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Modelling fungus dispersal scenarios using cellular automata

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ABSTRACT

This study focused on representing spatio-temporal patterns of fungal dispersal using cellular automata. Square lattices were used, with each site representing a host for a hypothetical fungus population. Four possible host states were allowed: resistant, permissive, latent or infectious. In this model, the probability of infection for each of the healthy states (permissive or resistant) in a time step was determined as a function of the host's susceptibility, seasonality, and the number of infectious sites and the distance between them. It was also assumed that infected sites become infectious after a pre-specified latency period, and that recovery is not possible. Several scenarios were simulated to understand the contribution of the model's parameters and the spatial structure on the dynamic behaviour of the modelling system. The model showed good capability for representing the spatio-temporal pattern of fungus dispersal over planar surfaces. With a specific problem in mind, the model can be easily modified and used to describe field behaviour, which can contribute to the conservation and development of management strategies for both natural and agricultural systems.

1. Introduction

Fungi are decomposer organisms, essential in maintaining the trophic equilibrium in ecosystems (Gadd, 1999; Wainwright, 1988). All species are heterotrophic, and as such, tend to deposit themselves on organic substrates, from which they obtain their nutrients. The 8000 fungi species are found in soil, plants, water, detritus, or inside animals (Agrios, 1997; De Lucca, 2007). However, most field species, although they are able to persist in a dormant phase for long periods of time in the soil, only develop when they reach a plant surface (usually leaves or fruits), whether that surface is dead or alive (Tortora et al., 2000).

The first model proposed to study fungal communities was published by Halley et al. (1994), which modelled competition among two pairs of species in different succession stages with cellular automata. Among the main results obtained, the displacement of species by a competitor as well as the coexistence of species in heterogeneous environment raise interesting points in terms of spatial heterogeneity, since the model projects variable spatial patterns in response to a regular resource input.

In spite of the ecological importance of these organisms in the conservation of natural areas, as well as for the planning and management of agroecosystems, there is little knowledge of the mechanisms that govern the epidemiological patterns of their dissemination. Well-founded models tend to describe colony formation, or their increase in biomass, beginning with the development of hyphae during the vegetative growth phase (Boswell et al., 2007; Lazlo and Silman, 1993; Meskauskas et al., 2004a,b). Other studies have given particular attention to mycelia (Boswell et al., 2003; Halley et al., 1994; Lamour et al., 2000) or have investigated hyphal growth (Regalado et al., 1997). Although several studies have been accomplished, some of them have focused on a continental or intercontinental scale. Several points remain to be investigated, such as the choice of scale, mainly considering spatial dynamics on a local level (Davidson, 2007a, b).

Few studies have proposed mathematical models for the dispersal of these organisms on an intermediate scale, in order to give attention to the epidemiological sense rather than the development of a colony. These studies are helpful in understanding, for example, disease proliferation in agroecosystems, or the ecology of forest fragments. Recent contributions have basically followed two pathways: they use differential equations (Boswell et al., 2003; Edelstein-Keshet and Ermentrout, 1989; Sapoukhina et al., 2010; Shigesada et al., 1995) or they introduce empirical growth curves to model time-series (Rowan et al., 1999). An alternative way of modelling fungus dispersal is to use a cellular automaton network, which has previously been used to describe fungus proliferation over a solid surface (Lazlo and Silman, 1993) or fungus biomass in soil (Boswell et al., 2007).

Cellular automata (CA) were introduced by John Von Neumann in 1948, in his attempt to simulate biological replication processes (Burks, 1966). They constitute simple mathematical models, in which space, time and variables are discrete. A regular lattice with an automaton in each site is set following a series of well-defined rules based

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on a pre-specified neighbourhood of these automata interacting in time. They allow the emergence of complex behaviour, including the most different spatio-temporal patterns on the most varied scales of observation (Wolfram, 2002). This highlights their capability of simulating and elucidating highly complex physical and biological phenomena.

The objective of the present study was to model the spatio-temporal pattern of fungal dispersal over flat surfaces, using a CA model. The spatial structure was taken into account on an intermediate scale, allowing the spreading behaviour of propagules in agroecosystems to be clarified.

2. Material & methods

2.1. The cellular automata model

The proposed model takes into account a finite two-dimensional square lattice with linear dimension I, in which the cellular automata are exhibited. The automata represent hosts for the fixation of propagules, which come from a hypothetical fungal population. Each automaton is assigned one of four possible states: permissive, resistant, latent or infectious. Permissive and resistant automata represent healthy hosts, which possess different degrees of susceptibility to propagule fixation. Latent automata represent infected hosts (or colonised substrate) during the phase of vegetative growth, in which the production of propagules is insignificant. Infectious automata are those that are capable of disseminating propagules to adjacent automata, which are a source of secondary infections. A cluster of automata is defined as a Moore neighbourhood. In the proposed model, the probability of infection of a healthy host depends on the probability that propagules will be transported to it, as well as its permissiveness. In each time step t, the automata are updated in a synchronous manner, under the following rules:

1 A healthy host (permissive or resistant) becomes latent with infection probability given by

$$p_i = F \times p_t, \tag{1}$$

with F being the host permissiveness and P_t the probability of propagules being transported to it at period Δt, which represents one day of simulation. All terms in expression (1) are defined in the [0,1] interval.

2 A latent host becomes infectious after Δ days, where Δ is the latency period.

3 An infectious host does not recover from the infection.

2.2. The transportation probability

The first step to evaluate the transportation probability is to model the release of propagules around the infectious hosts in a way that it is possible to determine the probability of a given propagule to fall in each region of the space. In appendix A, it is demonstrated that for each probability density distribution assumed by the range of the propagules, there is an isotropic correspondent distribution, which is helpful for this task.

In this study, the exponential distribution was introduced to model the range of each propagule, given by r. Its density is expressed by

$$g(r;\alpha) = \frac{1}{\alpha}\exp\frac{1}{\alpha}, \quad r \geq 0, \alpha > 0, \tag{2}$$

with α determining the average range. Let $d_i = d_i(x,y)$ be the Euclidean distance from a given point (x,y) situated in the Cartesian plane to a host fixed at $X_i = (x_i, y_i)$. Then, the isotropic density corresponding to the exponential reach may be written as

$$f(d_i;\alpha) = \frac{1}{2\pi\alpha^2}\exp\left(-\frac{d_i}{\alpha}\right). \tag{3}$$

Let the measuring unit of length be the size of the side of each reticulate cell. As each host occupies a unit area, a good approximation to the probability of the event – 'deposition of a propagule that came from X_i on a host located at (x,y)' – is the value of $f(d_i;a)$. Let the transportation probability for each host be defined as the probability that at least one propagule will be deposited during a given period. Let N infectious hosts be spread over the reticule, occupying positions $Z_1, Z_2, \ldots, Z_n$. Consider that all of the infectious hosts show the same frequency of propagule emission, releasing c of them at each time period Δt. Lastly, let all of the propagules be disseminated in an independent manner and according to the density $f(d_i;a)$. Then, the probability of transport to a host that is located at (x,y) in the time period Δt is

$$p_t = P(T \geq 1) = 1 - P(T = 0) = 1 - \prod_{i=1}^{N} [1 - f(d_i;\alpha)]^c, \tag{4}$$

in which T is the number of propagules transported to the target host and c is the daily load of propagules. Substituting Eq. (4) in Eq. (1), the probability of infection for a healthy host situated at (x,y) during time period Δt is obtained:

$$p_i = F \times \left\{1 - \prod_{i=1}^{N} [1 - f(d_i;\alpha)]^c\right\}. \tag{5}$$

2.3. The model incorporating seasonality

Propagule release is not constant over time, but rather increases in periods that are favourable for transport and fixation. Therefore, temporal functions that represent the influence of seasonality on propagule release are required. In order to include this effect in the model, it was determined that

$$c = c(t) = S(t) \times c_{max}, \tag{6}$$

with t being the time step (or day of the favourable period for infection), c_{max} is the maximum daily propagule load admitted, and $S(t)$ is the seasonality function with counter-domain limited in [0,1].

Different climates determine distinct seasonality functions. A temperate climate produces well-defined regimens, and consequently, fungi may have only one peak of sporulation, which occurs whenever environmental conditions are optimal. In contrast, fungi in a tropical (or sub-tropical) climate usually distribute their propagules in several periods of less-intense sporulation (Amorin, 1995). The influence of these sporulation patterns on the dispersal of tropical and temperate species was introduced into the model by the functions:

$$S_{temp}(t) = \exp - \frac{(t - t_0)^2}{s^2}, \tag{7}$$

$$S_{trop}(t) = A\left[\sin\left(\frac{2\pi t}{\Phi}\right) + 1\right], \tag{8}$$

which represent temperate and tropical climates, respectively, with $A \in [0,0.5]$. Representing the most favourable period for the spread of infection, 120 time steps were included. The parameter values used were $t_0 = 60$, $s = 10$, $A = 0.15$ and $\Phi = 20$. Comparison amongst the simulations generated by these functions is plausible, because

$$\int_0^{120} S_{temp}(t)dt \cong \int_0^{120} S_{trop}(t)dt. \tag{9}$$

2.4. Key experiments

Several scenarios were conceived in order to evaluate the model's robustness in describing the processes that may occur in the field.

Parametric sensitivity studies were performed by using permissive lattices in order to evaluate the effects of the propagule average range, the latency period, the propagule daily load, and the seasonality.

A question of interest is how the mixed arrangement of different permissiveness crops may prevent the spread of fungal disease in the fields. In order to evaluate the effect of the proportion of resistant hosts on disease control, three scenarios were considered:

- a reticule with permissive hosts only;
- fifty percent permissive and the remaining resistant;
- only resistant.

The spatial pattern and the number of infected were compared between the lattices. Besides the proportion of resistant hosts, another important question is whether specific spatial patterns of resistant and permissive hosts may favour disease control in the field (Bogs et al., 1996; Sapoukhina et al., 2010). In order to investigate this aspect, alternative spatial designs were proposed by intercalating bands of permissive and resistant hosts. The viability of this strategy was investigated by analysing different scenarios:

- a random mixture of permissive and resistant hosts;
- wide alternating bands of permissive and resistant hosts.

The final number of infected (latent and infectious) hosts was compared by using analysis of variance, performing 10 simulations for each scenario. The linear dimension of the squared network was fixed at 60 length units and the simulations started with 9 infectious hosts at its centre. Each simulation consisted of 120 time steps (days), representing the favourable days for the reproduction of a hypothetical fungal population. The F parameter was set at 0.003 for permissive hosts and 0.001 for resistant ones. The remaining parameter values used are shown in Table 1. The algorithm was implemented in C language, and the results were analysed with R software (R Development Core Team, 2011).

3. Results

3.1. Sensitivity studies

It was verified that the average range of the propagules determines the arrangement of the secondary infections, thus being a controlling factor in the spatial pattern of their spread. It was also noticed that lower values for α resulted in local dispersal, characterised by disconnected clusters in the network. On the other hand, when the average range was larger, the fungi spread through the entire network and the infected clusters coalesced. Moreover, the velocity of spread is a key factor for understanding of the dynamics. The results showed, as expected, a delay in fungal propagation that occurred in response to the latency period. Another factor that considerably affects the dynamics of fungal populations is their sporulation power. The results indicated a positive and non-linear relationship between the daily propagule load and the number of infected hosts (Fig. 1). The seasonality functions proposed to represent temperate and tropical climates were compared on a network of permissive hosts. According to the model, tropical fungi are characterised by a more efficient use of their propagules to cause secondary infections, leading to a major number of infected hosts in the final time step.

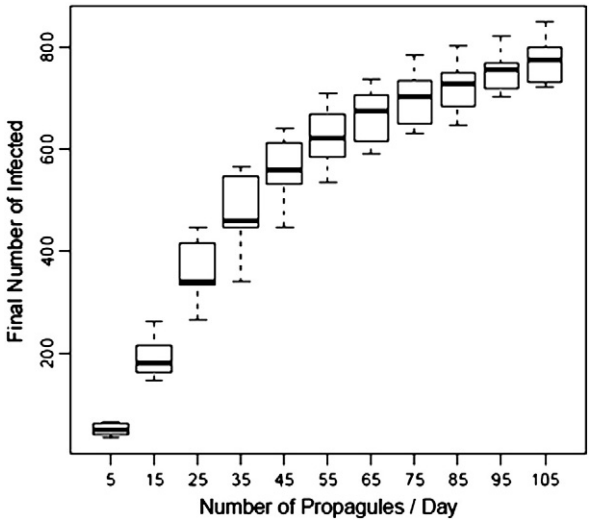

Fig. 1. Distribution of the number of infected hosts after 120 days, for different propagule daily loads.

3.2. Simulated scenarios

- It is observed that the permissive network led to the formation of clusters of infectious hosts, resulting in more rapid spread of infection and consequently facilitating the occurrence of epidemics (Fig. 2).
- The inclusion of resistant hosts in the network resulted in a lower spread rate (Fig. 3).
- The resistant network is characterized by point infections (Fig. 2).
- With respect to spatial arrangement of the mixed crops, it was verified that there were no significant differences ($p > 0.05$) among the final number of infected hosts in the different scenarios. Even bands with a width of 15 hosts, that is five times larger than propagule average range, showed no ability to prevent the fungal dissemination.

4. Discussion

From the proposed model, it is possible to understand the role of propagule range in the spatial structure of the system. It was observed that the lower α values determine a dispersion process which occurs via local interactions and enable the formation of larger clusters in the studied spatial pattern, while higher α values characterize isolated formations. The model also allows us to evaluate the effects of the latency periods in the dynamics. According to Amorin (1995), long periods of inoculation and reproduction result in a small number of generations per host cycle. The inverse reasoning is also valid. The simulations have shown that short latency periods accelerate the spread of fungal populations in the fields and this fact has important implications for producers, with respect to the development of strategies to prevent fungal disease.

Another interesting aspect is that the model allows us to comprehend how sporulation may act as a determinant of secondary infections. In order to start a new colony, the spores must survive the adverse conditions inherent to their transport, and must attach successfully to the

Table 1
Parameter values used in the simulations for each study.

Parameter	Permissiveness study	Propagule reach study	Latency period study	Propagule daily load study	Seasonality study
α	3.0	1.0, 3.0 and 5.0	3.0	3.0	3.0
Δ	10	10	0, 5, 10 and 15	10	10
c	40	40	40	5 to 105	–
c_{max}	–	–	–	–	270

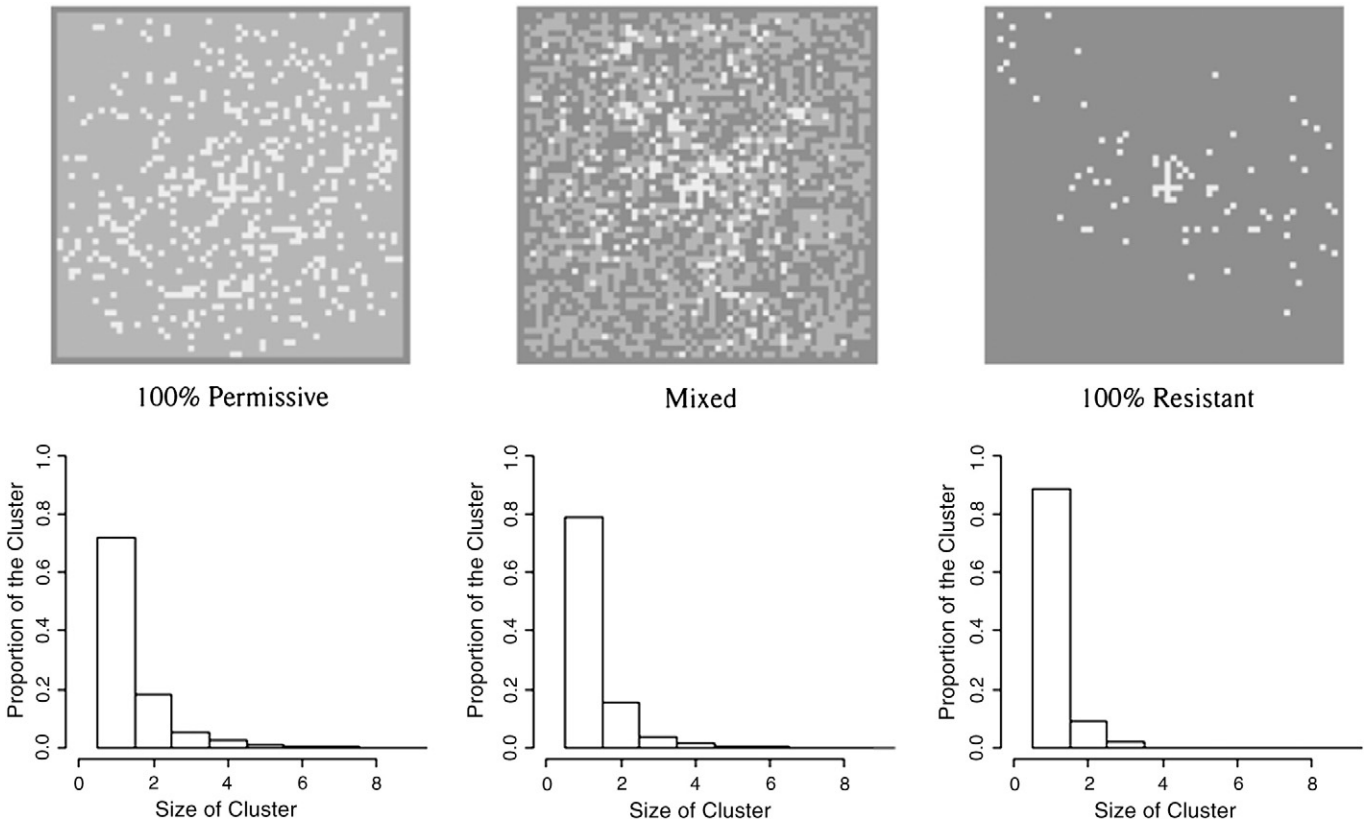

Fig. 2. Example of spatial pattern observed after 120 days (above). Permissive hosts in light grey, resistant in dark grey, and infected in white.

hosts. The propagules have a low probability of success, and a large number of propagules is required to ensure that some will survive. However, the daily propagule load and the number of infected hosts exhibited a non-linear relationship. Apparently, the propagule load influences the spread of infection, which slows after a certain point until the load effect saturation. This could be explained as a response to three interconnected factors. The increase of propagule load increases the probability of transport, which results in a larger number of secondary infections and a high level of space occupation. Finally, this decreases the probability of effective transport to healthy hosts.

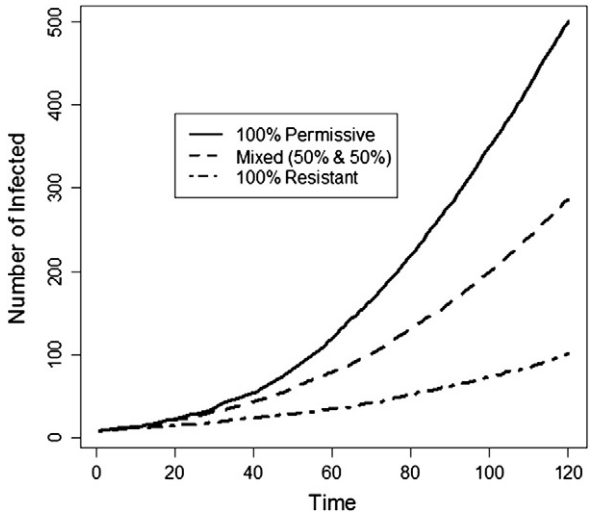

Fig. 3. Average number of infected hosts (latent and infectious) as a function of time. Results obtained from 10 simulations.

With respect to the inclusion of seasonality functions in the model's structure, the results make sense: it was verified that there was a larger number of infected hosts in the tropical climate. This fact was expected and the common occurrence of epidemics in the tropics supports this conclusion. The inclusion of seasonality in the model allows for the discussion of spatio-temporal patterns generated by different climates, as several of the parameters considered in the population dynamics, such as latency period and other host-pathogen interactions, might be affected by this.

The use of resistant and permissive hosts is a customary practice of crop producers, who generally mix permissive and resistant individuals in the field in an attempt to halt the spread of disease (Perrin, 1980; Sapoukhina et al., 2010). The flexibility of the proposed model allows us to evaluate the efficiency of these management strategies. The results indicated that resistant hosts were responsible for a lower spread rate of fungi, but the different spatial pattern of mixtures crops (random and with bands) showed no ability to prevent the fungal dissemination. According to the current model, the arrangement of the hosts in bands did not result in differences in the number of infected hosts in the observed time period. Sapoukhina et al. (2010) investigated the propagation of pathogenic fungi by using reaction–diffusion and stochastic models. In the model proposed by them, it is considered that the resistant hosts are immune to the disease, in a way that the propagules which contaminate resistant hosts are lost. They observed that the random mixture of permissive and resistant varieties exhibits better results in a short range and in a long range propagation scenario, the spatial structure may influence the velocity of disease spread.

Even though the spatial design did not influence the speed of fungal dispersal, there were differences with respect to spatial propagation. The permissive bands concentrated a larger number of infected hosts, facilitating optimised control of diseases with less use of chemical products (Sapoukhina et al., 2010). This would prevent additional waste and environmental damage due to residual effects.

In a similar approach, Bailey et al. (2000) investigated density and nutrient effects on the probability of fungi spread by using percolation theory to study the growth of *Rhizoctomia solani*, a soil-borne fungal plant pathogen. They used the colonisation profiles between donor and recipient sites to know threshold distances for expansion of *R. solani*. They conclude that percolation theory is a good approach to be used as a connection between growth of fungi and structuration of patches. The results found in this study have important implications for dynamics of soil colonisation in saprotrophic organisms.

The model presented in this work might reproduce the experimental results obtained by Bailey et al. (2000) when it is considered that resistant hosts are never infected by the fungus and, therefore, there must be a threshold for α values in which the percolation is or is not observed in the network.

Even though these studies are theoretical, they may serve as a new approach to modelling the dispersal of fungi. The structuration of the model's rules provides enough flexibility which allows for the simulation of more specific scenarios. For example, the different degrees of permissiveness allow us to build lattices that represent agricultural areas or native forests and its biodiversity; even edge regions could be focused, what would be helpful in order to understand the process of biological invasion. On the other hand, the model could be easily modified in a way to represent different species of fungi, allowing for studies of the fitness of these species in several simulated environments. It is also possible to apply the model's rules to simulate communities of fungi and study the consequences of competition, in an analogy of the work of Halley et al. (1994).

The model may also be used to evaluate the influence of the crops over the native biodiversity in an agroecosystem. The community dynamics in agricultural or forestry scenarios may also be investigated using the model proposed in this work. In addition, competition and co-existence among fungi species and/or strains with different epidemiological profiles, as well as the effects of spore range and latency period can be studied by using this approach.

Some studies have stressed the importance of spatial models to investigate risk of emerging infectious diseases of wildlife, including fungal pathogens, as essential tools for analysis emphasizing conservation (Murray & Skerrat, 2012). In special cases the presence of fungi, such as chytrid fungus can be associated with declines and extinctions of populations in amphibians, the most threatened vertebrates, mainly with respect to global biodiversity (Murray & Skerrat, 2012; Roedder et al., 2010). In that sense, the model may also be used to study effects of different biological invasion scenarios.

By modelling their field behaviour, it will be possible to understand more about fungal ecology and to predict the outcome of possible management approaches including the use of entomopathogenic fungi in biological control programmes (Desprez-Loustau et al., 2007). Fungi grow in many different environments, and they exhibit spatio-temporal heterogeneity (Boswell et al., 2003). Acting either as biological control agents or as pests, fungus species are continually dispersing in natural and agricultural systems, and the modelling of this dispersal will provide a new framework for decision-making regarding management strategies. Although the model is relatively 'simple', it is able to represent the spatio-temporal patterns observed in the field. The model allows us to study aspects of the biology/ecology in fungal species, and may contribute to the development of management strategies in agroecosystems.

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Appendix A

Let R be a random variable that represents the range of the propagules, with probability density function $g(\mathrm{r};\boldsymbol{\theta})$ being a smooth curve with positive support, for which $\boldsymbol{\theta}$ is the parameter vector that belongs to the parametric space $\boldsymbol{\Theta}$. Consider that the expected value for R exists and is denoted by $E(\mathrm{R})$. Then, the volume of the solid of revolution $V(S)$, given by the rotation of the parameterised curve $g(\mathrm{r};\boldsymbol{\theta})$ around the OZ axis, with $\mathrm{r}\in[0,\infty)$, is expressed by

$$V(S)=\lim_{b\to\infty}2\pi\int_0^b g(r;\boldsymbol{\theta})rdr=2\pi E(R). \tag{A.1}$$

Given that each observed range $\mathrm{R}=\mathrm{r}$ may be comprehended as the Euclidean distance between the point at which the infectious host is situated $(x_0\ y_0)$ and the site of propagule deposition $(x\ y)$, it is possible to show that the volume of interest $\mathrm{V}(S)$ can also be expressed, in Cartesian coordinates, by

$$V(S)=\iint_{R^2} g\left(\sqrt{\Delta x^2+\Delta y^2};\boldsymbol{\theta}\right)dxdy, \tag{A.2}$$

with $\Delta x=x-x_0$ and $\Delta y=y-y_0$. By setting expression (A.1) equal to expression (A.2), it follows that

$$\frac{1}{2\pi(R)}\iint_{R^2} g\left(\sqrt{\Delta x^2+\Delta y^2};\boldsymbol{\theta}\right)dxdy=1. \tag{A.3}$$

From expression (A.3), it is clear that

$$f\left(\sqrt{\Delta x^2+\Delta y^2};\boldsymbol{\theta}\right)=\frac{g\left(\sqrt{\Delta x^2+\Delta y^2};\boldsymbol{\theta}\right)}{2\pi E(R)} \tag{A.4}$$

determines a density function which is radially symmetric with respect to the point $(x_0\ y_0)$. Given the release of a propagule originated from the infectious host positioned at $(x_0\ y_0)$, the $f(.)$ function associates to all of the measurable region from the xy plane a transportation probability. Therefore, such a density meets the modelling goals of the process of propagule dissemination when isotropy (no preferential direction) is presumed. The function $f(.)$ will be denominated isotropic correspondent of $g(.)$.

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