**Title**

Parasite diversity-infection patterns: Potential drivers and recurrent patterns

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**Key words**

Amplification, co-infection, dilution, parasite aggregation

**Abstract**

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**I- INTRODUCTION**

**I.1- Infection-diversity patterns at the host level**

Evidence that species diversity influences infection likelihood and intensity has rapidly accumulated during the last 15 years (Johnson et al. 2015; Keesing and Ostfeld 2015; Young et al. 2017). In general, a change in the abundance of a host species ought to influence the diversity of species within a community, particularly so if a species is removed or introduced. In turn, the abundance and prevalence of parasites infecting those hosts ought to be affected too, because changes in host abundance and frequency influence parasite transmission. Yet, the particular direction of this relationship, whether biodiversity ought to increase (i.e. amplification effect) or decrease (i.e. the dilution effect hypothesis) infectious disease, remains the focus of much debate (e.g. Lafferty and Wood 2013; Wood et al. 2016; Young et al. 2017). Nonetheless, this debate and the search for a general relationship between biodiversity and infection has generated a momentous expansion of our understanding of disease dynamics [REFs?], and underscores that the processes that shape infection extend beyond single-host-single-parasite dynamics. Furthermore, whether and how biodiversity influences infection dynamics has broad ranging implications for human health, conservation and animal production (Johnson et al. 2015; Keesing and Ostfeld 2015). Ultimately, this search for broad patterns between host diversity and infection has brought to the forefront the notion that such emergent and general associations between diversity and disease may exist. Indeed, a recent metaanalysis shows that increasing host diversity is associated with lower infection, and that this association is widespread, occurring in different categorizations of parasites (micro vs. macro), types of study (experimental vs. observational), parasite life-cycles (simple vs. complex) and degrees of specialization (specialists vs. generalists).

[MAKE THE LINK TO IDEA OF EMERGENT PATTERNS]]

Assessments of biodiversity-infection relationships have been limited to the diversity of host species [REFs(but see Johnson et al. 2013)] and have ignored the diverse nature of parasite infections, which are often composed of multiple co-infecting species [REF]. Whether accounting for parasite diversity changes host biodiversity-infection patterns is unclear, not only because of the lack of direct studies but also because few studies have addressed the relationship between parasite diversity and infection. Here we evaluate whether there are recurrent parasite diversity-infection patterns, assess the potential mechanisms driving them, and discuss its implications for the understanding of parasitism and the role of diversity in disease.

[[[COULD WORK WITH TOM ON MODEL LOOKING AT HOW PARASITE INFECTION DIVERSITY PATTERNS LINK TO HOST INFECITON DIVERSITY PATTERNS]]]

It has been suggested that these patterns are widespread to non-free living species. Indeed, two papers support that this dilution effect may be general even among parasite interactions ...

And begs the question whether this is indeed the case and whether these patterns instead may drive (be behind) higher order free-living host dilution patterns. (WHAT parallels are there? – i.e. are there recurrent patterns at the parasite diversity-infection scale, and what are the mechanisms behind these patterns?)

Yet missing from increasingly studied/understood pattern are parasite infection diversity impacts. The above lack of integration/consideration is nonetheless surprising given that it is well established that parasites are “ubiquitous”, have major impacts on host populations and their communities, and that the infection behaviour of a given species of parasite can be drastically affected by other co-occurring parasites. (then note this is common) effects on communities.

[[Conclusion – amplification, not dilution seems to be the general effect - with few instances of dilution indeed occurring ]]]

Yet papers addressing this question are scarce (despite data to test it is abundant). We here review the patters from available literature, use freely accessible datasets to test further these relationships, and XXXXXXX, evaluate the direction and generality of para diversity-infection patterns, put forward mechanisms behind these patterns and address gaps in our understanding that should lead future research into the issue.

Indeed, asking whether parasite diversity can influence parasite infection (prevalence, abundance, etc) is warranted given that hosts are commonly affected by more than one parasite species...., these interactions are expected to be strong and influence parasite community composition, and can have major impacts in host community stability and dynamics....

Indeed there are compelling reasons to think that co-occurring parasites may influence each other...

And the exclusion of parasites from this picture is surprising given how widespread parasites are and their effects on community dynamics ....

**I.2- Co-infection**

Hosts are usually infected by more than one species of parasite concurrently – inferring patterns from one host species and one parasite species often does not match dynamics of multiparasite infections - because one parasite species can potentially influence other species directly or indirectly, even if these parasites form “proper” communities. An important confirmation here is to test whether these associations of parasites occur at random or occur by a process that suggests non-random assembly (biotic/abiotic) - one straight forward way to test this is to first assess whether associations of 0, 1, 2 etc. parasite species simply match what would be expected from each parasite species prevalence. If these distributions occur by random is it still appropriate to look for infection diversity patterns? Potentially yes- but a correlation (div-inf) would suggest that is not a diversity effect but just a random event/statistical event...

Interactions among parasites can directly (e.g. competition for space) or indirectly (e.g. use of host resources [food], effects on host immunity) influence each other and lead to positive or negative correlations (i.e. facilitation or competition). Indeed, strong evidence for pairwise species interactions which can lead to negative, positive or neutral impacts on other species. Such interactions not need be symmetric, can be directional (e.g. species located higher in rabbit intestines influence species ‘downstream’) and the outcome may depend on the context in which it occurs (i.e. pair-wise outcome is not always the same in different contexts – e.g. Holmes1961 *J Parasitol*, 47, 209-216; 1962 *J parasitol* 48,87-96; 1962 *J Parasitol* 48, 97-100; move from rat to hamster change dynamics).

Macro vs. macro, macro vs. micro, micro vs. micro....

Other approaches have tried to establish whether parasite assemblages are determined by among parasite interactions – as opposed to random assemblages – by assessing whether naturally occurring parasite communities differ from null models of random assembly. These studies show that XXXXXXXXXXXXXXX

How does differences in the environment influences the parasite interactions? How does temporal events influence the interactions (priority effects?) How does the interactions change through time? How much does the point at which the parasites are studied changes the interpretation of the outcome (temporal/seasonal fluctuations)?

Nonetheless, these studies cannot establish generality or emergent patterns that go beyond either the descriptive [null vs. commu] or the idiosyncratic (pair-wise interactions).

Nonentheless – parasite-parasite interactions can be one of a multicausal framework leading to infection-parasite diversity patterns (as we discuss below).

When a parasites affect each other, these interactions can be uni- or bidirectional and symmetric or asymmetric. Multiple mechanisms have been proposed to explain these patterns. For example... (immunity, phylogenetic relations, cross-immunity, overpowering host immunity, location in similar resource/area of body – increased comp or decreased? Is there evidence for resource/niche partitioning among parasites?

What do we know about parasite diversity – what are the drivers and what are the patterns.... (Poulin 2004, *Basic and Applied Ecology*)

[[[[Where to fit]]] surprising given that the little we know of this patterns (Although evidence is very limited Wood + Johnston 2016 *J Parasit*) is that the relationship is positive for H richness and P Richness (Wood + Johnston 2016 J Parasit; Kamiya et al. 2014 *Ecography*) which suggest parasite diversity shifts YET most importantly that does not imply (or is causally linked) to changes in parasite infection (abundance, prevalence etc) which is the crucial parameter we are interesting when assessing infection (this is what causes disease, morbidty, pop and ecological effects). Thus the key question we need to ask is whether parasite diversity influences parasite abundance.

Issues with community vs. simply assemblages... many reasons why set of co-infecting species could have occurred at random - key point in discriminating whether patterns are an outcome of biotic/abiotic processes or simply an statistical indirect effect of (say parasite aggregation)

What are the main potential mechanism for patterns (random assemblage, interactions, temporal accumulation/loss)?

ARE these patterns the same or do they change for infra-community, constituent-community and parasite fauna. – Scale dependency [[[[Perhaps touch on this on the scale section]]]]

[[Belden & Harris 2007 – Continuous interactions between host focal microbiota/communities and pathogenic invaders – environmental change can alter those dynamics and lead to changes in disease outbreaks – i.e. community dynamics between pathogens and non-pathogens too]]]

Despite clear evidence for pair-wise interactions the influence of parasite diversity on epidemiological dynamics is rarely accounted for, and few papers attempt a discussion of whether higher level processes can be simply projected from pair-wise outcomes or have emergent properties.

**I.3- Importance and need for parasite infection-diversity**

Same way that predictions obtained from one-host and one-parasite models are limited in their ability to infer outcomes of multiparasite dynamics (evidence –papers), host impacts (evidence for differential host survival, morbidity, etc in response to single vs. multiparasite infection) and epidemics (change in disease transmission at the host pop level between single vs. multi parasite infection), limiting the consideration of disease diversity patterns to one parasite species dynamics among multiple hosts may fail to explain (at the adequate level) patterns and predict outcome of changes in hosts or ecosystem... and management strategies.

Potential importance of dilution effect working at this level too (already evidence that diversity in hosts at genetic, population, etc also has a detrimental effect on parasites... suggesting a “multiscale” process)

II- PARASITE INFECTION-DIVERSITY STATE-OF-THE-ART

**II.1- Patterns**

Few published papers directly addressing the question (despite hundreds of papers quantifying parasite species in/on individual hosts), that are amenable to measures of both parasite loads or presence in individual hosts and parasite diversity (e.g. richness and measures of evenness) at the individual and population levels.

Measures of div used

Hosts and parasites

Despite commonality of microparasites in host level dilution effects – diversity-infection at the parasite level has concentrated only in macroparasites...

Many causes of parasite component community diversity – ecosystem/habitat characteristics (e.g. size/island), host diversity, parasite/host vagility, host range, – community composition similarity (and scales of para community – this is most at the component level – presumably host driven or parasite driven interactions will be stronger at the infracommunity level – not to say they wont have an effect at other levels and vice-versa – but resources and interactions might be more limited within the host rather than on an open habitat: spatially more constrained and depleteable RR)

[some of these will make infracommunity diversity just a sampling outcome – important to check for those]

II.2- Expanding the breadth of studies

II.3- Looking further

III- MECHANISMS AND MULTICAUSAL EXPLANATIONS

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III.2- Host-driven processes

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V.1- What is missing?

V.2- Random assemblages effects vs. interactions

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ROAD MAP

SECTION I -Introduction

1- Infection-diversity and the host scale

Insights, Importance

Overlooked for parasite diversity (why?), despite extensive research on co-infection – paras affecting each other (directly/indirectly)

2- Co-infection

Insights, Importance ~ Useful but noisy

What is missing? What are the potential benefits of Para Div-Infection approach

How common is co-infection? Any numbers?

[[[[despite most hosts being infected by more than one species of parasites at a time.... still understudied]]]]

3- Parasite Infection-Diversity

What we know and what/why it matters.

Basic idea and how it can be meaningful, provide insights

SECTION II – Parasite inf-div known and new

4- Patterns

Potential outcomes, what the literature suggests (other’s work, host, parasite types)

Potential effects

What is missing in terms of further approaches (link to last section of future directions)

5- Methods/Results

Using the para inf-div approach to look for insight in new datasets (potentially answer some of the above questions)

Could parse datasets to establish some general patterns (or simply do all parsing below)

Conclusions about Patterns (Intro of ideas for following section)

6- How to approach these studies (datasets out there) [Unsure about position and breath of this]

What is needed and how to deal with it

(cut-offs, species numbers, diversity metrics (more below))

SECTION III – Drivers/Mechanisms/Explanations

7- Overview

Potential drivers explanations – what drives recurrent positive correlations

Highlight importance of mechanistic approach – not just describing the patterns (Pedersen & Fenton 2006 TREE)

Subsection

8- Host-drive processes

Behaviour; Immune mediated; Condition, Stress, Vigour

Tests

-Strength of immune response will depend on (indirectly) the type of immune response and the degree of cross-reactivity among parasite species (Pedersen & Fenton 2006 TREE)

Subsection

9- Parasite-driven processes

By type; Interactive/Additive nature of pair-wise species interactions, Random associations

Cross-immunity

Effects of prevalence / Abundance?

Tests

Subsection

10- Environmentally-driven processes

Variation in Quality/Exposure; Community Complexity;

Effects on community, or direct effects on stages exposed to environment

SECTION IV- Statistical artifacts and other influences

Subsection

11- Scale effects

Drivers of differences in Community and Spatial Scale

Subsection [??]

12- Statistical Artifacts

Potential causes of apparent but non-biologically driven patterns

Subsection

13- Effects of diversity metrics ~ interpretation

Diffs b/w Richness/Identity/Evenness – Worth trying more than one, do they mean different things?

What each says, how to interpret; matching vs. non-matching results – how/why?

SECTION IV

14- Future directions and open questions

Data mostly available from field – Experimental rarer and much needed to address mechanisms

What is needed and how it should look like (exp / field) [PERHAPS]

Basic theory blocks available but underused [How would this theory look like - ]

Reminder of multi-causal explanations

Random assemblages effects vs. interactions (it is really a community)

Linking co-infection (lack) of patterns to parasite diversity-infection patterns (averages? Emergent/interactions)

Linking infection-diversity patterns at the host and parasite levels (Thieltges/Johnson...)

Invasive hosts and parasites (Young et al.)

Is this a law of parasite ecology? (yes: widespread, recurrent / No: no single or even clear mechanisms)

Theory and evidence linking parasite and host diversity-infection interactions.

Multi-parasite-multi-host dynamics

XX- Conclusions

The positive association between parasite diversity and infection we find recurrently independently of host/parasite taxa, habitat type, ETC underscores the need to include more nuances when discussing diversity-infection patterns. The current trend is to portray diversity as having a negative correlation with disease, but –whether one agrees or disagrees with that view [Contentions about dilution effect] – it should be made clear that such a pattern is limited to host diversity [perhaps even more specifically to certain types of hosts –find papers suggesting this is only for competitors, etc].

----Yet, this approach has failed to integrate the nature of parasite infections – which are often multiparasite – and is known that predictions/dynamics of single host-single parasite dynamics are different from multi-parasite ones.... In parallel, the analogy of what is gained from considering the community of hosts to understand the patterns and dynamics of disease can be gained by considering the community of parasites.

Notes from Poulin 2007 + comments and ideas.

Perhaps, numerical responses which are the basis of the infection-diversity response are less likely to show negative correlations than positive ones. For instance...

Of course, numerical responses (i.e. changes in infection rates caused by changes in rate of stage change, reproduction, accumulation/establishment, growth, survival within the host) are not the only potential response of parasites to the presence of another parasite species – instances of functional responses in which parasites engage in site segregation (Holmes 1973 *Can. J. Zool*) which is equivalent to ecological character displacement (Poulin 2007 pg. 197). These functional responses may interact or be independent of numerical responses (the fitness cost of moving to a less desired location or resource use within a host should be lower than the cost of competing with another species), in some instances leading to weaker or no apparent interaction (positive or negative) among parasite species. Therefore in some instances the impact of negative interactions may be toned down by functional responses decreasing the likelihood of these interactions influencing parasite diversity-infection correlations which may lead to negative outcomes. [[[IS THE CONVERSE TRUE – I.E. DO PARASITES WITH POSITIVE EFFECTS TEND TO OVERLAP MORE WITH EACH OTHER – MAKE THEIR RELAIZED NICHE THE OVERLAP OF THEIR FUNDAMENTAL NICHES?]]]]

Still – in nature it could be an illusion of site segregation if parasites can only occupy and survive in sections of their fundamental niche not already occupied by other parasites – i.e. numerical impact that appears as functional response (Poulin 2008 pg. 201).

If numerical effects are extreme – leading to competitive exclusion [should test for this] then it is also likely that it would appear that positive diversity-infection patterns are more often found than negative ones.

Combes 2001 book –cited by Poulin as giving many examples of niche reduction caused by competition.

Poulin 2007 (book – pg.193) raises the issue that due to the commonly aggregated distribution of parasites among hosts, there is a low likelihood of having high-enough numbers of more than one species infecting a host to lead to significant direct competitive impacts (resource or space) (indirect perhaps more likely since host might respond to low number of parasites) – and therefore negative impacts from one parasite species on another should be less often seen than in pair-wise competitive scenarios from laboratory experiments.

Despite this Lello et al. (nature/science?) show that these negative, as well as positive interactions are possible, and large numbers of co-ocurring parasites do occur in nature.

Specialists and Generalists

Expect less numerical and functional effects on specialists relative to generalists (except if they are both specialists in same niche – perhaps spatially isolated (geography).

Might come from intense past competition or other origins but coincidentally prevented competition (Poulin 2007 pg. 203).

Core parasites (abundant + prevalent) are expected to have narrower niches and interact (or have interacted) more with other species in the past – thus may be or have been under stronger numerical effects (negative). Evolution of niche should select for lower overlap if costly. Whereas satellite species (less prevalent and abundant) are expected to be distributed more randomly within the host and overlap with other species more often – present at low abundance and do not occur regularly – interactions may not be important or have not produced strong selection leading to niche shifts (Poulin 2008 pg. 204 – REFS there) due to low freq of occurrence. Low infrapop sizes and recruitment rates. Satellite species – if niche restrictions, likely evolved independently of the other satellite species (i.e. not in response to interaction).

Strong selection against negative interactions and selection in favour of positive interactions. Should weaken signal of negative interactions – Either selection in past to reduce negative numerical responses or to in instances of co-ocurrence to reduce negative impact by functional response (e.g. relocation).

Opportunities for intense competition might also be rare (Poulin 2007 pg. 205) and site specialization might be to facilitate interspecific encounters and mating (Rohde 1991); highly aggregated distributions of monogeneans reduce likelihood of large infrapopulations of various species co-occurring.

A key questions is whether in those diverse infracommunities with high intensity of one parasite –how often the other para sp have high intensities (that could lead to competition or direct strong effects among parasites)? Perhaps if they are generally high richness but low intensity of other sps (GIVEN removal of focal, high Shannon’s might also occur for other sps having low abundances while focal has high abundance) – this situation could easily lead to lower likelihood (if the species compete at all) of negative relationships at the pair-wise level that could affect the div-inf level.

“Niche restriction often serves to increase the chances of encountering a potential mate in low-density infrapopulations).

When comp effects do occur – numerical effects are often assymetrical

Poulin (2007 pg. 231) richness and composition of parasite infracommunities more likely caused by random assemblage (stochastic processes).

Yet WE SUGGEST CAUTION in assuming positive infection-div correlation indicate facilitation (at least until further data is assessed) given that we don’t know whether positive correlations are more likely to be obtained than negative ones.

-Test for infracommunity richness frequency relative to null model – Poulin 2007 pg. 213 – Algorithm by Janovy et al. 1995

Poulin 2007 pg. 213 – When sp interactions cause assemblages to depart from randomness (freq of assemblages with a given number of sps) – Sp-poor infracomm more frequent than by chance may mean strong competitive interactions; positive interactions can generate species rich infracommunities (presence of sps facilitating recruitment of others). BUT could still be host driven – heterogeneous response to infection.

Look at Simberloff and Moore 1997 – Evenness indexes in parasite diversity can be misleading! From Poulin – Parasites of diff species in the same infracommunity can be of diff sizes, even for those in the same guild... SO estimates of numerical dominance are of no importance when sizes differ considerably [[[Important point to make!!]]]] Pay attention to issue of numerical abundance vs. biomass!! And make note in paper that results may be misleading if this is not taken into account. Yet for my diversity-infection is not as problematic for richness, when sizes are relatively similar, and when looking at prevalence.

If dilution is common at host level and if Johnson and Hoverman 201X are correct that dilution is also a general outcome of parasite interactions (as it would seem that people believe due to lots of negative interactions) one would expect that loss of hosts and increase in the particular parasite studied would lead to decreased parasasitism through parasite the potential evenness or richness outcomes of the focal parasite increased abundance/prevalence – understanding what are the outcomes of these multi-host multi-parasite communities is ultimately the goal – our research seems to suggest that on the contrary, we should expect to see increased parasite abundance if host species loss leads to increases in the focal parasite species.

Also unknown which effect is strongest – loss of parasite species (at least the rare ones) due to loss of host species (host richness - parasite richness) OR loss increase in certain species due to typical dilution?

Guegan & Hugueny 1994 – relationship between fish size and infracommunity richness (monogenean ectoparasites of tropical fish). Tested for community nestedness (some species always present – rarer ones are add on). HOW DO I TEST FOR NESTEDNESS?? Size heterogeneity could provide structuring force for parasite communities – Confirmed by Poulin & Valtonen 2001a (some fish species endoparasite communities – but not others). CORRELATION MUCH STRONGER IN PARASITE COMMUNITIES SHOWING NESTED PATTERN THAN IN THOSE NOT SHOWING IT!!(Poulin 2007 pg. 217) (SAME AS WITH DILUTION EFFECTS – AT HOST LEVEL). Nestedness seems to occur when parasite sps are accumulated gradually and predictably related to host size / age (Poulin 2007). Measure nestedness – standardized index C, Wright & Reeves 1992.

Can the fact that most infracommunities show a random assembly pattern (at least according to Poulin e.g. pg. 219) (about a third only showing nestedness or antinestedness – equal ratio of both +/- Poulin & Guegan 2000) mean that diversity-infection patterns are a fluke? Or does nestedness and inf-div patterns do not interact in a simple way? How would this interaction look? Can we have random assembly and still get a div-infection pattern?

Poulin 2007 pg. 220 – methods used to detect pair-wise associations (co-occurrence) between species are more likely to detect positive, rather than negative, associations (citing Vickery & Poulin 2002).

Perhaps our methods are more likely to detect positive div-inf relations than negative ones.

Reasons to be wary of pairwise associations (perhaps in particular positive ones) Poulin 2007 pg. 223 (citing Lotz & Font 1994, Bush and Holmes 1986a). Hard to evaluate generality of interspecific associations based on pair-wise patterns since they are biased by samples of low prevalence (towards positive or negative spurious associations) and require large infracommunity samples (>100).

Citation for “patterns of pair-wise species associations are not consistent in space (p.g.224 top). Pair-wise patterns are also inconsistent among different subsets of the host pop (e.g. age, sex). [!]

Lello et al. (2004) consistent associations among intestinal helminthes of rabbits (23 years) might be a rare rather than common outcome.

Poulin 2007 pg. 224 – “interspecific associations may only become established in host pops where parasite sps occur at relatively high abundances – then contribute to community structure”

Unstated ideas behind infracommunity structure (Poulin 2007 – pg.224) – Paras join infracommunity independently and randomly; initial establishment and subsequent survival determined by host responses, presence of other paras, priority effects, and strength of interspecific interactions. Can arrive in packets through consumption of intermediate host. Can happen to simple life-cycle species too – e.g. packet of eggs from dif species released in feces

Pg. 225 – “One process that could provide structure to infracommunities in definitive hosts following the stacking of source communities (packets) would be for strong interspecific interactions to override the acquired structure”! Seems to be rare. Citing Lotz et al. 1995 – low or very high survival of one species affects the integrity of the association transfer from intermediate to definitive host – association is always stronger in intermediate. ALSO “positive associations in intermediate hosts are more readily transferred to definitive hosts than negative ones.” Apparently positive associations in intestinal helminthes of vertebrates tend to be more common than negative ones pg. 227 (cites Lotz & Font 1991,1994). – To distinguish between communities structured at the intermediate or definitive host level looking at both levels is required.

There seems to be no evidence for predictable patterns in parasite community structure (perhaps this has been predominantly done through sp absence/presence and rarely via abundance (p.g 227).

Pg. 227- Typical infracommunity – one or few numerically dominant species (most individuals from them) and few to several species with a low number of individuals. NICE GRAPH AND LOTS OF LIT IN FIGURE ON FOLLOWING PAGE. Relative abundance (average of percentage of infracommunity of that species) by rank figure – steepness of curve reflects dominance of one species over others. Read Tokeshi 1999 book.

-Comparisons of pair-wise interactions and community structures that deviate from random assembly – suggest that parasite interactions may play a minor role in community structuring.

Pg. 231 – Citing Mouillot 2005: “...negative relationship between variability in total parasite biomass among infracommunities in a component commu, and the mean species richness of these infracommunities. The higher the richness the less variability in mean biomass ... HOW COULD THIS INFLUENCE THE DIVERSITY-INFECTION CORRELATION?? “Not necessarily associated with greater structure or predictability”. “High sp richness can produce more homogeneous infracommunities” (so Shannon should show this (?)). “Likelihood that community is influenced and structured by its species may depend on its productivity).

-Experiments are crucial because (p.g.232) “infracommunities can only be understood by examining the way in which new parasites are recruited”

-pg. 235 Relationship between parasite fauna richness and max component community richness is a saturating positive function – at least for helminthes (citing Kennedy & Guegan 1994). In contrast with non-saturating but positive (+/- 1/2) of infracommunity average richness by component community richness. Reasons (p.g. 234) for saturation – 1) limited available niches w/n host (yet no evidence that new species cannot be accommodated), 2) component communities unable to sample the whole sp pool of parasite fauna (cannot sample through entire range – parasites have non-overlapping ranges).

p.g. 235 – distance among component communities tends to be a good predictor of species composition similarity and species richness- but not universal phenomenon.

-p.g. 236-237 Increased mobility reduces effect of community similarity decay with distance (comparison sea vs. freshwater fish); but habitat preferences and specialized diets can lead to predictable community species composition (freshwater fish) along a broad range.

Parasite species colonizing ability can also be influenced not by host movement but by movement of intermediate, definitive or paratenic hosts (e.g. fish parasites transmitted by birds among water bodies – Esch et al. 1988). [IMPORTANT FOR STICKLEBACKS] Thus, allogenic parasites are more likely to have predictable communities whereas autogenic ones are less likely (stronger effect of community similarity with distance) – but some exceptions: parasites with migratory hosts such as eels or trout (p.g. 238).

- p.g. 238 Habitat characteristics can also be correlated with parasite component community richness, can be a cause or a spurious association. W/n same host species but among populations component community composition can correlate with physical characteristics of the environment. Larger water bodies have the potential to be colonized by more parasite species (extinction might be less likely, potentially more species of hosts too) and therefore should have higher component community richness. Example given of pH impact – snail intermediate hosts missing from low calcium ion concentration lakes and thus digenean parasites they transmit (Curtis and Rau 1980). Biological characteristics also matter (i.e. host richness).

- p.g. 238 Unionid fauna more diverse (linear) with increasing number of fish host species. But among localities within same river system the relationship is much weaker (citing Watter 1992).

-p.g. 239 The parasite communities of newly introduced species/ displaced communities are expected to be species poor.

-p.g. 240 As spatial scale increases, parasite species richness increases too BUT relative number of specialist species decreases (!) (from Barker et al. 1996). Specialists (which are few) are well represented across – whereas each component community only contains a few generalists generalists (non-specialists). Non-specialists might not spread to other component communities.

-p.g. 240 Role of habitat or host can be masked by stochastic events (chance colonizations / extinctions).

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