Reproduction of Fungal Epidemic Visualizations

Deniz Ovalioglu

deniz.ovalioglu@student.auc.nl

05/05/2017

Word count: 2013

Ecology – Soil to Society, Simulating Fungal Epidemics,

Cor Zonneveld

Abstract

The aim of this paper is to reproduce the curves in the study “Emerging fungal threats to animal, plant and ecosystem health” by Fisher et al. The original study investigates the obscured effects of fungal epidemics on ecosystems via various dynamics. The information provided in Box 1 and Figure 2 of the original study is used to recreate the ecological simulations in Stella Architect. The data collected from Stella models are used to create the corresponding graphs in Microsoft Excel. The results of the reproduced models matched with the original results.

Based on the reproduction of the models, it is observed that the fungal epidemics show different results at inter and intra-specific levels. These effects on the ecosystem originate from various dynamics hidden in the ecological concepts within the system. These ecological concepts are investigated as the initial population size, length of a free-living stage of the pathogen and saprophytic growth rate. It is concluded that the Figures 2a, 2b, and 2c are confirmed to be accurate via the reproduction of the results while only the Stella model for Figure 2d could be reproduced.

Introduction

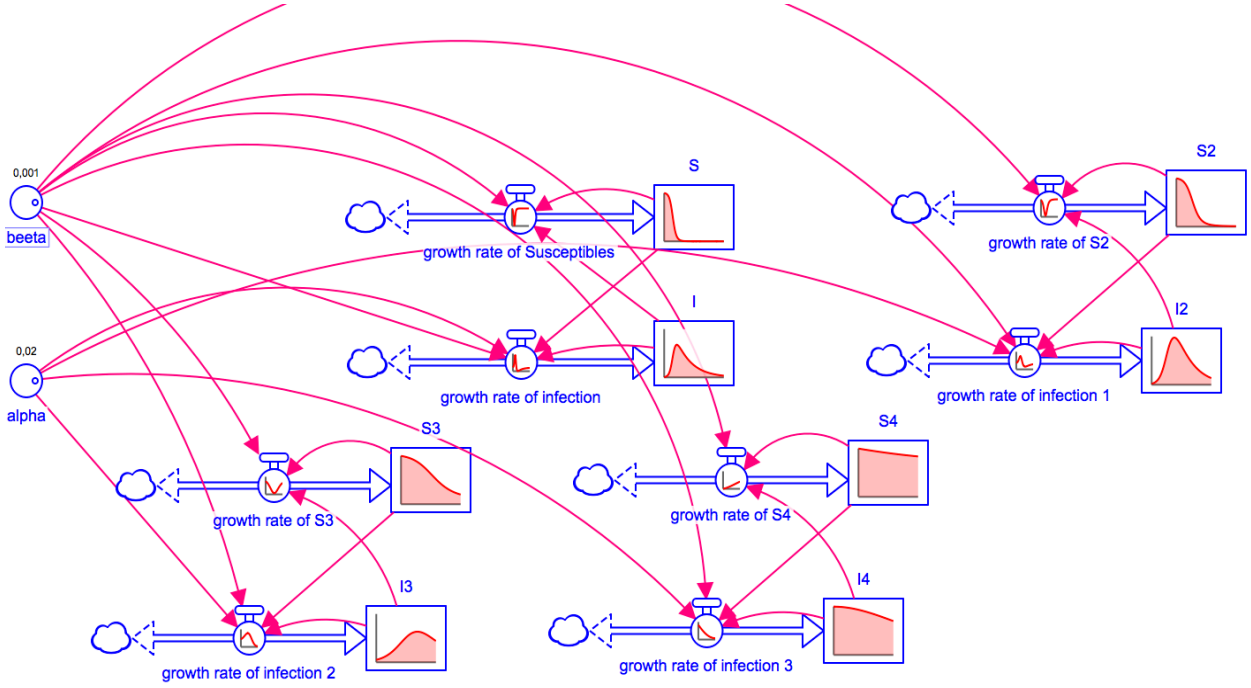
Fungal infections can cause the extinction of animal and plant species resulting in the loss of biodiversity. Fisher et al. studied the impact of fungal infections on animals, plants and ultimately also on ecosystems in 2012. In this paper, causes and results of the fungal epidemics are explained under six different sections, namely: increasing risk of biodiversity loss by Fungi, fungal-disease dynamics leading to host extinction, promotion of globalization of fungi via trade and transport, accelerated evolution of virulence in pathogenic fungi, environmental change as a driver of fungal EIDs, fungal EIDs impact on food security and ecosystem services and mitigation of fungal EIDs in animals and plants. The main method used to show the effects of fungal EIDs on different populations with various initial sizes is modeling. Using a susceptible-infected model, sizes of susceptible host populations with different initial sizes are simulated initially. Following this, the effect of a pathogen’s free-living stage on the fraction of the killed host population is modeled. Equilibrium host densities and free-living spore densities versus the rate of saprophytic growth are modeled in the third graph. In the final graph, susceptible host species’ density versus tolerant host species’ density is modeled. The conclusion drawn by Fisher et al. states that the direct causal relationship is uncertain in some of these diverse host–pathogen relationships. However, it is observed that pathogenic fungi are having a pronounced effect on the global biota.

In this paper, aforementioned four graphs in Figure 2 of the paper by Fisher et al. are reproduced using the information provided by the authors.

Materials & Methods

The materials used to reproduce the graphs are “Emerging fungal threats to animal, plant and ecosystem health” by Fisher et al., Stella Architect software and Microsoft Excel. All of the equations are copied from Box 1 of Fisher et al.’s study and the majority of the initial values are taken from the legend of Figure 2 in the original study. Except the first graph showing the susceptible host population sizes with various initial population populations, the graphs are generated in Microsoft Excel using the data collected from the simulations in Stella Architect. The first graph is produced completely in Stella Architect.

Results



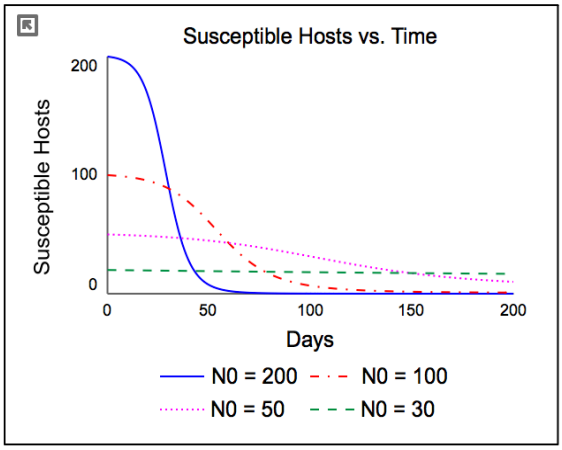


Figure 1

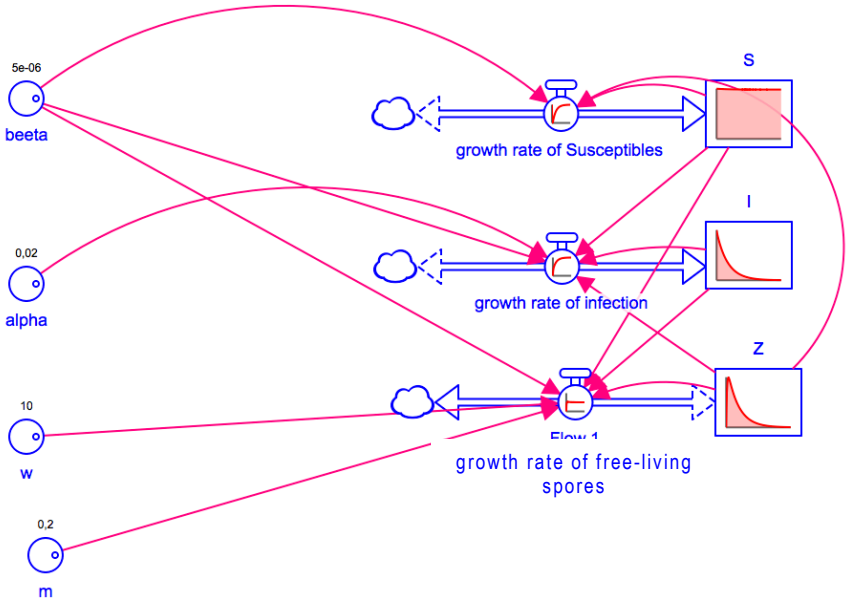
Stella Model to Graph Susceptible Hosts vs. Time (Days)
In the graph is it observed that in all the cases except N0 = 30, the host species go extinct due to the disease outbreak. Greater sizes of host population cause extinction faster because of the greater the population size the more rapid the transmission of the disease. All cases (N_0 = 200, N_0 = 100, N_0 = 50, N_0 = 30) have the same two equations for growth rate of susceptible and infected populations. The formula used for the growth rate of susceptible population, dS/dt, is –beeta*S*I, where beeta is the pathogen transmission rate. The growth rate of infected population, dI/dt, is beeta*S*I–alpha*I, where alpha is the disease-induced death rate. (beeta = 0.001 per individual per day; alpha =

Table 1

Table of the average duration of the free-living stage ($1/\mu$) and fractions of host population killed for different initial population sizes
μ values from 0.02 to 0.1 are used to calculate $1/\mu$ values listed. The values listed under different N_0 values are calculated by $(N_0 - S_{400})/ N_0$ where N_0 is the initial population and S_{400} is the final population left.

$1/\mu$	$N_0 = 200$	$N_0 = 50$	$N_0 = 20$
0	0.00	0.00	0.00
5	0.87	0.03	0.02
10	0.99	0.24	0.05
15	0.99	0.66	0.11
20	0.99	0.87	0.21
25	0.99	0.94	0.34
30	0.99	0.97	0.48
35	0.99	0.99	0.60
40	0.99	0.99	0.70
45	1.00	0.99	0.78
50	1.00	0.99	0.83
55	1.00	0.99	0.87
60	1.00	0.99	0.90
65	1.00	0.99	0.92
70	1.00	0.99	0.94
75	1.00	0.99	0.95
80	1.00	0.99	0.96
85	1.00	0.99	0.97
90	1.00	0.99	0.97
95	1.00	0.99	0.98
100	1.00	0.99	0.98

a


5e-06
beeta
0,02
alpha
10
w
0,2
m
S
I
Z
growth rate of Susceptibles
growth rate of infection
growth rate of free-living spores

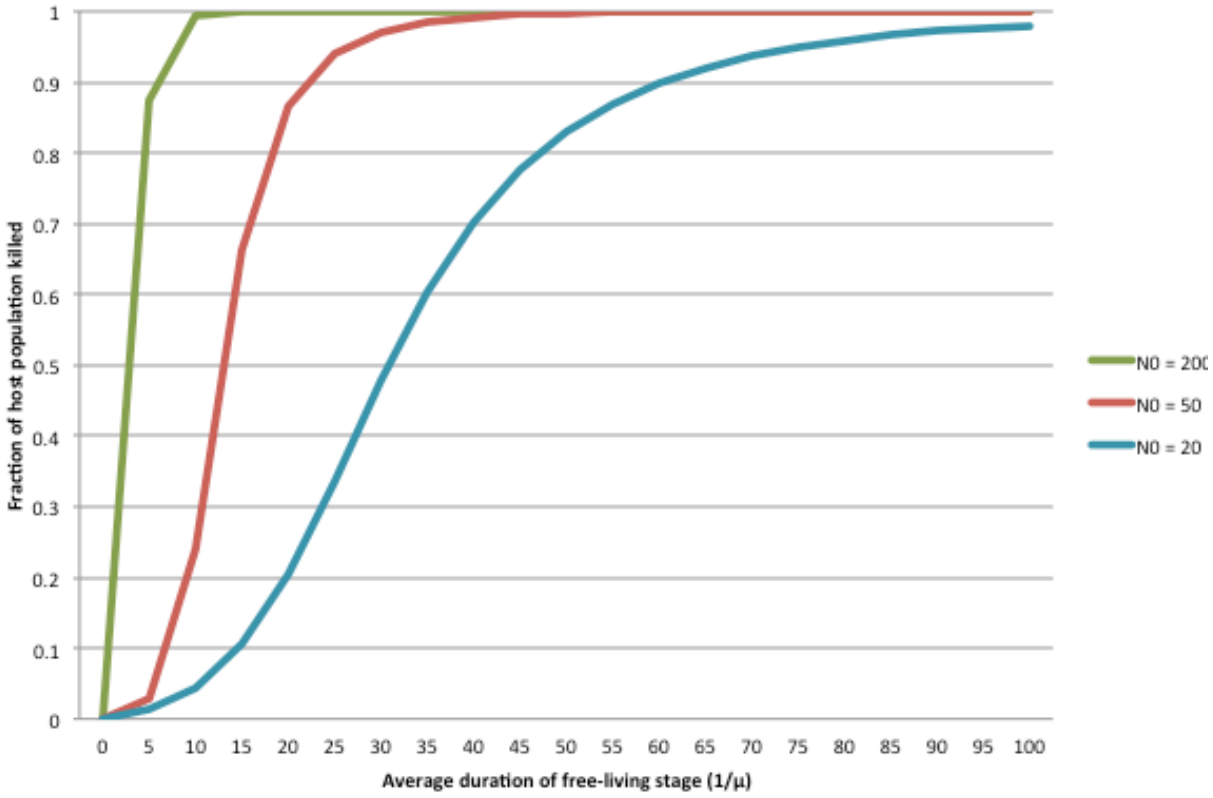

Fraction of host population killed
Average duration of free-living stage (1/μ)
N0 = 200
N0 = 50
N0 = 20

Figure 2

a An example of the Stella Architect model for the effect of a pathogen's free-living stage on the fraction of the killed host population reproduced using the information provided in Box 1 of the original study. dS/dt, is –beeta*S*I; dI/dt, is beeta*S*I–alpha*I; dZ/dt, is w*I - μ*Z - beeta*(S+I)*Z where w is the rate of release of spores from infected host (beeta = $5*10^{-6}$; alpha = 0.02; w = 10; I = 1 & S = N_0).

b Graph of Fraction of host population killed vs. Average duration of free-living stage ($1/\mu$)

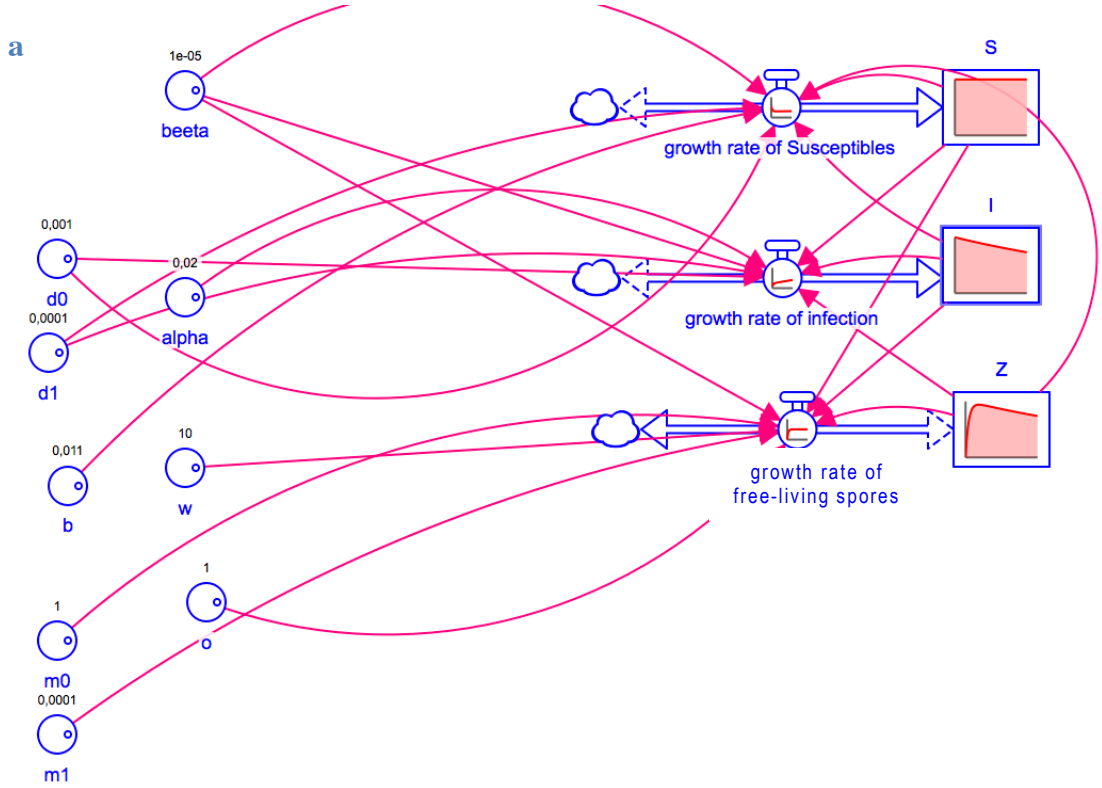
Figure 2b is representing the data from Table 1. The longer the infectious stages the higher the potential for host extinction. In this graph, the killed host fraction in an epidemic is shown as a function of the duration of the free-living infectious spore stage.

Table 2

Table of susceptible (S), infected (I) and free-living spore (Z) populations in relation to different saprophytic growth rates (o) ($S_0 = 99$, $I_0 = 1$, $Z_0 = 0$).

Saprophytic Growth (O)	S	I	Z
0.00	100	0	0
0.25	100	0	0
0.50	100	0	0
0.75	100	0	0
1.00	92	12	1089
1.25	1	1	2053
1.50	0	0	5000
1.75	0	0	7000
2.00	0	0	10000

a


1e-05
beeta
S
growth rate of Susceptibles
0,001
d0
0,02
alpha
I
0,0001
d1
growth rate of infection
Z
0,011
b
10
w
growth rate of
free-living spores
1
m0
1
o
0,0001
m1

b

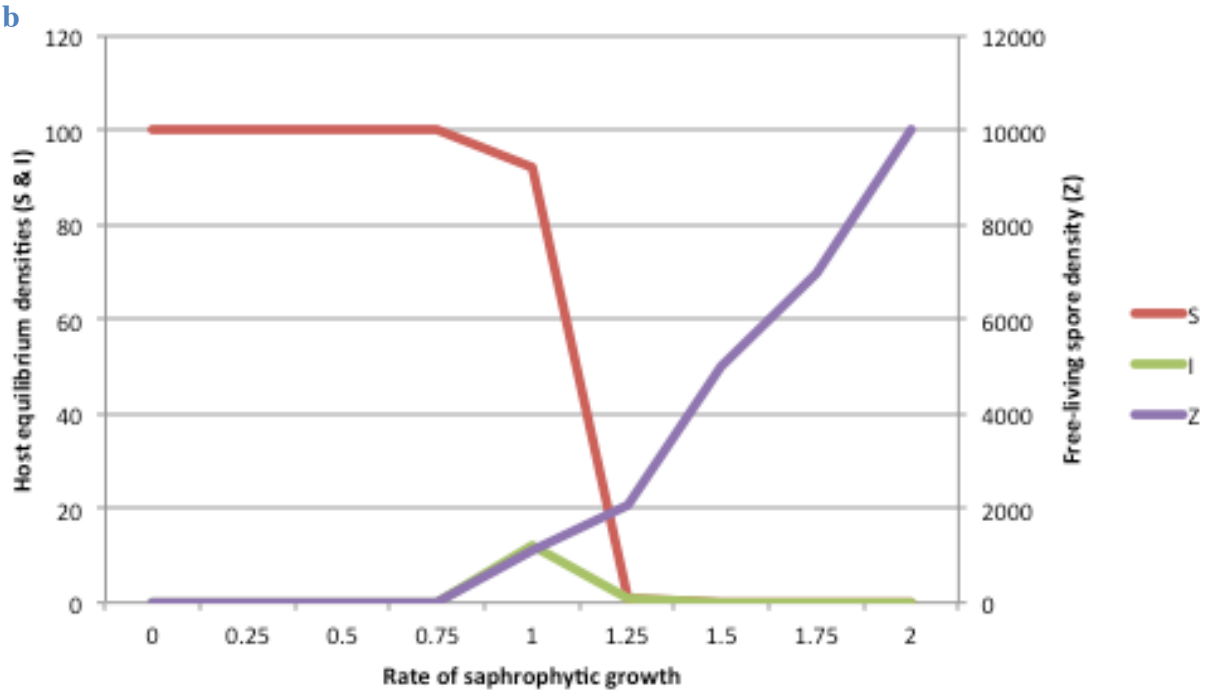

120
100
80
60
40
20
0
12000
10000
8000
6000
4000
2000
0
0
0.25
0.5
0.75
1
1.25
1.5
1.75
2
Host equilibrium densities (S & I)
Free-living spore density (Z)
Rate of saphrophytic growth
S
I
Z

Figure 3

Graph of host equilibrium and free-living spore densities versus saprophytic growth rate

a An example of the Stella Architect model reproduced using the information provided in Box 1 of the original study. The growth rate of susceptibles is b*(S+I) – (d_0+d_1(S+I))*S – beeta*S*Z, where *b* is the density-independent host reproduction, d_0 is density-independent host death rate and d_1 is the strength of density dependence in host death rate. The growth rate of the infected population is beeta*S*Z – alpha*I – (d_0+d_1(S+I))*I. Finally, the growth rate of the free-living spores is w*I + o*Z – (μ_0+μ_1(Z))*Z – beeta*(S+I)*Z, where *w* is the rate of release of spores from infected hosts, *o* is the rate of saprophytic growth, μ_0 is the rate of density-independent spore mortality and μ_1 is the strength of density-dependence in spore mortality rate.

b A pathogen's saprophytic growth may cause the host become extinct and may allow the pathogen to persist in the absence of the host. In the study by Fisher et al., free-living infectious spores are released from infected hosts with rate *w* and can increase in abundance through saprophytic growth. Host equilibrium densities S, I, and Z are calculated by the formula S_f / (S_f + I_f + Z_f), I_f / (S_f + I_f + Z_f), and Z_f / (S_f + I_f + Z_f) respectively, where X_f indicates the final value.

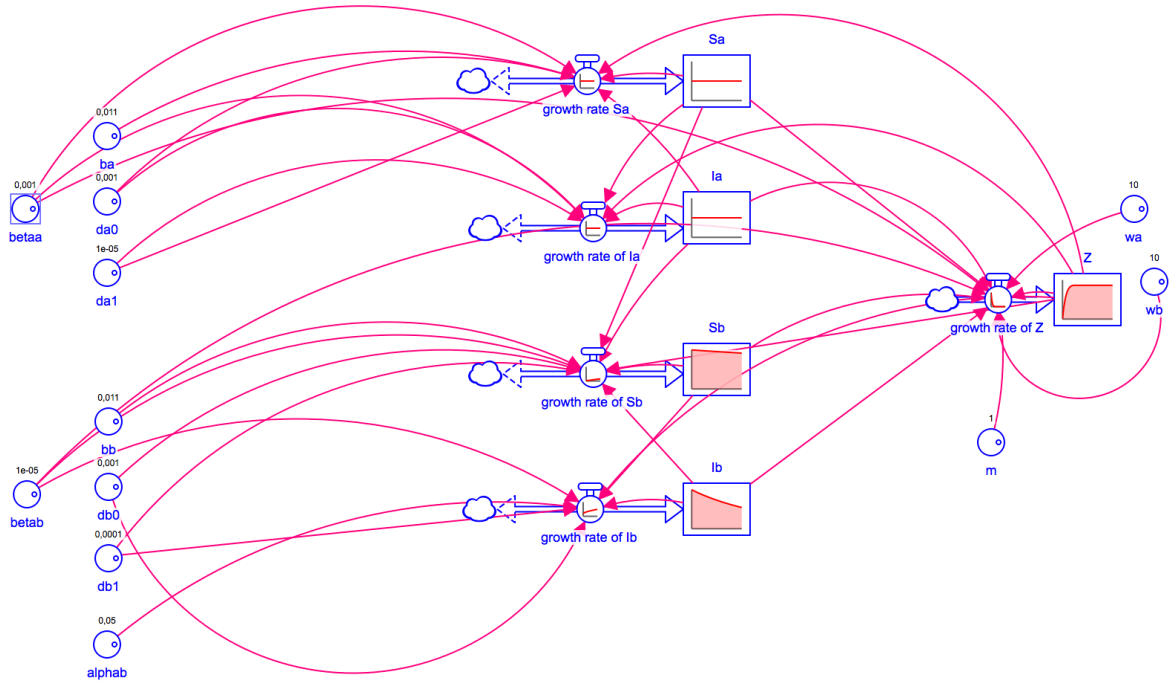


Figure 4

An example of the Stella Architect model reproduced using the information provided in Box 1 of the original study. The model aims to simulate the interactions in the presence of two host species, one tolerant and one susceptible, which may result in the extinction of the susceptible host species if the tolerant host species has high densities. In this model, *Sb* represents the susceptible host species while *Sa* is the tolerant host species. All parameters used in the following equations are defined previously in the paper and lowercase *a* and *b* following S, I, beta and alpha indicate which parameter belongs to which species:

Growth rate of Sa = ba*(Sa + Ia)– (d_{A0}+d_{A1}*(Sa + Ia))*Sa– betaa*Sa*Z
Growth rate of Ia = betaa*Sa *Z– (d_{A0}+d_{A1}*(Sa + Ia))* Ia
Growth rate of Sb = bb*(Sa + Ia)– (d_{B0}+d_{B1}*(Sb + Ib)) * Sb – betab* Sb *Z
Growth rate of Ib = betab* Sb *Z– (d_{B0}+d_{B1}*(Sb + Ib))* Ib – alphab* Ib
Growth rate of Z = wa* Ia + wb* Ib – m*Z – betaa*(Sa + Ia)*Z– betab*(Sb + Ib)*Z

Sufficient information to reproduce figures 2a, 2b and 2c of the original paper is provided. Figure 2d of the original paper by Fisher et al. cannot be reproduced because the information provided is insufficient. The initial values of *Sa, Ia, Sb, Ib* and the value of *betab* are not provided to the reader.

The effects of fungal epidemics on ecosystems were not investigated significantly until this study in 2012. The article highlights how human activities cause the dispersal of pathogenic fungi and interact with key fungal characteristics resulting in the emergence of diseases.

In the paper, fungal-disease dynamics leading to host extinction are explained as high virulence, long-lived environmental dispersal stage, and variation of the susceptibility of host species. Virulence is a measure of the relative capacity of a microbe to cause damage to a host, and high virulence is associated with rapid intra-host growth rates, ultimately leading to rapid inter-host transmission (Fisher et al. 2012). Figure 1 shows the effects of population size on the transmission rate of the disease and similar to the Figure 2a of the original paper, higher initial population size causes the earlier extinction of the species. The ability to survive independently as a free-living saprophyte or as durable spores in the environment is crucial to drive the emergence of pathogenic fungi, through the increased risk of transporting the inocula to naive hosts (Fisher et al. 2012). Table 1 created using the simulation in Figure 2a, shows that the increase in the average duration of free-living stage increases the fraction of the population killed regardless of the population size. However, as the graph in Figure 2b depicts, lower population size increases the tolerance against this phenomenon. Pathogenic fungi with a saprophytic stage can lead to host extirpation because their growth rate is decoupled from host densities and opportunistic fungi with long-lived environmental stages cause many fungal diseases threatening natural populations (Fisher et al. 2012). Running the simulation in Figure 3a for different values of saprophytic growth rate, Table 2 is created. In Figure 3b, which is the visualization of Table 2, it is observed that the

host species can tolerate the saprophytic until a certain rate after which the free-living spore density tremendously increases. Figure 1, 2b and 3b agree with the statements of the original study.

The final fungal-disease dynamic described is the variation of the susceptibility of host species and this concept was visualized in Figure 2d of the original paper. Host species that can tolerate high infection loads while serving as a source of infectious stages, act as community 'super spreaders' by maintaining persistent infectious stages in the system (Fisher et al. 2012). The graph in the original study supports this statement by showing that the rise in the tolerant host population results in the decrease and eventual extinction of the susceptible host population even when the pathogen transmission rate is decreased. However, due to the lack of information regarding the pathogen transmission rate for susceptible species, the conclusion of the paper cannot be reproduced. Figure 4 shows the Stella Architect model created using the information provided in the paper. However, the model cannot be operated due to lack of input. As a scientific paper, the information on the method should be provided completely in order to ensure that the others can obtain the same results. However, because the others are encouraged to come up with approximate initial population values and pathogen transmission rate, the credibility of the model cannot be tested.

Discussion

Based on the reproduction of the virtual models in "Emerging fungal threats to animal, plant and ecosystem health", the same results are observed. These results indicated that the fungal epidemics cannot be evaluated separately since they are connected to various dynamics that alter the ecosystems. In Figures 1, 2b and 3b, these dynamics are studied as the initial population size of the host, free-living stage of the pathogen and the saprophytic growth rate. The interaction of susceptible and tolerant host species could not be reproduced, however, a detailed model to operate this simulation is provided. The results obtained through the reproduction the original models match with the results of the original study by Fisher et al.

References

Fisher MC, Henk DA, Briggs CJ, Brownstein JS, Madoff LC, Mccraw SL, Gurr SJ. *Emerging fungal threats to animal, plant and ecosystem health*. 2012; 484: 186–194.