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CAN HELMINTH COMMUNITY PATTERNS BE AMPLIFIED WHEN TRANSFERRED BY PREDATION FROM INTERMEDIATE TO DEFINITIVE HOSTS?

William L. Vickery and Robert Poulin*

Département des Sciences Biologiques, Université du Québec à Montréal, C.P. 8888, Succursale Centre-Ville, Montreal, Quebec, Canada H3C 3P8. *e-mail: robert.poulin@stonebow.otago.ac.nz*

ABSTRACT: Helminth communities in definitive hosts are formed by the acquisition of packets of larvae arriving each time an intermediate host is consumed. It is thus possible that associations between parasite species or other aspects of community structure get transferred from intermediate to definitive hosts. Earlier computer simulations showed that associations between 2 parasite species, in particular positive associations, could be transferred up the food chain. Here, we alter some of the assumptions of previous models and generate new simulations of several ways in which source infracommunities in intermediate hosts can be transferred to target infracommunities in definitive hosts. In particular, we introduced nonrandom selection of intermediate hosts by predatory definitive hosts, to mimic the phenomenon of host manipulation by parasites; this consisted in biasing predation toward intermediate hosts harboring a certain parasite species. Overall, our results show that positive covariances between 2 parasite species can not only be transferred but can also be amplified during transmission to definitive hosts; significant covariance between parasite species can even appear in the definitive hosts when none existed in the intermediate hosts. Negative covariance was not as readily transferred to definitive hosts and amplified, in part because of properties of the presence–absence covariance index. Amplification of covariance results from intermediate host manipulation as well as from other processes taking place during transmission. These results suggest that the patterns of association between helminth species in definitive hosts cannot be taken to reflect the processes acting inside those hosts: they may simply be inherited, with amplification, from intermediate hosts.

Many empirical studies of parasite community ecology have focused on the structure of gastrointestinal helminth infracommunities in vertebrate hosts (see Esch et al., 1990; Sousa, 1994; Poulin, 1997). Attempts to understand the forces structuring these infracommunities are usually based solely on inferences from the observed patterns of species co-occurrences in the vertebrate hosts. Any departure from a random species assemblage is often interpreted as evidence that interactions are at work between parasite species within the vertebrate host. In an important article Lotz et al. (1995) made the point that parasites are not recruited singly into vertebrate definitive hosts; instead, they arrive in ''packets,'' i.e., they are acquired in groups of larval parasites belonging to 1 or more species and are contained within an intermediate host that gets ingested by the definitive host (see also Bush et al., 1993). Using computer simulations, Lotz et al. (1995) showed that if the ''source'' infracommunities in intermediate hosts are structured, i.e., if there are positive or negative associations between parasite species in intermediate hosts, then this structure can be transferred to ''target'' communities in definitive hosts. Lotz et al. (1995) simulated the passage of 2 intestinal parasite species from ''source packets'' in an intermediate host to a target definitive host. Their model predicted that helminths that had a positive covariance in the intermediate host population would also have a positive covariance in the subsequent host. Negative covariance could also be passed from the intermediate to the definitive host, but not as readily. Further, they concluded that this process would never amplify the covariance; covariance in the definitive host was always of equal or lesser amplitude than in the intermediate host. On the basis of these results, Lotz et al. (1995) proposed as a null hypothesis that there will be neither more nor stronger associations in target than in source infracommunities; departures from this null model could indicate that posttransmission processes, such as interspecific competition, structure target infracommunities in the definitive host.

We have extended the model of Lotz et al. (1995) to evaluate the possibility that, under some circumstances, the trophic transmission process can amplify the covariance between species in parasite communities. Of particular interest is the possibility that positive covariances might be amplified more than negative covariances because an excess of positive covariances between pairs of species is often observed in intestinal helminth communities in definitive hosts (Lotz and Font, 1991, 1994).

The model of Lotz et al. (1995) begins by generating a population of 1,000 source packets, i.e., intermediate hosts containing 2 parasite species, each with a negative binomial distribution of abundance among hosts. Initially, the abundance of the 2 helminths was independent. Positive covariance was created by separating the 2 species, sorting both by abundance, and then pairing the 2 species in the new sorted order. Negative covariance involved sorting 1 helminth in ascending order and the other in descending order. A total of 1,000 source packets were then consumed by a predator (definitive host). The predator chose randomly among packets at any given time, and mortality occurred between consumption events among parasites in the predator's infracommunity. Lotz et al. (1995) then treated the 1,000 target host infracommunities thus produced as though they were 1,000 independent hosts and calculated the covariance of the 2 helminth species over the 1,000 target hosts.

The model presented by Lotz et al. (1995) provided parasite community ecologists with a general framework in which to study the structure of the infracommunity. However, their methods involved a number of assumptions. First, they supposed that the sequence of 1,000 target infracommunities that they generated were the equivalent of 1,000 independent communities. Second, their source packets contained the maximum possible covariance given the number of infected packets. Thus, their conclusions do not necessarily apply to cases in which the source packets have lower covariance. Third, they assume that each predatory definitive host eats just 1 source packet per time interval. Lotz et al. (1995) based some of these assumptions on simulations of a single parasite species being passed from prey to predator.

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* To whom correspondence should be addressed. Department of Zoology, University of Otago, P.O. Box 56, 340 Great King Street, Dunedin, New Zealand.

TABLE I. Proportion of simulations in which covariance between 2 parasite species was amplified during transmission from intermediate to definitive hosts, as a function of whether host manipulation was involved or not, and of other simulation procedures.*

	Without manipulation		With manipulation	
	Maximum covariance	Reduced covariance	Maximum covariance	Reduced covariance
Negative covariance				
Sequential method	0.18	0.23	0.25	0.22
Independent method	0.17	0.20	0.33	0.29
Poisson-Pascal (mean size = 1)	0.05	0.04	0.03	0.03
Zero covariance				
Sequential method	0.04, 0.04	0.04, 0.06	0.03, 0.06	0.04, 0.05
Independent method	0.01, 0.03	0.02, 0.03	0.02, 0.04	0.02, 0.04
Poisson-Pascal (mean size = 1)	0, 0.52	0.002, 0.54	0, 0.64	0, 0.68
Positive covariance				
Sequential method	0.20	0.20	0.28	0.29
Independent method	0.21	0.22	0.34	0.36
Poisson-Pascal (mean size = 1)	0.21	0.23	0.40	0.48

* A covariance was considered amplified if its amplitude in the target host exceeded that in the source intermediate host; in the case of 0 covariance in the source host, the proportions of significant covariances (negative and positive, respectively) found in the target host are shown. Each proportion is based on a total of 1,800 simulations.

Another of the model's assumptions is likely to be violated in many natural systems. Lotz et al. (1995) assumed that source infracommunities were recruited at random, from the pool that is available, into target infracommunities. In other words they assumed that definitive hosts select individual prey at random from those present in the population. In fact, many helminths can alter the behavior, coloration, or other aspect of the phenotype of their intermediate hosts in ways that render them more susceptible to predation by definitive hosts (Moore and Gotelli, 1990; Poulin, 1995). In a review of the effect of phenotype-manipulating helminths, Thomas et al. (1998) found that predation rates on intermediate hosts harboring manipulative helminths were on average 30% higher than those on uninfected conspecifics, and sometimes they were much higher (data from 13 field and laboratory studies). In nature, these manipulating parasites often share their intermediate and definitive hosts with other helminths with identical life cycles (see table II in Thomas et al., 1998 for review). For instance, Bush and Holmes (1986) found that the intestinal helminths of scaup consist of 2 suites of species, each using a different intermediate host species. One suite of 7 helminth species used 1 amphipod species as intermediate host, and the other suite of 4 species used another amphipod. Each suite includes an acanthocephalan known to alter the behavior of its amphipod host in ways that could make the host more susceptible to predation by scaup (Bethel and Holmes, 1973). In target infracommunities in scaup there were strong positive associations among helminth species belonging to the same suite (Bush and Holmes, 1986). These associations may only mirror those in source infracommunities, but they could also be amplified by the nonrandom recruitment of source infracommunities into target infracommunities that results from host phenotype manipulation.

Here, we test these assumptions explicitly in order to evaluate their effect on the possible amplification of parasite covariance. We modified the model of Lotz et al. (1995) in many ways, in particular to include the nonrandom recruitment of source infracommunities into target infracommunities. We thus examine how nonrandom recruitment affects the transfer of positive or negative associations between a manipulator parasite species and another, nonmanipulating species. Manipulation of intermediate host phenotypes may be the rule in any system including 1 or more acanthocephalans (Moore, 1984) and in other communities; our results thus provide an important adjustment to the null model proposed by Lotz et al. (1995) to assess the influence of posttransmission processes in structuring target infracommunities.

MATERIALS AND METHODS

We used simulation modeling based on the model of Lotz et al. (1995) to evaluate the transmission of covariance among parasites from intermediate to definitive hosts. We, therefore, do not repeat the description of the procedures detailed in Lotz et al. (1995); instead, we only specify what modifications have been made to their model. The intermediate host, or source packets, contained 2 parasite species, each aggregated among their hosts after a negative binomial distribution. We used the parameter values of figure 7 in Lotz et al. (1995) as a starting point for the simulations and then selected combinations of parameter values of the negative binomial distribution that we suspected might amplify covariance in the target infracommunities (Table I). Survival rates followed those used in figure 7 of Lotz et al. (1995), except that we evaluated our models not only for survival rates of 0.01, 0.1, 0.3, 0.5, and 0.7, as they did, but also for a higher rate of 0.9. For each combination of parameter values and for each type of simulation procedure (see later), we ran 5 replicate simulations. This study is thus based on a total of 64,800 simulations.

We allowed definitive hosts to ''eat'' the source (intermediate host) communities in order to generate target infracommunities. We proceeded in 2 ways. First, we used the sequential method of Lotz et al., in which a new target infracommunity is created each time a meal is consumed (after equilibrium conditions have been reached). This assumes that infracommunities within the sequence are independent of each other at equilibrium. Second, we simulated 1,000 communities independently. This involved repeating the sequential method of Lotz et al. 1,000 times and retaining only the final parasite infracommunity in each simulation. In this case, sequences were continued only until the infracommunities reached equilibrium, i.e., about as many simulations as the mean ex-

pected equilibrium parasite population size (see Lotz et al., 1995, p. 14), instead of up to 1,000 time periods.

Next, we altered our source packets so that their covariance was no longer maximized. We did this by first using the method used by Lotz et al. for sorting each parasite species by its abundance and then pairing the 2 species in packets according to their ranking (same ranking for positive covariance and opposite ranking for negative covariance). This produced maximal covariance (see Appendix I). To reduce this covariance we chose 250 source packets at random and switched the abundance of 1 of the parasites in that packet with that from another packet also chosen randomly.

Another modification to the model of Lotz et al. (1995) involved simulating prey (source packet) consumption as a Poisson variable. This allowed predatory definitive hosts to eat a variable number of prey in each time interval, i.e., variable meal size. Lotz et al. (1995) showed that for a single parasite species, this process (known as Poisson–Pascal) produces a negative binomial distribution of parasites among definitive hosts if they are also distributed in this way in intermediate hosts. Lotz et al. did not, however, explore the effect of variable meal size on the covariance between 2 parasite species.

We simulated 1,000 meals per target definitive host, with a mean meal size of 1 source packet. These simulations used the same parameter values as did the sequential and independent simulations described earlier.

Finally, we simulated parasite manipulation of intermediate host behavior. For each of the simulations described above, we ran a corresponding simulation with host manipulation. In the latter simulations, source packets infected with parasite species A were twice as likely to be eaten by definitive hosts as those which were not infected by parasite species A. The presence or absence of the second parasite species did not affect consumption of source packets.

RESULTS

In contrast to Lotz et al. (1995) we find that the transfer, through the food chain, of parasite communities from source to target hosts can amplify the covariance among parasite species. Of the 21,600 simulations that started with a positive covariance between 2 parasite species in the source intermediate host, 6,146 (about 28%) had an even higher covariance in the target definitive host. The remaining simulations generally showed a positive covariance in the target host but with lower magnitude than in the source host. Negative covariances were amplified (became more negative) in 3,624 of the 21,600 simulations (about 17%), with the remaining simulations producing a negative covariance of lesser magnitude than that in the source host. When the parasite species do not covary in the intermediate host, they also rarely (but see next paragraph) covary in the target host. Only 1,059 of the 21,600 simulations (about 5%) produced significant covariance in the target host when there was none in the source host.

The method of simulation did have some effect on covariance amplification (Table I). Whereas the sequential method differed very little from independent simulations, the latter did amplify covariance more often when manipulation of intermediate host behavior was included. The full Poisson–Pascal simulations, in which meal size varied as a Poisson variable, often differed from the sequential and independent simulations, generally amplifying positive covariances more often and negative covariances less often than did the other simulations. In addition, the Poisson–Pascal simulation is the only simulation that consistently generates positive covariance in the target host where there was none in the source host (no simulation ever produced a negative covariance in the definitive host when no covariance existed in the intermediate host). Poisson–Pascal simulations tended to amplify positive covariances but not negative ones.

Table I also shows that manipulation of intermediate host behavior can both generate covariance where none existed in the source packets and amplify positive covariance. Furthermore, in 215 cases the combination of intermediate host manipulation and Poisson–Pascal simulation produced significant positive covariance in the target host when there was a negative covariance in the source intermediate hosts. This represents about 5% of the Poisson–Pascal simulations, with manipulation that began with negative covariance in the source packets. This phenomenon was not seen in any other simulation. Nor was the converse, positive covariance in the source hosts generating significant negative covariance in the target host, ever seen.

Occasionally, i.e., 258 times or about 1% of the simulations with positive covariance in the source packets, a positive covariance in the source host did produce a small (but not statistically significant) negative covariance in the target host. On the other hand, negative covariance in the source host resulted in positive covariance (usually not statistically significant) in the target host 6,307 times (about 29% of the time). Clearly, there is a tendency to produce positive covariances as parasites pass through the food chain from intermediate to definitive hosts.

To see which factors contribute to this amplification of covariance, we look at each of the parameters that we varied separately in our simulations. Amplification varies with k, the aggregation parameter of the negative binomial distribution (aggregation of parasites among hosts increases as k decreases), but without producing a consistent pattern (Table II). Similarly, mean parasite abundance (Table II) appears related to covariance amplification without producing a clear pattern.

The factor that is most clearly associated with covariance amplification is the covariance in the source intermediate host (Table II). Clearly, smaller covariances in the source host are easily amplified in the target host, whereas larger covariances are not. Also, positive covariances are more likely to be amplified than negative ones. This occurred despite the fact that the negative covariances generally had a smaller amplitude than did the positive ones (median covariance values, -0.03 vs. $+0.10$). Overall, when positive covariance in the source host was small (less than the median value), covariance was amplified in 57% of the simulations. But when positive covariance in the intermediate hosts was large (greater than the median value), only 4% of the simulations showed amplification. Similarly, with large negative covariance (less than the median value), only 1% of the simulations showed amplification, whereas 29% of the small negative covariances (closer to 0 than to the median value) were amplified.

When the 2 parasite species were distributed independently in the source packets, i.e., covariance = 0, significant covariance was generally not found in the target host, except for Poisson–Pascal simulations.

Survival rates within the target host do affect the transmission of covariance. As survival increases, covariance is less easily transmitted from source to target host. Not only are covariances less likely to be amplified (Fig. 1) but absolute covariance also decreases as survival approaches 100% (results not shown). Our initial simulations included survival rates of 0.99, but these were abandoned when we realized that this almost always produced 0 covariance. At this high survival rate all individuals in the target host population eventually become

TABLE II. Amplification of covariance between 2 parasite species during transmission from intermediate to definitive hosts, as a function of the parameters of the negative binomial distribution describing their occurrence in intermediate hosts.*

k	Mean parasite abundance	Negative covariance at source	Positive covariance at source	No. of negatives amplified	No. of significant values obtained from 0 covariance	No. of positives amplified
0.2	5.0	−0.21	0.24	2	150	87
0.1	2.5	−0.09	0.20	70	112	154
0.067	1.0	−0.03	0.12	455	79	795
0.01	0.25	−0.001	0.03	1,062	2	1,701
0.2	2.5	−0.16	0.24	11	167	44
0.2	1.0	−0.11	0.21	118	207	246
0.2	0.1	−0.01	0.07	884	0	1,474
0.1	5.0	−0.11	0.21	19	115	57
0.067	5.0	−0.06	0.18	80	93	185
0.01	5.0	−0.004	0.05	859	111	1,378

* A covariance was considered amplified if its amplitude in the target host exceeded that in the source intermediate hosts; in the case of 0 covariance in the source host, the numbers of significant covariances found in the target host are shown. Each value is based on a maximum possible total of 2,160 simulations.

infected with both parasite species, thus producing a presence–absence covariance of 0.

We also found a pattern noted by Lotz et al. (1995) that covariance is best transmitted when survival rates of the 2 parasite species in the definitive host are nearly equal. In their study this produced the greatest tendency to conserve covariance. This was also the case in our simulations (results not shown); Figure 1 shows, however, that covariances are most often amplified when survival rates are equal.

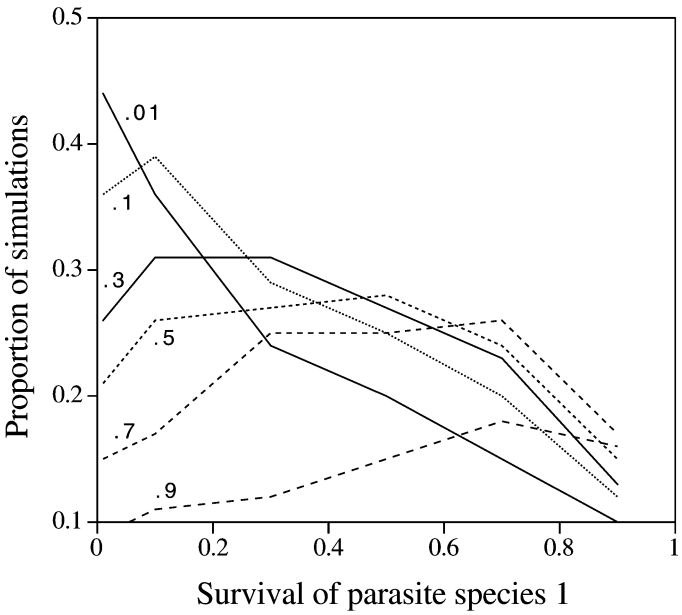

FIGURE 1. Proportion of simulations in which covariance between the 2 parasite species was amplified as a function of their survival rates after their recruitment into target (definitive) host infracommunities. The values on the curves represent the survival rate of the second parasite species. A covariance was considered amplified if its amplitude in the target host exceeded that in the source (intermediate) hosts; simulations in which the initial covariance was nil are not included here. Proportions are based on a total of 1,200 simulations for each combination of survival rates of the 2 parasite species.

DISCUSSION

Our simulations show that not only can covariance in parasite infracommunities be passed from prey (intermediate hosts) to their predators (definitive hosts), as suggested by Lotz et al. (1995), but these covariances can also be amplified, i.e., increase in magnitude, during this process.

The manipulation of intermediate host behavior by parasites can act to amplify the covariance between parasite species as they are transmitted from intermediate to definitive hosts. Our results show that the presence of a manipulating species in source infracommunities can, under a range of simulation conditions, amplify covariance between parasite species, particularly with positive associations. Thus, many positive associations observed among parasite species in definitive hosts may be explained by both pretransmission and transmission processes, without the need to invoke posttransmission interactions. Several recent studies have exposed significant associations in intermediate hosts between parasite species with identical life cycles, including many cases where 1 parasite is a known manipulator (e.g., Thomas et al., 1997, 1998; Lafferty, 1999; Dezfuli et al., 2000; Lafferty et al., 2000). In these systems nonmanipulative parasites, often referred to as ''hitch-hikers,'' associate with the manipulator inside intermediate hosts to gain a better chance of reaching their common definitive host. This can amplify their covariance through the food chain. For instance, Poulin and Valtonen (2001) have shown that covariances in fish hosts between pairs of helminth parasites, including known or suspected manipulators, are clearly higher when these helminths come from a common invertebrate intermediate host. Our simulations support the postulated role of manipulation in the amplification of covariances.

This amplification can occur for a number of other reasons as well. First, eating multiple prey per meal can amplify covariance among parasites (Appendix II). This occurs in our simulations using the explicit Poisson–Pascal process. When a definitive host eats more than 1 intermediate host per meal, it acquires the sum of all the source infracommunities it eats in that meal. The sums of the abundances of the 2 parasite species

thus become more correlated in the definitive host than they were in the intermediate host.

Second, variable meal size can increase covariance (see Appendix III for a simple illustration). If the probability of eating a meal is sufficiently large, covariance will increase (rather than be amplified) as a result of the trophic process. This may explain the results of our Poisson–Pascal simulations, in which positive covariances were often amplified, null covariances became positive, and negative covariances were rarely amplified and sometimes became positive, even significantly so.

The result of the latter 2 factors is that amplification of covariance occurs more readily for positive than for negative covariance. This occurs because of the use of presence–absence covariances in this study. When the covariance in the source packets is negative, but definitive hosts eat several prey per meal, the probability that they will acquire at least 1 parasite of each species actually increases as the number of prey eaten increases, even if the initial covariance is negative. Thus, 2 opposing processes are at work here, one creating a larger negative covariance as negatively covarying species are accumulated and the other pushing the presence–absence covariance in a positive direction as the frequency at which both parasites are present in the definitive host increases. Of course, for a positive covariance in the source packet, these forces both act in the same direction, producing greater positive covariance.

The cases in which covariance was amplified in our results were mainly of 2 types: (1) those in which mean parasite abundance increased in the definitive hosts compared with the source packets, leading to an increase in the maximum covariance possible as well as the observed covariance in the target infracommunities; and (2) those in which we reduced the initial covariance below the maximal values used by Lotz et al. (1995). We chose to explore cases in which parasite prevalence was less than 50% in the source packets so that there would be a potential for increased prevalence and, thus, covariance amplification in infracommunities of definitive hosts.

As a consequence, our results disagree with those of Lotz et al. (1995). The latter's conclusions are illustrated in their figure 7. However, the parameters used to generate that figure leave little chance for the amplification of covariance. Covariance in the source packets is so close to its theoretical maximum of 0.25 that there is no room for increase. When we used these parameters in our simulations, we found amplification only twice in 2,880 simulations. Amplification was much more common under the other parameter values.

Our findings also differ from those of Lotz et al. (1995) in that there is much more variability in our results. In some cases, for instance, negative covariances in source infracommunities become positive in target infracommunities. In other cases positive covariances become negative (although not significantly so). Thus, the relation between source and target infracommunity structure is not easily predicted.

One secondary objective of this study was to find the source(s) of positive covariance within parasite infracommunities. One possible source is suggested by the covariances in the source communities in Table II. In all cases positive covariances have greater absolute value than do the corresponding negative covariances. This suggests that there is a bias in the way in which presence–absence covariance is calculated, and this tends to produce larger positive covariances. This bias may make it easier to detect positive covariances than negative ones when the majority of the host population is uninfected (Appendix IV) and could thus explain the observation of Lotz and Font (1991, 1994) that positive covariances are found more often than negative covariances in parasite infracommunities.

It has been pointed out before that most of the methods commonly used to detect interspecific associations between parasites are more likely to detect positive associations than negative ones of comparable strength (Haukisalmi and Henttonen, 1998). To avoid this bias it might be preferable to measure species association using a correlation coefficient, based on the numbers of parasite individuals of each species in each host, rather than presence–absence covariance. Whereas this may not be feasible for some parasite species, it may produce more reliable results for those species where it is possible. To evaluate the advantages of such an approach, we repeated our simulations using the Pearson product–moment correlation rather than the presence–absence covariance. These results are not yet fully analyzed, but a preliminary evaluation shows that both positive and negative correlations can be amplified and that positive amplification is not more frequent than negative amplification.

The effects of the 2 parameters of the negative binomial distribution, k and the mean number of parasites per host, appear minimal because it is the initial covariance they generate in source infracommunities that is of greatest importance. However, there may be small effects. Table II suggests that as k decreases (as aggregation increases among intermediate hosts), the frequency of covariance amplification increases. Similarly, when the mean parasite load is low, the frequency of amplification is higher. Both these observations may result from the lower prevalence in the source packets, leaving more room for prevalence and thus covariance to increase in the target host.

In summary, host manipulation and other processes can not only facilitate the transfer of but also amplify the associations between parasite species as they are acquired by definitive hosts from intermediate hosts. Because infracommunities in definitive hosts thus begin with an acquired structure, one would need direct evidence of posttransmission processes within the definitive host to conclude that they occur. We must, therefore, strongly caution against drawing inferences from the observed patterns of species associations within definitive hosts. Such patterns have too many possible origins, including pretransmission, transmission, and posttransmission events, as well as combinations of these.

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APPENDIX I—RANGE OF THE PRESENCE–ABSENCE COVARIANCE

The presence–absence covariance has a limited range, which depends on parasite prevalence.

Proof: Let p be the proportion of hosts infected by parasite 1; then $1 - p$ is the proportion of uninfected hosts. Similarly, let q and $1 - q$ be the proportions of hosts infected and uninfected by parasite 2, respectively (p and q are the parasite prevalences). Let X_i be 1 if parasite 1 is present in host i and 0 if it is not, and similarly for Y_i. The presence–absence covariance between X and Y is given by

$$\mathrm{PACOV}(X,Y) = (\Sigma X_i Y_i - npq)/n$$

where n is the total number of hosts. Now, consider the maximum possible value of $\Sigma X_i Y_i$, given that X_i and Y_i can take on only values of 0 and 1, and $\Sigma X_i Y_i$ cannot exceed the lesser of ΣX_i and ΣY_i. Suppose, without any loss of generality, that $\Sigma X_i < \Sigma Y_i$. Then, the maximum value of the presence–absence covariance is

$$\mathrm{MAX}(\mathrm{PACOV}(X,Y)) = (np - npq)/n = p(1 - q).$$

Thus, the presence–absence covariance is a function of the parasite prevalences p and q.

We can determine the maximum possible value of the presence–absence covariance as a function of p and q. Taking the derivatives with respect to p and q, it can be seen that the presence–absence covariance increases as p increases but decreases as q increases. The maximum value will occur when p is highest and q is lowest. As $p < q$, this means the maximum occurs at $p = q$. Further, it is easily seen that the overall maximum will occur when $p = q = 1/2$, at which point $\mathrm{PACOV}(X,Y) = 1/4$. It can be shown in a similar way that the minimum value is $-1/4$. Thus, the value of the presence–absence covariance is constrained between $-1/4$ and $+1/4$.

We note that figure 7 in Lotz et al. (1995), illustrating the transmission of parasite covariance, uses source packets with covariances of $+0.24$ and -0.22. Thus, it is not surprising that the trophic transmission process does not amplify the covariance because there is almost no room for an increase in the absolute values of these covariances.

We observe that for many of our simulations and some of the simulations of Lotz et al. (1995), covariance in the definitive host infracommunities was maximized when survival rates were equal. Given that both parasites had the same distribution in the source packets, this amounts to showing that the covariance in definitive host infracommunities reaches its maximum when the 2 parasites have equal prevalence (as predicted previously).

APPENDIX II—LARGE MEALS SHOULD INCREASE COVARIANCE

Suppose that a predator (definitive host) eats n prey (intermediate hosts) per meal, and the prey contain 2 parasite species having a covariance c. Then, the covariance of the parasites within a meal will be given by the sum of the covariances within the prey eaten. Thus, if

$$\mathrm{COV}(X_i, Y_i) = c \text{ then}$$

$$\mathrm{COV}(\Sigma X_i, \Sigma Y_i) = nc$$

However, our simulations deal with variable meal sizes, so we do not expect exactly this result; we do expect covariance to rise with meal size.

Also, the above result applies to normal covariances, not just presence–absence covariances. Our simulations use presence–absence covariance, which is dependent on prevalence. Prevalence will increase with meal size. Thus, when prevalence is high, presence–absence covariance may actually decrease in cases where normal covariance would increase (an anonymous reviewer suggests that this decrease occurs when prevalence is greater than 0.5).

APPENDIX III—VARIABLE MEAL SIZE CAN INCREASE COVARIANCE

Consider the simplest case, in which a predatory definitive host will include either 0 or 1 source packet (intermediate host) in a given meal. Let v be the probability of eating a source

packet in a meal ($1 - v$ is then the probability of eating no source packet in that meal). Suppose that a proportion r of the source packets contain both parasites, whereas a proportion s contain only parasite 1, and a proportion t contain only parasite 2. Thus, the presence–absence covariance in the source packets is

$$C_s = r - (r + s)(s + t).$$

Now, we calculate the covariance in the target definitive host after a meal:

$$C_p = vr - v(r + s)v(r + t).$$

The covariance increases in the target (definitive host) species if $C_p > C_s$, that is, if $vr - v(r + s)v(r + t) > r - (r + s)(s + t)$, which means that $1 + v > r/((r + s)(r + t))$.

Thus, if the probability of eating a source packet in a given meal is sufficiently large, the covariance in the target host species will be larger than that in the source host. Note that the right-hand side of this inequality is the ratio of the proportion of source packets having both parasites to the product of the proportion that has parasite 1 and the proportion that has parasite 2. In this case it is not amplification that occurs but a simple increase in covariance. Thus, positive covariance will become larger, whereas negative covariance will approach 0 (perhaps even become positive).

APPENDIX IV—DETECTION OF POSITIVE VERSUS NEGATIVE COVARIANCE

Consider our 2 parasite species. Suppose that parasite 1 is present in a proportion a of hosts, and parasite 2 is present in a proportion b. Let us find the greatest possible positive and negative covariances among these species. With no loss of generality, we assume $a < b$.

The greatest positive covariance occurs when parasite 1 is found only in host individuals that have parasite 2. This gives a covariance of $c_{maxp} = a - ab = a(1 - b)$.

The greatest negative covariance has 2 possibilities depending on whether $a + b > 1$. If $a + b < 1$, then $c_{minp} = 0 - ab$. But when $a + b > 1$, $c_{minp} = a + b - 1 - ab$.

First, consider $a + b < 1$ and $c_{minp} = 0 - ab$. c_{minp} has an absolute value of ab, whereas c_{maxp} has a value of $a(1 - b)$. Thus, when $1 - b > b$ (i.e., $b < 1/2$), the maximum positive covariance will have a greater absolute value than the maximum negative covariance (and vice versa when $b > 1/2$).

Now, when $a + b > 1$ and $c_{minp} = a + b - 1 - ab$, the absolute value of c_{minp} is $(1 - a)(1 - b)$. Again, this is less than $a(1 - b)$, the absolute value of the maximum positive covariance provided that $a > 1/2$.

Combining these 2 results, we see that the maximum possible positive covariance will be greater than the minimum possible negative covariance either when a and b are both less than 1/2 or when a and b are both greater than 1/2. In these cases positive covariance will be easier to detect than negative covariance, and thus positive covariances will appear to be more common than negative ones. When 1 of the parasite prevalences, a or b, is less than 1/2 and the other is greater, the converse will be true. However, negative binomial parasite distributions within hosts tend to produce distributions where most parasites are found in a small proportion of hosts, thus leading to low prevalences and a bias toward the detection of positive covariances.