

# Disease spread in small-size directed networks: Epidemic threshold, correlation between links to and from nodes, and clustering

Mathieu Moslonka-Lefebvre, Marco Pautasso, Mike J. Jeger

# ► To cite this version:

Mathieu Moslonka-Lefebvre, Marco Pautasso, Mike J. Jeger. Disease spread in small-size directed networks: Epidemic threshold, correlation between links to and from nodes, and clustering. Journal of Theoretical Biology, 2009, 260 (3), pp.402. 10.1016/j.jtbi.2009.06.015 . hal-00554629

# HAL Id: hal-00554629 https://hal.science/hal-00554629

Submitted on 11 Jan 2011

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers. L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

# Author's Accepted Manuscript

Disease spread in small-size directed networks: Epidemic threshold, correlation between links to and from nodes, and clustering

Mathieu Moslonka-Lefebvre, Marco Pautasso, Mike J. Jeger

PII:S0022-5193(09)00267-7DOI:doi:10.1016/j.jtbi.2009.06.015Reference:YJTBI 5597

To appear in: Journal of Theoretical Biology

Received date:13 January 2009Revised date:15 April 2009Accepted date:7 June 2009



www.elsevier.com/locate/yjtbi

Cite this article as: Mathieu Moslonka-Lefebvre, Marco Pautasso and Mike J. Jeger, Disease spread in small-size directed networks: Epidemic threshold, correlation between links to and from nodes, and clustering, *Journal of Theoretical Biology*, doi:10.1016/j.jtbi.2009.06.015

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting galley proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

- 1 Disease spread in small-size directed networks: epidemic
- 2 threshold, correlation between links to and from nodes, and
- 3 clustering
- 4
- 5 Mathieu Moslonka-Lefebvre<sup>1,2,3</sup>, Marco Pautasso<sup>3\*</sup>, & Mike J. Jeger<sup>3</sup>
- 6
- 7 1 Department of Biology, Université Paris-Sud XI, 91405 Orsay, France
- 8 2 Biochemistry and Bio-engineering Department, ENS Cachan, 61 av President Wilson,
- 9 94230 Cachan, France
- 10 3 Division of Biology, Imperial College London, Silwood Park, Ascot, SL5 7PY, UK
- 11 \*correspondence: m.pautasso@ic.ac.uk, ++44 020 759 42533
- 12
- 13 Revised for: Journal of Theoretical Biology
- 14 Running head: Threshold, in- and out-degree, and clustering
- 15
- 16 Word Count: ~ 4,000
- 17 Five tables, four figures
- 18

#### 18 Abstract

19 Network epidemiology has mainly focused on large-scale complex networks. It is unclear 20 whether findings of these investigations also apply to networks of small size. This 21 knowledge gap is of relevance for many biological applications, including meta-22 communities, plant-pollinator interactions and the spread of the oomycete pathogen 23 *Phytophthora ramorum* in networks of plant nurseries. Moreover, many small-size biological 24 networks are inherently asymmetrical and thus cannot be realistically modelled with 25 undirected networks. We modelled disease spread and establishment in directed networks 26 of 100 and 500 nodes at four levels of connectance in six network structures (local, small-27 world, random, one-way, uncorrelated and two-way scale-free networks). The model was 28 based on the probability of infection persistence in a node and of infection transmission 29 between connected nodes. Regardless of the size of the network, the epidemic threshold 30 did not depend on the starting node of infection but was negatively related to the 31 correlation coefficient between in- and out-degree for all structures, unless networks were 32 sparsely connected. In this case clustering played a significant role. For small-size scale-free 33 directed networks to have a lower epidemic threshold than other network structures, there 34 needs to be a positive correlation between number of links to and from nodes. When this 35 correlation is negative (one-way scale-free networks), the epidemic threshold for small-size 36 networks can be higher than in non scale-free networks. Clustering does not necessarily 37 have an influence on the epidemic threshold if connectance is kept constant. Analyses of 38 the influence of the clustering on the epidemic threshold in directed networks can also be 39 spurious if they do not consider simultaneously the effect of the correlation coefficient 40 between in- and out-degree.

41

# 42 Keywords

43 Commercial transport, disease management, food webs, initial conditions, spread of ideas,
44 Susceptible-Infected-Susceptible (SIS)

- 45
- 46

## 46 Introduction

47 Epidemic models assuming regularly or randomly connected individuals are now 48 involving more complex networks (Keeling, 2005; May, 2006; Jeger et al., 2007). Compared 49 to regular lattices, epidemics in small-world networks are facilitated by long-distance 50 connections (Moore and Newman, 2000). In scale-free networks of infinite size, epidemics 51 lack a threshold, which implies that even pathogens with a low probability of transmission 52 will persist (Pastor-Satorras and Vespignani, 2001). Whether these findings also apply to 53 complex networks of small size is still unclear. This is an important knowledge gap as 54 small-size networks are relevant to many epidemics spreading within subgroups of 55 individuals (Liu et al., 2004; Guimarães et al., 2007; Sun and Gao, 2007; Pellis et al., 2009). 56 Networks are not only relevant for epidemiology but have found application in a variety of 57 biological systems (Proulx et al., 2005; Bascompte, 2007; Jeger et al., 2007). Networks with 58 size of the order of magnitude of hundreds of nodes, in turn, are relevant for many current 59 issues in ecology. Examples include closely interacting primate groups (Dunbar, 1993), 60 social networks of manakins (Ryder et al., 2008), cavity-nesting community webs (Blanc 61 and Walters, 2007), diseases of bumble bee colonies (Otterstatter and Thomson, 2007) and 62 vascular epiphytes on host tree species (Lobel et al., 2006; Burns, 2007). The importance of 63 small-size networks in biology is further shown by plant-pollinator interactions (e.g. 64 Olesen et al., 2006; Nielsen and Bascompte, 2007), mycorrhiza, rhizomorphs and plant 65 pathosystems (Southworth et al., 2005; Lamour et al. 2007; Brooks et al., 2008), and food 66 webs (e.g. Montoya and Solé, 2002; Neutel et al., 2007).

67 Although networks of small size are relevant both for epidemiology and for ecology and 68 evolution (e.g. Hanski and Ovaskainen, 2000; Brooks, 2006; Ings et al., 2009), there has been 69 surprisingly little theoretical work investigating whether results obtained for large-size and 70 infinite networks also apply to small-size networks. We know for example that 71 heterogeneity in the contact structure can markedly lower invasion thresholds in networks 72 of large size (Boguna et al., 2004; Colizza and Vespignani, 2007; Jeger et al., 2007), but there 73 is still only patchy knowledge about the dynamics and properties of small-size networks of 74 various structures. This is of concern, given the several applications of small-size networks

75 in natural systems and also given that the rapid globalization and structural changes of 76 trade interactions are increasing the relevance of complex networks of small size for the 77 invasion biology of exotic organisms (Jones and Baker, 2007; Dehnen-Schmutz et al., 2007; 78 Brenn et al., 2008). A real world application of directed networks of small size in epidemic 79 controlling are trade movements of infected material amongst plant nurseries (e.g. 80 Bandyopadhyay and Frederiksen, 1999; Anderson et al., 2004; Slippers et al., 2005). A 81 recent example is given by the spread in regional networks of plant nurseries and garden 82 centres of *Phytophthora ramorum*, the oomycete causing Sudden Oak Death in the West 83 Coast of the USA and leaf blight and dieback in many ornamental shrubs both in America 84 and Europe (Werres et al., 2001; Prospero et al., 2007; Xu et al., 2009). Given the wide range 85 of species affected, movements of infected plant material in the horticultural trade have the 86 potential to make this emerging plant disease outbreak even more widespread 87 (Holdenrieder et al., 2004; Frankel, 2008; Grünwald et al., 2008). There is thus the necessity 88 to control the network of plant nurseries and retail outlets trading ornamental species 89 susceptible to *P. ramorum* in an effective and efficient way.

90 In this study, we investigated whether heterogeneity in the contact structure and the 91 presence of short-cuts as in small-world networks still make a difference to epidemic 92 development in small-size, directed networks. Directed networks, given the more 93 complicated adjacency matrices, have been used to model epidemics relatively rarely 94 (Newman et al., 2001; Meyers et al., 2006; Park and Kim, 2006; Kenah and Robins, 2007), 95 but are relevant to many real-world situations with asymmetries in contact structures, and 96 deserve more study for various network sizes, structures and levels of connectance. We 97 have shown elsewhere (Pautasso and Jeger, 2008) that heterogeneity in the contact 98 structure still affects the epidemic threshold even in the case of networks of one hundred 99 nodes, but inquire here whether variations in the epidemic threshold (the boundary 100 between no epidemic and an epidemic) for different network structures and at different 101 levels of connectance can be explained by the correlation coefficient between links to and 102 from nodes and by the clustering coefficient of the network. Previous work in percolation 103 theory suggests that the correlation coefficient between links to and from nodes is 104 important in the case of heterogeneity in the contact structure (Schwartz et al., 2002;

105 Woolhouse et al., 2005; Kao et al., 2006), but it is unclear whether or not this importance is

106 maintained for small-size networks with different (i) size, (ii) structure (local, random,

107 small-world and scale-free), (iii) levels of connectance, and (iv) clustering.

#### 108 Materials and Methods

109 We simulated disease spread and establishment in networks of 100 and 500 nodes. For both 110 network sizes, we used six kinds of structure: (1) local (nearest-neighbour transmission), (2) 111 random (nodes connected with probability p), (3) small-world (local networks rewired 112 with short-cuts), and scale-free structure (see Jeger et al., 2007 for a visualization). For 113 scale-free networks, we considered separately networks with in- and out-degree of nodes 114 (4) positively, (5) not and (6) negatively correlated. The networks were directed, i.e. a link 115 from node a to node b did not imply the reverse connection (as e.g. in Newman et al., 2001; 116 Boguna and Serrano, 2005; Meyers et al., 2006; Park and Kim, 2006). Directed networks are 117 realistic approximations of many real world systems; wherever a directed link does not 118 entail the reverse connection (asymmetrical interactions), from food webs to plant-animal mutualistic networks, from infectious disease epidemiology to the spread of information 119 120 (Bascompte et al., 2003; da Gama and Nunes, 2006; Bode et al., 2008; Thebault and 121 Fontaine, 2008).

122 For each network structure, 100 replicates were built in MATLAB at each level of 123 connectance (for 100 nodes: 100, 200, 400, and 1000 links; for 500 nodes: 500, 2500, 5000, and 124 10000 links; Table 1). Rather than keeping connectance constant between the two network 125 sizes, we needed a lower level of connectance for 500 nodes (500 links, i.e. connectance = 126 0.002) to obtain results similar to the lower level of connectance for 100 nodes (100 links, i.e. 127 connectance = 0.01). To obtain a disappearance of a significant correlation between 128 epidemic threshold and correlation links in and out for the larger network size (500 nodes), 129 it was necessary to use a lower connectance level that at network size equal 100 nodes. We 130 also left out results from the highest level of connectance for 500 nodes (25000 links, i.e. 131 connectance = 0.1) as in this case networks were overly connected so that differences 132 between network structures were likely to be small. Local networks were built starting 133 from a regular ring with 100 (for 100 nodes) and 500 (for 500 nodes) links more than the

134 target number of links and by randomly generating 100 or 500 gaps, respectively. Random 135 digraphs were generated using the G(N,M) model where M directed links are placed 136 randomly and independently between the N nodes of the graph. Small-world networks 137 were built with the Watts and Strogatz (1998) algorithm and a rewiring coefficient of 0.25. 138 This rewiring coefficient allowed the construction of small-world networks with clustering 139 intermediate between those of random and of local networks. Small-world networks are 140 networks with high clustering compared to random networks and small shortest path 141 length compared to local networks, and these conditions were respected in our case. Scale-142 free networks were built with a preferential attachment algorithm, starting with a seed 143 network and based on five parameters adding nodes and/or links depending on the in-, 144 out-, and total degree of existing nodes: 145 1) The parameter a added both a node i and an arrow (i,j) according to the in-degree of 146 an existing node j. This step was repeated a-times. 2) The parameter b added both a node i and an arrow (j,i) according to the out-degree 147 148 of an existing node j. This step was repeated b-times. 149 3) The parameter c added both a node i and an arrow (i,j) or (j,i) according to the total 150 degree of an existing node j. The choice of the direction of each arrow added, i.e. (i,j) 151 or (j,i), was equally probable. This step was repeated c-times.

- 4) The parameter d only added an arrow (i,j) and not a node according to the outdegree of an existing node i and to the in-degree of an existing node j (i≠j). This step
  was repeated d-times.
- 155 5) The parameter e only added an arrow (i,j) and not a node according to the total
  156 degrees of existing nodes i and j (i≠j). This step was repeated e-times.

Epidemic development was deterministic, with discrete time-step and governed by the probabilities of infection transmission between nodes ( $p_t$ ) and of infection persistence in a node ( $p_p$ ). The transmission probability  $p_t$  was either zero (unconnected nodes) or a value constant for different links in a network but variable amongst network replicates (in order to work at the threshold conditions in each replicate; see Pautasso and Jeger, 2008). The

162 persistence probability  $p_p$  combined in one single parameter the length of infectiousness, 163 detection and control measures. We also set  $p_p$  to be the same for all nodes. Both  $p_t$  and  $p_p$ are real variables, going from 0 to 1. This can be a realistic assumption for many ecological 164 165 networks, wherever persistence and transmission are not either switched on or off, but can 166 assume any value between these two extremes. We assumed all nodes to be of equal 167 capacity and kind (differences between nodes are thus entirely due to their in- and out-168 degree). At each iteration, the contact structure of the network realization was maintained 169 exactly the same. Networks were not necessarily fully connected, so it is possible that at the 170 lower levels of connectance not all nodes could be reached from all nodes.

For each iteration, we obtained the infection status of a given node  $P_{i(x)}$  in the following way:

173  $P_{i(x)} = \sum p_{t(x,y)} P_{i(y)}$ 

174 for y going from 1 to 100, where pt refers to the connection of the node x from a node y, and  $P_{i(y)}$  is the infection status of the node y at the previous iteration. At the beginning of the 175 176 epidemic  $P_{i(x)}$  was set to zero for all nodes except for the starting node of the epidemic, 177 with  $P_{(i)} = 1$ . For the connection of a node with itself,  $p_p$  was used instead of  $p_t$ . The 178 biological motivation for self-loops is that nodes which have become infected by a 179 pathogen have a certain probability to remain infected due to the persistence of inoculum 180 through time. The model was thus a SIS (Susceptible-Infected-Susceptible) model. This can 181 be a realistic assumption for many epidemiological systems, wherever nodes are still at risk 182 even after eradication of a disease outbreak if complete immunization is not possible and if 183 there is a continuing trade or contact with susceptible material or inoculum (Jeger et al., 184 2007).

The development of the epidemic was assessed on the basis of the sum of  $P_{i(x)}$  across all nodes and on the basis of the number of nodes with  $P_{i(x)}$  higher than an arbitrary value (0.01). The epidemic was started with a single infection of a single node, as the threshold conditions were not affected by whether epidemics are started with a single or with multiple infections (unpublished observations). Also, results were consistent using a different starting probability of infection. Although the starting node had a marked

- 191 influence on the epidemic size at equilibrium (Pautasso and Jeger, 2008), making the
- 192 epidemic start from different nodes did not affect the threshold conditions  $(p_p^* \text{ and } p_t^*)$
- 193 which define a boundary between no epidemic and an epidemic. Given that there is a
- linear threshold in a graph of  $p_{p}^{*}$  as a function of  $p_{t}^{*}$  (Pautasso and Jeger, 2008), we worked
- 195 at  $p_p^* = 0$  and assessed the threshold only in terms of  $p_t^*$ .

196 The clustering coefficient C<sub>i</sub> of a node i which is part of a digraph characterizes the extent

197 to which nodes adjacent to any node i are adjacent to each other. More precisely,

$$198 \qquad C_i = \frac{Card(E_{\Gamma i})}{Card(V_{\Gamma i}) \times [Card(V_{\Gamma i}) - 1]}$$

199 where *Card* (*X*) symbolyses the cardinality of the set *X*, i.e. the number of elements of *X*. 200 The neighbourhood  $\Gamma_i$  of a vertex *i* is the digraph that consists solely of the set  $V_{\Gamma i}$  of 201 vertices connected from and/or to *i* (not including *i* itself) and of the set  $E_{\Gamma i}$  of all arrows 202 connecting such vertices. The average clustering *C* of a digraph is the average of the 203 clustering of each node of this digraph.

Analysis of variance (ANOVA) of the epidemic threshold and of the correlation coefficient 204 205 between in- and out-degree of the 100 nodes of the network replicates for the different 206 levels of connectance (within a network structure) and for the different types of network 207 structure (at a given level of connectance) was carried out in SAS 9.1 (proc ANOVA). The 208 same package (proc GLM) was used for multi-variate regressions of the threshold  $p^{*}_{t}$ 209 against the correlation coefficient between in- and out-degree and the average clustering 210 coefficient of the 100/500 nodes of the network replicate for a given network structure and 211 level of connectance.

#### 212 Results

The threshold  $p^*_t$  significantly decreased with increasing connectance for all structures and with both network sizes (Fig. 1a, b). With the exception of the lowest connectance level for both network sizes, two-way scale-free networks showed a significantly lower and oneway scale-free networks a significantly higher threshold than all other structures (Fig. 1c,

217 d). For network size of 100 nodes, random networks showed a significantly lower 218 threshold than local networks, but not at the highest connectance (Fig 1c). This result was 219 not confirmed at the larger network size, where random networks had an epidemic 220 threshold not significantly different than the one of local networks (Fig. 1d). Small-world 221 networks showed a threshold not significantly different from random networks (except at 222 the lowest connectance for both network sizes). The threshold of uncorrelated scale-free 223 networks, at all connectance levels and for both network sizes, lay between those for two-224 way and one-way scale-free networks (Fig. 1c, d).

The correlation coefficient between in- and out-degree of the nodes of the networks was, by definition, positive for two-way scale-free networks, not significantly different from zero for uncorrelated scale-free networks (except, by chance, at some levels of connectance; Fig. 1e, f), and negative for one-way scale-free networks. This coefficient was also clustered around zero for local, small-world and random networks. There was a significant increase in the correlation coefficient between in- and out-degree for two-way scale-free networks with increasing connectance for both network sizes (Fig. 1g, h).

232 With the exception of the lowest connectance, there were generally significantly negative 233 relationships between threshold  $p^*_t$  and the correlation coefficient between in- and out-234 degree for all structures and with both network sizes (Fig. 2; Tables 2, 3). At the lowest 235 level of connectance, these relationships were significant only for small-world and two-236 way scale-free networks for the network size of 100 nodes. The proportion of variance in 237 threshold  $p^{*}_{t}$  explained by the correlation between the in- and the out-degree increased 238 with connectance for all structures and for both network sizes, but with exception of two-239 way scale-free networks. Apart from one-way scale-free networks for 500 nodes and two-240 way scale-free networks for both network sizes, at the highest connectance these 241 proportions of variance were substantial (between 0.84 and 0.93 for 100 nodes, and 242 between 0.56 and 0.95 for 500 nodes). However, apart from one-way scale-free networks at 243 the network size of 100 nodes, the slopes of these relationships tended to become flatter 244 with increasing connectance (Tables 2, 3). This is a consequence of the overall lower 245 threshold  $p^*_t$  at higher connectance (Fig. 2).

246 Including the clustering as an additional explaining factor in the regression of the 247 threshold  $p^*_t$  against the correlation coefficient between links to and from nodes did not affect the previous results. The clustering increased with increasing level of connectance for 248 249 all structures and for both network sizes (Figs. 3, 4). By definition, at a given connectance, 250 the clustering was higher in local than in small-world networks, and in small-world 251 compared to random networks (Figs. 3, 4). The clustering also decreased from two-way to 252 uncorrelated and to one-way scale-free networks (Figs. 3, 4). For both network sizes, the 253 clustering was significantly negatively related to the threshold pt at the lowest level of 254 connectance for local and small-world networks, and at all levels of connectance for two 255 way scale-free networks (Fig. 3; Tables 2, 3).

For both network sizes, the relationship of the correlation coefficient between links to and from nodes with the clustering was significantly positive for all scale-free networks (except at the lowest level of connectance; Fig. 4; Tables 4, 5). However, the proportion of variance in the correlation coefficient between in- and out-degree explained by the clustering was substantial for uncorrelated and one-way, but not for two-way scale-free networks. For both network sizes, there was also a significantly positive correlation between these two variables for random networks at the two highest connectance levels (Tables 4, 5).

#### 263 Discussion

264 Networks of small size have biological significance in a variety of ecological fields. 265 Examples include meta-populations, mutualistic and antagonistic interactions (Dunne et 266 al., 2002; Lundgren and Olesen, 2005; Brooks, 2006; Pautasso et al., 2008; Thebault and 267 Fontaine, 2008). In spite of the relevance of small-size networks for many issues in natural 268 sciences, it is not clear whether theoretical results derived from analyses of large-scale 269 complex networks apply also to small-size networks (Guimarães et al., 2007). Moreover, 270 much work in network epidemiology has focused on undirected networks (e.g. Keeling, 271 2005; Shirley and Rushton, 2005; May, 2006), whereas many of the biological networks 272 mentioned in the Introduction are inherently asymmetrical and thus can only be 273 realistically modelled with directed networks.

274 Our analysis shows that in SIS models of epidemics in directed networks of small size, the 275 threshold is lower for scale-free network structures only if there is a positive correlation 276 between in- and out-degree of nodes (Fig. 2). This finding is in good agreement with results 277 obtained in percolation theory for large-size networks (Schwartz et al., 2002). Our work 278 further shows that when this correlation between in- and out-degree of nodes is negative 279 (one-way scale-free networks), for small-size networks the epidemic threshold is higher 280 than in non scale-free networks. This result is broadly independent of the connectance level 281 and of the network size, although it breaks down in case of sparsely connected networks. 282 For sparsely connected networks, differences in clustering amongst networks can become 283 important.

284 Clustering has been shown to be a network feature relevant to the development and 285 control of epidemics (e.g. Eames and Keeling, 2003; Keeling, 2005; Kiss et al., 2005; 286 Hartivgsen et al., 2007; Turner et al., 2008; Miller, 2009). More clustered networks are 287 believed to be less prone to invasion by a pathogen, as this will be likely to become confined inside clusters of connected nodes (Keeling, 2005; Naug, 2008). Clusters can also 288 289 slow down epidemic development in the first phases of epidemics (e.g. Szendroi and 290 Csanyi, 2004). In scale-free networks of infinite size, the presence of high local clustering 291 has been shown to make it possible for a non-null epidemic threshold to be present 292 (Eguiluz and Klemm, 2002; but see Serrano and Boguna, 2006). Clustering in large-scale 293 scale-free networks has been shown to decrease the size of epidemics, but also to decrease 294 their threshold (Newman, 2003).

295 In the case of our small-size networks, the influence of clustering on the epidemic 296 threshold is dwarfed by the one of the correlation coefficient between in- and out-degree. 297 The negative correlation between epidemic threshold and clustering for uncorrelated and 298 one-way scale-free networks at the highest levels of connectance (Fig. 3) can be explained 299 by the positive correlation of the correlation coefficient between in- and out-degree with 300 the clustering in the same networks (Fig. 4). Unlike Eames (2008) for undirected networks, 301 we do not observe any differences in the influence of clustering on epidemic development 302 depending on whether contacts are regular or random. In our small-size, directed

networks, with the exception of sparsely connected local networks, clustering has noinfluence on the epidemic threshold within both local and random networks (Fig. 3).

305 Our main conclusion is that, in directed networks, analyses of the influence of clustering on 306 the epidemic threshold can be spurious if they do not consider simultaneously the effect of 307 the correlation coefficient between in-and out-degree. In some cases (sparsely connected 308 local, small-world and two-way scale free networks), we observe a negative correlation 309 between epidemic threshold and clustering and no significant effect of the correlation 310 coefficient between in- and out- degree on the threshold, suggesting that in extreme 311 situations the correlation coefficient between in- and out-degree may not play the same 312 role as for large-scale networks. We also point out that analyses which show that clustering 313 is negatively related to the epidemic threshold (e.g. Britton et al., 2008) need to take the 314 connectance level into account, as (i) the clustering increases with increasing connectance, 315 (ii) the epidemic threshold decreases with increasing connectance, so that (iii) if 316 connectance increases the epidemic threshold is bound to be negatively correlated with the 317 clustering. Our results show, however, that, when keeping connectance constant, clustering 318 and epidemic threshold are not necessarily related.

Epidemics in scale-free networks are now commonly investigated (e.g. Boccaletti et al., 319 320 2006; Masuda and Konno, 2006; Colizza and Vespignani, 2008), but there has been less 321 attention to directed scale-free networks. In this analysis, we confirm the importance of the 322 correlation coefficient between in- and out-degree for epidemics in directed scale-free 323 networks (Woolhouse et al., 2005). We also provide evidence for a different behaviour of 324 the clustering in directed scale-free networks where there are different correlation 325 coefficients between in- and out-degree. Although there is a positive correlation of the 326 clustering with the correlation coefficient between in-and out-degree for all scale-free 327 network types (except at the lowest connectance level), only for uncorrelated and one-way 328 scale-free networks has this correlation a substantial r<sup>2</sup> (for 500 nodes: between 0.54 and 329 0.79, Table 5). For two-way scale-free networks, the correlation between clustering and 330 correlation coefficient between links in and out of nodes is significant but negligible (again 331 for 500 nodes, r<sup>2</sup> values are between 0.01 and 0.08, Table 5). Further work is needed to

investigate whether results obtained for undirected scale-free networks apply to all typesof directed scale-free networks.

334 Our results are essentially independent of the network size used (100 and 500 nodes). We 335 have avoided using a lower number of nodes than 100 as it is likely that differences 336 between network structures will tend to become blurred for even smaller networks. 337 However, an interesting question would be at which small size heterogeneity in the contact 338 structure stops having a significant influence on network properties and epidemic 339 processes. A remarkable result is also that network breakdown at the lowest connectance 340 level (which causes for example the disappearance of a significant relationship between 341 epidemic threshold and correlation coefficient between in- and out-degree) happens at two 342 different connectance levels (0.01 for 100 nodes and 0.002 for 500 nodes, Table 1) for the 343 two network sizes. More research is needed to assess which other processes are not 344 independent of the interaction between network size and connectance. 345 In conclusion, our study confirms the result obtained for large-size networks that 346 regardless of the size of the network, the epidemic threshold is negatively related to the 347 correlation coefficient between in- and out-degree for all structures. However, this does not 348 hold if small-size networks are sparsely connected. In this case, clustering plays a 349 significant role (Table 2). In small-size networks, when the correlation between links in and 350 out is negative (one-way scale-free networks), the epidemic threshold can be higher than in 351 non scale-free networks. Contrary to what is found in analyses of large-size networks, 352 clustering does not necessarily have an influence on the epidemic threshold of small-size 353 networks if connectance is kept constant. Analyses of the influence of the clustering on the 354 epidemic threshold in directed networks can also be spurious if the effect of the correlation 355 coefficient between in- and out-degree is disregarded.

# 356 Acknowledgments

357 Many thanks to T. Harwood, O. Holdenrieder, J. Parke, M. Shaw, J. Tufto, F. Van den Bosch and X. Xu for

358 discussions and insights, and to T. Matoni and anonymous reviewers for helpful comments on a previous

359 version of the ms. This study was funded by the Department for Environment, Food and Rural Affairs, the

360 Rural Economy and Land Use Programme, UK, and the French Ministry of National Education and Research.

#### 361 **References**

- 362 Anderson, P.K., Cunningham, A.A., Patel, N.G., Morales, F.J., Epstein, P.R., Daszak, P., 2004. Emerging
- 363 infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. Trends Ecol.
- 364 Evol. 19, 535-544.
- 365 Bandyopadhyay, B., Frederiksen, R.A., 1999. Contemporary global movement of emerging plant diseases.
- 366 Ann. New York Acad. Sci. 894, 28-36.
- 367 Bascompte, J., 2007. Networks in ecology. Basic Appl. Ecol. 8, 485-490.
- 368 Bascompte, J., Jordano, P., Melian, C.J., Olesen, J.M., 2003. The nested assembly of plant-animal mutualistic
- 369 networks. Proc. Natl. Acad. Sci. USA 100, 9383-9387.
- 370 Blanc, L.A., Walters, J.R., 2007. Cavity-nesting community webs as predictive tools: where do we go from
- 371 here? J. Ornithology 148, S417-S423.
- 372 Boccaletti, S., Latora, V., Moreno, Y., Chavez, M., Hwang, D.U., 2006. Complex networks: structure and
- 373 dynamics. Phys. Rep. 424 (4-5), 175-308.
- 374 Bode, M., Burrange, K., Possingham, H.P., 2008. Using complex network metrics to predict the persistence of
- 375 metapopulations with asymmetric connectivity patterns. Ecol. Model. 214, 201-209.
- Boguna, M., Serrano, M.A., 2005. Generalized percolation in random directed networks. Phys. Rev. E 72,
  016106.
- Boguna, M., Pastor-Satorras, R., Vespignani, A., 2004. Cut-offs and finite size effects in scale-free networks.
  Eur. Phys. J. B 38, 205-209.
- 380 Brenn, N., Menkis, A., Grünig, C.R., Sieber, T.N., Holdenrieder, O., 2008. Community structure of
- 381 *Phialocephala fortinii* s. lat. in European tree nurseries, and assessment of the potential of the seedlings as
- 382 dissemination vehicles. Mycol. Res. 112, 650-662.
- 383 Britton, T., Deijfen, M., Lageras, A.N., Lindholm, M., 2008. Epidemics on random graphs with tunable
- 384 clustering. J. Appl. Prob. 45, 743-756.
- Brooks, C.P., 2006. Quantifying population substructure: extending the graph-theoretic approach. Ecology 87,
  864-872.
- Brooks, C.P., Antonovics, J., Keitt, T.H., 2008. Spatial and temporal heterogeneity explain disease dynamics in
  a spatially explicit network model. Am. Nat. 172, 149-159.
- Burns, K.C., 2007. Network properties of an epiphyte metacommunity. J. Ecol. 95, 1142–1151.
- Colizza, V., Vespignani, A., 2007. Invasion threshold in heterogeneous metapopulation networks. Phys. Rev.
   Lett. 99, 148701.
  - 6/19/2009

- 392 Colizza, V., Vespignani, A., 2008. Epidemic modeling in metapopulation systems with heterogeneous
- 393 coupling pattern: theory and simulations. J. Theor. Biol. 251 (3), 450-467.
- da Gama, M.M.T., Nunes, A., 2006. Epidemics in small world networks. Eur. Phys. J. B 50, 205-208.
- Dehnen-Schmutz, K., Touza, J., Perrings, C., Williamson, M., 2007. A century of the ornamental plant trade
   and its impact on invasion success. Divers. Dist. 13, 527-534.
- 397 Dunne, J.A., Williams, R.J., Martinez, N.D., 2002. Food-web structure and network theory: the role of
- 398 connectance and size. Proc. Natl. Acad. Sci. USA 99, 12917-12922.
- 399 Eames, K.T.D., 2008. Modelling disease spread through random and regular contacts in clustered
- 400 populations. Theor. Pop. Biol. 73, 104-111.
- 401 Eames, K.T.D., Keeling, M.J., 2003. Contact tracing and disease control. Proc. R. Soc. London B 270, 2565-2571.
- 402 Eguiluz, V.M., Klemm, K., 2002. Epidemic threshold in structured scale-free networks. Phys. Rev. E 89,
- 403 108701.
- 404 Frankel, S.J., 2008. Sudden oak death and *Phytophthora ramorum* in the USA: a management challenge. Aust.
- 405 Plant Path. 24: 282-284.
- 406 Grünwald, N.J., Goss, E.M., Press, C.M., 2008. *Phytophthora ramorum*: a pathogen with a remarkably wide host
- 407 range causing sudden oak death on oaks and ramorum blight on woody ornamentals. Mol. Plant Path. 9, 729-408 740.
- 409 Guimarães, P.R., de Menezes, M.A., Baird, R.W., Lusseau, D., Guimaraes, P., dos Reis, S.F., 2007.
- 410 Vulnerability of a killer whale social network to disease outbreaks. Phys. Rev. E 76, 042901.
- Hanski, I., Ovaskainen, O., 2000. The metapopulation capacity of a fragmented landscape. Nature 404, 755-758.
- 413 Hartivgsen, G., Dresch, J.M., Zielinski, A.L., Macula, A.J., Leary, C.C., 2007. Network structure, and
- vaccination strategy and effort interact to affect the dynamics of influenza epidemics. J. Theor. Biol. 246, 205-213.
- Holdenrieder, O., Pautasso, M., Weisberg, P.J., Lonsdale D., 2004. Tree diseases and landscape processes: the
  challenge of landscape pathology. Trends Ecol. Evol. 19, 446-452.
- 418 Ings, T.C., Montoya, J.M., Bascompte, J., Bluethgen, N., Brown, L., Dormann, C.F., Edwards, F., Figueroa, D.,
- 419 Jacob, U., Jones, J.I., Lauridsen, R.B., Ledger, M.E., Lewis, H.M., Olesen, J.M., van Veen, F.F.J., Warren, P.H.,
- 420 Woodward, G., 2009. Ecological networks beyond food webs. J. Anim. Ecol. 78, 253-269.
- 421 Jeger, M.J., Pautasso, M., Holdenrieder, O., Shaw, M.W., 2007. Modelling disease spread and control in
- 422 networks: implications for plant sciences. New Phytol. 174, 279-297.

- 423 Jones, D.R., Baker, R.H.A., 2007. Introductions of non-native plant pathogens into Great Britain, 1970-2004.
- 424 Plant Pathol. 56, 891-910.
- 425 Kao, R.R., Danon, L., Green, D.M., Kiss, I.Z., 2006. Demographic structure and pathogen dynamics of the
- 426 network of livestock movements in Great Britain. Proc. R. Soc. London B 273, 1999-2007.
- 427 Keeling, M.J., 2005. The implications of network structure for epidemic dynamics. Theor. Pop. Biol. 67, 1-8.
- 428 Kenah, E., Robins, J.M., 2007. Network-based analysis of stochastic SIR epidemic models with random and
- 429 proportionate mixing. J. Theor. Biol. 249, 706-722.
- Kiss, I.Z., Green, D.M., Kao, R.R., 2005. Disease contact tracing in random and clustered networks. Proc. R.
  Soc. London B 272, 1407-1414.
- 432 Lamour, A., Termorshuizen, A.J., Volker, D., Jeger, M.J., 2007. Network formation by rhizomorphs of
- 433 *Armillaria lutea* in natural soil: their description and ecological significance. FEMS Microb. Ecol. 62, 222-232.
- 434 Liu, J.Z., Wu, J.S., Yang, Z.R., 2004. The spread of infectious disease on complex networks with household-
- 435 structure. Physica A 341, 273-280.
- 436 Lobel, S., Snall, T., Rydin, H., 2006. Metapopulation processes in epiphytes inferred from patterns of regional
- 437 distribution and local abundance in fragmented forest landscapes. J. Ecol. 94, 856-868.
- 438 Lundgren, R., Olesen, J.M., 2005. The dense and highly connected world of Greenland's plants and their
- 439 pollinators. Arct. Antarct. Alp. Res. 37, 514-520.
- 440 Masuda, N., Konno, N., 2006. Multi-state epidemic processes on complex networks. J. Theor. Biol. 243, 64-75.
- 441 May, R.M., 2006. Network structure and the biology of populations. Trends Ecol. Evol. 21, 394-399.
- 442 Meyers, L.A., Newman, M.E.J., Pourbohloul, B., 2006. Predicting epidemics on directed contact networks. J.
- 443 Theor. Biol. 240, 400-418.
- Miller, J.C., 2009. Spread of infectious disease through clustered populations. J. Roy. Soc. Interface, in press.
  doi:10.1098/rsif.2008.0524
- 446 Montoya, J.M., Solé, R.V., 2002. Small world patterns in food webs. J. Theor. Biol. 214, 405-412.
- 447 Moore C, Newman MEJ (2000) Epidemics and percolation in small-world networks. Phys. Rev. E 61, 5678448 5682.
- 449 Naug, D., 2008. Structure of the social network and its influence on transmission dynamics in a honeybee
- 450 colony. Behav. Ecol. Sociobiol. 62, 1719-1725.
- 451 Neutel, A.M., Heesterbeek, J.A.P., van de Koppel, J., Hoenderboom, G., Vos, A., Kaldeway, C., Berendse, F.,
- 452 de Ruiter, P.C., 2007. Reconciling complexity with stability in naturally assembling food webs. Nature 449,
- 453 599-602.

- 454 Newman, M.E.J., 2003. Properties of highly clustered networks. Phys. Rev. E 68, 026121.
- 455 Newman, M.E.J., Strogatz, S.H., Watts, D.J., 2001. Random graphs with arbitrary degree distributions and
- 456 their applications. Phys. Rev. E 64, 026118.
- 457 Nielsen, A., Bascompte, J., 2007. Ecological networks, nestedness and sampling effort. J. Ecol. 95, 1134–1141.
- 458 Olesen, J.M., Bascompte, J., Dupont, Y.L., Jordano, P., 2006. The smallest of all worlds: pollination networks. J.
- 459 Theor. Biol. 240, 270-276.
- 460 Otterstatter, M.C., Thomson, J.D., 2007. Contact networks and transmission of an intestinal pathogen in
- 461 bumble bee (*Bombus impatiens*) colonies. Oecologia 154, 411-421.
- 462 Park, S.M., Kim, B.J., 2006. Dynamic behaviors in directed networks. Phys. Rev. E 74, 026114.
- Pastor-Satorras, R., Vespignani, A., 2001. Epidemic spreading in scale-free networks. Phys. Rev. Lett. 86, 32003203.
- 465 Pautasso, M., Jeger, M.J., 2008. Epidemic threshold and network structure: the interplay of probability of
- 466 transmission and of persistence in small-size directed networks. Ecol. Compl. 5, 1-8.
- 467 Pautasso, M., Harwood, T., Shaw, M.W., Xu, X., Jeger, M.J., 2008. Epidemiological modeling of *Phytophthora*
- 468 *ramorum*: network properties of susceptible plant genera movements in the nursery sector of England and
- 469 Wales. Proc Sudden Oak Death III Science Symposium, PSW-GTR-214, USDA Forest Service, pp. 257-264.
- 470 Pellis, L., Ferguson, N.M., Fraser, C., 2009. Threshold parameters for a model of epidemic spread among

471 households and workplaces. J. R. Soc. Interface, in press doi:10.1098/rsif.2008.0493

- 472 Prospero, S., Hansen, E.M., Grunwald, N.J., Winton, L.M., 2007. Population dynamics of the sudden oak
- 473 death pathogen *Phytophthora ramorum* in Oregon from 2001 to 2004. Mol. Ecol. 16, 2958-2973.
- 474 Proulx, S.R., Promislow, D.E.L., Phillips, P.C., 2005. Network thinking in ecology and evolution. Trends Ecol.
  475 Evol. 20, 345-353.
- 476 Ryder, T.B., McDonald, D.B., Blake, J.G., Parker, P.G., Loiselle, B.A., 2008. Social networks in the lek-mating
  477 wire-tailed manakin (*Pipra filicauda*). Proc. R. Soc. London B 275, 1367-1374.
- Schwartz, N., Cohen, R., ben-Avraham, D., Barabasi, A.L., Havlin, S., 2002. Percolation in directed scale-free
  networks. Phys. Rev. E 66, 015104.
- 480 Serrano, M.A., Boguna, M., 2006. Percolation and epidemic thresholds in clustered networks. Phys. Rev. Lett.481 97, 088701.

<sup>482</sup> Shirley, M.D.F., Rushton, S.P., 2005. The impact of network topology on disease spread. Ecol. Compl. 2, 287483 299.

- 484 Slippers, B., Stenlid, J., Wingfield, M.J., 2005. Emerging pathogens: fungal host jumps following
- 485 anthropogenic introduction. Trends Ecol. Evol. 20, 420-421
- 486 Southworth, D., He, X.H., Swenson, W., Bledsoe, C.S., Horwath, W.R., 2005. Application of network theory to
- 487 potential mycorrhizal networks. Mycorrhiza 15, 589-595.
- 488 Sun, H.J., Gao, Z.Y., 2007. Dynamical behaviors of epidemics on scale-free networks with community
- 489 structure. Physica A 381, 491-496.
- 490 Szendroi, B., Csanyi, G., 2004. Polynomial epidemics and clustering in contact networks. Proc. R. Soc. London
  491 B 271, S364-S366.
- Thebault, E., Fontaine, C., 2008. Does asymmetric specialization differ between mutualistic and trophicnetworks? Oikos 117, 555-563.
- 494 Turner, J., Bowers, R.G., Clancy, D., Behnke, M.C., Christley, R.M., 2008. A network model of *E. coli* O157
- transmission within a typical UK dairy herd: the effect of heterogeneity and clustering on the prevalence of
- 496 infection. J. Theor. Biol. 254, 45-54.
- 497 Watts, D.J., Strogatz, S.H., 1998. Collective dynamics of 'small-world' networks. Nature 393, 440-442.
- 498 Werres, S., Marwitz, R., Veld, W.A.M.I., De Cock, A.W.A.M., Bonants, P.J.M., De Weerdt, M., Themann, K.,
- 499 Ilieva, E., Baayen, R.P., 2001. *Phytophthora ramorum* sp nov., a new pathogen on *Rhododendron* and *Viburnum*.
- 500 Mycol. Res. 105: 1155-1165.
- 501 Woolhouse, M.E.J., Shaw, D.J., Matthews, L., Liu, W.C., Mellor, D.J., Thomas, M.R., 2005. Epidemiological
- 502 implications of the contact network structure for cattle farms and the 20-80 rule. Biol. Lett. 1, 350-352.
- 503 Xu, X.M., Harwood, T.D., Pautasso, M., Jeger, M.J., 2009. Spatio-temporal analysis of *Phytophthora ramorum*
- 504 cases in England and Wales (2003-2006). Ecography, in press

Accei

505 **Table 1.** Number of links (L) and connectance ( $C = L/N^2$ ) for the two network sizes studied

506 (number of nodes = N).

N	100			
L	100	200	400	1000
С	0.01	0.02	0.04	0.1
N	500			
L	500	2500	5000	10000
С	0.002	0.01	0.02	0.04

507

508

6/19/2009

508 **Table 2.** Proportion of variance explained, slope and associated p value of the regression of 509 threshold probability of transmission as a function of (a) the correlation coefficient between 510 in- and out-degree and (b) the clustering for the 100 replicates of the six network structures 511 (local, random, small-world, two-way, uncorrelated, and one-way scale-free) at four levels

512 of connectance (100, 200, 400 and 1000 links; network size = 100 nodes).

		100			200			400			1000	
	r <sup>2</sup>	а	b	r <sup>2</sup>	а	b	r <sup>2</sup>	а	b	r <sup>2</sup>	a	b
1	0.17	+0.05	-5.45	0.33	-0.06	+0.09	0.58	-0.01	+0.03	0.85	-0.001	+0.000
	р	0.30	0.001		0.001	0.15		0.001	0.03		0.001	0.98
r	0.02	-0.07	-0.76	0.54	-0.25	+0.04	0.74	-0.06	+0.02	0.86	-0.009	-0.003
	р	0.23	0.41		0.001	0.89		0.001	0.79		0.001	0.86
SW	0.22	-0.16	-4.63	0.27	-0.11	-0.02	0.62	-0.02	-0.00	0.84	-0.002	+0.001
	р	0.003	0.001		0.001	0.86		0.001	0.82		0.001	0.45
sf2	0.11	-0.17	-3.24	0.55	-0.23	-0.30	0.34	-0.09	-0.02	0.25	-0.04	-0.01
	р	0.008	0.04		0.001	0.001		0.001	0.05		0.001	0.001
sf0	0.01	-0.04	-0.90	0.28	-0.59	-0.27	0.59	-0.69	+0.28	0.93	-0.27	+0.02
_	р	0.49	0.48	CK	0.001	0.61		0.001	0.33		0.001	0.45
sf1	0.04	+0.12	-1.52	0.03	-0.86	+1.66	0.23	-3.31	+4.09	0.87	-0.92	+0.09
	р	0.27	0.15		0.12	0.24		0.001	0.005		0.001	0.35

513

Table 3. Proportion of variance explained, slope and associated p value of the regression of
threshold probability of transmission as a function of (a) the correlation coefficient between
in- and out-degree and (b) the clustering for the 100 replicates of the six network structures

- 517 (local, random, small-world, two-way, uncorrelated, and one-way scale-free) at four levels
- 518 of connectance (500, 2500, 5000 and 10,000 links; network size = 500 nodes).

		500			2500			5000			10000	
	r <sup>2</sup>	а	b	r <sup>2</sup>	a	b	r <sup>2</sup>	а	b	r <sup>2</sup>	a	b
1	0.15	+0.00	-32.7	0.66	-0.01	-0.00	0.48	-0.001	-0.001	0.56	-0.0001	-0.0001
	р	0.97	0.001		0.001	0.63		0.001	0.68		0.001	0.25
r	0.02	-0.21	+3.43	0.77	-0.04	-0.02	0.87	-0.01	-0.03	0.94	-0.002	-0.003
	р	0.21	0.50		0.001	0.89		0.001	0.31		0.001	0.66
SW	0.24	-0.13	-26.7	0.66	-0.01	+0.00	0.68	-0.002	-0.003	0.87	-0.0004	-0.0001
	р	0.20	0.001		0.001	0.85		0.001	0.15		0.001	0.76
sf2	0.08	-0.20	-18.1	0.50	-0.06	-0.09	0.60	-0.03	-0.02	0.38	-0.04	-0.004
	р	0.06	0.03		0.001	0.001		0.02	0.001		0.001	0.001
sf0	0.01	-0.12	-6.20	0.41	-3.66	+5.74	0.83	-0.64	+0.44	0.95	-0.16	+0.07
	р	0.48	0.44	C	0.001	0.01		0.001	0.001		0.001	0.001
sf1	0.01	-0.12	-5.11	0.15	-4.16	+2.59	0.55	-2.28	+0.75	0.40	-0.63	+0.11
	р	0.38	0.49		0.001	0.33		0.001	0.07		0.001	0.07

519

520

521 **Table 4.** Proportion of variance explained, slope and associated p value of the regression of

522 the correlation coefficient between in- and out-degree as a function of the clustering for the

523 100 replicates of the six network structures (local, random, small-world, two-way,

524 uncorrelated, and one-way scale-free) at four levels of connectance (100, 200, 400 and 1000

525 links; network size = 100 nodes).

		100			200			400		1000			
	r <sup>2</sup>	slope	р	<b>r</b> <sup>2</sup>	slope	р	r <sup>2</sup>	slope	р	r <sup>2</sup>	slope	р	
1	0.06	-7.40	0.01	0.00	-0.01	0.99	0.00	+0.26	0.85	0.00	-0.93	0.88	
r	0.00	-0.65	0.69	0.01	+0.14	0.37	0.07	+5.37	0.008	0.06	+10.1	0.01	
sw	0.00	-0.84	0.68	0.01	-0.49	0.41	0.03	+1.55	0.09	0.00	+0.33	0.83	
sf2	0.00	+1.46	0.56	0.18	+1.13	0.001	0.04	+0.16	0.04	0.06	+0.05	0.01	
sf0	0.00	-0.03	0.98	0.13	+1.81	0.001	0.65	+2.61	0.001	0.70	+1.83	0.001	
sf1	0.05	-2.02	0.03	0.08	+0.71	0.005	0.42	+1.50	0.001	0.69	+1.37	0.001	

526

527 **Table 5.** Proportion of variance explained, slope and associated p value of the regression of

528 the correlation coefficient between in- and out-degree as a function of the clustering for the

529 100 replicates of the six network structures (local, random, small-world, two-way,

- 530 uncorrelated, and one-way scale-free) at four levels of connectance (500, 2500, 5000 and
- 531 10000 links; network size = 500 nodes).

		500			2500			5000			10000		
	r <sup>2</sup>	slope	р	r <sup>2</sup>	slope	р	r <sup>2</sup>	slope	р	r <sup>2</sup>	slope	р	
1	0.05	-45.5	0.03	0.00	+1.61	0.48	0.00	-3.40	0.46	0.00	-4.90	0.52	
r	0.01	+3.08	0.32	0.02	+7.42	0.21	0.09	+22.4	0.002	0.06	+25.0	0.01	
SW	0.00	+2.67	0.59	0.00	+0.05	0.96	0.00	+0.02	0.98	0.00	-0.84	0.60	
sf2	0.00	-0.37	0.96	0.08	+0.19	0.005	0.09	+0.04	0.002	0.01	+0.01	0.33	
sf0	0.00	+2.82	0.55	0.73	+3.33	0.001	0.79	+2.18	0.001	0.71	+1.71	0.001	
sf1	0.00	-1.63	0.75	0.54	+1.60	0.001	0.74	+1.10	0.001	0.55	+0.45	0.001	
	Accepted												

532

#### 533 Figure legends

- **Fig. 1:** Threshold probability of transmission for the different (a, b) levels of connectance
- 535 and (c, d) network structures, and correlation coefficient between in- and out-degree of the
- 536 nodes of the 100 replicates for the different (e, f) levels of connectance and (g, h) network
- 537 structures. Network size = 100 (a, c, e, g) and 500 (b, d, e, h) nodes. Error bars are standard
- 538 deviations of 100 replicates. Different letters show significant differences (ANOVA, p <
- 539 0.05) within (a, b, e, f) levels of connectance for a given network structure, and (c, d, g, h)
- 540 network structures for a given level of connectance.
- 541 Fig. 2: Threshold probability of transmission for the different network replicates as a
- 542 function of the correlation coefficient between in- and out-degree of the nodes for the six
- 543 network structures. Network size = 100 nodes (number of links = (a) 100, (b) 200, (c) 400,
- 544 and (d) 1000) and 500 nodes (number of links = (e) 500, (f) 2500, (g) 5000, and (h) 10000).
- 545 **Fig. 3:** Threshold probability of transmission for the different network replicates as a
- 546 function of the average clustering coefficient of the nodes for the six network structures.
- 547 Network size = 100 nodes (number of links = (a) 100, (b) 200, (c) 400, and (d) 1000) and 500

548 nodes (number of links = (e) 500, (f) 2500, (g) 5000, and (h) 10000).

- 549 Fig. 4: Correlation coefficient between in- and out-degree for the different network
- replicates as a function of the average clustering coefficient of the 100 nodes for the six
- 551 network structures. Network size = 100 nodes (number of links = (a) 100, (b) 200, (c) 400,
- 552 and (d) 1000) and 500 nodes (number of links = (e) 500, (f) 2500, (g) 5000, and (h) 10000).





ACCEPTED MANUSCRIP

# threshold probability of transmission



# threshold probability of transmission





threshold probability of transmission

# threshold probability of transmission



# correlation coefficient between in- and out-degree



# correlation coefficient between in- and out-degree

