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1 **Disease spread in small-size directed networks: epidemic**
2 **threshold, correlation between links to and from nodes, and**
3 **clustering**

4

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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
119 mutualistic networks, from infectious disease epidemiology to the spread of information
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 than 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.
- 147 2) The parameter b added both a node i and an arrow (j,i) according to the out-degree
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.
- 152 4) The parameter d only added an arrow (i,j) and not a node according to the out-
153 degree of an existing node i and to the in-degree of an existing node j ($i \neq j$). This step
154 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 ($i \neq j$). This step was repeated e -times.

157 Epidemic development was deterministic, with discrete time-step and governed by the
158 probabilities of infection transmission between nodes (p_t) and of infection persistence in a
159 node (p_p). The transmission probability p_t was either zero (unconnected nodes) or a value
160 constant for different links in a network but variable amongst network replicates (in order
161 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
 164 are real variables, going from 0 to 1. This can be a realistic assumption for many ecological
 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.

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

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

174 for y going from 1 to 100, where p_t refers to the connection of the node x from a node y , and
 175 $P_{i(y)}$ is the infection status of the node y at the previous iteration. At the beginning of the
 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).

185 The development of the epidemic was assessed on the basis of the sum of $P_{i(x)}$ across all
 186 nodes and on the basis of the number of nodes with $P_{i(x)}$ higher than an arbitrary value
 187 (0.01). The epidemic was started with a single infection of a single node, as the threshold
 188 conditions were not affected by whether epidemics are started with a single or with
 189 multiple infections (unpublished observations). Also, results were consistent using a
 190 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^* and p_t^*)
 193 which define a boundary between no epidemic and an epidemic. Given that there is a
 194 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_i 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,

$$198 \quad C_i = \frac{Card(E_{\Gamma_i})}{Card(V_{\Gamma_i}) \times [Card(V_{\Gamma_i}) - 1]}$$

199 where $Card(X)$ symbolyses the cardinality of the set X , i.e. the number of elements of X .
 200 The neighbourhood Γ_i of a vertex i is the digraph that consists solely of the set V_{Γ_i} of
 201 vertices connected from and/or to i (not including i itself) and of the set E_{Γ_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.

204 Analysis of variance (ANOVA) of the epidemic threshold and of the correlation coefficient
 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

213 The threshold p_t^* significantly decreased with increasing connectance for all structures and
 214 with both network sizes (Fig. 1a, b). With the exception of the lowest connectance level for
 215 both network sizes, two-way scale-free networks showed a significantly lower and one-
 216 way 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).

225 The correlation coefficient between in- and out-degree of the nodes of the networks was, by
226 definition, positive for two-way scale-free networks, not significantly different from zero
227 for uncorrelated scale-free networks (except, by chance, at some levels of connectance; Fig.
228 1e, f), and negative for one-way scale-free networks. This coefficient was also clustered
229 around zero for local, small-world and random networks. There was a significant increase
230 in the correlation coefficient between in- and out-degree for two-way scale-free networks
231 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
248 affect the previous results. The clustering increased with increasing level of connectance for
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 p_t 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).

256 For both network sizes, the relationship of the correlation coefficient between links to and
257 from nodes with the clustering was significantly positive for all scale-free networks (except
258 at the lowest level of connectance; Fig. 4; Tables 4, 5). However, the proportion of variance
259 in the correlation coefficient between in- and out-degree explained by the clustering was
260 substantial for uncorrelated and one-way, but not for two-way scale-free networks. For
261 both network sizes, there was also a significantly positive correlation between these two
262 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
288 confined inside clusters of connected nodes (Keeling, 2005; Naug, 2008). Clusters can also
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

303 networks, with the exception of sparsely connected local networks, clustering has no
304 influence 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.

319 Epidemics in scale-free networks are now commonly investigated (e.g. Boccaletti et al.,
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^2 (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^2 values are between 0.01 and 0.08, Table 5). Further work is needed to

332 investigate whether results obtained for undirected scale-free networks apply to all types
333 of 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.

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- 505

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
C	0.01	0.02	0.04	0.1
<hr/>				
N	500			
L	500	2500	5000	10000
C	0.002	0.01	0.02	0.04

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Accepted manuscript

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 ²	a	b	r ²	a	b	r ²	a	b	r ²	a	b
l	0.17	+0.05	-5.45	0.33	-0.06	+0.09	0.58	-0.01	+0.03	0.85	-0.001	+0.000
p	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
p	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
p	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
p	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
p	0.49	0.48		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
p	0.27	0.15		0.12	0.24		0.001	0.005		0.001	0.35	

513

514

514 **Table 3.** Proportion of variance explained, slope and associated p value of the regression of
 515 threshold probability of transmission as a function of (a) the correlation coefficient between
 516 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 ²	a	b	r ²	a	b	r ²	a	b	r ²	a	b
l	0.15	+0.00	-32.7	0.66	-0.01	-0.00	0.48	-0.001	-0.001	0.56	-0.0001	-0.0001
p	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
p	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
p	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
p	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
p	0.48	0.44		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
p	0.38	0.49		0.001	0.33		0.001	0.07		0.001	0.07	

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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 ²	slope	p	r ²	slope	p	r ²	slope	p	r ²	slope	p
l	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

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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 ²	slope	p	r ²	slope	p	r ²	slope	p	r ²	slope	p
l	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

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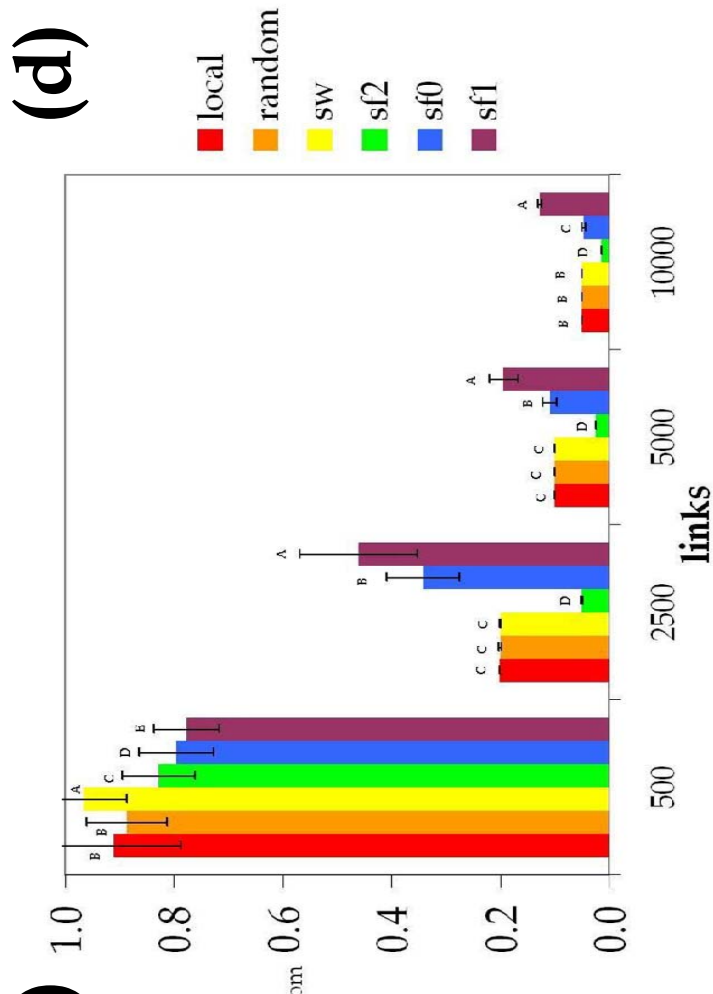
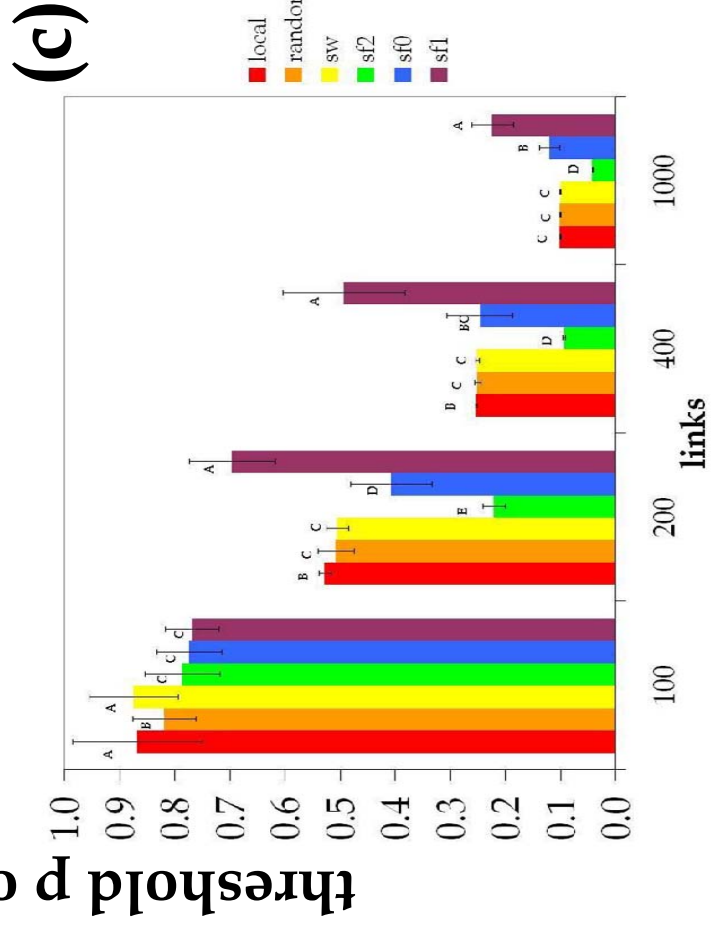
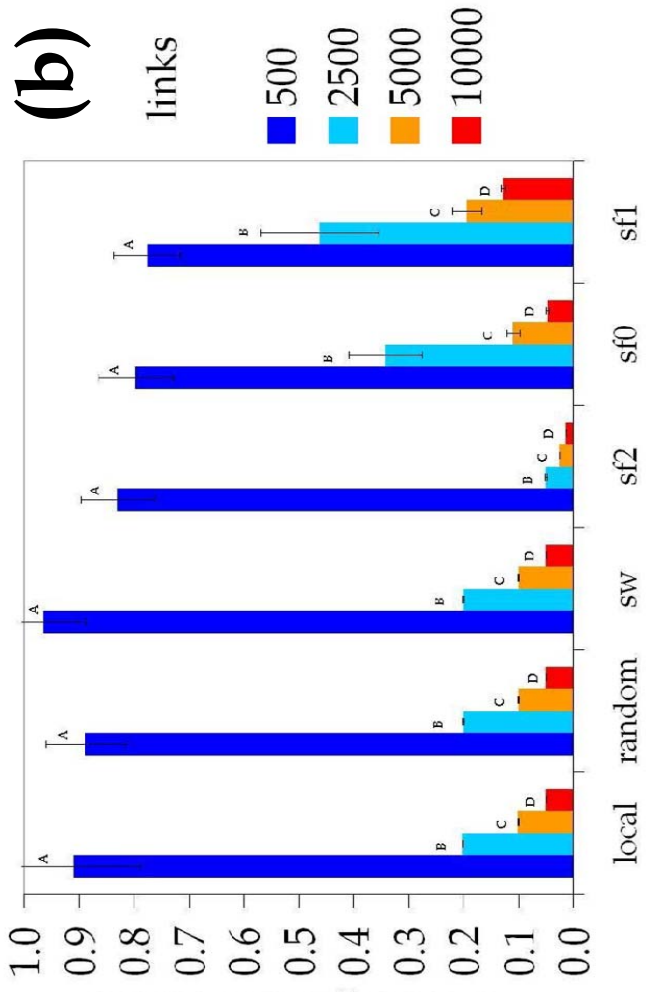
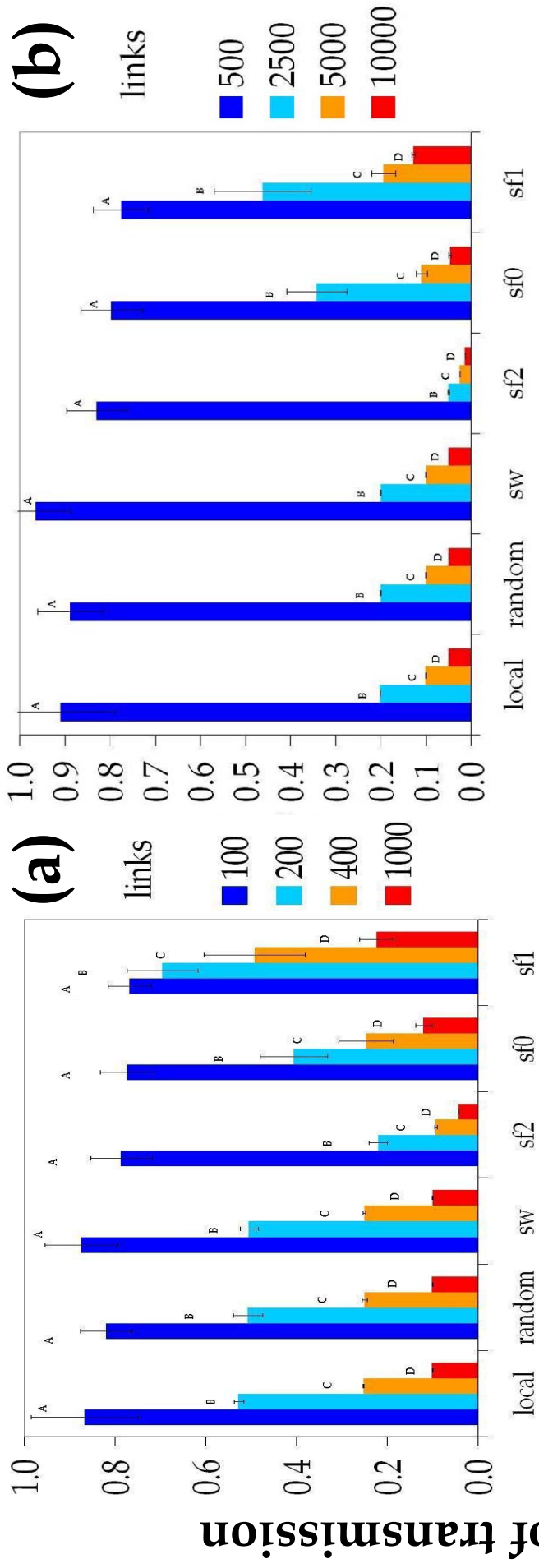
533 Figure legends

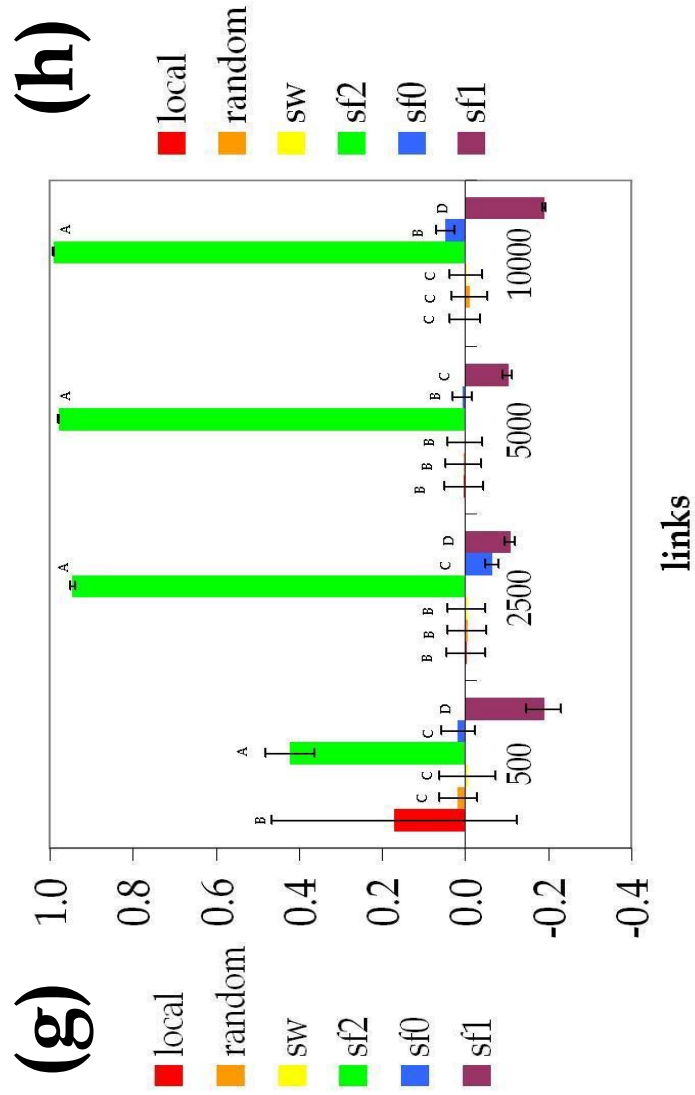
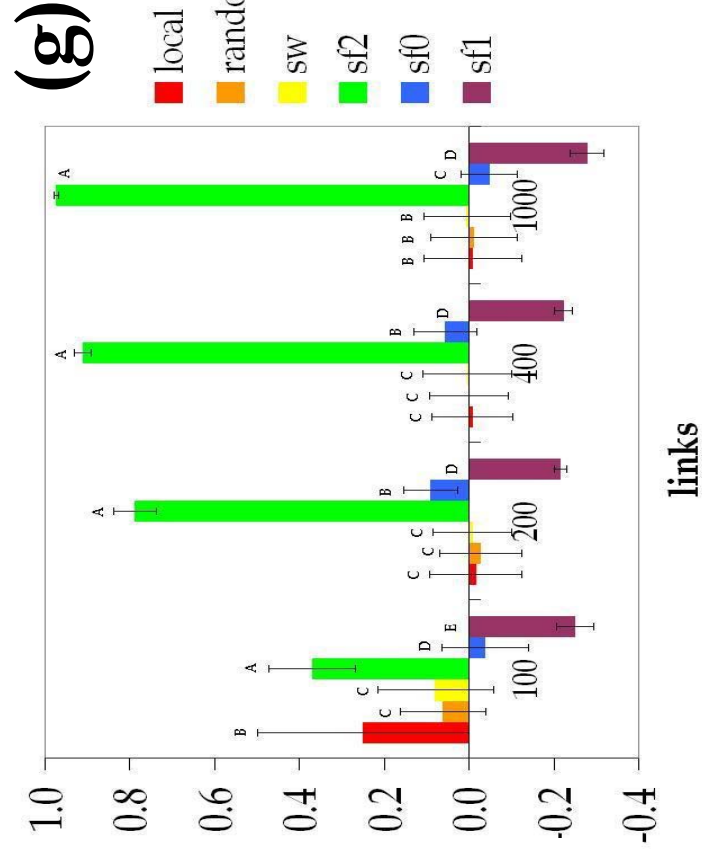
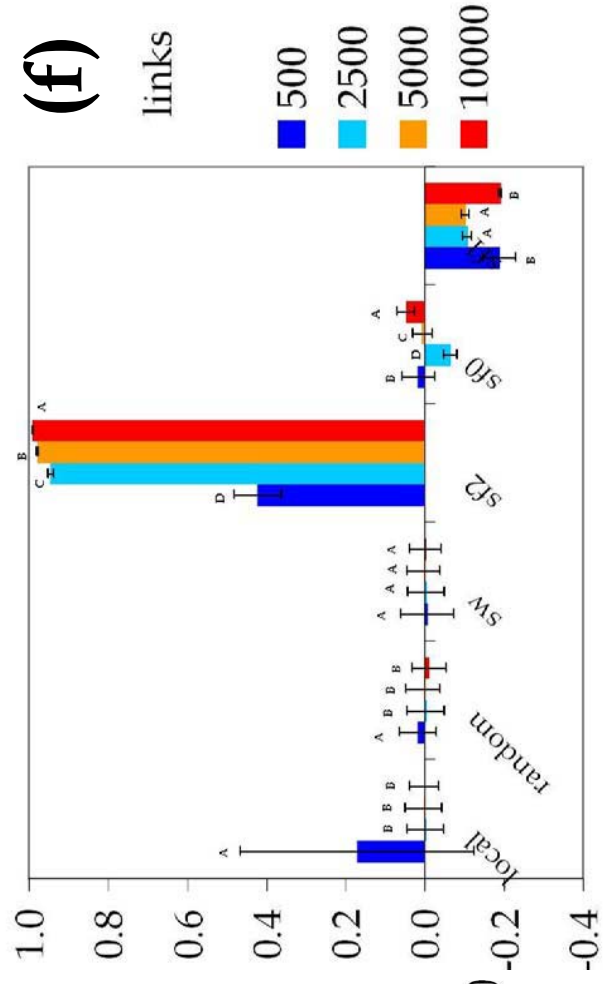
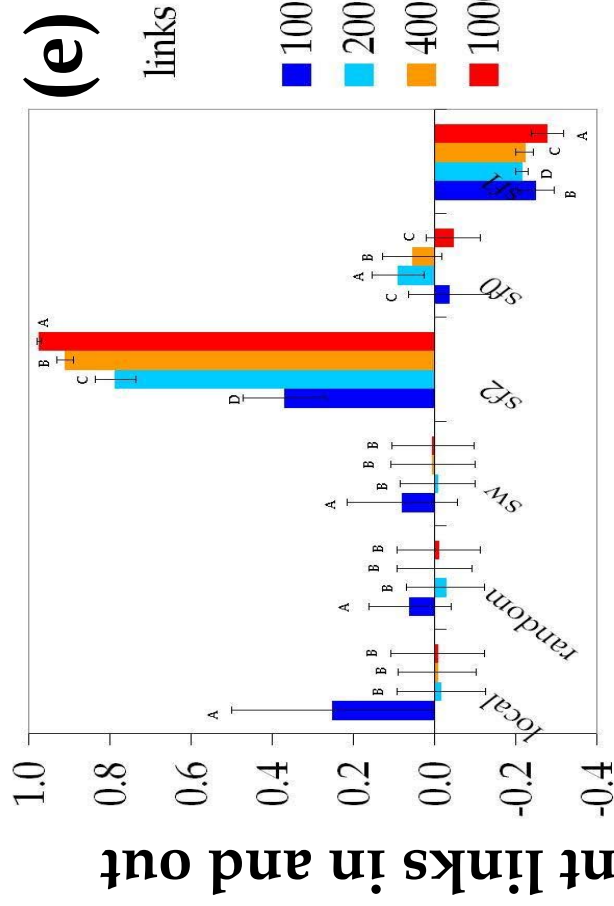
534 **Fig. 1:** Threshold probability of transmission for the different (a, b) levels of connectance
535 and (c, d) network structures, and correlation coefficient between in- and out-degree of the
536 nodes of the 100 replicates for the different (e, f) levels of connectance and (g, h) network
537 structures. 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.

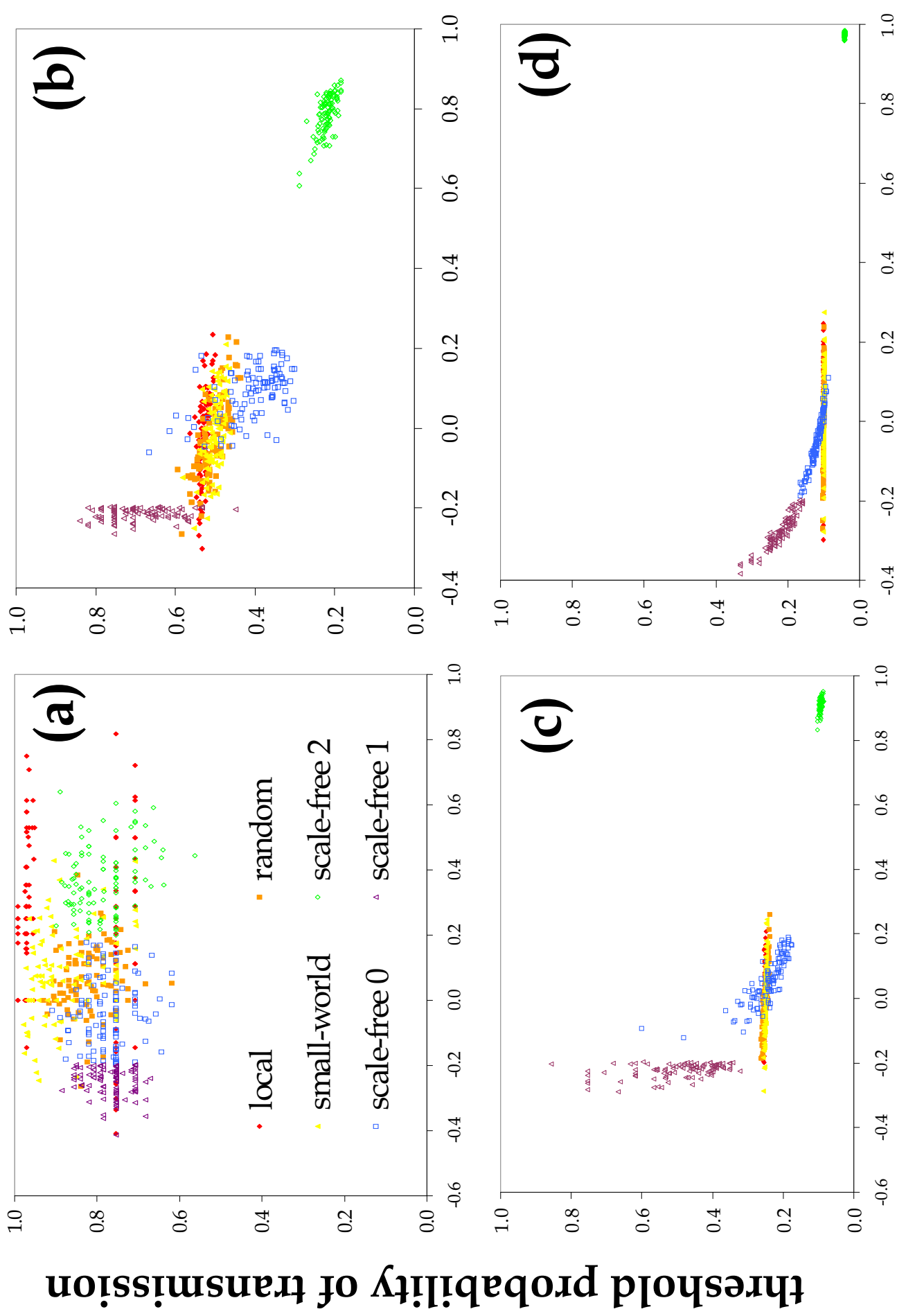
541 **Fig. 2:** Threshold probability of transmission for the different network replicates as a
542 function of the correlation coefficient between in- and out-degree of the nodes for the six
543 network structures. Network size = 100 nodes (number of links = (a) 100, (b) 200, (c) 400,
544 and (d) 1000) 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).

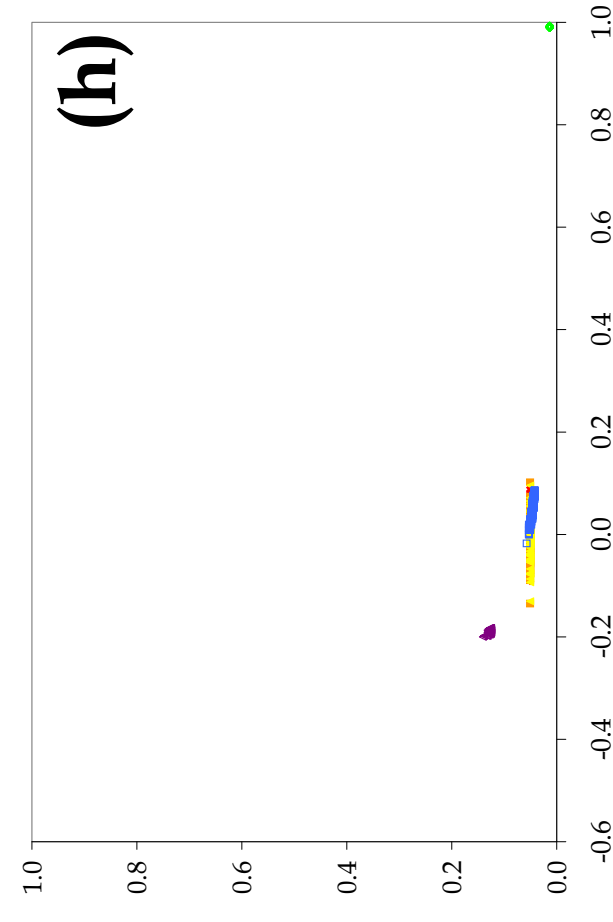
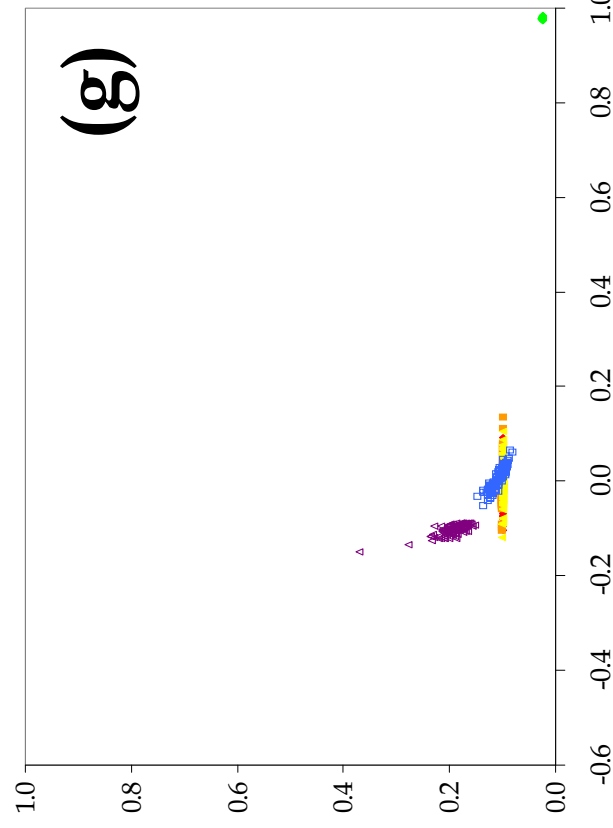
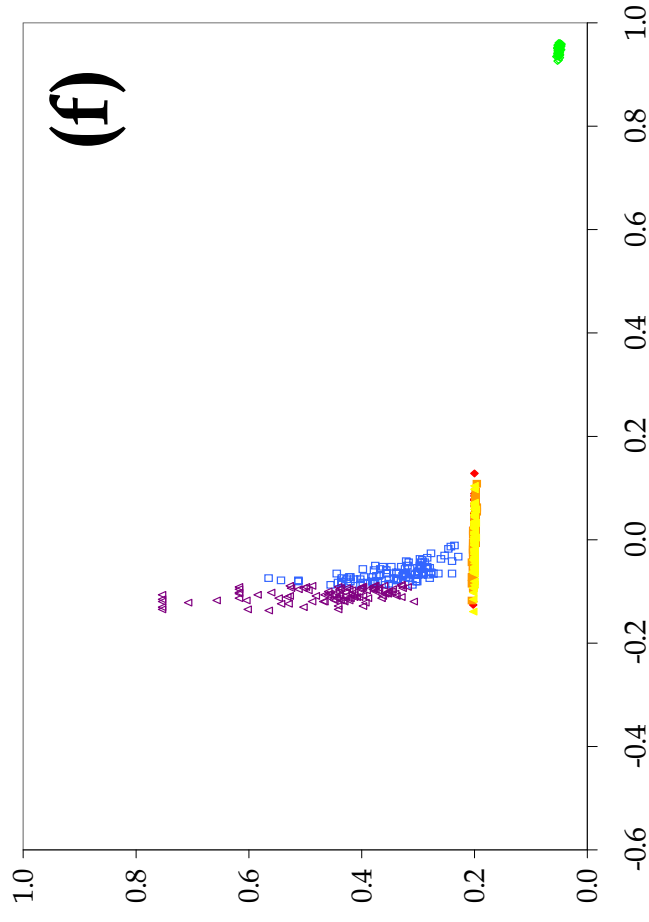
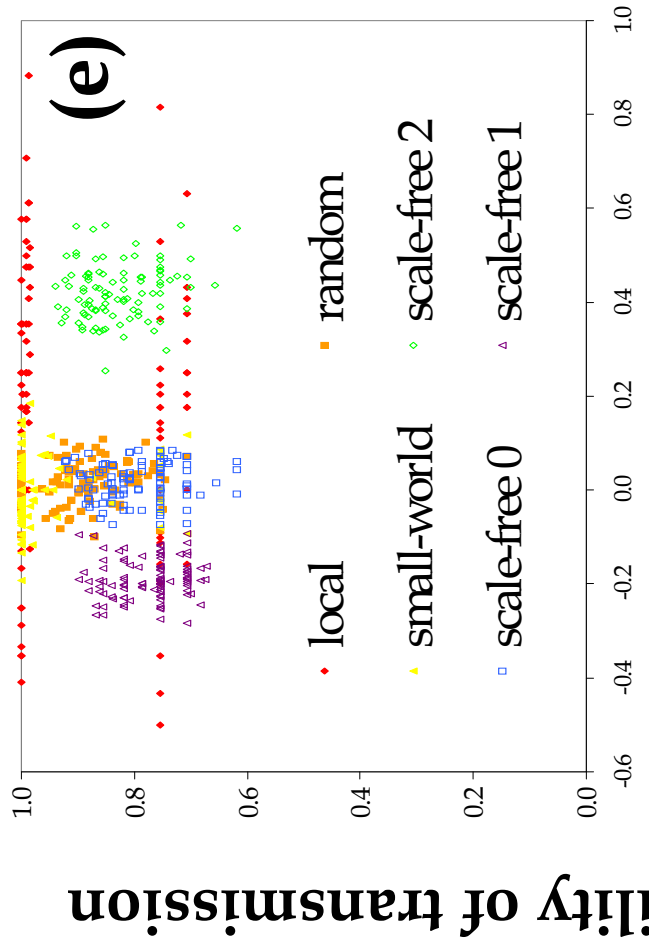
549 **Fig. 4:** Correlation coefficient between in- and out-degree for the different network
550 replicates as a function of the average clustering coefficient of the 100 nodes for the six
551 network structures. Network size = 100 nodes (number of links = (a) 100, (b) 200, (c) 400,
552 and (d) 1000)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



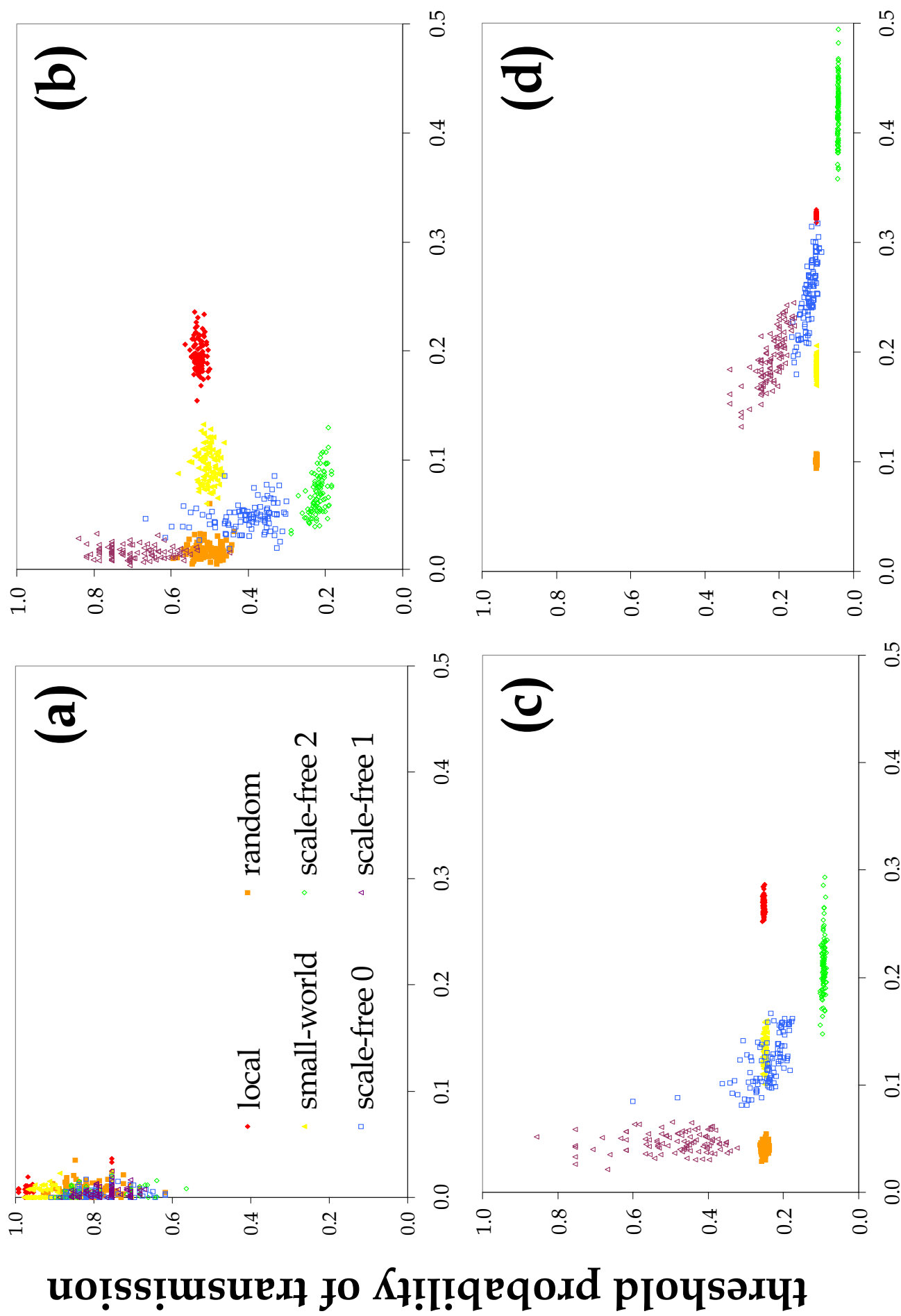




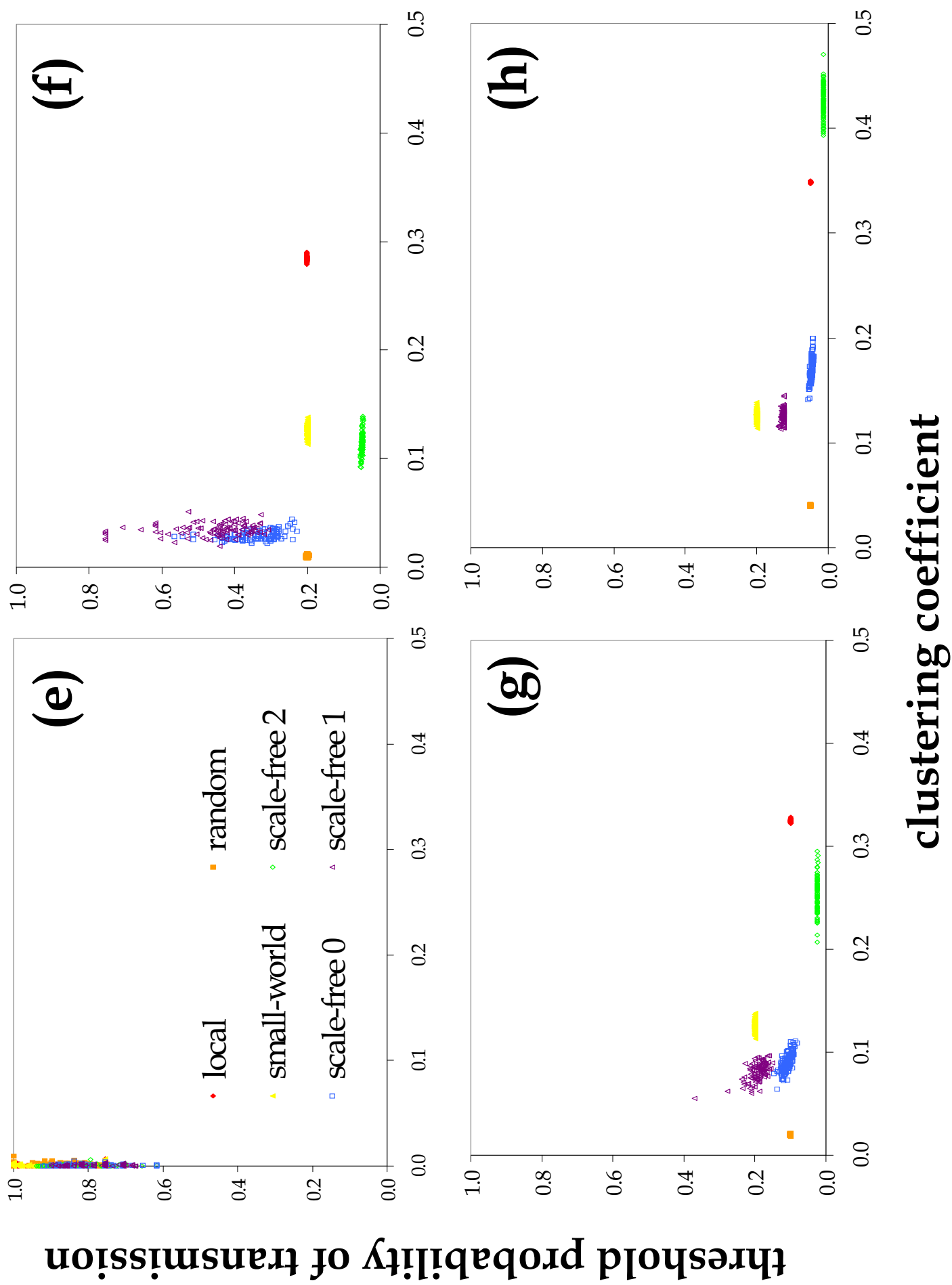
correlation coefficient between in- and out-degree

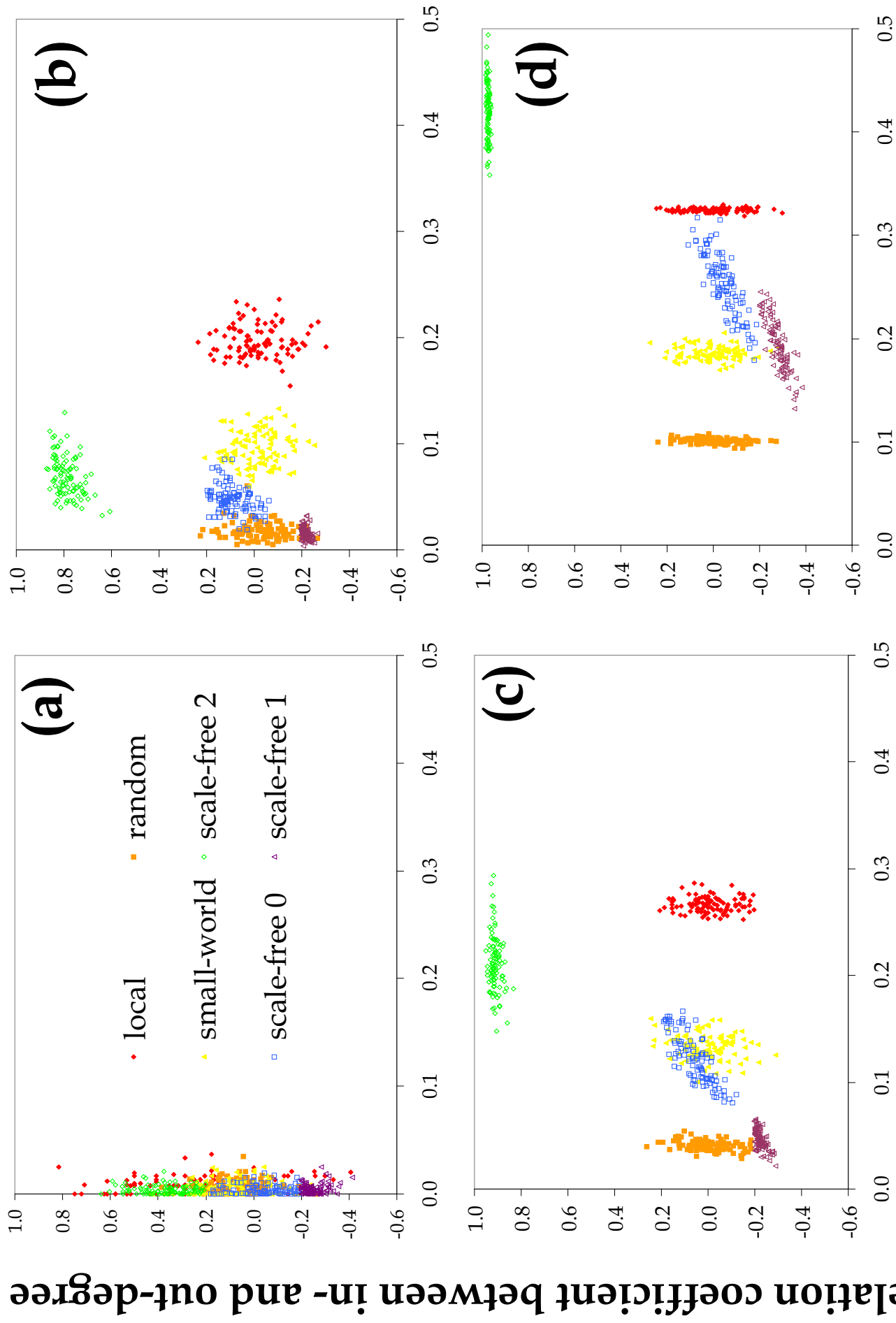


correlation coefficient between in- and out-degree



clustering coefficient





clustering coefficient

correlation coefficient between in- and out-degree

