate variance (4TLQV); Nine-term local quadrate variance (9TLQV) and Two-term local quadrate covariance (TTLQC).

The TTLQV uses variance data in a similar manner to the BQV method although it has a more refined "blocking scheme" in its calculation to overcome the BQV's limitation. It calculates the average squared difference between pairs of adjacent blocks for a range of block size. Peaks in the plot of the variance are indicative of scales of pattern in the data.

Unlike earlier methods, TTLQV is not restricted to detecting pattern on a scale of $2n$ blocks. However, TTLQV has some limitations. Formulas for quantification of variance for 3 TLQV, 4TLQV, 9TLQV worked out by Mathur (2014).

In the PQV method, pair of quadrats is selected at a specified spacing or distance along the belt to estimate variance. Since the quadrats are of fixed size, the spacing or distance between the quadrats is the only component contributing to the variance, rather than both spacing and size as observed in BQV. In the PQV method, variances are calculated for all possible paired quadrats at a given spacing. Examples of PQV and TTLQV are found in studies of Tobacco-*Meloidogyne incognita, Tylenchornhynchus claytoni, Helicotylenchus dihystera* and *Crinonemella ornate* (Noe and Campbell 1985); *Lactuca sativa-Sclerotinia sclerotiorum* (Chitrampalam and Pryor 2013) and *Quercus castaneifolia-Biscogniauxia mediterranea* (Karami *et al.,* 2015).

Distance methods

Distance sampling (or called plot less sampling) depends partly or wholly on distances from randomly selected points to the nearest host or from a randomly selected plant to its nearest neighbor. Distance sampling is considerably more efficient then quadrate sampling when individuals are sparse and widely scattered. Most of the distance sampling methods are based on the random selection of m points with uniform distribution in a sampling frame S lying slightly within the region A in order to avoid edge effects. Some distance methods can be based on systematic samples of m points, rather than random samples.

Hopkins index

Hopkins (1954) have suggested a coefficient of aggregation (CA) for detecting non-randomness based upon the squared distance (x) from a random point to the nearest plant, and the squared distance (y) from a randomly chosen individual to its neighbor (Equation 13).

The significance of the departure of CA values from 1 is assessed by calculating the parameters = CA/ (1+CA) which has a mean of 0.5 for random distribution. Armstron (2006) have proposed a significance test for this index (Equation 14). This method adopted by Leps and Kindlmann (1987), Condes and Martinez (1998), Shaukat *et al.,* (2009) Shaukat *et al.,* (2012) and Khojasteh *et al.,* (2013). Poushinsk and Basi (1984) used this index for study of distribution and sampling of soybean plant infected with *Pseudomona syringae* pv. *glycine.*

Clark and Evans Index

Clark and Evans (1954) were the first to suggest a method for analyzing pattern for spatial maps (Equation 15). Usually, the interpretation of R values is as follows: R>1 if the pattern has a tendency of regularity, R= 1 if it is completely random (Poisson) and R<1 if there is clustering in the patter. *R* approaches an upper limit around 2.15. Poushinsk and Basi (1984) used this for soybean plant infected with *Pseudomona syringae* pv. *glycinea* similarly, Palacio-Bielsa *et al.,* (2012) used this index to quantify the spatial spread of *Erwinia amylovora* on pome fruit.

Kernel Estimation of Density (k mean)

Kernel density estimation is an interpolation technique that generalizes individual point locations or events, s_i, to an entire area and provides density estimates, $\lambda(s)$, at any location within the study region *R*. From a visual point of view it can be thought of a three-dimensional sliding kernel function *K* (•) that 'visits' every location *s*. Distances to each observed event *si* that lies within a specified distance *b*, referred to as the bandwidth, are measured and contribute to the intensity estimate at *s* according to how close they are to *s* (Bailey and Gatrell, 1996). This produces a more spatially smooth estimate of variations in $\lambda(s)$ than could be attained by using a fixed grid of quadrates. There are a number of different kernel functions. However, since the result of an analysis is not strongly influenced by the chosen function as long as the function is symmetric (Burt and Barber 1996) there are no binding rules concerning the choice of an appropriate function. The most common kernel function is the normal distribution function (Jansenberger and Steinnocher, 2004). Choice of appropriate bandwidth (b) is the most crucial step in kernel density estimation (Mathur 2014). There are several

methods which attempt to optimize the value of *b* given the observed pattern of event location. Basically, the bandwidth can either be fixed (fixed kernel estimation) or adaptive (adaptive kernel estimation). Detail attributes of this technique are explained by Kelly and Meentemeyer (2002), Liu *et al.,* (2007) and Rieux *et al.,* (2014). With respect to plant pathogen Carrasco *et al.,* (2010) used this approach for eradication programmes of western corn rootworm in Europe.

Nearest Neighbour Index (NNI)

The Nearest neighbour index measures the degree of spatial dispersion in the distribution based on the minimum of the inter-feature distances, i.e. it is based on the distance between adjacent point features. Such that the distance between points features in a clustered pattern will be smaller than in a scattered (uniform) distribution with random falling between the two.

The values of NNI range between two theoretical extremes, 0 and 2.1491. When all the points in a pattern fall at the same location, the pattern represents the theoretical extreme of spatial concentration, in this case NNI = 0. The more closely the points are clustered together, the closer to 0 NNI will be, since the average nearest neighbour distance decreases. The closer NNI gets to 1, the more randomly spaced the points are. The sample mean of nearest neighbour distance can be calculated by equations 16, 17, 18 and 19.

Palacio-Bielsa *et al.,* (2012) used this to quantify the spatial spread of *Erwinia amylovora* on pome fruit. Gidoin *et al.,* (2014) have used this approach in cooco agro-ecosystems for black pod disease, caused by *Phytophthora megakarya*. Kamu *et al.,* (2015) have utilized this approach to quantify the distribution of infected oil palms with *Ganoderma* basal stem root disease. In their

analysis this tool showed that spatial distribution of the infected palm in all the studied area was clustered.

Spatial autocorrelation

'Spatial autocorrelation' is the correlation among values of a single variable strictly attributable to their relatively close locational positions on a two-dimensional (2-D) surface, introducing a deviation from the independent observations assumption of classical statistics. Reynolds and Madden (1988) have provided the theory of spatio-temporal autocorrelation. According to them autocorrelation tool like the Moran I statistic and others conserve the spatial information content of a filed sampling scheme by making use of information on the location of each sample point. Modjesk and Rawling (1983) and Madden *et al.,* (1987) provided the information's about advantages of spatial autocorrelation coefficients for a series of sample time that can be used in conjugation with spatial pattern statistics such as Lloyd's mean crowding and patchiness indices to characterize changes in spatial aggregation over time. Gent *et al.,* (2006) had utilized first and second-order spatial autocorrelation to quantify the similarity or dissimilarity of disease incidence among cones of neighboring plants with a transect by following Turechek and Madden (1999). Some predominant techniques of autocorrelation are as follows

Moran's I

Moran's I is defined as a measure of the correlation among neighboring observations in a pattern (Boots and Getis 1988). Computation of Moran's I is achieved by division of the spatial co variation by the total variation. Resultant values are in the range from approximately -1 to 1. Positive signage represents positive spatial autocorrelation, while the converse is true for negative signage

with a Zero result representing no spatial autocorrelation. Nicot *et al.,* (1984) have provided the advantages of using the Moran I statistic to characterize the spatial pattern of disease, while Noe and Campbell (1985) used this technique to characterize the spatial pattern for nematode distribution. Mihail (1989) used this tool for spatio-temporal dynamics related with This index approached for *Euphorbia lathyris-Macrophomina phaseolina,* Pineapple hear rot-*Phytophthora nicotianae* var. *parasitica, Lycopersicon esculentum* and *Cyamopsis tetragonoloba - Meloidogyne javanica Solanum melongena-Meloidogyne javanica, Lycopersicon esculentum tetragonoloba -Meloidogyne javanica* systems.

Geary's C (contiguity) ratio, General G-statistic and Getis and Ord's G are the other indices of spatial autocorrelation's. Computation of Geary's C is similar to Moran's I whose values ranges from 0 to 2. With zero being a strong positive spatial autocorrelation, through to 2, which represents a strong negative spatial autocorrelation? Both Moran's I and Geary's C indicates clustering or positive spatial autocorrelation if high value (e.g. high host density) cluster together (often called hot spots) and/or if low values cluster together (host with low density), but they cannot distinguish between these situations. However, the General G statistic distinguishes between hot and cold spots. It identifies spatial concentrations. Basically G is relatively large if high values cluster together while G is relatively low if low values cluster together. In addition to this Getis and Ord (as described by Mathur, 2014) provide a new index know as Getis and Ord's G and this index not only provide hypothesis testing to determine clustering has occurred within a dataset, but also provide information on the extent to which above and below average values cluster more strongly and identify local concentration of clustering. Statistical tests for above

mentioned three indices are depicted by author in his previous detail works (Mathur 2014).

Variogram approach

An empirical variogram is a plot of half the squared difference between two observations (the semi-variance) against their distance in space, averaged for a series of distance classes. A simple variogram model is defined by the model family and the parameters sill (the average half squared difference of two independent observations), range (the maximum distance at which pairs of observations will influence each other), and nugget (the variance within the sampling unit).

A correlogram is a graph in which autocorrelation values are plotted, on the ordinate, against distance classes among sites on the abscissa. Correlograms provide evidence for the autocorrelation intensity, the size of the zone of influence and the type of spatial pattern of the variable under study. The shape of a correlogram gives indications about the spatial pattern of the variable, as well as about the underlying generating process (Sokal 1986; Legendre and Fortin 1989).

This approach used for following host-pathogen: *Valeriana saliina-Uromyces valeiana,* Sugar beets-*Apera spica-venti*, Citrus-*Citrus tristeza virus* (CTV), Apple-*Monilinia fructigena*, *Prunus armeniaca* and *Prunus persica* Sharka disease of stone fruits caused by the plum pox potyvirus (PPV) Pineapple hear rot-*Phytophthora nicotianae* var. *parasitica, Phaseolus vulgaris-Sclerotinia scleotiorum*, Canary-grass - *Pratylenchus penetrans* and other nematodes, Cauliflower-*Verticillium dahliae*, *Humulus lupulus* cones with powdery mildew-*Podosphaera macularis*, *Triticum aestivum* L. 'WPB926-*Rhizoctonia oryzae*, *Pinus elliottii* var. elliottii Engelm- *Fusarium circinatum*, *Lycopersicon esculentum* and *Cyamopsis tetragonoloba* -

Meloidogyne javanica, *Solanum melongena-Meloidogyne javanica*, *Lycopersicon esculentum tetragonoloba - Meloidogyne javanica Grapes-Phomopsis viticola*, *Humulus lupulus- Pseudoperonospora humuli* and *Glycine max- Macrophomina phaseolinan.* Jamie-Garcia *et al.,* (2001) used this to study spatial and temporal progress of *Phytophthora infesstans* genotypes in tomatoes and potatoes, while Kallas *et al.,* (2003), used in the study of area with probability of occurrence of *Armillaria* spp in roots of ponderosa pine and Zas *et al.,* (2007) used variogram and kriging for screening *Pinus pinaster* resistant to *Armillaria ostoyae* in field conditions. Calonnec *et al.,* (2009) used variogram for spatiotemporal spread of powdery mildew epidemics in the vineyard. Alves and Pozza (2010) studied indicator kriging modeling epidemiology of common bean anthracnose. Azahar *et al.,* (2011) used semivariogram for temporal analysis of basal stem rot disease in oil palm plantations.

Segment C

Multi-variant approaches (refers to any statistical technique apply to analyze data that arises from more than one variable) basically carried out for four types of research questions viz., degree of relationship between the variables, measure significant differences between group means, predicting membership in two or more groups from one or more variables and to explaining underlying structure. In present study Frequency Distribution (FD) and Agglomerative Hierarchical Clustering (AHC) were performed to identify the patterns pertains to use of various indices for different host-pathogen systems by different researchers across the globe. Binomial data set (Supplementary material 2) was prepared with attributes like researcher, aim of the study, area, results, host-pathogen complex and indices.

Table.1 Spatial pattern studies for various host-pathogen complexes

Studied by	Host-Pathogen System
Gottwald *et al.,* (1996)	Citrus-*Citrus tristeza* virus
Bassanezi and Laranjeira (2007)	Citrus- *Citrus leprosis* virus and *Brevipalpus phoenicis*
Gottwald *et al.,* (2007)	Citru*s-Xanthomonas axonopodis* pv. *citri*
Sposito *et al.,* (2008)	Citrus black spot-*Guignardia citricarpa*
Batista *et al.,* (2008)	Citrus-*Citrus tristeza* virus
Sule *et al.,* (2012)	*Citrus suhuiensis-Diaphorina citri*
Rouse *et al.,* (1981)	*Triticum aestivum-Erysiphe graminis* f. sp. *tritici*
Paulitz *et al.,* (2003)	*Triticum aestivum* L. 'WPB926- *Rhizoctonia oryzae*
Kristensen *et al.,* (2006)	*Triticum aestivum-Sinapis arvensis*
Nordmeyer (2009)	*Triticum aestivum*, barley and Sugar beets-*Apera spica-venti*
Gosme *et al.,* (2007)	*Triticum aestivum - Gaeumannomyces graminis* var. *tritici*
Bentley *et al.,* (2009)	Crown rot of wheat and barley-*Fusarium pseudograminearum*
Gottwald *et al.,* (1995)	*Prunus armeniaca* and *Prunus persica* Sharka disease of stone fruits caused by the *pulm pox potyvirus* (PPV)
Leeuwen *et al.,* (2000)	Apple- *Monilinia fructigena*
Xu *et al.,* (2001)	Apple- *Monilinia fructigena*
Dallot *et al.,* (2003)	*Prunus persica-Plum pox* virus
Biggs *et al.,* (2008)	Apple-*Erwinia amylovora*
Everhart *et al.,* (2012)	*Prunus persica-Monilinia fructicola*
Everhart *et al.,* (2011)	*Prunus cerasus-Monilinia laxa*
Spolti *et al.,* (2012)	Apple-Sooty blotch/flyspeck disease (SBFS) complex and bull's eye and bitter rots
Alves and Pozza (2010)	*Phaseolus vulgaris-Colletotrichum lindemuthianum*
Johnson *et al.,* (1988)	*Humulus lupulus - Pseudoperonospora humuli*
Gent *et al.,* (2006)	*Humulus lupulus -Podosphaera macularis*
Gent *et al.,* (2007)	*Humulus lupulus- Podosphaera macularis*
Gent *et al.,* (2008)	*Humulus lupulus -Podosphaera macularis*
Kelly and Meentemeyer (2002)	*Lithocarpus densiflorus- Phytophthora ramorum*
Liu *et al.,* (2007)	*Lithocarpus densiflorus- Phytophthora ramorum*
Poushinsky and Basu (1984)	*Glycine max-Pseudomona syringe* pv. *glycinea*
Taliei *et al.,* (2013)	*Glycine max- Macrophomina phaseolinan*
Schuh *et al.,* (1986)	*Sorghum bicolour-Peronosclerospora sorghi*
Noe and Campbell (1985)	Tobacco-*Meloidogyne incognita, Tylenchornhynchus claytoni, Helicotylenchus dihystera* and *Crinonemella ornate*
Gray *et al.,* (1986)	*Cucumis melo* var. *reticulates-Mosaic* virus 2 (WMV 2)
Chellemi *et al.,* (1988)	Pineapple heart rot-*Phytophthora nicotianae* var. *parasitica*
Mihail (1989)	*Euphorbia lathyris-Macrophomina phaseolina*
Wallace and Hawkins (1994)	Canary-grass forage field at the University of Minnesota- *Pratylenchus penetrans* and other nematodes
Xiao *et al.,* (1997)	Cauliflower- *Verticillium dahliae*
Giblert *et al.,* (1997)	*Trichilia tuberculata-Scolecopeltidium maayteni*
Turecheck and Madden (1999)	Strawberry plant (*Fragaria × ananassa*)- *Phomopsis obscurans*
Siddiqui and Shaukat (2002)	*Solanum melongena-Meloidogyne javanica*
Nita (2005)	Grape-*Phomopsis viticola*
Laine and Hanski (2006)	*Plantago lanceoloata-Podosphaera plantaginis*
Solla and Camarero (2006)	*Abis alba- Melampsorella caryophyllacearum*
Lopez-Zamora *et al.,* (2007)	*Pinus elliottii* var. *elliottii - Fusarium circinatum*
Carisse *et al.,* (2008)	Onion-*Botrytis squamosa*
Ericson *et al.,* (1999)	*Valeriana saliina-Uromyces valeianae*
Bonnot *et al.,* (2010)	*Cocos nuciferai-Phytoplasma*
Moradi-Vajargah *et al.,* (2011)	*Medicago sativa-Hypera postica*
Michereff *et al.,* (2011)	*Capsicum annuum - Cercospora capsici*
Jones *et al.,* (2011)	*Phaseolus vulgaris-Sclerotinia scleotiorum*

Chitrampalam and Pryor (2013)	*Lactuca sativa-Sclerotinia sclerotiorum*
Parashurama *et al.,* (2013)	*Centella asiatica-Cercospora centella*
Shaukat *et al.,* (2014)	*Lycopersicon esculentum* and *Cyamopsis tetragonoloba - Meloidogyne javanica*
Karami *et al.,* (2015)	*Quercus castaneifolia-Biscogniauxia mediterranea*
Husain *et al.,* (2015)	*Lycopersicon esculentum tetragonoloba -Meloidogyne javanica*
Kamu *et al.,* (2015)	*Elaeis guineensis- Ganoderma boninese*
Taliei *et al.,* (2013)	*Glycine max-Macrophomia phaseolina*
Keske *et al.,* (2013)	*Prunus persicae-Monilinia fructicola*
Azahar *et al.,* (2011)	*Elaeis guineensis-Ganoderma*
Bassanezi *et al.,* (2003)	Citrus sudden death- *Citrus tristeza clostero*virus

Table.2 Frequency Distribution Parameters

Skewness (Pearson)	**0.838**
Kurtosis (Pearson)	-0.797
Kolmogorov-Smirnov test (at alpha 0.05 level)	0.224
Chi-Square test (at alpha 0.05 level)	0.014

Table.3 Various attributes of Agglomerative Hierarchical Cluster analysis

Class	Clusters					
	1	**2**	**3**	**4**	**5**	**6**
Objects	21	6	10	1	1	1
Within-class variance	1.59	1.4.	1.77	0.0	0.0	0.0
Minimum distance to centroid	1.02	0.79	0.89	0.0	0.0	0.0
Average distance to centroid	1.25	1.07	1.23	0.0	0.0	0.0
Maximum distance to centroid	1.43	1.40	1.73	0.0	0.0	0.0
	1,2,4,5,6,8,10, **12**,13,14,15,16,17,18, 19,20,23,31,32,36,40	3,7,**11**, 21,26, 37	9,**25**,27,28 ,29,30,34, 35,38,39	22	24	33

*Bold numbers are center object 1. *Phaseolus vulgaris-Colletotrichum lindemuthianum*, 2. *Phaseolus vulgaris-Colletotrichum lindemuthianum*, 3. *Cocos nucifera-Phytoplasma*, 4. Citrus- *Citrus Leprosis Virus* (CiLV) and *Brevipalpus phoenicis*, 5. Citrus sudden death- *Citrus tristeza clostero* virus, 6. Citrus-*Citrus tristeza* virus, 7. Crown rot of wheat and barley-*Fusarium pseudograminearum,* 8. Apple-*Erwinia amylovora*, 9. *Lactuca sativa-Sclerotinia sclerotiorum*, 10. *Prunus persica-Plum pox* virus, **11. *Prunus cerasus- Monilinia fructigena,* 12. *Humulus lupulus -Podosphaera* macularis**, 13. *Humulus lupulus -Podosphaera macularis,* 14. *Humulus lupulus-Podosphaera macularis,* 15. Wheat- *Gaeumannomyces graminis* var. *tritici,* 16. *Prunus armeniaca* and *Prunus persica* Sharka disease of stone fruits - *pulm pox poty*virus *(PPV)* pulm pox potyvirus (PPV), 17. Citrus-*Citrus tristeza* virus (CTV), 18. *Citrus-Xanthomonas axonopodis* pv. *Citri,* 19. *Humulus lupulus - Pseudoperonospora humuli,* 20. *Phaseolus vulgaris-Sclerotinia scleotiorum,* 21. *Elaeis guineensis-Ganoderma*, 22. *Quercus castaneifolia-Biscogniauxia mediterranea,* 23. *Prunus persicae-Monilinia fructicola,* 24. *Triticum aestivum-Sinapis arvensis,* **25. Apple- *Monilinia fructigena*,** 26. *Lithocarpus densiflorus- Phytophthora ramorum,* 27. *Capsicum annuum-Cercospora capsici,* 28. *Medicago sativa-Hypera postica,* 29. *Medicago sativa-Hypera postica,* 30. *Triticum aestivum*, barley and Sugar beets-*Apera spica-venti,* 31. *Centella asiatica-Cercospora Centella,* 32. *Triticum aestivum* L. 'WPB926- *Rhizoctonia oryza,* 33. *Glycine max-Pseudomona syringe* pv. *Glycinea,* 34. *Solanum melongena-Meloidogyne javanica,* 35. *Solanum melongena-Meloidogyne javanica,* 36. Apple-Sooty blotch/flyspeck disease (SBFS) complex and bull's eye and bitter rots, 37. Citrus black spot-*Guignardia citricarpai,* 38. *Citrus suhuiensis-Diaphorina citri* 39. *Cauliflower- Verticillium dahlia* and 40. Apple- *Monilinia fructigena*

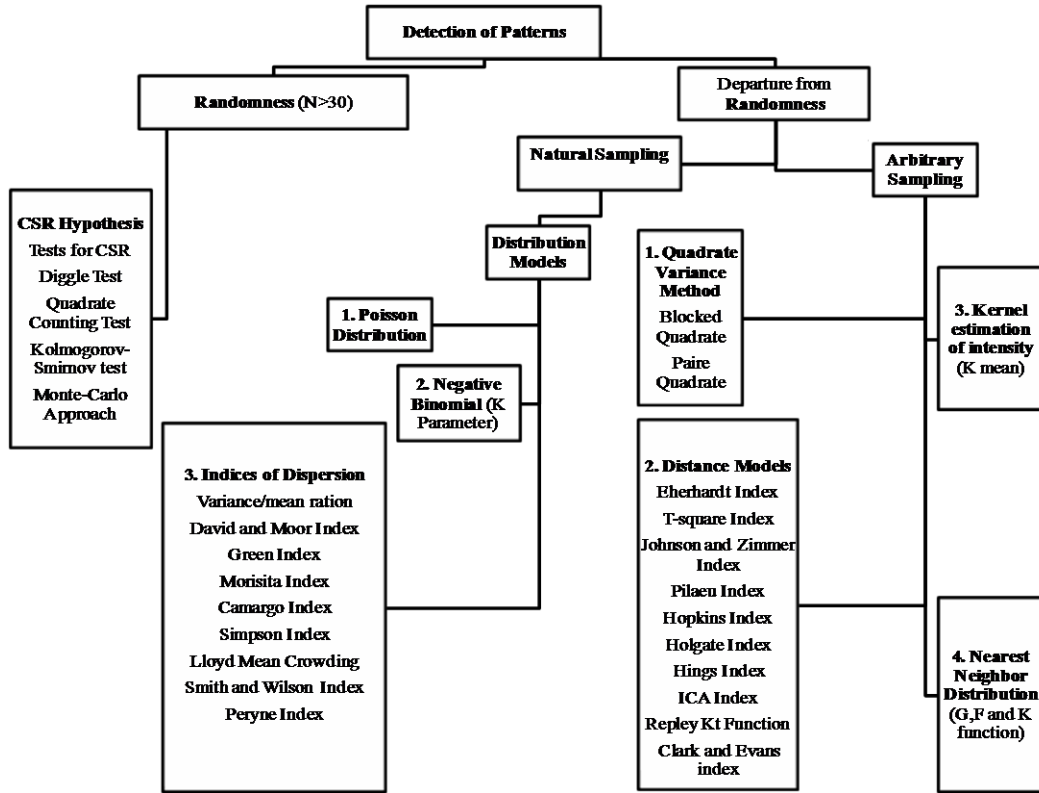**Fig.1** Graphical Presentation of Spatial Distribution Detection

Fig.2 Frequency distributions of spatial pattern indices utilized in 40 different studies
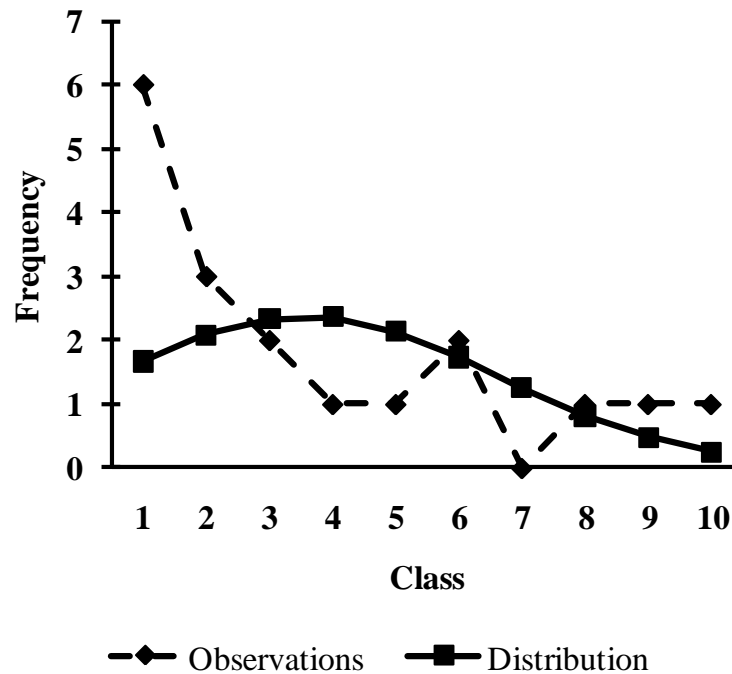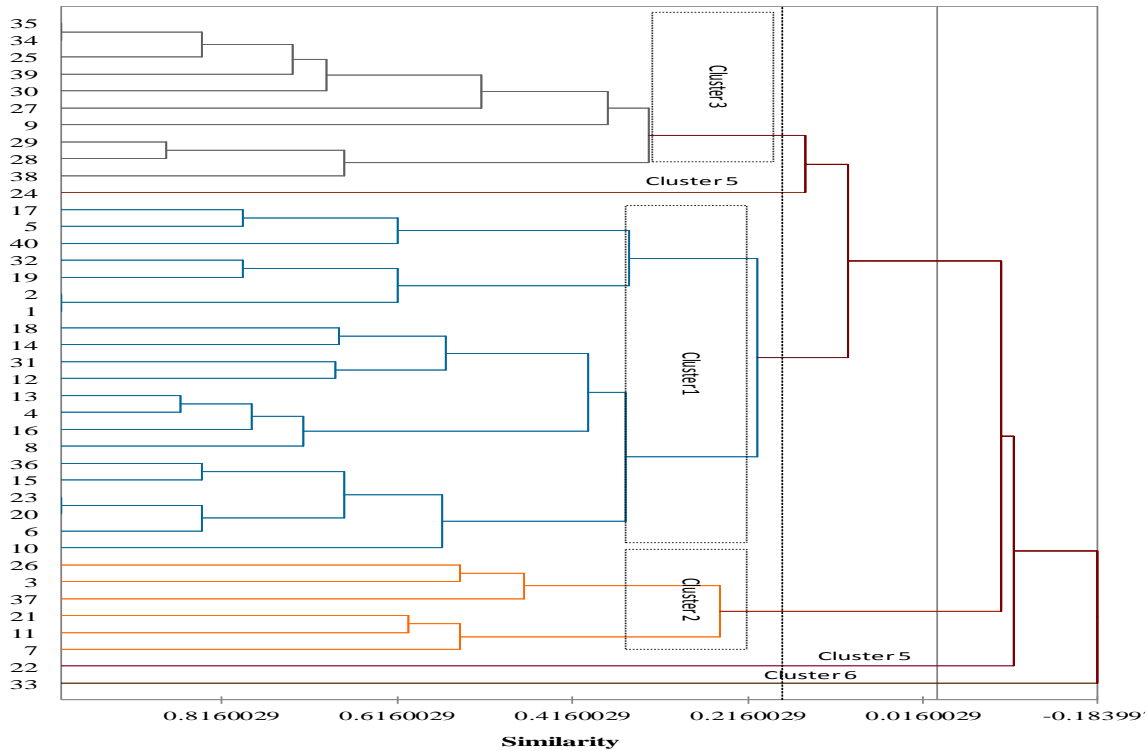
Fig.3 Agglomerative hierarchical cluster analysis

Study revealed the regional dominance for such studies headed by the America, the Brazil, and few from the Iran, Spain and Malaysia. While from India, Germany and Netherland only one study was reported. These studies were conducted from various land uses like cropland, yards, plantation field and forest. Major aims of these studies were to identify the spatial variability of disease incidence on various crops, to quantify the spread of disease within field, to study the effect of planting or population densities on distribution patterns of pathogen, to identify the degree of dependency among infected and disease plant on different spatial scales, to provide more specific recommendations for applying various control measures like (Biggs *et al.,* 2008), to evaluate the effectiveness of eradication policy for citrus plantation (Gottwald *et al.,* 2007) and linked crop loss with spatial pattern of pathogen (Leeuwen *et al.,* 2000).

In these studies nineteen different spatial indices were utilized for forty different host-pathogen complexes. Among these indices, Index of dispersion (I_D)>spatial autocorrelation (Moran *I*, variogram, semi-variogram, Geary G)>Taylor power law> beta binomial distribution CSR>LIP>Ordinary run > Morisita index were utilized by many researchers while, other indices were approached in less than five studies. Results of frequency distributions (FD) interpreted through skewness, kurtosis, Kolmogorov-Smirnov and chi-square tests and results are depicted in table 2. Skewness is a measure of symmetry. A data set is symmetric if it looks the same to the left and right of the center point. The skewness for a normal distribution is zero and a symmetric data should have skewness near zero. A symmetrical distribution with a long tail to the right (higher value) has a positive skew and an asymmetrical distribution with a long tail to

the left (lower values) has a negative skew. If the skewness is less than -1 or greater than +1, the distribution is highly skewed. In present study it was 0.838 reveled positive skeweness. Kurtosis quantifies whether the shape of the data distribution matches the Gaussian distribution, in general, a Gaussian distribution has a kurtosis of zero and a flatter distribution has a negative kurtosis.

A normal distribution has kurtosis exactly 3 is called mesokurtic, <3 called platykurtic (its central peak is lower and broader) and >3 called leptokurtic (central peak is higher and sharper). For present analysis it was recorded -0.797 which indicated platkurtic type (Table 2). As the computed p-values of Kolmogorov-Smirnove and Chi-square tests recorded higher than the significance level (alpha =0.05) that indicates the samples not follows normal distributions. Thus, all four parameters suggested that FD of uses of nineteen indices for different host-pathogen was not normal (Table 2 and Figure 2).

Cluster analysis attempted to subdivide or partition a set of heterogeneous objects into relatively homogeneous groups. The objective of cluster analysis is to develop sub grouping such that objects within a particular subgroup are more alike than those in a different subgroup. AHC was carried out through un-weighted pair-group average with Pearson correlation similarity coefficient. Hierarchical clustering do not only cluster sample, but also cluster the various clusters that were formed earlier in the clustering process. Agglomerative clustering algorithms start by treating each sample or variable as a cluster of one. The closest two clusters are joined to form a new cluster. Cluster analysis classified the forty host-pathogen into six different clusters (Figure 3). Number of host-pathogen systems and various distances from centroid are depicted in table 3. Host-pathogens grouped under cluster one are similar with

each other in respect of use of indices like binary power law, beta binomial, spatial autocorrelation, I_D, Ordinary runs and CSR, while, for cluster two species CSR, point pattern, Monte Carlo Test and Ripley K Function indices were utilized mostly. Cluster three which is the second largest cluster composed by host-pathogen system for which LIP, I_D, spatial auto-correlation, binary form and Iwo's Patchiness were studied. For Host-Pathogen systems of clusters 4, 5, 6 spatial indices like TTLQV, Morisita and Pielou, Clark and Evans, Hopkins and Skellam, respectively.

Segment D

Based on above cited studies, gaps and related preliminary works are as follows.

Some indices still not utilized for pathogens spatial patterns detection

For example spatial indices for that are not use in any study are Cole's Index of Dispersion (I), Kuno Index (Ca), Iwao's Method, Camargo's Index (E'), Simpson's measures of evenness, $E_{1/D}$ (Variant of Simpson 1949) and Smith and Wilson Index (E_{var}) and for arbitrary sample T-Square Index, Hines and Hines Index (1979), Johnson and Zimmer Index (1985) of Dispersion, Eberhardt's Index (I_E), Hopkins Index Hopkins-Skellam aggregation index, Pervasiveness index, Pielou-Mountford aggregation index, Hamill and Wright Test, Pielou's Index of Segregation (S) Individual-centered analysis (ICA) and Contagion Index (Mathur 2014).

Additional information's about eighty different indices are provided in supplementary file 3. Application of these indices may provide bittern spatial behavior of pathogen and their subsequent management options.

A generalized framework still requires for phylogenetic connection between hosts or with its family and with distribution patterns of pathogen

Gilbert and Webb (2007) had explored the importance of phylogenetic signal in plant pathogen host complex. According to them the likelihood that a pathogen can infect two plant species decreases continuously with phylogenetic distance between the plants. Their nursery (in Panama), forest (semi-deciduous low land moist tropical forest) sites and 53 necrotrophic plant pathogenic fungi based study revealed that rate of spread and ecological impacts of a disease through a natural plant community will depend strongly on the phylogenetic structure of the community itself and that current regulatory approaches strongly underestimate the local risks of global movement of plant pathogens or their hosts**.** Latter meta-analysis studies by Gilbert *et al.,* (2015) further added that phylogenetic distance is an informative surrogate for estimating the likely impacts of a pest or pathogen on potential plant hosts, and may be particularly useful in early assessing risk from emergent plant pests, where critical decisions must be made with incomplete host records. Garcia-Guzman and Morales (2007) have studied the life-history strategies of plant pathogen in relation to their distribution patterns and phylogenetic analysis. They concluded that in tropical region although distribution of obligate pathogens among plant families not strongly follows any patterns, except that smut pathogens are particularly dominant on Poaceae and Asteraceae, and rust are most common on Fabaceae. Such types of studies are requires for other pathogens producing high economic damage like, damping off, root rot and scab. In case of differential spatial patterns pertains to native and introduce species, Mitchell *et al.,* (2010) suggested that host geographic range size, agricultural uses

and time since introduction are the major controlling traits while biological traits are non-significant for such differences. Additionally adaptive evolution approach for pathogen (ability to infect) and host (tolerance of infection) has been discussed by Gilbert and Parker (2010).

Inter and intrasteric (pathogen and host) factors controlling spatial patterns of pathogen rarely approached

Gomez-Aparicio *et al.,* (2012) have quantified the spatial pattern analysis of soil born pathogens *Phytophthora cinnamomi, Pythium speculum* and *Pythium* spp. They concluded that pathogen abundance in the forest soil was not randomly distributed, but exhibited spatially predictable patterns influenced by both abiotic and biotic factors. According to them pathogen abundance is the multiplier of average potential pathogen abundance, abiotic effect, tree effect and shrub effect. Further hypothesis of decline development in oak forest suggested that loss of fine roots by soil-borne pathogen may translate into a loss of leaf area aboveground. The opening of the canopy trigger a series of environmental changes (e.g. reduced organic matter content, microbial activity and higher soil temperature) that might in turn favor pathogen development giving rise to a feedback loop where pathogen under the trees produce changes at the canopy level that favor the build-up of larger pathogen loads, eventually killing the tree. For example depending on the soil properties *Heterobasidion annosum* may either directly colonize the bark of live roots (in low -H sols) or grow ectotrophically (Garbelotto and Gonthier, 2013) under the outer bark scales (in high-pH soils). Earlier Korhonen and Stenlid (1998) correlated its distribution patterns with stand composition, thus, identification of such pattern types with associated factors may enhance our

management approaches. Disease transmission depends on two primary processes: dispersal of infective proagules and successful infection of host by those propagates (Roche *et al.,* 1995). In case of sessile hosts, the resultant process will be aggregated.

Dispersal process largely depends on distance from the source of inoculums and consequently it develops the disease foci (primary or secondary). Success of long distance dispersed disease can be evaluated through evaluating the data of contact distribution (i.e. the probability that a disease propagule released at one location will be deposited at another location). Three complementary approaches are generally used to study the wind dispersed propagules such as pollen, seeds or spores. First approach relies on mechanistic models that include a fine modeling of physical process contributing to the release, transport and deposition of propagules (Tufto *et al.,* 1997; Aylor 2003; Soubeyrand *et al.,* 2008 and Burie *et al.,* 2011). The second approach is an indirect approach related with genetic differentiation among populations or individuals. The third approach is based on direct measurements of dispersal distances by tracking propagule movement. The magnitude and frequency of Long Distance Dispersal (LDD) events, may affects the shape of tail of the dispersal kernel. In principle, dispersal kernels can experimentally be estimated from the real-time tracking of propaguls (Lagrangian methods) or from the amount and/or diversity of propagules observed at different distance from a source (Eulerian approach). Basically Lagrangian methods have mostly been applied to animals or animal-dispersed propagules, using mark/recapture or tracking designs. They can hardly be applied to wind-dispersed pathogen propagules because of small size. On the other side, Eulerian approach is more suited

in determining how many propagules originating from an isolated or identifiable source of inoculums arrive at specific distant location. Thus, quantitative evaluation of factors associated with pathogens distributions are need to generalized.

Spatiotemporal aspects of pattern detection are also not fully explored

For airborne inoculums, analysis of spore dispersal mechanism, spore and disease gradient, and long range spore dispersal are major approaches, while at filed scale, spatial analysis have primarily concentrated on soil borne inoculums (McCartney and Fitt, 1998). Carisse *et al.,* (2008) have identified the knowledge gaps related with quantitative aspects of spatial and temporal relationship that develop between airborne inoculums and disease intensity during the course of aerially spread epidemics. They have also identified the causes of such space which are related with difficulty in measuring airborne fungal spores and the lack of knowledge on the relationship between airborne inoculums and disease development. For example airborne inoculums play a crucial role in the development of *Botrytis* leaf blight epidemic (cause by *Botrytis squamosa*) on onion. This pathogen overwinters as sclerotia and in spring, these sclerotia are serving as primary sources of inoculums germinate and produce conidia that are dispersed through wind. Under favorable weather conditions, these conidia can infect onion leaves causing lesions and maceration of leaf tissue, resulting in leaf dieback and blighting. The secondary spread of the disease is caused by airborne conidia produced on necrotic tissues. Sutton *et al.,* (1978) have reported a significant correlation between airborne conidium concentration with severity of this disease, while, Vincelli and Lorbeer (1988) reported that increases in *Botrytis* leaf blight severity were associated with days with more than 10

conidia per m^3 of air. Carisse *et al.,* (2005) have assessed the usefulness of airborne inoculums measurements as an aid for improving *Botrytis* leaf blight management. To detect spatial patterns of disease incidence (*Cocos nucifera-Phytoplasma,* Lethal yellowing) at six assessment times, Bonnot *et al.,* (2010) had utilized binary power law and spatial autocorrelation and based on their permutations, new infection appeared in proximity of previously diseased host and a spatially structured pattern of times of appearance of lethal yellowing within clusters of infected host that suggested secondary spread of the disease. Sehajpal *et al.,* (2015) have correlated meteorological parameters with progression of Botrytis blight on *Gladiolus grandiflorus*

Very few studies have approached management practices based on spatial patterns of pathogens

In present study around fifty nine studies were evaluated and it revealed that only three studies have documented disease management strategies. These includes: *Citrus-Xanthomonas axonopodis* pv. *citri* (Gottwald *et al.,* 2007); Apple-Sooty blotch/flyspeck disease (SBFS) complex and bull's eye and bitter rots (Spolti *et al.,* 2012) and *Lactuca sativa-Sclerotinia sclerotiorum* (Chitrampalam and Pryor, 2013). In addition to these, Gonthier *et al.,* (2012) employed geostatistical and statistical analytical tools for distribution patterns of *Heterobasidion* irregular in the Italy. Based on its patterns, they recommended least stump treatment and a prescription to minimize wounding included in the management plants of all pine stands within 500 m from infested oak or pine forest. Thus, many more effort still requires. Further some of the other open ended tasks are: What is the line of difference between specialized and non-specialized pathogen for their spatial pattern? Is the availability of high inoculums

ensure uniform pattern (if all other conditions are conducive)? Information's on year to year variations of pathogen spatial patterns with similar crop/host. And majority of the studies were restricted to control conditions or site dominated with single species and their pattern behaviors within mixed population are rarely explore.

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