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Effects Of Isolation On Metapopulation Dynamics In Small-world Networks

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EFFECTS OF ISOLATION ON METAPOPULATION DYNAMICS IN SMALL-WORLD NETWORKS

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ABSTRACT

Simulation models are valuable for making predictions that may be tested in natural systems and for understanding observed patterns. The simulation model developed for this thesis evaluates the effects of spatial network architecture, including organism dispersal patterns and isolation of habitats, on metapopulations. Two fields were merged throughout this project: metapopulation biology and small-world network theory. Small-world networks are characterized in their extremes as scale-free or single-scale. These models potentially simulate the networks of habitats and corridors in which metapopulations operate. Small-world network theory has been used to describe systems as diverse as rivers, the world-wide-web, and protein interactions, but has not been used as an experimental treatment for metapopulation dynamics. I tested the effects of growth rate, dispersal pattern, network architecture (scale-free and single-scale), attack type (targeted or random), and attack severity (0, 5, 10, 20, or 40% attacked populations) on metapopulation size and inter-population variation in a simulated system designed to be relevant to conservation biology and ecology. Metapopulation size and inter-population variation changed due to combinations of dispersal pattern, growth rate, and attack severity. Specifically, metapopulations were most affected by a combination of unidirectional dispersal and low growth rate in both metapopulation number and inter-population variation. However, a significant difference between scale-free and single-scale metapopulations was not found due to a low connectivity in the modeled networks as well as limitations of experimental assumptions. However, future studies that alter the model's assumptions could improve understanding of the influence of landscape structure on at-risk metapopulations.

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CHAPTER ONE: INTRODUCTION

"The key to prediction and understanding lies in the elucidation of mechanisms underlying observed patterns" (Levin 1992)

Metapopulation and landscape concepts

A metapopulation is an ensemble of populations distributed in space that interact with each other via dispersal (Levins 1969, Hanski and Gilpin 1991, Hastings & Harrison 1994). Each population within this metapopulation structure has its own population dynamics, which in combination with extinction and recolonization events determine metapopulations dynamics. Metapopulation dynamics are seen in the behavior of the interacting populations (Hanski and Gilpin 1991) and can be measured by the change in metapopulation size over time and inter-population variation (Hanski 1999).

Much of metapopulation biology has been summarized in a series of books edited or written by Hanski and colleagues (Hanski and Gilpin 1991, Hanski and Gilpin 1997, Hanski & Gaggiotti 2004, Erlich & Hanski 2004). However, the term has evolved from the traditional extinction-recolonization model (Levins 1969) to include a focus on extant, dispersal-linked populations (Hastings & Harrison 1994). Metapopulation concepts have been applied to study inbreeding and extinction patterns in a large fragmented butterfly metapopulation (Saccheri et al. 1998), endangered species management (Beissinger and Westphal 1998, Hanski & Ovaskainen 2000),

dispersal (Cain et al. 2000), and landscape fragmentation questions (Fahrig & Merriam 1994).

Landscape structure affects the connectivity of the landscape and species' use and movement within landscape patches (Tischendorf & Fahrig 2000, Dunning et al. 1992). Monkkonen & Reunanen (1999) note that connectivity is a "characteristic determined by the interaction between the movement potential of each species and landscape structure." However, only a few metapopulation studies have addressed the importance of large scale landscape structure (Vuorinen et al. 2004, May 2006) for metapopulation dynamics, including metapopulation response to fragmentation and isolation. Spatial or landscape studies have addressed patch connectivity patterns (Peltonen & Hanski 1991, Hastings & Harrison 1994, and Fahrig 2003) but there is still a need to research the effects of large scale spatial structure on metapopulation dynamics. Fragmentation is an important landscape process, and the literature is dense and diverse in defining fragmentation. Saunders et al. (1991) define fragmentation as the remnant landscape consisting of "native vegetation surrounded by a matrix of agricultural or other developed land." However, this definition fails to incorporate the connections between populations, or corridors. Fahrig (2003) acknowledged this distinction and stated that fragmentation is typically "defined as a landscape-scale process involving both habitat loss and the breaking apart of habitat." For clarity, I here distinguish fragmentation as consisting of (1) habitat loss and (2) isolation. In this thesis I will focus on isolation effects; habitat loss is expected to affect populations more strongly (Fahrig 1997), but isolation of extant populations most directly addresses the effects of landscape structure, whereas habitat loss also involves population extirpation. It is difficult to tease apart the relative importance of habitat loss and isolation in natural systems, but this distinction is possible in simulation. In my study, habitats

remained intact, and only habitat isolation was experimentally introduced by removing connections (corridors) between discrete habitats.

Beyond isolation, landscape structure can also affect metapopulation dynamics by determining the direction of dispersal and the degree of connectivity among populations within a metapopulation. Unidirectional dispersal only allows individuals to move one-way, and thus establishes a source/sink interaction between two populations (Pulliam 1988). Bidirectional dispersal forces a mirrored two-way dispersal pattern with both populations sending and receiving individuals. A compromise between these two extremes (and likely a more realistic scenario) may be represented as random direction, where dispersal has both one-way and two-way dispersal directions within metapopulation architecture. Network concepts provide a usable framework to study the effects of spatially heterogeneous landscapes, including dispersal direction and isolation, on metapopulation dynamics.

Network concepts

The degree of connectivity among locations has been well-characterized in the rapidly-developing field of small-network theory (Watts & Strogatz 1998, Barabasi & Albert 1999, Amaral et al. 2000, Dorogovtsev & Mendes 2002). Small-world networks have been the focus of recent studies because of their potential to model complex systems having scale-free, broad-scale, and single-scale network architectures (Amaral 2000, Amaral et. al 2000). Scale-free networks are identified by a vertex connectivity distribution that grows by preferential attachment based on a probability of connecting that depends on the number of connections

($P(k)$) producing a frequency distribution of nodes with different number of connections that decreases by a power law as the number of connections (k) increases (Figure 1). Single-scale networks have random connectivity and show a normal frequency distribution of nodes with different numbers of connections and therefore have fewer well-connected vertices than expected in a scale-free power-law distribution (see Table 1 for summary). Broad-scale networks are an intermediate between scale-free and single-scale networks and will not be addressed in this experiment. Examples of small-world networks can be seen in the world-wide-web (Huberman & Adamic 1999), airplane and highway maps (Barabasi and Bonabeau 2003), social interactions (e.g., 6 degrees of separation; Wasserman 1994), and river drainage basins (Anderson and Jenkins, submitted).

In addition to network architecture, each network can be defined by how it responds to attack, which I define for this study as a permanent disturbance (consistent with Albert et al. 2000) that removes corridors, thus leaving populations isolated. Network attack has been a popular subject (e.g, Albert et al. 2000, Barabasi & Bonabeau 2003, Crucitti et al. 2003, Gallos et al. 2005, and Deng et al. 2007), but studies have not addressed how species living within complex systems respond to the attack. In my study, the attack occurs on the corridors that link two populations to each other, leaving populations isolated from one another but intact. This scenario is consistent with the concept of population isolation and distinct from that of habitat loss, as discussed earlier. Albert et al. (2000) described disturbances (termed “attacks” in network terminology) as either targeted or random. Targeted attacks limit dispersal to and from the most highly connected populations (hubs), whereas random attacks affect randomly chosen populations (hubs or non-hub nodes). Typically, single-scale networks show a similar metapopulation change given

random or targeted attacks (Barabasi & Bonabeau 2003), because there is a normal distribution of connections. On the other hand, scale-free networks respond differently to random attacks than to targeted hub attacks (Barabasi & Bonabeau 2003) due to the greater heterogeneity in the number of links per node. Isolation of weakly-linked populations in a scale-free network would hardly alter the system, but the removal of hubs would greatly alter the network's connectivity, thus greatly affecting the metapopulation structure.

Merging Metapopulations and Networks

Few studies have mapped large scale connectivity in metapopulations (Hanski & Thomas 1994, Vuorinen et al. 2004), but none have tested the effects of network pattern (landscape and dispersal pattern) on metapopulation dynamics. If network theory is to be applied to spatial ecology, then clear expectations are needed for the effects of network structure and changes in that structure on metapopulations. I experimentally evaluated the effects of network pattern (dispersal pattern, metapopulation architecture, and permanent disturbance events) and population growth rate on metapopulations dynamics. I used numerical simulations (in Matlab 7.0) to assess the effects of small-world network architectures (scale-free, single-scale, or no connections), dispersal regimes (bidirectional, random, or unidirectional), and population growth rates ($r = 0.005$, 0.05, or 0.5) on metapopulation dynamics. I then also assessed the effects of different disturbance patterns (targeted or random) and attack severity (% attack= 0, 5, 10, 20, 40) on metapopulation dynamics. I used the results to evaluate the importance of network structure in metapopulation dynamics and more broadly to examine the applicability of network concepts to metapopulation biology.

Practical application of network theory in the natural world could assist managers in making important conservation decisions in human-dominated environments. An example includes using small-world networks to classify naturally occurring metapopulation architecture to identify and protect important nodes (habitat patches). Depending on network design, important habitats and corridors could be prioritized as management targets. Network designs also help to determine the pattern of patch connectivity, via corridors, in nature and can therefore be used to help plan for fragmentation.

Hypotheses

Undisturbed Metapopulations: Network pattern (scale-free, single-scale, no connections) will interact with dispersal (Unidirectional, Bidirectional, Random) and growth rate (r = 0.005, 0.05, 0.5) to cause a difference in undisturbed metapopulation size and coefficient of variation among populations.

Disturbed Metapopulations: Network pattern (scale-free, single-scale, no connections) will interact with dispersal (Unidirectional, Bidirectional, Random), growth rate (r=.005, .05, .5), attack type (Targeted or Random), and attack percent (0, 5, 10, 20, 40) to cause a difference in metapopulation size and coefficient of variation among populations.

Table 1. Major differences between single-scale and scale-free networks.

SINGLE-SCALE	SCALE-FREE
Random connectivity distribution	Preferential attachment to highly connected populations creating hubs
The distribution of connections per population is described well by the average (a normal distribution around the mean)	The range of connections is large and the distribution is skewed to fit a power curve. Many populations have a small number of links and a few populations have a large number of links
Probability of connecting to other populations does not change as the number of connections increases.	Probability of connecting to other populations increases as the number of connections increases. For example, if a population has >2 connections, the probability of it connecting to another population is higher than for a population having only one.
Random attacks disrupt the network	Targeted attacks disrupt the network

CHAPTER TWO: METHODS

All modeling was conducted using Matlab 7.0. The following is a narrative description of the model, its assumptions and experiments conducted with the model: see appendix A for annotated model code.

Metapopulation architecture

Networks were built of 500 nodes and were either scale-free or single-scale in their degree distributions (Figure 1). Networks were assembled using these rules (mostly based on network literature; Amaral et al. 2000 & Barabasi and Albert 1999):

(1) In scale-free networks, nodes were defined first and then connected to each other with a probability of connection (*p)*, so as randomly chosen nodes become more connected, *p* increased. This algorithm created a scale-free network consisting of a few highly connected populations (hubs), some moderately-connected populations, and many isolated populations, consistent with a scale-free frequency distribution (consistent with Barabasi & Bonabeau 2003).

(2) To build the single-scale networks, *p* remained the same for each node, creating a random chance of any one node connecting to another node. The value of *p* was adjusted so that both network types had similar total number of connections (i.e, if scale-free had 100 connections, *p* was changed until single-scale also had around 100 connections). Single-scale metapopulations having 500 populations required a *p* value of .995 to have a similar network connection size (Figure 1). Hubs may develop by chance in a single-scale network, but the majority of nodes will have the average number of connections for the entire network, whereas in the scale-free network the nodes have very different connection numbers. Both single-scale and scale-free

network architectures were checked with degree distribution plots to confirm the correct architecture (Figure 2).

Population Growth

Population growth was accomplished with a rounded logistic/discrete growth model (round($a_{t+1} = a_t e^r(1-(a_t/k))$), where *k* was set at 100 and intrinsic growth rate *r* was experimentally varied. Rounding was employed because simulation was intended to represent real metapopulations dispersing whole organisms. The rounded logistic model obtained metapopulation sizes that were lower than those of a standard logistic model, but this difference was consistent among experimental treatments and did not affect the relative rank of the treatment effects (Figure 3). Data from all treatment combinations that showed effects (unidirectional and random dispersal x slow and medium growth) were tested to confirm this pattern.

Attack Program

The random attack program was written to remove connections randomly and the targeted attack program was written to preferentially remove connections of the most highly-connected nodes, or hubs. The number of connections in each population (network node) was calculated for both programs. Populations were then sorted by the number of connections for targeted attacks, so the hubs in scale-free network and highly connected populations in single-scale networks could be identified by the program. Random attacks were accomplished by random selection of populations that had connections. The number of attacked connections depended on the percent of attack, which was defined as either 0, 5, 10, 20, or 40 percent of nodes with connections.

Thus the extent of isolation was based on the percent of populations selected. The algorithm simply removed connections for those nodes but did not extirpate selected populations; thus isolated populations were permitted to maintain and grow according to the programmed growth rate.

Preliminary Experiments

Preliminary tests were conducted to make sure the model performed correctly and to identify the importance of various factors on metapopulation dynamics. Several conclusions were reached from these results:

(1) Initial population size greatly affected variation between populations. Initial population sizes needed to be > 10 individuals to control variation between populations due to demographic effects that masked the effect of other variables.

(2) Attacks needed to be done while populations were still growing (population size < carrying capacity) so that the effect of attack could be seen since populations reaching k were stabilized and were not affected by attack. This effect was related to simple isolation of populations (i.e., isolated populations continued to contribute to total metapopulation size).

(3) Population growth rates were important and needed to be a variable in experiments, because different growth rates led to different metapopulation dynamics in each of the network architectures.

(4) Bidirectional dispersal was overly simplistic and not considered representative of real-world scenarios, so other dispersal types (unidirectional and random) needed to be incorporated into the final model.

(5) The total number of connections in scale-free and single-scale metapopulations needed to be

similar so comparisons were for equally-dense networks.

(6) A sufficient number of populations was needed in the metapopulation to see an effect of growth rate, dispersal, and attack. This is related to the need for scale-free networks to be sufficiently large for rare hubs to occur.

Based on the preliminary experiments, conditions (assumptions) for the primary experiments were:

(1) Initial populations consisted of fifty individuals per population; thus occupied sites were not established sequentially. This was enacted to focus the study on the dynamics of an existing metapopulation, rather than a global colonization event.

(2) Homogeneous population parameters of fixed growth rates, carrying capacity, and initial number of individuals within a metapopulation. This assumption avoided the added complication of habitat heterogeneity within a metapopulation, given the complicated experimental design, and essentially assumes that population biology is driven by species life history rather than local conditions.

(3) All populations within a metapopulation had identical initial population size, growth rates and carrying capacities: the only simulation parameters able to generate heterogeneity in the system were dispersal direction and connectivity. Preliminary results indicated that a fast growth rate ($r = 0.5$) overwhelmed the effect of network and dispersal patterns, so slower growth rates ($r = 0.05$ and 0.005) were added to the primary experiment.

(4) Dispersal events occurred in even-numbered time steps. This effectively introduced a time lag after a dispersal event to develop a population. Preliminary tests revealed that a time lag was needed to prevent populations from homogenizing due to excess dispersal events.

(5) Only one individual was moved per connection for each dispersal event to concentrate the study on direction of dispersal and connectivity patterns rather than the effects varying numbers of dispersers.

(6) Attacks removed corridors, thus fragmenting and isolating metapopulations. This focused study outcomes on the effects of network fragmentation on metapopulation biology, separately from any added effects due to habitat loss and population extirpation. Populations that were experimentally isolated could still contribute to metapopulation size if the population remained viable.

(7) Standard parameters were used based on preliminary results: 500 populations, maximum time=100, initial population sizes=50, five replicates per treatment, attacks occurred at time=5 (see preliminary results for explanation). Metapopulations composed of 500 populations were considered more relevant to ecological structures than internet-scale networks typically analyzed in network studies (e.g., internet, which has 6,209 nodes and 12,200 connections; Barabasi et al. 2000, Cohen et al. 2000, Albert et al. 2000).

(8) A low number of connections in each metapopulation existed due to the restrictions set by *p* in a scale-free network. A single scale network's *p* value was adjusted to approximate matching a scale-free network for the total number of connections (Figure 2). This setting was important relative to other analyses of networks with more connections (e.g., internet; Barabasi et al. 2000, Cohen et al. 2000, Albert et al. 2000). The relatively low number of connections meant that not all populations in a metapopulation were connected, even before attacks (Table 2), and thus simulated networks represented metapopulations that were already partially fragmented. Scale-free metapopulations had higher numbers of isolated populations because hubs held most of the connections (Table 2).

(9) In some conditions, a source node may have more connections to receiver nodes than individuals to disperse. In this scenario, the program randomly selected connections until the number of connections equaled the number of available dispersers, and then sent individuals on the selected connections to receiver nodes. Therefore, some connected populations may not have received dispersers despite an existing connection in the network. This condition for a receiver node did not affect that node's ability to send dispersers and was enacted as needed at each dispersal event.

Table 2. Number of connections in scale-free and single-scale networks (500 populations each) with three different dispersal types. Values represent the ranges of total connections per network and the number of populations isolated pre-attack (N=15).

Network	Bidirectional	Random	Unidirectional
Scale-free			
Connected populations	1092 – 914	569 – 479	546 – 471
Isolated populations	203 - 174	286 - 257	195 - 171
Single-scale			
Connected populations	1372 - 1196	677 - 580	661 - 591
Isolated populations	67 - 31	167 - 123	193 - 170

Experimental Design

The main experiment was conducted as a five-way factorial design to test the effects of network architecture, population growth rate, dispersal mode, attack type, and attack severity on metapopulation size and variation among populations within a metapopulation. Interactions among treatments were analyzed (Table 3). Each treatment combination was replicated 5 times.

Data were collected from the program's output and transferred to SPSS (version 11.5) for statistical analysis and graphing. Statistical analyses included analysis of variance (ANOVA), but this approach was considered secondary for a modeling study with ultimately-controllable treatment settings and replication. Primary analyses were by examination of plots through time (means $\pm$ 95% confidence intervals) and box plots were used to evaluate the effect of each treatment.

Table 3. Experimental Design. A full factorial of all listed treatments was conducted. Attacks consisted of habitat isolation but not habitat loss.

Treatment	Levels
Network	no connections, single-scale, scale-free
Growth rate (r)	0.005, 0.05, 0.5
Dispersal direction	unidirectional, random, bidirectional
Attack type	targeted, random
Attack severity	0, 5, 10, 20, and 40 % of nodes isolated

CHAPTER THREE: RESULTS

Pre-Attack

An expected difference between population growth rates occurred (Figures 4 and 5) and affected all treatments and interactions. Importantly, fast growth (r = 0.5) exhibited essentially no variation within metapopulations, (expressed as coefficient of variation %, or CV) or among treatments because populations rapidly reached k and maintained large population sizes thereafter (Figure 5). This occurred because populations were not extirpated during attacks on network links. Therefore, fast population growth will be ignored hereafter while low (r = 0.005) and medium (r = 0.05) growth rates will be discussed as a modifier of other treatments.

Two dispersal modes (unidirectional and random) affected metapopulation size and CV. Bidirectional dispersal exhibited little effect because reciprocal dispersal events homogenized populations. Therefore, narrative results below emphasize unidirectional and random dispersal modes. Networks assembled with random and unidirectional dispersal attained very similar connection densities, and networks constructed with bidirectional dispersal direction had approximately twice the connections (Table 2).

In contrast with expectations based on network-attack literature, attack type (targeted or random) did not change the metapopulation size or CV in either scale-free or single-scale networks (Figure 6). The three remaining main treatments (network architecture, dispersal mode, and attack severity) are addressed in their entirety below, with growth rate and dispersal mode considered as modifiers of those treatments.

Network Architecture

Network architecture significantly affected both metapopulation size (Figure 7; $p = 0.03$) and CV (Figure 8; $p = 0.03$). Un-connected "null" metapopulations reached a collective carrying capacity by time = 35 in a medium growth rate (Figure 7 C, D). Relative to this "null" system, metapopulations in single-scale and scale-free networks grew differently, depending on dispersal mode and growth rate. Random dispersal directions slightly modified metapopulation size in scale-free and single-scale networks relative to non-connected systems (Figures 7 B, D) but single-scale and scale-free networks did not differ. The effect of network architecture was strong in unidirectional dispersal (Figures 7 A, C); at low growth rates, unconnected populations did not grow, but both scale-free and single-scale metapopulations grew (Figure 7A). However, the benefit of being connected due to dispersal was greatly diminished at medium growth rate ($r = 0.05$; Figure 7C); scale-free metapopulations grew only slightly more than unconnected networks, and single-scale metapopulations grew less than those in unconnected networks. These effects demonstrated that at a fine level (a) scale-free and single-scale metapopulation sizes are increased by unidirectional dispersal for species with low growth rates, but that this benefit may be hidden at faster growth rates and (b) at a broad level network architecture and dispersal direction matter for slow- and medium-growing metapopulations.

Network connections introduced variation in all treatment combinations, but the magnitude of that variation depended on the network, dispersal mode, and growth rate (Figures 7 & 8). No inter-population variation occurred in unconnected metapopulations; all populations were

identical. Metapopulations with unidirectional dispersal were usually more patchy than those with random dispersal and patchiness (CV) was greater at low growth rates (Figure 8). Metapopulations in single-scale networks were more patchy given unidirectional dispersal, but this difference was mitigated with random dispersal (Figure 9). Also, this variation was usually consistent among replicate experiments, as evidenced by tight confidence intervals (Figures 7 & 8), with the sole exception being single-scale networks with random dispersal and growing at r = 0.005 (Figures 6D, 8B, & 10B). This exception was related to the relatively strong influence of randomized dispersal direction among populations and randomized network architecture at low growth rates. All other combinations of treatments exhibited no variation between replicate runs of the experiment or between populations.

Dispersal Mode

The direction of dispersal within a metapopulation significantly affected metapopulation size (Figure 9; $p = 0.02$). Unidirectional dispersal had larger metapopulation sizes (Figures 9 A, B, and C), except in single-scale networks with medium growth rates (Figure 9D). Metapopulations with unidirectional dispersal also had greater inter-population variation, except in scale-free networks at slow growth rate, where unidirectional and random directions exchanged places over time as most-varied (Figure 10A). Metapopulation patchiness reached especially high levels in single-scale networks with unidirectional dispersal and slow growth (Figures 5D, 8B, & 10B). Bidirectional dispersal always had the lowest CV, due to the homogenizing nature of two-way dispersal. These results indicate that (a) metapopulation size and patchiness depend on the directionality of dispersal among populations, and that this effect

in turn depends on population growth rates, (b) more bidirectional dispersal among populations can reduce metapopulation patchiness, and (c) the effects of dispersal direction in a metapopulation are roughly consistent in both single-scale and scale-free networks.

Attack Severity

The sequential increase in attack severity (0, 5, 10, 20, and 40% of habitats isolated) significantly affected metapopulation size and variation ($p = 0.04$; (Figures 11 & 12). The magnitude of isolation's affect was determined by dispersal and growth rate, and the most extreme response was in metapopulations with unidirectional dispersal and slow growth rate (Figures 11A, B and 12 A, B). In comparison, attack severity only slightly decreased metapopulation size in random-dispersing metapopulations (Figures 11E & F). The highest degree of attack (40%) had the lowest CV (Figure 12); as attack severity increased the CV decreased toward that of a completely disconnected metapopulation. Slow growth rate caused high variation between populations in both networks (Figure 12A, B, E, F), but tight confidence intervals indicate that variation between replicate metapopulations was low. All other combinations exhibited no variance between replicates, no inter-population CV, and no change in metapopulation size due to attack severity.

Isolation was also assessed by its effects on the number of connections lost in the network, similar to customary network analyses. The number of connections lost in networks increased as the percent attack increased (Table 4). Networks with bidirectional dispersal lost the most connections, followed by those with random, then unidirectional dispersal (Table 4), related to the number of available connections. The expected sensitivity of scale-free networks to targeted

attacks was observable only at 5% and 10% severity; at greater attack rates this treatment combination was similar to others (Table 4). Dispersal direction, network architecture, and form of attack all affected the effect of attack severity on connections lost. Networks with bidirectional dispersal lost more connections than those with random or unidirectional dispersal, as expected. Scale-free networks with random directionality seemed to be more sensitive to targeted attack than other random or unidirectional networks, but this difference faded as attack severity increased. Otherwise, systems were roughly comparable in the network effects of attacks.

In summary, metapopulation size and CV were affected by dispersal direction (unidirectional & random) and attack severity at low to moderate growth rates. Contrary to expectations from network theory, single-scale and scale-free networks did not differ in response to random or targeted attacks, but this study focused on metapopulation parameters rather than network structure as response variables. Metapopulations with bidirectional dispersal and fast growth were unaffected by attacks. Minor differences were seen between single-scale and scale-free metapopulations, but the similarity of the two could be attributed to low connectivity within simulated networks thus making the two network types more similar.

Table 4. Effects of habitat isolation on network connections. Values are number of connections lost, sorted in decreasing order for the most severe attack (40%).

			Attack Severity				
Direction	Network	Attack	0 %	5 %	10 %	20 %	40 %
Bi	Scale-free	Random	0	157	277	522	847
Bi	Single-scale	Random	0	141	269	497	844
Bi	Scale-free	Targeted	0	441	516	612	760
Bi	Single-scale	Targeted	0	161	250	438	726
Random	Scale-free	Random	0	91	170	310	483
Random	Single-scale	Random	0	80	152	284	469
Random	Scale-free	Targeted	0	184	223	289	371
Random	Single-scale	Targeted	0	78	124	219	361
Uni	Single-scale	Random	0	74	137	469	469
Uni	Scale-free	Random	0	71	125	257	397
Uni	Single-scale	Targeted	0	69	119	216	355
Uni	Scale-free	Targeted	0	78	132	183	284

CHAPTER FOUR: DISCUSSION

Though metapopulation theory has been active for over a decade (Hanski and Gilpin 1991), it has only recently begun to address the effects of complex spatial pattern on metapopulations (Fagan 2002 , Vandermeer & Carvajal 2001, Thomas & Kunin 1999). The recent popularity of network theory (Albert 2005, Newman 2003, Dorogovtsev and Mendes 2002) suggests it offers a means to advance the study of metapopulation biology, especially because it may represent spatial systems that are realistic and behave predictably (Jeong et al. 2003, Barabasi & Bonabeau 2003, Albert et al. 2002 , Vuorinen et al. 2004). This study combined metapopulation biology with network concepts to address three features of spatial structure: patterns of landscape connectivity (scale-free and single-scale), dispersal pattern (bidirectional, unidirectional, and random) within networks, and isolation (attack type and percent) of habitats in the networks.

Habitat fragmentation changes spatial configuration of a metapopulation and has attracted much attention in conservation biology (Noss 1990, Saunders et al. 1991, Andren 1994, Fahrig 2002, Fahrig 2003). Fragmentation consists of habitat loss and the loss of dispersal among habitats (Fahrig 2003). It is difficult to separate the effects of these two components in natural systems, but simulation offers an opportunity to evaluate the effects of corridor loss separately from habitat loss on metapopulations. As stated earlier, this simulation model tested for the effects of spatial network architecture, including organism dispersal patterns and isolation of habitats, on metapopulations. Three mechanisms (network architecture, dispersal direction, and population growth rate) affected metapopulation size and heterogeneity given specific combinations of treatments, including isolation. My findings indicate that metapopulations having slow growth

rates and unidirectional or random dispersal are most sensitive to network architecture. Metapopulations having bidirectional dispersal or fast growth rates were not affected by network types or attack severity, because population growth compensated for emigration from populations.

Network connectivity decreased as attack percent increased, but the severity of the metapopulation response primarily depended on dispersal direction. Even in metapopulations having unidirectional or random dispersal, only a modest effect of strong attacks (40% fragmentation) was observed on metapopulation size, and the type of attack (targeted or random) did not matter. Metapopulations in scale-free networks were expected to reduce in size and increase in CV given targeted attacks because of the large reduction in the network connectivity. Likewise, metapopulations in single-scale networks were expected to be most affected by random attacks. The lack of such effects in my study was apparently because the number of populations and dispersal connections were low relative to studies of attack in the world-wide web (Albert et al. 2000; 6,209 nodes and 12,200 connections). Compared to larger and more densely-connected networks, the small networks used in this study increased the probability of a random attack hitting hub connections. This factor made my study relevant to ecological studies but may have contributed to the similarity of random and targeted attacks. In addition, other studies of network attack (Barabasi & Bonabeau 2003, Albert et al. 2000) usually remove the node *and* connections during attacks (Deng et al. 2007), where this study only removed the connections during attacks. This approach focused the study on isolation as a component of habitat fragmentation rather than habitat loss, but contributed to a muted response of metapopulation size to attacks because isolated populations remained viable. Further

experiments including (a) ephemeral habitat loss to induce extinction/recolonization in populations and (b) permanent habitat loss will permit comparisons of the relative importance of isolation and habitat loss for metapopulations undergoing fragmentation, but it is expected that habitat loss impacts metapopulations more than isolation alone (Fahrig 1997).

Central populations are thought to support the metapopulation more than peripheral populations (Hastings & Harrison 1994 and Peltonen & Hanski 1991). This is consistent with an expected resistance of scale-free networks to random habitat fragmentation (Barabasi & Bonabeau 2003, Albert et al. 2000), but was not supported in my study because scale-free and single-scale networks responded identically to targeted and random attacks and differed only slightly in response to attack severity. Because this outcome was due to small networks, my results suggest that threatened or endangered metapopulations (e.g., those with < 500 habitats) are already too small to exhibit strong effects of central hub populations on the metapopulation. Clearly, this possibility implies strong limits to the value of network theory as a predictive tool for conservation biology and should be tested further. Future experiments to selectively attack peripheral non-hub population connections or to exclude hubs from random attacks may help determine the effect of protecting the hub versus the peripheral populations.

Increasing isolation reduced metapopulation size and variation when dispersal was unidirectional or random and populations grew at slow and medium rates. This effect was not due to diminished carrying capacity within habitats or population growth rates as these factors were constants. Instead, reduced metapopulation size and patchiness was due to isolation (i.e., limited metapopulation dispersal) only. Therefore, species with relatively slow population growth rates

and limited dispersal would appear to be at greatest risk of habitat fragmentation, due to isolation effects alone. Population growth *and* dispersal propensity are relevant for conservation biology and together may be a more sensitive indicator of risks for species than population growth alone.

Metapopulations with unidirectional dispersal were more sensitive to attack than those with random dispersal when populations grew slowly. Isolation from a source population is important for a receiver population's growth, and if a metapopulation is solely composed of unidirectional dispersal, many populations are receivers. This result suggests that natural populations that disperse in one direction between any two habitats and grow slowly (e.g., corals, molluscs, barnacles, etc.) should be prioritized by conservation managers, as they are most sensitive to fragmentation. Conversely, this result also suggests that metapopulations with at least some bidirectional dispersal and moderate growth ($r = 0.05$) are robust to habitat isolation, provided that habitats are not lost. However, further research needs to be done with different sets of assumptions (e.g., density-independence) to address the importance of dispersal directionality, growth rate, and network configuration for metapopulation stability.

In summary, metapopulations having unidirectional or random dispersal and slow or medium growth were most affected by isolation. This simulation study therefore found that growth rate and dispersal play a key role in determining the metapopulation size and inter-population variation. A significant difference between metapopulations existing in single-scale and scale-free networks was not found, but this could be attributed to the model scale and assumptions. Given different metapopulation assumptions, especially assumptions that match network literature such as large network size and high connectivity, an effect of network architecture is

expected.

Future suggested research directions with this simulation model include:

(1) adding density-independent mortality to allow colonization/extinction cycles

(2) adjusting assumptions to fit selected species life history traits (e.g., growth rate)

(3) addressing the differences between highly connected metapopulations

(4) adding in dispersal rates (number of dispersers per dispersal event)

(5) applying variation to the growth rate so each population has a unique growth

(6) making hubs a source or sink to determine the effects of "central" populations

(7) sequential metapopulation growth versus established growth that was modeled in this experiment

(8) sequential isolation of populations versus defining a specific time to attack.

Due to the complexity of metapopulations (dispersal & growth/death within a population), application of network concepts should be studied in many different contexts (various growth rates, dispersal directions, dispersal rates, population sizes, etc.). Upon further examination of complex systems in spatially complex landscapes, networks could be used as a tool to better study and protect natural metapopulations. In addition, use of network concepts could assist conservation managers to prioritize populations and metapopulations with various life history traits.

APPENDIX A: PROGRAM DETAILS

A main program was built that took data from five additional models: scale free and single scale building programs, random and targeted attack programs, an analysis program. Steps to run the program included: (1) User opens either targeted or random network program, depending on which attack type is needed for experiment, (2) User defines which network the program will build (scale-free, single-scale, or no connections), (3) User types in parameters that would stay constant throughout replicates (carrying capacity, number of populations in each metapopulation, the initial number of individuals in each population, number of replicates, and attack time), (4) Tested parameters are inserted into the program (growth rate, percent attack, and dispersal type), and (5) Run the program.

Example code from main program. The "%" sign indicates that values were ignored or silenced during the programs runs, thus allowing both annotation and ghost variables to be in the program without contributing to the code. Other programs contributing to this program (network building program, attack program, and analysis program) are bolded, but not shown in this example.

```
% PROGRAM network_model_dispersal_targetedattack: Disturbance & Dispersal Experiment

clear all
close all
clc
%*****************************PARAMETERS*************************************
*
K=100;                  %carrying capacity
r=0.5;                  %population growth rate
%vari=.145;             %growth rate variation
Tmax=100;               %max time
pop=20;                 %number of populations in network
init=50;                %number of individuals in each population initially (only time 1)
runs=1;                 %number of replicates in the program
perc=0.40;              %attack severity (percent 0, 5, 10, 20, 40)
attacktime=5;
```

```
dir=1; %sets dispersal program; Unidirectional dir==3, Random dir==2, Bidirectional==1
output=zeros(Tmax,pop,runs);    %empty 3D matrix for the output (N)
NetSum=[];
%**************************NETWORK REPLICATES**************************
for rep=1:runs
%**************************NETWORK MODELS*****************************
%Net=zeros(pop,pop);      %network with no connections
Net=scalefree(pop,dir);      %scalefree (hub) network w/ number of populations
%Net=singlescale(pop,dir);    %singlescale (random) network w/number ofpopulations
%Net=ones(pop,pop);       %network with all connections
%**********************************************************************
   Ninit=ones(1,pop)*init;
   N=zeros(Tmax,pop);
   Net1=zeros(pop,pop);
   conn=sum(Net);                rand('state',sum(100*clock));
   randn('state',sum(100*clock));
   for t=1:Tmax;
      if t==1
        N(t,:)=Ninit;
      else
        %Growth loop
        for i=1:pop
           G=r;%+randn*vari;
           Ks=K; %+randn*3;
           N(t,i)=round(N(t-1,i)*exp(G*(1-(N(t-1,i)/Ks))));
           if mod(t,2)==0
                %Migration Scenario no problem
                if N(t,i)>=conn(i);
                   Net1(i,:)=Net(i,:);
                %Migration Scenario not enough individuals for the number of connections
                elseif N(t,i)<conn(i) & N(t,i)~=0;
                   comp=(conn>0);
                   k=0;
                   z=[];
                   w=rand(1,pop
                   x=comp.*w;
                   while k~=N(t,i)
                      y=x==(max(x)); %
                      z=[z;y==1];
                      m=x~=(max(x));
                      x=m.*x;
                      k=sum(sum(z));
                   end
                    rr=size(z);
                    if rr(1)>1
```

```
                    y=sum(z);
                elseif rr(1)==1
                    y=z;
                end
                Net1(i,:)=y(1,:);
                clear m z
            end
        end
        end
        %Adding or subtracting individuals loop
        for i=1:pop
            for j=1:pop
                N(t,i)= N(t,i) + Net1(i,j);
                if N(t,j)>0
                    N(t,j)= N(t,j) - Net1(i,j);
                end
            end
        end
        Net1=zeros(pop,pop);
      end
   if t==attacktime
   preNetconnections=sum(Net,2);
   Net=targetedattack(Net,perc,pop);
   end
   end
connections=sum(Net,2);
NetSum=[NetSum, preNetconnections, connections];
   output(:,:,rep)=N;
end
analysis
```

APPENDIX B: FIGURES

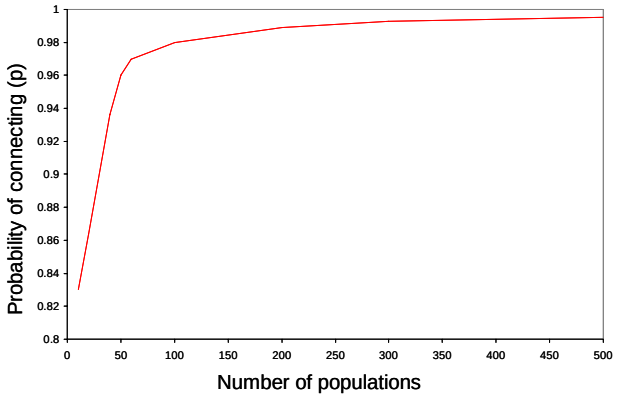

Figure 1: Change in single-scale probability of connection, *p*, based on the number of populations. As the number of populations increases, *p* also increases. A *p* value of 0.995 was selected, therefore limiting the number of connections in a single-scale system. This figure was derived from preliminary test where the number of connections in scale-free network was compared to the connections in a single-scale network, until the compared connections were not significantly different (done with a T-test). Thus, in this study scale-free and single-scale networks were similar though not identical in their total number of connections.

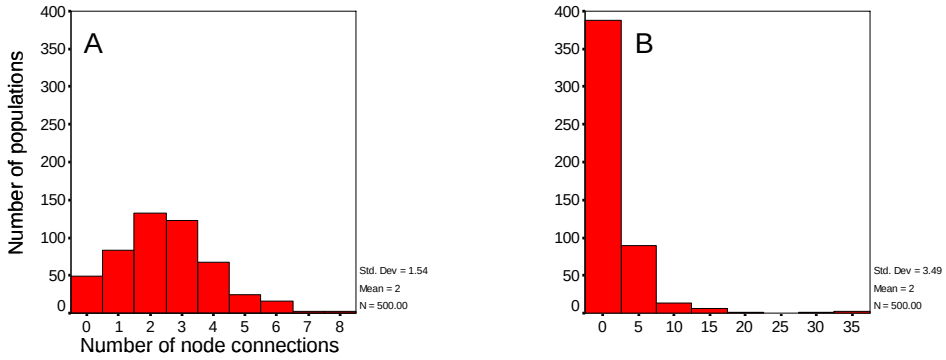

Figure 2: Single-scale (A) versus scale-free (B) networks. Single-scale networks are well defined by an average, where scale-free networks are not. Preferential attachment in scale-free networks leads to a few populations having many connections and many populations having low numbers of connections.

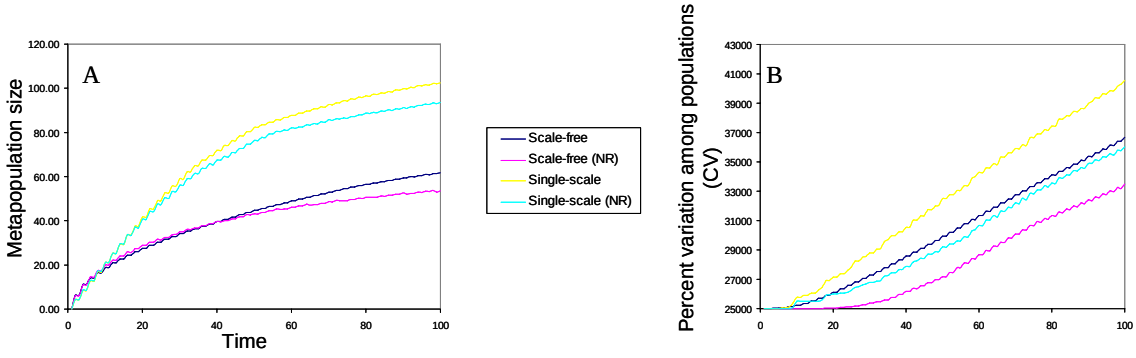

Figure 3: The difference between rounded and non-rounded logistic models. Data shown in both figures is from Unidirectional x r = .005 x Scale-free, because this treatment combination had significant effects throughout all test. The rounded logistic model obtained metapopulation sizes (A) and CVs (B) that were lower than those of a standard logistic model, but the difference was consistent among experimental treatments and did not affect the relative rank of the treatment effects.

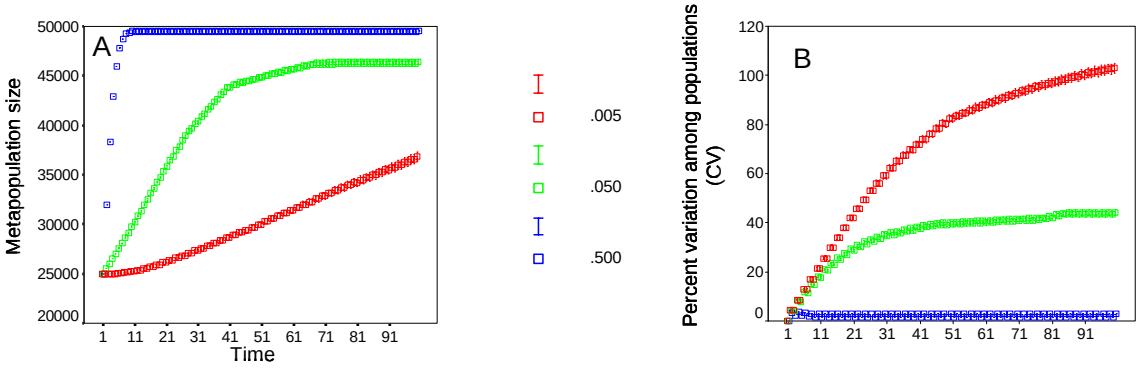

Figure 4: Effect of growth rate on metapopulation size (A) and inter-population variation (B). Symbols in figures are ± 95% confidence intervals. Two figures are shown to represent all networks and dispersal types since analysis was the same for all treatment combinations. Fast growth rates (r=0.5) always had the largest metapopulation size and lowed CV, where slow growth rates (r=0.005) always had the smallest metapopulation size and highest CV.

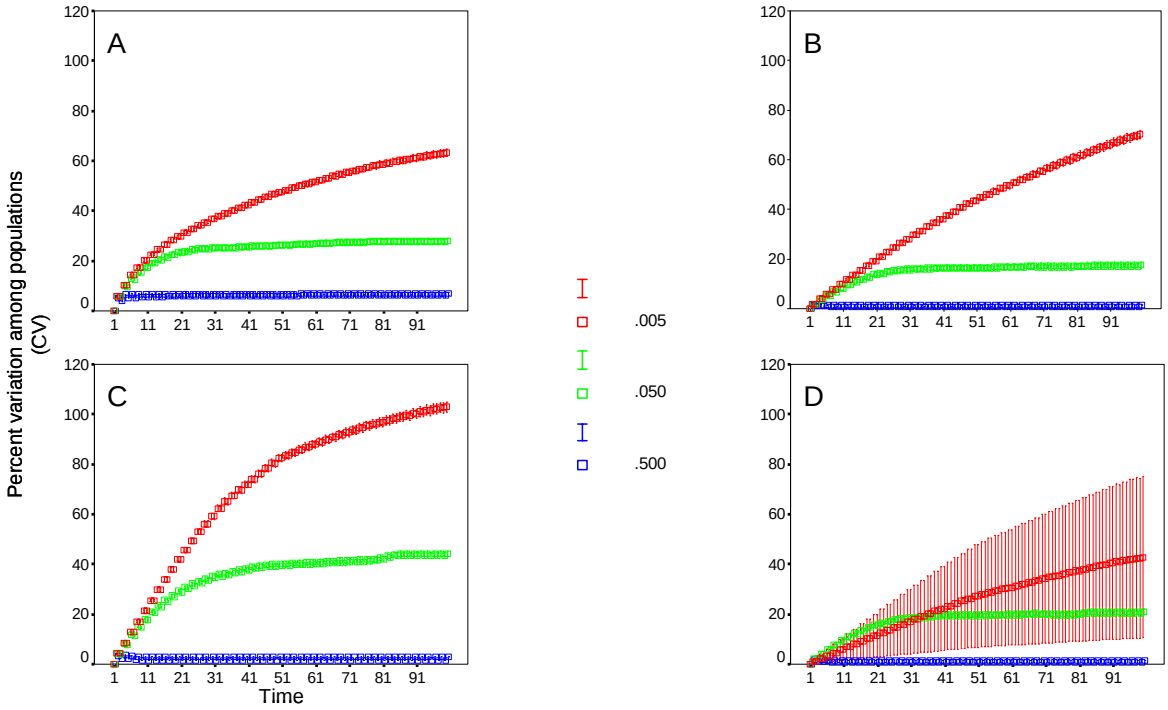

Figure 5: Effect of population growth rate on inter-population variation. A: Unidirectional x Scale-free, B: Random x Scale-free, C. Unidirectional x Single-scale, D: Random x Single-scale. Symbols in figures are ± 95% confidence intervals. Low growth rate (r=0.005) had the largest CV and fast growth rates (r=0.5) had the lowest CV (A-D). Medium growth rates fell in between. Low growth rate in single-scale networks having random dispersal had large variation between replicates (D, consistent with figure 7B).

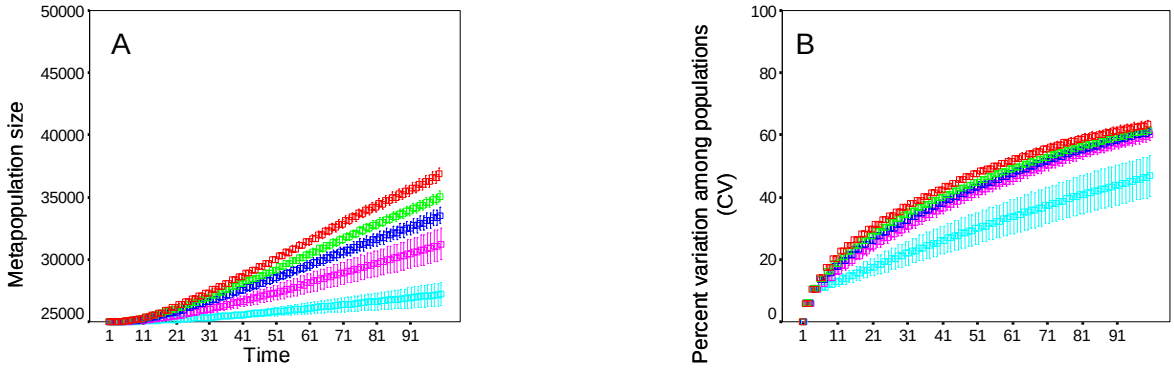

Figure 6: Effect of targeted and random attacks on metapopulation size (A) and CV (B). Symbols in figures are ± 95% confidence intervals. This graph shows the similarity of attack type and network type for any one attack severity because all data for severity, attack type, and network are plotted together. Metapopulation sum (A) shows no overlap in confidence intervals for each severity, thus indicating a significant difference between attack severity. CV (B) does show overlap in confidence intervals in all attack severities but 40%, indicating that there is no difference between lower attack severity. However, both figures do not show large confidence intervals around the mean signifying that data plotted (both network types and attack types) are similar.

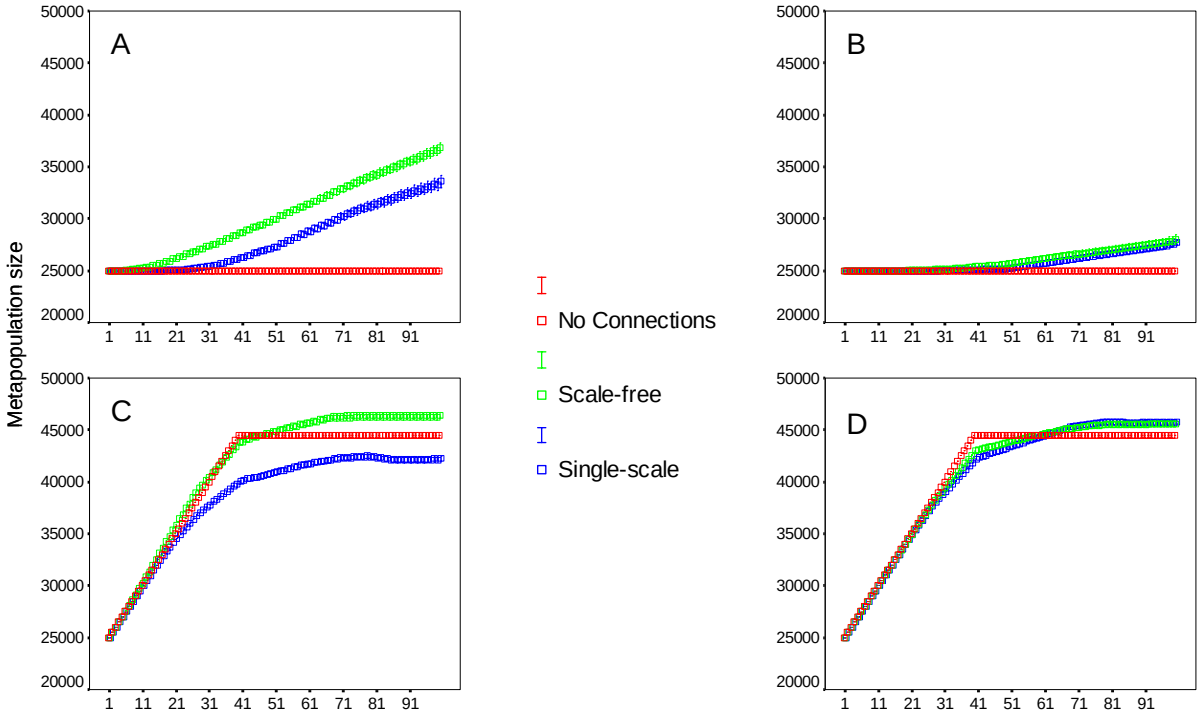

Figure 7: Effect of network on metapopulation size. A: Unidirectional x r=0.005, B: Random x r=0.005, C: Unidirectional x r=0.05, D: Random x 0.05. Symbols in figures are ± 95% confidence intervals. Three distinct networks were seen in unidirectional dispersing populations, with scale-free having the largest metapopulation size (A and C). Randomly dispersing populations had larger metapopulation sizes in both networks compared to the control (no connections), but the networks did not differ from each other (B and D).

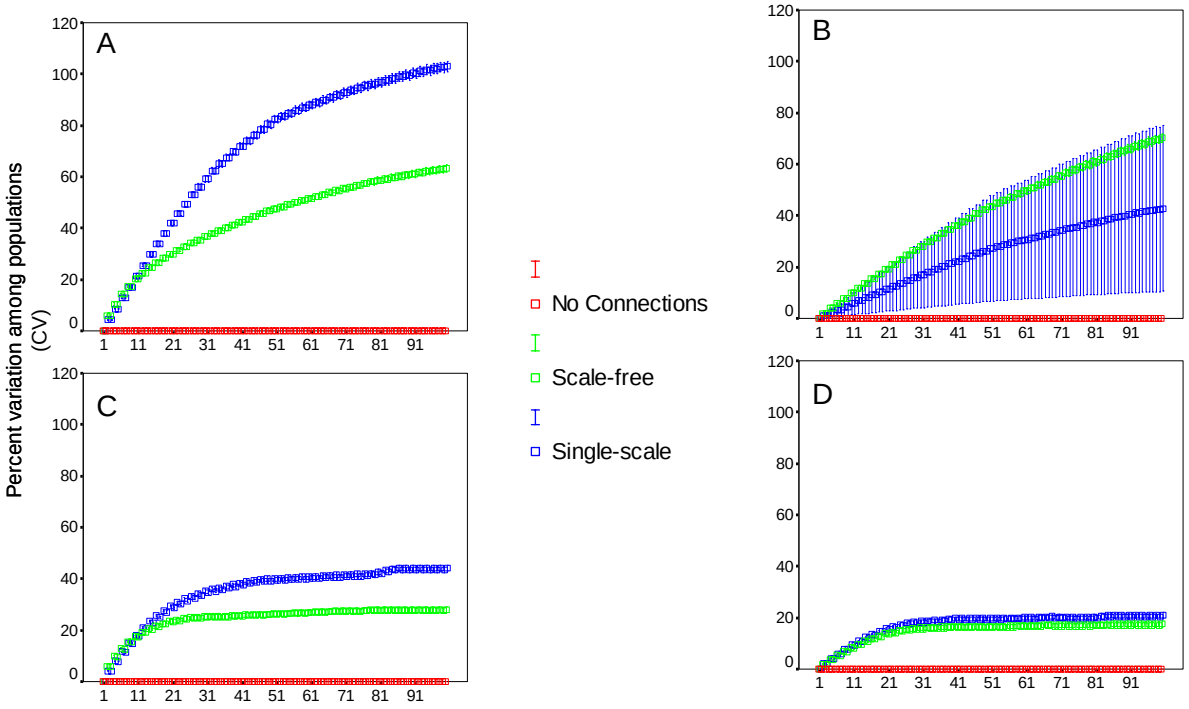

Figure 8: Effect of network on population variation. A: Unidirectional x r=0.005, B: Random x r=0.005, C: Unidirectional x r=0.05, D: Random x 0.05. Symbols in figures are ± 95% confidence intervals. Single-scale had the most variation (A, C, and D) except in random dispersal and low growth rate (B). No connections always had 0% CV (A-D). Bidirectional dispersal and high growth rates had 0% CV and are therefore not shown.

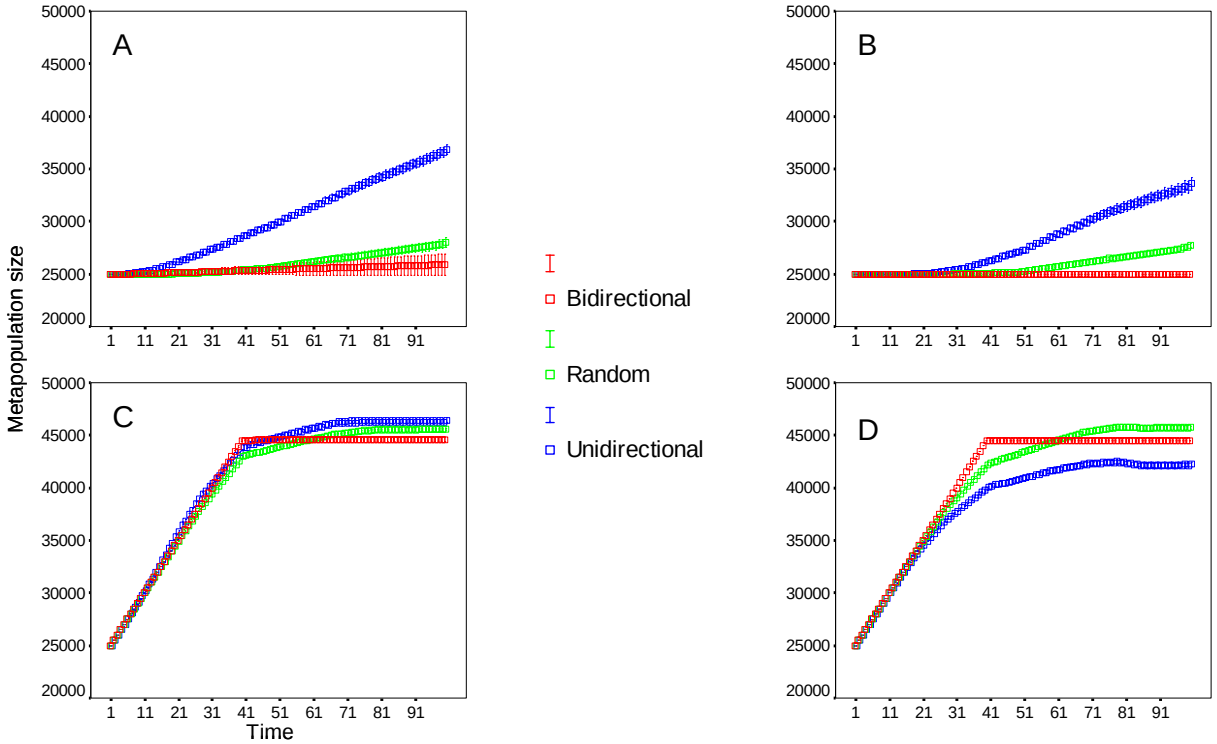

Figure 9: Effect of dispersal type on metapopulation size. A: Unidirectional x r=0.005, B: Random x r=0.005, C: Unidirectional x r=0.05, D: Random x 0.05. Symbols in figures are ± 95% confidence intervals. Unidirectional dispersal had larger metapopulation sizes (A, B, and C), except in single-scale networks with medium growth rates (D). Low growth rates in both networks had three distinct metapopulation sizes due to the dispersal type (A and B).

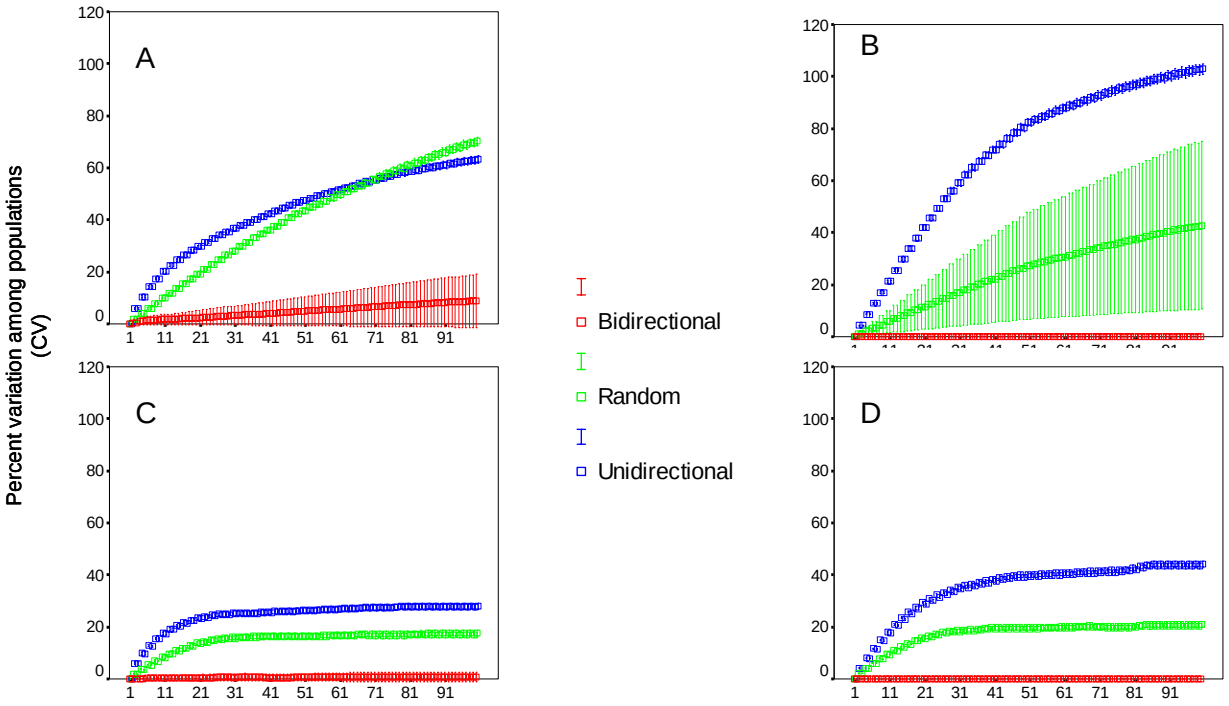

Figure 10: Effect of dispersal on population variation. A: Unidirectional x r=0.005, B: Random x r=0.005, C: Unidirectional x r=0.05, D: Random x 0.05. Symbols in figures are ± 95% confidence intervals. CV was highest in unidirectional dispersal and lowest in bidirectional dispersal (A-D). Bidirectional always had the lowest variation. Random dispersal showed lots of variation between replicates in single-scale networks having a low growth rate (B). Bidirectional dispersal and high growth rates had 0% CV and are therefore not shown.

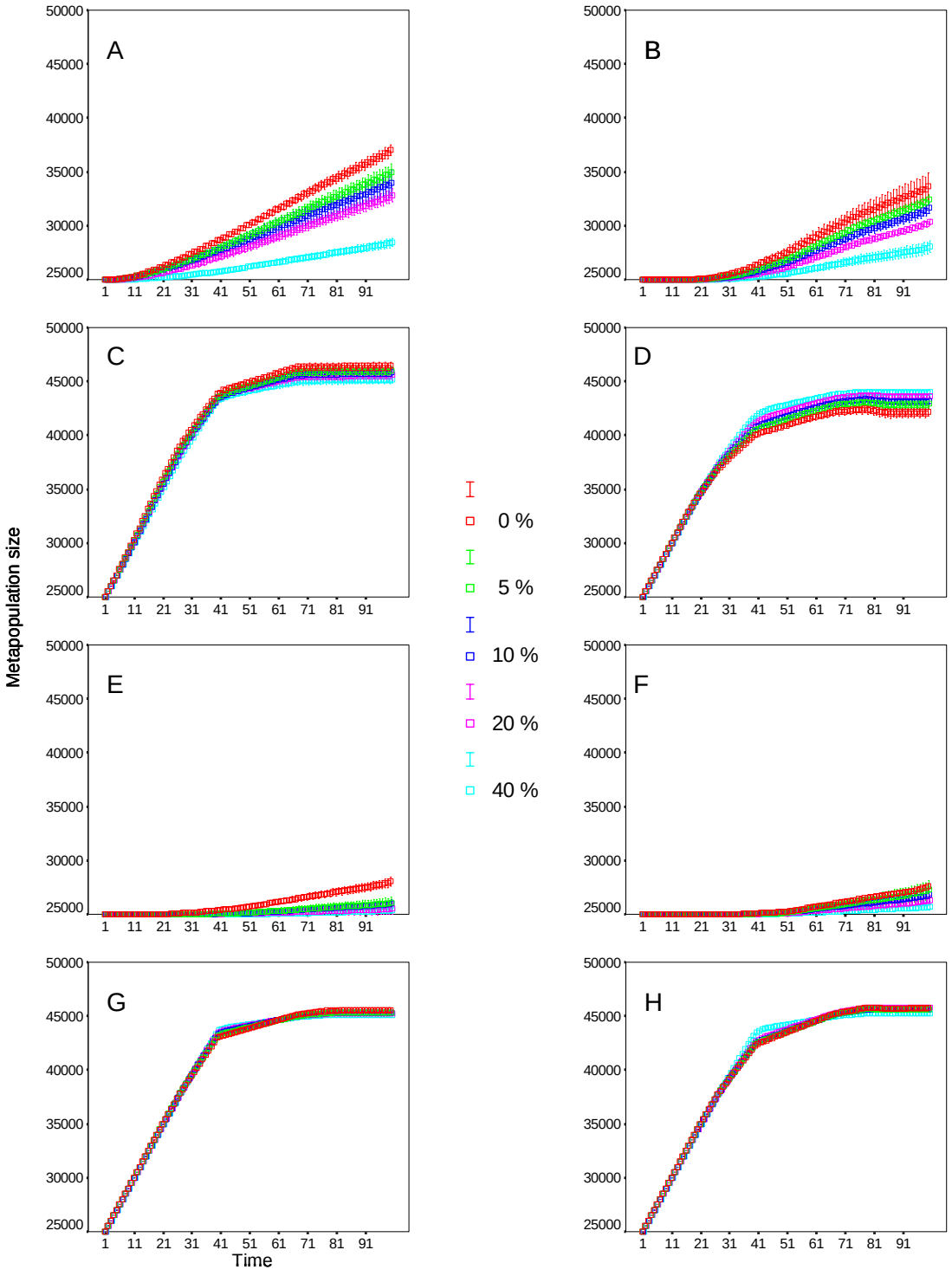

Figure 11: Effect of attack severity (represented as percent) on metapopulation size. A: Unidirectional x Scale-free x r=0.005, B: Unidirectional x Single-scale x r=0.005, C: Unidirectional x Scale-free x r=0.05, D: Unidirectional x Single-scale x r=0.05, E: Random x Scale-free x r=0.005, F: Random x Single-scale x r=0.005, G: Random x Scale-free x r=0.05, H: Random x Single-scale x r=0.05. Symbols in figures are $\pm$ 95% confidence intervals. As attack percent increased, the metapopulation size decreased most in unidirectional dispersal with slow and medium growth rates. This combination also yielded a gradient, in which population sizes decreased as number of connection losses increased (A, B). A similar though weaker gradient also occurred in randomly connected networks (E, F). Attack percent did not change the size in bidirectional dispersal and is therefore not shown.

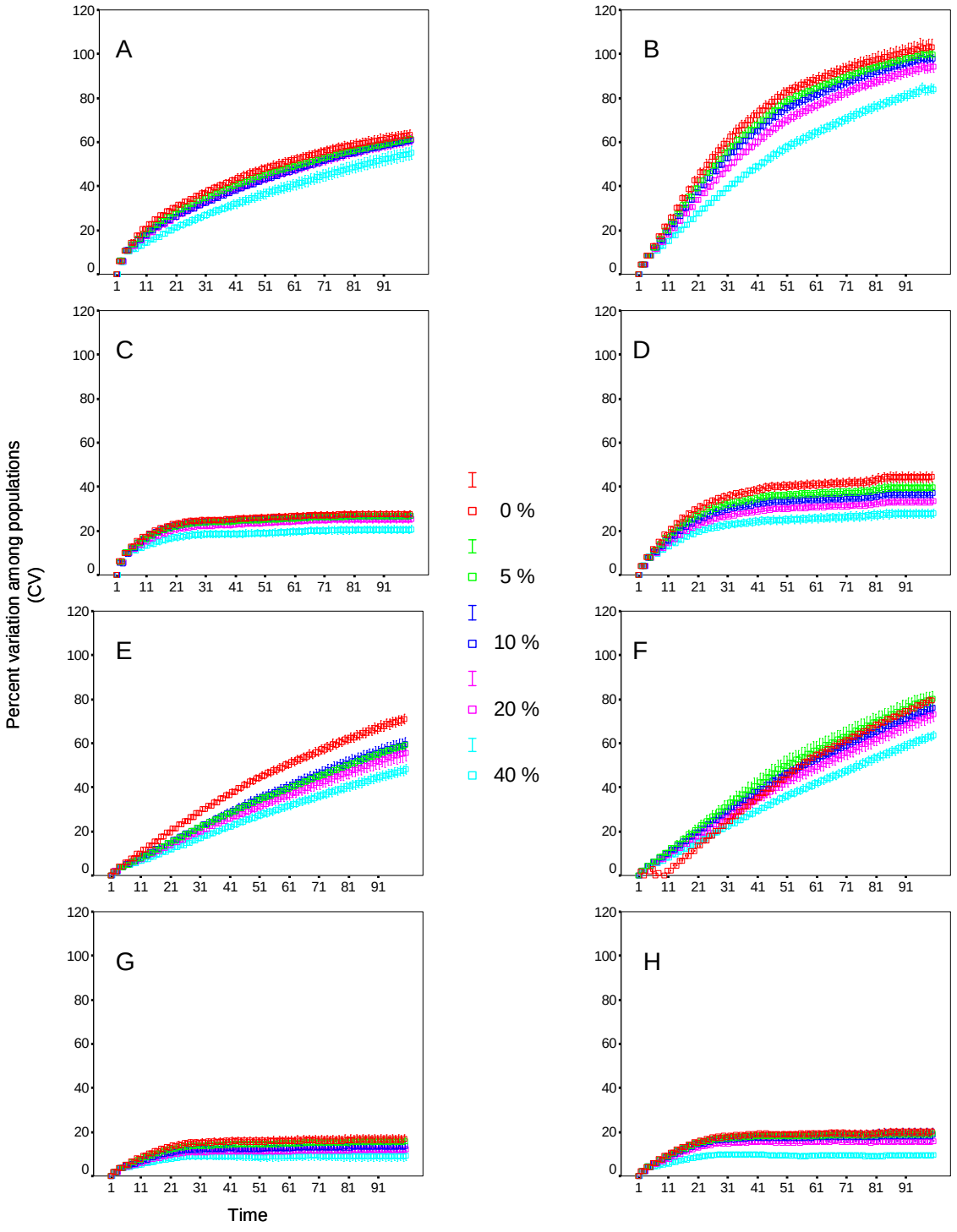

Figure 12: Effect of attack severity (represented as percent) on interpopulation variation (CV). A: Unidirectional x Scale-free x r=0.005, B: Unidirectional x Single-scale x r=0.005, C: Unidirectional x Scale-free x r=0.05, D: Unidirectional x Single-scale x r=0.05, E: Random x Scale-free x r=0.005, F: Random x Single-scale x r=0.005, G: Random x Scale-free x r=0.05, H: Random x Single-scale x r=0.05. Symbols in figures are $\pm$ 95% confidence intervals. Single-scale networks with unidirectional dispersal had the largest CV (B), followed by Scale-free (unidirectional, A, and random, E) and single-scale (random dispersal, F). Low growth rates in both dispersal types and in both networks had low CV (C, D, G, and H). Bidirectional dispersal and fast growth rates showed no difference in the CV in various percent attacks and are therefore not shown.

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