

1 *Running head:* Disease-mediated nutrient dynamics

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4 **Disease-mediated nutrient dynamics: coupling host-pathogen interactions with ecosystem**
5 **elements and energy**

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28 **Abstract**

29 Autotrophs play an essential role in the cycling of carbon and nutrients, yet disease-ecosystem
30 relationships are often overlooked in these dynamics. Importantly, the availability of elemental
31 nutrients like nitrogen and phosphorus impacts infectious disease in autotrophs, and disease can
32 induce reciprocal effects on ecosystem nutrient dynamics. Relationships linking infectious
33 disease with ecosystem nutrient dynamics are bidirectional, though the interdependence of these
34 processes has received little attention. We introduce disease-mediated nutrient dynamics (DND)
35 as a framework to describe the multiple, concurrent pathways linking elemental cycles with
36 infectious disease. We illustrate the impact of disease-ecosystem feedback loops on both disease
37 and ecosystem nutrient dynamics using a simple mathematical model, combining approaches
38 from classical ecological (logistic and Droop growth) and epidemiological (susceptible and
39 infected compartments) theory. Our model incorporates the effects of nutrient availability on the
40 growth rates of susceptible and infected autotroph hosts and tracks the return of nutrients to the
41 environment following host death. While focused on autotroph hosts here, the DND framework
42 is generalizable to higher trophic levels. Our results illustrate the surprisingly complex dynamics
43 of host populations, infection patterns, and ecosystem nutrient cycling that can arise from even a
44 relatively simple feedback between disease and nutrients. Feedback loops in disease-mediated
45 nutrient dynamics arise via effects of infection and nutrient supply on host stoichiometry and
46 population size. Our model illustrates how host growth rate, defense, and tissue chemistry can
47 impact the dynamics of disease-ecosystem relationships. We use the model to motivate a review
48 of empirical examples from autotroph-pathogen systems in aquatic and terrestrial environments,
49 demonstrating the key role of nutrient-disease and disease-nutrient relationships in real systems.
50 By assessing existing evidence and uncovering data gaps and apparent mismatches between

51 model predictions and the dynamics of empirical systems, we highlight priorities for future
52 research intended to narrow the persistent disciplinary gap between disease and ecosystem
53 ecology. Future empirical and theoretical work explicitly examining the dynamic linkages
54 between disease and ecosystem ecology will inform fundamental understanding for each
55 discipline and will better position the field of ecology to predict the dynamics of disease and
56 elemental cycles in the context of global change.

57

58 *Keywords:* autotrophs, feedbacks, global change, infectious disease, nitrogen, nutrient recycling,
59 pathogens, phosphorus, primary producers

60

61 **1. Introduction**

62 Host-pathogen interactions and elemental cycles are linked through a suite of direct and
63 indirect connections that span levels of ecological organization. Elemental resources such as
64 nitrogen (N) and phosphorus (P) can impact host chemistry as well as growth, birth, and death
65 rates, scaling up to determine population attributes or community composition, and shaping
66 infection patterns across scales (Dordas 2008, Aalto et al. 2015, Borer et al. 2016, Civitello et al.
67 2018). In turn, pathogens are consumers, limited by both the energy and nutrients provided by
68 their hosts (Smith 2007) and can alter the physiology, defenses, and ecological function of
69 individual hosts (Hatcher et al. 2012). Scaled up to host populations and communities, infection
70 can impact ecosystem-level nutrient pools and fluxes (Ruardij et al. 2005, Suttle 2007, Eviner
71 and Likens 2008, Preston et al. 2016, Fischhoff et al. 2020). These reciprocal processes occur
72 simultaneously, creating the potential for feedback loops; yet most studies focus on individual,
73 unidirectional relationships between elemental cycles and disease. However, the few studies that

74 have specifically focused on these reciprocal effects demonstrate that failing to account for the
75 feedbacks linking disease and nutrients may lead to fundamental shortfalls in ecological
76 predictions (Narr and Frost 2016, Borer et al. 2021).

77 Given the ubiquity of pathogens in nature, understanding the mechanistic links between
78 disease and ecosystem nutrient dynamics has the potential to fundamentally enhance two vibrant
79 sub-disciplines of ecology: disease ecology and ecosystem ecology (Borer et al. 2021). Disease
80 ecology is rooted in understanding the dynamics of host populations (Anderson and May 1979),
81 whereas ecosystem ecology, with its focus on elemental fluxes and pools, arose from
82 geosciences, oceanography, and limnology (Chapin et al. 2011). The disparate origins of these
83 lineages have exacerbated a persistent, disciplinary divide between ecosystem ecology and
84 disease ecology, hindering progress in understanding the role of the disease-ecosystem
85 relationships that span these disciplines (Ostfeld et al. 2008, Preston et al. 2016). Furthermore,
86 shifts in elemental cycles that range in scale from the local eutrophication of ecosystems to
87 global changes in nutrient availability (Bhaduri et al. 2000, Fenn et al. 2003, Ackerman et al.
88 2019) are occurring simultaneously with shifts in the spatial extent and prevalence of infectious
89 disease (Anderson et al. 2004, Jones et al. 2008, Fisher et al. 2012), yet we lack a general
90 understanding of the interactions that dynamically link these changes.

91 Because autotrophs play an essential role in the cycling of Earth's carbon and nutrients,
92 we examine disease-mediated nutrient dynamics in this context. We simultaneously consider the
93 broad suite of processes linking elemental cycling with host-pathogen interactions and disease,
94 the reciprocal effects of pathogens on elemental cycling, and the potential for dynamic feedback
95 loops connecting infection with resource supply and recycling. To do this, we develop a new
96 model of disease-mediated nutrient dynamics that combines approaches from ecological and

97 epidemiological theory (section 2), synthesizing past unidirectional approaches into a unified
98 framework. By focusing on the role of feedbacks, we examine how the coupling of disease and
99 nutrients has the potential to alter both disease and ecosystem dynamics. We then use this model
100 to guide a literature review examining the biology underpinning the links between disease in
101 autotrophs and elemental cycles (section 3), from sub-cellular processes to host communities.
102 We illustrate the role of nutrient supply in infection and infection in nutrient cycling using
103 examples from a broad range of autotroph-pathogen systems in aquatic (freshwater, marine) and
104 terrestrial (grassland, forest, agricultural) environments as well as a wide array of pathogen taxa
105 (viruses, fungi, bacteria) and strategies (e.g., specialists and generalists, biotrophs and
106 necrotrophs). Taken together, this synthesis provides a conceptual and mathematical framework
107 and identifies gaps and future directions for advancing understanding at the intersection of
108 disease and ecosystem ecology.

109

110 **2. Disease-mediated nutrient dynamics**

111 The disease-mediated nutrient dynamics framework unites two sets of processes that have
112 traditionally been studied separately (Figure 1): the bottom-up effects of environmental nutrient
113 availability on disease (Dordas 2008, Aalto et al. 2015, Borer et al. 2016, Civitello et al. 2018)
114 and the reciprocal, top-down effects of disease on ecosystem nutrient dynamics (Eviner and
115 Likens 2008, Preston et al. 2016, Fischhoff et al. 2020). Studying these relationships as
116 unidirectional processes is empirically convenient and a prerequisite to a broader perspective that
117 encompasses many processes linking disease and ecosystem function. However, pathogens and
118 nutrient availability can simultaneously impact host stoichiometry, growth rate, and mortality,
119 scaling up to generate dynamic impacts on nutrients as well as host populations and communities

120 (Vannatta and Minchella 2018, Borer et al. 2021). Thus, these concurrent effects can interact to
121 qualitatively alter the dynamics of hosts, pathogens, and nutrients.

122

123 **2.1 Rethinking consumer-driven nutrient recycling for autotrophs and disease**

124 Traditional models of ecosystem function emphasize the importance of autotrophs and
125 environmental microbes as the major biotic drivers of productivity, decomposition, and
126 elemental cycling (Rastetter and Shaver 1992). The concept of consumer-driven nutrient
127 dynamics (CND) has been built around the explicit recognition that consumer egestion and
128 excretion influence ecosystem N and P dynamics via effects on nutrient uptake by autotrophs and
129 via effects of nutrient availability on consumer performance (Sturner 1990, Elser and Urabe
130 1999, Atkinson et al. 2017). Importantly, the CND framework accounts for the mismatch
131 between the elemental content of autotrophs and the dietary needs of herbivores. In response to a
132 mismatch, the consumer will retain more of the element at lowest supply and will excrete more
133 of the element in excess relative to dietary needs, thereby altering the ecosystem-scale cycling
134 rates of elements (Sturner and Elser 2002). While the focus of the CND literature has been on
135 free-living consumers, with the recent addition of parasites that infect herbivores (Vannatta and
136 Minchella 2018), pathogens of plants represent an important group of consumers for elemental
137 cycling. In fact, the ubiquity of pathogens in nature and the degree to which infection can impact
138 host physiology and survival points to the importance of pathogens for mediating ecosystem
139 processes, including nutrient cycling, across Earth's environments (Suttle 2007, Sanders and
140 Taylor 2018).

141 Disease-mediated nutrient dynamics (DND), the broad suite of pathways linking host-
142 pathogen interactions with ecosystem-level storage and flux of nutrients like N and P, falls

143 within the scope of CND. However, pathogens differ substantially from free-living consumers in
144 terms of life history, movement patterns, physiology, nutrient acquisition strategy, and
145 relationship to the environment (with a host often serving as a pathogen's immediate
146 environment). Therefore, a shift to examine pathogens requires refocusing the CND framework.
147 Some components of the CND framework can be reframed by analogy. For example, pathogens
148 do not excrete elements in the same way as free-living consumers, although they typically have
149 higher N:C and P:C than their autotrophic hosts. This discrepancy can lead to differential
150 excretion of C relative to nutrients from infected hosts (Frenken et al. 2021). However, unique
151 characteristics of pathogen biology require a novel model structure that differs from CND
152 models. For example, infected hosts often continue to reproduce, a dynamic that differs from
153 victims of predation. Spatial considerations also differ between CND and DND. Many free-
154 living consumers move across space, potentially ingesting and egesting nutrients in different
155 locations (Capps and Flecker 2013). Plant pathogens typically only infect a single stationary
156 host, but pathogen populations can reproduce and disperse to 'consume' additional hosts on a
157 much faster timescale (i.e., during an epidemic). Thus, focusing on pathogen-host interactions in
158 a nutrient dynamic framework explicitly allows examination of rapid population-level responses
159 of the pathogen, reproductive contributions by infected hosts, and a sharpened focus on the
160 importance of pathogen biomass nutrients and C:nutrient excretions from infected hosts.

161 From an ecosystem perspective, pathogens of autotrophs are even more likely than
162 consumers to impact nutrient cycling. In particular, most autotrophic biomass does not come in
163 contact with free-living consumers (e.g., herbivores) (Chapin et al. 2011), whereas pathogens of
164 autotrophs are ubiquitous (Burdon and Laine 2019). The DND model developed here (section 2)
165 focuses on pathogens of autotrophs but is intentionally similar in structure to CND models (e.g.,

166 Elser and Urabe 1999, Atkinson et al. 2017), with most parameter meanings redefined by
167 analogy. For example, transmission in the DND model is analogous to ingestion (e.g., grazing) in
168 a CND model, with nutrient-dependence of transmission analogous to a shift in ingestion rate
169 with food quality or stoichiometric mismatch. However, the relative parameter values for DND
170 fall outside those describing free-living consumers, leading to new ranges of model dynamics
171 and empirical predictions. Even the relatively simple DND model analyzed here produces
172 surprisingly complex behavior (section 2, Figures 3-5). Taken together, the model and empirical
173 review of DND highlight the many ways in which host-pathogen interactions differ from
174 consumer-autotroph interactions, laying the groundwork for future empirical and theoretical
175 exploration (section 4).

176

177 **2.2 Dynamic model describing disease-nutrient feedback loops**

178 Feedback loops in dynamic models linking disease with elemental cycles have received
179 very little attention to date (but see Borer et al. 2021). Yet predicting the relationships between
180 disease and ecosystem function, particularly in a changing nutrient environment, requires explicit
181 attention to the interplay of these processes. To examine the dynamic effects that arise from
182 coupling infection in primary producers with ecosystem nutrient recycling, we developed a
183 simple dynamic model of disease-mediated nutrient dynamics (Figure 2A). Our framework
184 merges ecological modeling of nutrient-dependent population dynamics (logistic and Droop
185 growth) with epidemiological modeling (susceptible and infected compartments), building
186 conceptually from consumer-driven nutrient dynamics theory (Sternner 1990, Elser and Urabe
187 1999, Atkinson et al. 2017). The resulting model links disease and nutrient dynamics via
188 multiple pathways (Figure 2B).

189 In the disease-mediated nutrient dynamics (DND) model, host growth rate depends on
190 environmental nutrients. This dependence, in turn, impacts host tissue chemistry, susceptible
191 host population size, density-dependent pathogen transmission, and the resulting prevalence of
192 infection (Figure 1, arrow 1). Pathogens reduce host growth rate and increase host mortality rate,
193 impacting the rate at which nutrients are returned to the environment (Figure 1, arrow 2). Host
194 population dynamics are coupled with instantaneous environmental nutrient supply using a
195 Droop-like formulation (Droop 1973), in which the nutrient uptake rate increases asymptotically
196 with environmental availability (Table 1). Hosts grow logistically, limited by low internal
197 nutrient quotas at low environmental nutrients and by light availability at high environmental
198 nutrients (see similar approach in Loladze et al. 2000). Nutrient uptake rate depends on whether
199 or not a host is infected, reflecting empirical evidence for a wide range of autotrophs (Dordas
200 2008, Fones and Gurr 2017, Table 1).

201 We use this disease-mediated nutrient dynamics model (*DND model*) to examine the
202 consequences of disease-nutrient feedback loops for host population density, host phenotype (as
203 stoichiometric quota), disease dynamics, and environmental nutrient pools and fluxes. To
204 examine the role of feedback loops, we compare the DND model to a simplified model in which
205 host growth is independent of nutrient availability (*decoupled growth model*), thus allowing a
206 direct comparison of dynamics with and without a disease-nutrient feedback loop (Figure 2,
207 dashed lines). For illustration, we use parameter values describing a deciduous forest (Table 1;
208 see Borer et al. 2021 for parameterization details). However, the relative simplicity of this model
209 allows it to apply broadly to a wide array of autotroph-pathogen systems.

210

211 **2.3 Dynamics arising from disease-nutrient feedback loops**

212 Model simulations comparing decoupled growth to the DND model with disease-nutrient
213 feedbacks demonstrate the importance of this coupling for the dynamics of infection prevalence
214 and the distribution of nutrients between autotrophs and the environment (Figure 3). Infection
215 prevalence oscillates after pathogen introduction, with or without the model feedback (Figure
216 3A). In the decoupled growth model, these oscillations dampen, and prevalence quickly reaches
217 a stable equilibrium. In contrast, cycles of infection prevalence and environmental nutrients
218 persist in the DND model, although this feedback does not strongly impact the mean distribution
219 of nutrients between the environment and host populations (Figure 3B). In the same nutrient
220 environment, both *per capita* growth rates (Figure 3C) and organismal C:N stoichiometry
221 (Figure 3D) settle to lower equilibria in the DND model than the decoupled growth model, with
222 convergent oscillations persisting longer in the DND model. These examples clarify that the
223 disease-nutrient feedback is destabilizing relative to the decoupled growth model, particularly
224 under high nutrient conditions.

225 Environmental nutrient availability interacts with pathogen transmission rates to
226 determine host and infection dynamics. In Figures 4A and 4B, with disease mediated nutrient
227 recycling (DND model), R_0 increases with nutrients until host growth is no longer limited by
228 nutrients; at this point, host growth and infection become light-limited. However, with nutrient
229 recycling in the DND model, ecosystems with very low nutrient availability (e.g., oligotrophic
230 lakes) do not support a sufficient *per capita* host growth rate or reach sufficient host biomass to
231 sustain density-dependent pathogen transmission. When recycled nutrients affect host growth
232 rate (DND model), pathogens with low transmission rates require substantially more nutrient-
233 rich conditions to support the *per capita* host growth rate that sustains infection ($R_0 = 1$)
234 compared to pathogens with higher transmission rates. In contrast, for these same parameter

235 values in the decoupled growth model (Table 1), the *per capita* growth rate is always high
236 enough to sustain infection, even under low nutrient conditions.

237 With disease-mediated nutrient recycling (DND model), environmental nutrients also
238 interact with transmission rates to control cycling dynamics of infection prevalence (Figures 4C
239 and 4D). In particular, the DND formulation with a high transmission rate leads to cycling
240 dynamics at elevated nutrients, whereas the decoupled growth model does not cycle (Figure
241 4D). Like prevalence, nutrient availability impacts host dynamics differently at low and high
242 transmission rates (Figures 4E and 4F). At high transmission and elevated nutrients, both
243 susceptible and infected hosts in the DND model cycle, whereas neither transmission nor
244 nutrients induce cycles in the decoupled growth model (Figure 4F).

245 From an empirical perspective, these results suggest that infection prevalence, even
246 within the same host species or ecosystem type, could become destabilized, taking on an
247 extremely wide range of values at high ecosystem nutrient supply, with long time periods of
248 either high or low prevalence arising from small shifts in nutrient availability, as in the ‘paradox
249 of enrichment’ (Rosenzweig 1971). Similarly, while the pathogen generally benefits from greater
250 nutrient availability, these cycles also could lead to its stochastic extinction. Thus, this
251 exploration suggests that in a system with disease-mediated nutrient dynamics, empirical data
252 should reflect an increase in the basic reproductive number (R_0) and infection prevalence from
253 low to intermediate nutrient availability. However, under high transmission rates and high
254 nutrient availability, prevalence and R_0 may appear to become decoupled from environmental
255 nutrient supply due to complex dynamics arising from the disease-ecosystem feedback. Highly
256 transmissible pathogens also may shift between stable, endemic infections and large amplitude,
257 epidemic cycles, depending on the environmental nutrient supply (Figure 4D and 4F).

258 This model comparison also demonstrates how disease-mediated nutrient recycling
259 impacts mean host tissue chemistry, host growth rate and density, and infection prevalence. Host
260 tissues in the DND model exhibit lower stoichiometric C:nutrient ratios (Figure 3D) and *per*
261 *capita* growth is slower (Figure 3C) than for hosts in the decoupled growth model, even though
262 the assumptions and parameterization of the nutrient uptake and release rates are the same in
263 these models. These differences arise because of the nutrient limitation of growth in the DND
264 model. Nutrient limitation reduces *per capita* growth rates in the DND model compared to the
265 decoupled growth model, with convergence of *per capita* growth rates (Figure 4A & 4B) and
266 host density (Figure 4E & 4F) only under high nutrient supply, when nutrients no longer limit
267 growth. The models do not always converge, however. With high transmission rates in the DND
268 model, as nutrients increase, host densities begin to oscillate and, on average, hosts remain
269 limited by nutrients, even at high nutrient supply (Figure 4F). Because of this, host growth rates
270 remain lower than those in the decoupled growth model, even at high environmental nutrient
271 concentrations (Figure 4B). Thus, failing to account for dynamic feedbacks between disease and
272 nutrients can lead to substantially different predictions for both disease and nutrient dynamics.

273 We also used the model to examine nutrient dynamics as a function of disease
274 transmission (Figure 5). In particular, investment in defense modifies pathogen transmission,
275 often trading off with growth investment in autotrophs (section 3.1), and this investment can
276 covary with the stoichiometry of an organism (section 3.3). However, our model allows us to
277 decouple these effects to examine the independent influence of defense on pathogen dynamics
278 and nutrient recycling along a gradient of transmission success (Figure 2 “ β ”). The DND model
279 demonstrates that when transmission rates are very low, the pathogen cannot persist in the
280 environment, and all hosts remain healthy. When the transmission rate crosses the threshold for

281 the pathogen to persist ($R_0 = 1$) in the DND model, the C:nutrient content of individual host
282 tissues (Q_s^{-1}, Q_I^{-1}) declines with increasing transmission rate until the transmission is high
283 enough that the nutrient content no longer varies with further increases in transmission (Figure
284 5A). With increasing transmission, nutrient recycling in the DND model induces instability in
285 infected hosts, but this oscillatory behavior does not occur in the decoupled growth model.
286 Further, at low transmission rates, virtually all environmental nutrients are taken up by hosts
287 (N_E). However, with increasing transmission in the DND model, host nutrient content
288 experiences a bifurcation in which a small change in transmission leads to wide swings in
289 environmental nutrients (Figure 5B). In short, transmission rate – and any host defenses that
290 modify this rate – in the DND model controls host population density and stoichiometric
291 phenotype, both of which contribute to the distribution of nutrients among infected hosts,
292 susceptible hosts, and the abiotic environment.

293 While the simplest DND formulation leads to oscillatory dynamics associated with both
294 transmission and nutrient supply, small, biologically motivated changes can alter these dynamics.
295 For example, the addition of even a small amount of reproduction by infected hosts is stabilizing
296 in the DND model, causing the oscillation amplitudes to quickly dampen to stable equilibria
297 (Figure 6). While this reflects the biology of a wide range of pathogens (with the obvious
298 exception of complete infection by castrating pathogens, Clay and Schardl 2002, Hartmann et al.
299 2019), the CND-DND analogy breaks down here, qualitatively changing the dynamics of hosts,
300 infection, and nutrient recycling.

301 This model of disease-mediated nutrient dynamics is intentionally simple to clarify the
302 importance of feedbacks linking the dynamics of a host, an environmentally transmitted
303 pathogen, and elemental nutrients (Figure 2). Despite the model's structural simplicity, these

304 results demonstrate that feedbacks between disease and nutrients can generate a surprisingly
305 wide range of host elemental content and population dynamics, infection patterns, and
306 environmental nutrients, and these results diverge from dynamics lacking this feedback (Figure
307 3). Even this simple coupling causes nutrients to influence disease prevalence, the pathogen's
308 basic reproductive number, and host density (Figure 4). The rate of transmission (and host
309 defenses that may reduce this) influences the distribution of nutrients among organisms and the
310 abiotic environment (Figure 5), inducing instability where environmental nutrient supply (Figure
311 4) and transmission (Figure 5) are high.

312 In the following sections, we review a wide range of empirical autotroph-pathogen
313 examples ranging from sub-cellular to host community scales. Many of the necessary data do not
314 yet exist to fully link environmental nutrient supply with many of the dynamics uncovered in this
315 modeling exercise. However, we use this review, spanning a wide range of real systems, to
316 highlight key relationships between environmental nutrient supply and recycling, hosts, and
317 pathogens that converge in the DND framework (Figure 1).

318

319 **3. The cycle from nutrient supply to host-pathogen interactions and ecosystem nutrient** 320 **dynamics**

321 For pathogens infecting autotrophs, nutrient-induced changes to a host's phenotype may
322 impact a pathogen's infection cycle through a range of pathways (Figure 1, arrow 1). The DND
323 model simulations demonstrated that feedbacks and interactions among these changes can
324 control the dynamics of nutrient cycling and disease. As documented in the following sections,
325 plasticity in host growth rate, size, defense investment, and tissue elemental content may alter
326 pathogen replication based on the quality of the host as a resource, and nutrient-induced shifts in

327 host defense investment may alter host susceptibility, tolerance, or competence for a pathogen.
328 Evidence from many host-pathogen systems demonstrates that individual-level shifts in
329 characteristics such as growth rate, defense, or tissue elemental content, when examined across
330 host populations and communities, play a key role in pathogen transmission and disease
331 outcomes. Additional processes operating at the population and community scales alter infection
332 dynamics as a function of environmental nutrient supply.

333 Shifts in infection within hosts can, in turn, lead to impacts on nutrient dynamics,
334 particularly when the hosts are autotrophs, by altering individual host chemistry, physiology, and
335 demographic rates (Borer et al. 2021, Figure 1, arrow 2). Although infection is often associated
336 with elevated mortality, pathogens can alter ecosystem function via both sub-lethal, trait-
337 mediated effects or lethal, density-mediated effects (Preston et al. 2016, Fischhoff et al. 2020),
338 including decoupling elemental flows into and out of infected hosts (Frenken et al. 2021).
339 Because autotrophs sit at the nexus between the abiotic world of elements and energy and biotic
340 food webs, disease-mediated variation in host traits such as growth rate, defense, tissue
341 chemistry, and competitive ability, can control nutrient pools and fluxes through ecosystems
342 when scaled up to the level of host populations or communities. Here, we synthesize a broad
343 range of examples that, taken together with the DND model, point to the likelihood that nutrient
344 supply and nutrient feedback loops play an important role in the nutrient and disease dynamics of
345 natural systems.

346

347 **3.1 Growth rate and size**

348 All autotrophs share common biochemical machinery that requires N and P for growth,
349 metabolic functions, and reproduction (Sterner and Elser 2002). Because of this, we focus on

350 nutrient impacts to host growth rate (Figure 2, “Growth”). By influencing organismal and
351 species-level functioning, nutrient supply constrains species growth rates (Figure 2A, dashed
352 arrow), ultimately shaping the diversity and composition of host communities (Harpole et al.
353 2016). Thus, nutrient supply and recycling through death and decomposition are critical
354 processes fueling biological systems. The supply of growth-limiting nutrients can change host
355 phenotype by increasing organism size for unicellular autotrophs or increasing total biomass or
356 investment in specific tissues for multicellular autotrophs (Fay et al. 2015, Garcia et al. 2016).
357 For pathogens, size differences among hosts can represent variation in space for colonization or
358 replication (Kuris et al. 1980, Holfeld 2000, Rasconi et al. 2012). Metabolic rates, generally
359 declining with host body size (Makarieva et al. 2008), can set limits on rates of within-host
360 pathogen replication (Cable et al. 2007, Banerjee et al. 2017). However, there is some evidence
361 that cellular nucleotide content, which generally increases with host cell size (Machado et al.
362 2021), also may limit pathogen replication and the number of pathogen particles released into the
363 environment (Machado et al. 2021).

364 Infection also can interact with nutrients to generate countervailing effects on host growth
365 rate and size. In particular, infection can be energetically expensive for a host, feeding back to
366 slow host growth and metabolic rate and diverting energy and nutrients to other functions
367 (Berger et al. 2007), ultimately slowing population growth rate and reducing host density (Figure
368 4E & 4F). Reduced photosynthesis and increased respiration rate in response to infection leads to
369 reduced primary productivity in autotrophic hosts (Kohli et al. 2021), ultimately reducing
370 biomass. Following host death, this biomass becomes organic matter that eventually
371 decomposes, recycling carbon and nutrients.

372 The biology of a pathogen also can interact with plant growth and development to
373 determine impacts on nutrient recycling, and these impacts can vary with plant growth rate
374 (Häffner et al. 2015). Necrotrophic pathogens that derive nutrients from dead host cells, for
375 example, can manipulate hosts to speed development, inducing an earlier onset of senescence
376 (Mengiste 2012), which leads to more rapid nutrient recycling. Biotrophs, on the other hand,
377 derive nutrients from living host tissues, and infection can slow host senescence (Newton et al.
378 2010, Häffner et al. 2015). Hosts and their pathogens have a wide range of strategies to control
379 the signaling molecules that determine the host's metabolic rate and development, many of
380 which alter nutrient mobilization within living hosts and recycling following senescence
381 (Häffner et al. 2015).

382 Studies of pathogens and their autotrophic hosts provide evidence of host size and
383 growth rate impacting pathogens with concurrent impacts of pathogens on host size and growth
384 as a function of environmental nutrients. For example, experimentally elevated N supply to
385 grasses generally increases individual growth rate, which is subsequently associated with altered
386 viral titer, a measure of pathogen concentration within host tissues (Whitaker et al. 2015, Lacroix
387 et al. 2017). For this group of grass hosts and their barley and cereal yellow dwarf viral
388 pathogens, nutrients indirectly modify virus concentrations via effects on host size and growth
389 rate, and virus infection interacts with nutrients to modify host traits associated with growth (e.g.,
390 leaf thickness, Lacroix et al. 2017). This influence of nutrients on host-pathogen interactions may
391 be due to a general tradeoff between investment in growth and investment in defense. In
392 particular, crops are often bred to maximize growth and nutrient responsiveness while
393 minimizing investment in defense (Huot et al. 2014), which means that N supply to crops
394 generally enhances growth rate and size (Luo et al. 2020). Because of this tradeoff within and

395 among species in the use of nutrients for growth or defense, faster-growing, larger individuals
396 tend to experience more infection (Huot et al. 2014, Heckman et al. 2019). These relationships
397 are similar for algae (Holfeld 2000).

398

399 **3.2 Defense**

400 Although control of growth and senescence via signaling pathways is a key battleground for
401 autotrophs and their pathogens (Häffner et al. 2015), autotrophs also defend themselves from
402 pathogens and other consumers using both chemical and structural defenses, the production of
403 which depends on the nutrient environment (Lerdau et al. 1994, Chen and Ni 2011). By reducing
404 the transmission success of pathogens (Figure 2A “ β ”), investment in defense can slow nutrient
405 recycling by retaining nutrients in living tissue and reducing tissue or whole organism
406 senescence.

407 Both structural and chemical defense can vary as a function of the nutrient environment,
408 impacting transmission success. A host’s cell wall serves as a first line of defense against
409 invading pathogens, representing a less costly defense investment than host-induced cell or tissue
410 death (Underwood 2012). However, elevated environmental nutrient supply can reduce cell wall
411 lignin and cellulose, reducing disease resistance (Ogden et al. 2018). While cell wall thickness
412 can serve as a constitutive defense, cell walls also can be reinforced by papillae, an induced
413 structural defense that is rapidly laid down on cell walls in response to the sensing of a wide
414 range of fungal or bacterial pathogens (Ogden et al. 2018). While the general relationship with
415 environmental nutrient supply is unclear, plant tissue C:N regulation is related to papillae
416 formation (Maekawa et al. 2014). Thus, elevated nutrients likely cause reduced investment in
417 structural defenses, leading to increased infection and more rapid nutrient recycling.

418 Although nutrients such as N generally reduce physical defenses, elevated N supply can
419 increase defense-related enzymes, proteins, and gene expression in plants (Sun et al. 2020). For
420 example, N-rich oligopeptides such as microcystins, produced by cyanobacteria, may defend
421 against fungal pathogens (Rohrlack et al. 2013), and production of cyanobacterial toxins with
422 similar molecular structure can increase with higher relative N availability (Van de Waal et al.
423 2009). However, elevated N supply also has been shown to downregulate defensive chemical
424 production in terrestrial plant species ranging from crops like soybeans, grapes, and rice to trees
425 such as beech and Norway spruce (Sun et al. 2020). Importantly, investment in defense often
426 trades off with growth investment in autotrophs (section 3.1) and can covary with organismal
427 chemistry (section 3.3).

428

429 **3.3 Tissue chemistry**

430 From the perspective of pathogens, plant elemental composition, including stoichiometric
431 ratios among multiple elements, can represent host quality. In autotrophs, tissue chemistry can
432 vary, often responding to elevated environmental nutrient supply with uptake that shifts the
433 elemental composition of plant tissues (Figure 2, “ N_S ”). Ecological stoichiometry predicts that
434 imbalanced ratios between consumers, such as pathogens, and their host resources will decrease
435 consumer growth and fitness (Sterner and Elser 2002, Frenken et al. 2021). This prediction has
436 been tested extensively for herbivores, which are generally more homeostatic in elemental
437 composition than autotrophs (Hillebrand et al. 2009). While the effects of elemental imbalance in
438 pathogen-host interactions have not been studied as extensively (Sanders and Taylor 2018,
439 Frenken et al. 2021), these effects may be strong since most pathogens depend on their hosts for
440 all chemical resources. Additionally, the high growth rates of many pathogens relative to their

441 hosts correspond to high demands for elemental nutrients to support the synthesis of nucleic
442 acids and proteins (Sterner and Elser 2002, Clasen and Elser 2007).

443 Increased host N or P content or reduced C:N or C:P may directly alleviate nutrient
444 limitation of pathogen population growth via direct use of stored host nutrients (Fatima and
445 Senthil-Kumar 2015). Alternatively, host nutrient uptake may indirectly benefit pathogen
446 reproduction via macromolecules produced by the host (Sun et al. 2020) or via increased host
447 investment in growth, providing the cellular machinery also required for some pathogens (e.g.,
448 viruses) to replicate (Smith 2007, Cuomo et al. 2012). In one example of P supply stimulating
449 rapid pathogen growth, a *Chlorella* virus infecting green algal hosts (*Chlorella*) benefited from
450 decreased host C:P (Clasen and Elser 2007). In this case, P supply and host C:P limited virus
451 replication and assembly. More recently, both N- and P-limitation of algal growth have been
452 demonstrated to reduce viral replication within their phytoplankton hosts by up to 90% (Maat
453 and Brussaard 2016). N supply limits growth of fungal pathogens infecting autotrophic hosts as
454 wide-ranging as cyanobacteria (Frenken et al. 2017b) and grasses (Mitchell et al. 2003),
455 clarifying that the impact of a change in the C:N:P nutrient environment on infection depends, at
456 least in part, on the stoichiometric requirements of – and mismatch between – hosts and their
457 pathogens (Frenken et al. 2021).

458 Although nutrients that increase host growth rate can increase pathogen reproduction
459 (also see section 3.1), the nutrient environment limiting pathogen growth also can differ from
460 that limiting host growth. For example, in the experiment with a virus and *Chlorella* algal hosts,
461 even though the host growth rate and reproduction were independent of P supply, the increase in
462 the P content of cells due to algal elemental plasticity alleviated the pathogen's nutrient
463 limitation, leading to increased pathogen replication (Clasen and Elser 2007). Similar effects

464 occurred in a grassland field experiment, where N addition increased grass-host biomass
465 accumulation, but P addition increased the prevalence of a viral pathogen in these hosts (Borer et
466 al. 2010).

467 Pathogens can stimulate host nutrient uptake (Figure 2A, “ N_I ”) while simultaneously
468 competing with their host for these resources. These interactions between infection and nutrient
469 supply can alter autotrophic host tissue chemistry and decomposition rates, ultimately regulating
470 the storage and recycling of nutrients in ecosystems. When a host’s growth-limiting resources are
471 depleted by pathogen infection, this can lead to reduced host growth, lifespan, and lifetime
472 reproduction (Smith and Holt 1996, Smith 2007). For example, infection by a phloem-limited
473 virus of grasses reduces nutrient concentrations (N, Mg, Ca) of crop leaves (Riedell et al. 2007).
474 For stoichiometrically-driven host-pathogen interactions, elevated CO₂ can increase host
475 C:nutrient ratios, potentially exacerbating the impact of infection on growth and reproduction
476 (Mitchell et al. 2003). For example, mortality of European beech trees infected with an oomycete
477 was substantially increased under elevated CO₂ and low N conditions, when C:N was very high
478 (Fleischmann et al. 2010). However, at ambient CO₂, survival was high under all N conditions.
479 Analogous to CND (Sternner 1990, Elser and Urabe 1999, Atkinson et al. 2017), higher
480 requirements of N or P of the pathogen relative to host tissue may lead to greater retention of
481 these nutrients in infected biomass (e.g., compare red and black lines Fig. 3D), thereby
482 enhancing limitation of these nutrients and possibly increasing the recycling of non-limiting
483 nutrients and C. Many empirical studies have found infection-induced increases in host tissue
484 nutrients, ranging from increased N content in response to oomycete infection in bay laurel and
485 European beech (Fleischmann et al. 2002, Wang et al. 2003), to increased N (Borer et al. 2015)
486 and P content (Rúa et al. 2013) in response to viral infection in grasses. Importantly, these

487 chemical signatures of infection can persist past death, influencing litter chemistry,
488 decomposition (Cobb and Rizzo 2016), and nutrient recycling (Hobbie 2015).

489

490 **3.4 Variation within and across host populations**

491 The effects of nutrient supply can scale up, modifying the population size of an
492 autotrophic host species (Figure 1, Host population) via changes in growth rate (section 3.1,
493 Figure 2A, dashed arrow), tissue chemistry (section 3.3, Figure 2A, “ N_s ” and “ N_I ”), and
494 investment in defense (section 3.2, Figure 2A, “ β ”), all of which can interact with infection.
495 Since transmission of many pathogens requires host density to exceed a minimum threshold
496 (Anderson and May 1981), population density, *per se*, can impact infection dynamics (Burdon
497 and Chilvers 1982). Importantly, small host populations that increase due to nutrient supply,
498 crossing this threshold, become vulnerable to the spread of infections (Figure 4A & 4B, DND
499 model). Within a host population, transmission heterogeneity, arising from variation in host
500 characteristics, such as age, nutrition, defense, or genetics, can amplify (or slow) disease
501 transmission within or among populations. While transmission heterogeneity has received far
502 more attention in animal hosts (Lloyd-Smith et al. 2005, Paull et al. 2012), experimental work
503 with the oomycete pathogen, *Phytophthora ramorum*, has uncovered evidence of substantial
504 genetic variation in infection susceptibility among bay laurel host individuals (Anacker et al.
505 2008).

506 Infection can, of course, feed back to reduce host population size via reduced
507 reproduction or increased mortality (Burdon 1991), subsequently affecting rates of nutrient
508 recycling. For multicellular autotrophs, the impacts of infection on reproduction can be both
509 indirect and direct. Host population size can be reduced indirectly when infection reduces growth

510 and investment in reproduction. For example, infection by viruses in the barley and cereal yellow
511 dwarf virus group reduces biomass, the number of inflorescences, and seed production in a wide
512 range of crops and wild grasses (Malmstrom et al. 2005a, Riedell et al. 2007, also see section
513 3.1). However, some pathogen groups attack anthers (Falloon et al. 1988, Hartmann et al. 2019)
514 or seed heads (Alderman et al. 1998, Clay and Schardl 2002), directly reducing lifetime
515 reproduction and survival of their hosts. Both direct and indirect impacts of infection on host
516 population growth can interact with environmental nutrient supply to control host population size
517 in a wide range of autotrophic hosts (Alexander 2010).

518 Infection also can impact nutrient dynamics at the population scale through impacts on
519 mortality. Perhaps the most well-known example of infection-induced nutrient recycling is that
520 of viruses in marine systems, where estimates suggest that about 20% of all microbial biomass is
521 killed by viruses daily, controlling biogeochemical cycling in oceans (Fuhrman 1999, Suttle
522 2005, 2007). In both freshwater and marine environments, infection by chytrid fungi also can
523 cause mass mortality of phytoplankton, reducing or terminating algal blooms, and playing a
524 major role in the recycling of carbon and nutrients (Frenken et al. 2017a). In terrestrial systems,
525 mass mortality in forest stands from fungal and oomycete infections also can alter nutrient
526 dynamics. For example, in Hawai'i, a ceratocystis fungal infection first reported in 2010 has
527 already killed hundreds of thousands of *Metrosideros polymorpha* trees, the most abundant
528 native tree species in the Hawaiian Islands (Barnes et al. 2018). Historically, chestnut blight
529 (*Cryphonectria parasitica*) led to an almost complete loss of the iconic American chestnut
530 (*Castanea dentata*), declining from a cover of 36% across eastern North America to less than 1%
531 (Elliott and Swank 2008). As in aquatic systems, forest mortality from infection can have
532 substantial consequences for nutrient and carbon recycling (Matson and Boone 1984, Hobara et

533 al. 2001, Cobb et al. 2013). For example, infection by the oomycete pathogen *Phytophthora*
534 *ramorum* can increase forest litterfall mass by 1-2 orders of magnitude, and decomposing
535 litterfall from infected hosts can increase soil N availability (Cobb et al. 2013, Cobb and Rizzo
536 2016).

537

538 **3.5 Variation among species in a community**

539 By altering the competitive environment for autotrophs, nutrient supply can induce turnover in
540 local species composition (Huberty et al. 1998, Cleland and Harpole 2010, Harpole et al. 2016,
541 Lehtinen et al. 2017), shifting the relative abundance of hosts and non-hosts (Figure 1, Host
542 community). Autotroph diversity can amplify or reduce community-wide disease risk, depending
543 on the characteristics of the community members (Keesing et al. 2006, Seabloom et al. 2018).
544 Experiments simultaneously manipulating host richness and nutrient supply have demonstrated
545 that the richness, composition, and relative abundance of species can be an even stronger
546 predictor of fungal infection severity than nutrient supply or foliar nutrient content (Mitchell et
547 al. 2003, Cappelli et al. 2020). Similarly, a study in a Tibetan grassland found that fertilization
548 increased the community-wide pathogen load primarily via host compositional change; disease
549 susceptible host species tended to be favored by fertilization whereas more resistant species were
550 extirpated from the community (Liu et al. 2017). Pathogen life history (biotroph vs necrotroph)
551 and host specialization also are key predictors of host-pathogen interactions and responses to
552 nutrients in multi-species communities (Woolhouse et al. 2001, Woolhouse et al. 2005, Keesing
553 et al. 2006, Moury et al. 2017, Liu et al. 2020). Thus, in addition to individual-level responses to
554 nutrients (sections 3.1-3.3), pathogen biology and the impact of nutrient supply on the richness,

555 abundance, and identities of host and non-host species will jointly determine the impact of
556 nutrients on disease dynamics in communities.

557 Pathogens also can control host community composition and nutrient recycling through
558 differential impacts of infection among community members (van Donk and Ringelberg 1983,
559 Hennes et al. 1995, Mordecai 2011). When these interactions alter the dominant traits of species
560 in a community, they are most likely to impact ecosystem-scale processes, such as nutrient
561 cycling (Litchman et al. 2015). For example, an infection-induced reversal in competitive ability
562 has been implicated in the community trait shift in California grasslands from domination by
563 perennial grasses to domination by lower biomass and C:nutrient annual grasses (Malmstrom et
564 al. 2005b, Borer et al. 2007). This compositional shift dramatically reduced ecosystem-scale soil
565 carbon storage (Koteen et al. 2011) and increased soil N availability (Parker and Schimel 2010).
566 Differential parasitism by chytrids in plankton communities can alter community composition,
567 speeding nutrient recycling by causing higher mortality in large, inedible algal cells (Holfeld
568 2000) and species (Rasconi et al. 2012). Similarly, species differences in pathogen-induced
569 mortality in mixed forest stands increased nutrient recycling via changes in community
570 composition (Metz et al. 2012), litterfall mass and chemistry, and soil N availability (Cobb et al.
571 2013).

572

573 **4. A case study of virus-mediated nutrient dynamics in marine phytoplankton**

574 While our model (section 2) and biological examples (section 3) suggest that the feedback loops
575 integral to disease-mediated nutrient dynamics are likely general and dynamically important,
576 systems for which we have the data to paint a more holistic view of these feedbacks remain rare.

577 Here, we synthesize work on marine phytoplankton and viruses to illustrate disease-mediated
578 nutrient dynamics and feedbacks in one system-focused example (Figure 7).

579 Marine phytoplankton, responsible for half of Earth's primary production (Field et al.
580 1998), play a key role in global biogeochemical cycles, and viral infections modify these cycles,
581 from manipulation of individual algal host metabolism (Monier et al. 2017) to extensive
582 mortality terminating massive algal blooms (Suttle 2005). With increasing N and P supply
583 (Figure 7, arrow 1), more virus particles are produced per host (increased burst size) and the time
584 from infection to host cell lysis (latent period) is reduced (e.g., Maat and Brussaard 2016).
585 Infected hosts leak and excrete organic matter even before cell lysis, altering elemental cycling
586 (Figure 7, arrow 2, Sheik et al. 2014). Viral infection also increases nutrient uptake rates (Figure
587 7, arrow 1), shifting the quantity and ratio of carbon and nutrients released from infected cells
588 upon lysis (Figure 7, arrow 2, Monier et al. 2017).

589 These effects of infection on the physiology and mortality of marine phytoplankton cells
590 can control population and community dynamics, scaling up to impact ecosystem nutrient
591 recycling (Figure 7, feedback to arrow 1). For example, field studies of blooms of the dominant
592 algal species, *E. huxleyi*, found up to 50% of the population was infected by viruses (Brussaard
593 et al. 1996), and 25-100% of mortality could be attributed to infection (Bratbak et al. 1993).
594 Because many of these viruses have narrow host ranges, viral-mediated mortality also can play
595 an important role in the turnover of traits and species succession (Brussaard 2004). At the
596 community scale, infection-induced shifts in traits such as growth rate, size, and tissue chemistry
597 impact the cycle rate and amplitude as well as the regional nutrient recycling feedback
598 (Brussaard 2004, Litchman et al. 2015).

599

600 **5. Emerging themes and future directions for disease-mediated nutrient dynamics**

601 The DND model (section 2, Figure 2) illustrates the impact of disease-ecosystem feedbacks,
602 highlighting the ways in which model dynamics change with host and pathogen traits and with
603 shifts in the abiotic nutrient environment. Despite the relative simplicity of the DND model (e.g.,
604 nutrient-independent transmission rates, similar nutrient uptake dynamics by infected and
605 uninfected hosts), this formulation and parameterization exhibit surprisingly complicated
606 dynamics (e.g., Figures 3-6). While the DND model highlights the exciting dynamical
607 consequences of disease-mediated nutrient recycling for both disease and ecosystems, taken
608 together with the biological examples, a range of important gaps and future directions are
609 brought into focus.

610 We focus here on the dynamics – and shifts in dynamics – that arise from the structure,
611 assumptions, and parameterization of the DND model, and use these to plot a path forward for
612 the exploration of disease-mediated nutrient dynamics in both theory and empirical systems. As a
613 starting point, the nutrient feedbacks that arise from consumer-resource interactions in both the
614 DND and CND frameworks generate dynamics not seen in unidirectional models (section 2,
615 Elser and Urabe 1999, Atkinson et al. 2017). State transitions are possible where stable equilibria
616 shift to sustained oscillatory dynamics with very small changes in environmental nutrients. In
617 addition, the large cycles in host density, pathogen prevalence, and environmental nutrients only
618 occur when nutrients are recycled, and these cycles also are damped by the addition of small
619 amounts of reproduction by infected hosts even when nutrient recycling is included. The ability
620 of infected hosts to contribute offspring to the host pool is a key biological difference between
621 the CND framework, with consumption and death of prey, and the DND framework, with
622 infection of living hosts. The simple DND model structure thus highlights the role of the

623 feedback loop in the system dynamics, increasing mechanistic understanding (Rastetter 2017).
624 These dynamic dependencies on small changes in environmental nutrients or small, biologically
625 motivated, departures from the CND model structure also open the door to a new set of
626 knowledge gaps and new questions for nutrient dynamics in empirical host-pathogen systems
627 and the theory of disease-mediated nutrient dynamics.

628 From an empirical perspective, the predicted cycles also are an important area of focus.
629 Despite the ubiquity of infection, the large magnitude cycles of hosts, infection prevalence, and
630 environmental nutrients predicted by the simple DND model are not frequently documented in
631 natural systems. This apparent mismatch may reflect the general absence of DND-mediated
632 cycles, the importance of additional biological relationships that are not included in this initial
633 model, or may reflect empirical observations that miss key points in temporal dynamics or
634 average across spatial dynamics. While infection can reduce the total mass (Seabloom et al.
635 2017) and carbon flux rates (Kohli et al. 2021) at the scale of autotroph communities, and can
636 increase environmental nutrients (Cobb et al. 2013), the links between pathogens of autotrophs
637 and temporal cycling of environmental elements are rarely documented. The review of the
638 biology of host-pathogen systems (section 3) points to a wide array of deviations from the simple
639 DND model structure that may be necessary to predict the dynamics of specific empirical
640 systems. Whether these pathogen-mediated nutrient cycles are rare or simply undocumented, the
641 uncertainty about the relationship between the model predictions and empirical dynamics of
642 DND uncovers gaps in knowledge and points to promising future research directions.

643 As a starting point, the DND model made the simplifying assumption of a single pathogen
644 species infecting a single host species. While building from simple CND models and providing
645 an important starting point for redefinition by analogy of the CND plant-consumer parameters to

646 host and disease definitions (section 2), this assumption ignores the multi-species context of
647 ecological systems (section 3.5) and the potential for important dynamic consequences. For
648 example, in a system with two species, there is the potential for compensatory dynamics (Holt
649 and Pickering 1985) which could allow the epidemic-stimulated release of nutrients from a
650 highly susceptible host species to be rapidly taken up by a less susceptible (or non-host) species,
651 damping the cycles of environmental nutrients. Even within species, if genetic variation exists
652 that favors a subset of host or pathogen genotypes under elevated nutrients, this could also
653 generate compensatory, stabilizing dynamics with ecological and evolutionary implications
654 (Alexander 2010, Burdon and Laine 2019, French and Holmes 2020). Insect vectors of
655 pathogens may differ in nutritional requirements from the pathogens they carry, introducing a
656 new set of stoichiometric constraints on uptake, excretion, and cycling of nutrients (Borer et al.
657 2010). Pathogens interacting within hosts also may modify host population dynamics, potentially
658 damping or exacerbating cycles (Seabloom et al. 2015). Although not captured in the simplest
659 DND formulation presented here, single host species are frequently infected by multiple
660 pathogens, sometimes highly related (e.g., co-infection by pathogens in the same genus) and
661 sometimes distinctly different (e.g., biotrophic and necrotrophic pathogens). Nutrients also can
662 concurrently increase pathogens while reducing mutualists (e.g. mycorrhizal fungi), shifting
663 nutrient uptake and supply to a host and its pathogens (Lekberg et al. 2021). Further, although
664 we focused here on infection of autotrophic hosts because of their enormous importance for
665 global biogeochemical cycles, models of DND could readily be expanded to examine the
666 dynamic consequences of pathogens at higher trophic levels (Vannatta and Minchella 2018).
667 Thus, the consequences of the community context of host-pathogen interactions for nutrient
668 dynamics represent a rich future direction. The conditions under which these new model

669 structures would dampen or eliminate cycles will require new model development, empirical
670 parameterization, and analysis.

671 Although the simple DND model presented here assumes homogeneous mixing, species and
672 nutrients are not homogeneously mixed, even in most aquatic systems, thus raising the important
673 consideration of spatial variation modifying the predicted dynamics. The stability of host-
674 pathogen interactions is strongly influenced by spatial structure, relative dispersal distances, and
675 connectivity of host and pathogen populations (Thrall and Burdon 1997). A careful empirical and
676 theoretical exploration of the conditions under which factors such as spatial heterogeneity of
677 nutrient availability, system mixing, vector movement, or relative dispersal distances of hosts
678 and pathogens determines the stability or cycling of environmental nutrients would advance
679 understanding of DND.

680 In addition to the dynamic consequences of nutrient-dependent host growth examined here,
681 our biological review revealed other key rates that depend on nutrients. For example, host
682 defense, one component of pathogen transmission, varies as a function of environmental nutrient
683 availability (section 3.2). The rate of host nutrient uptake also can decline or increase, depending
684 on the host and type of infection (section 3.3), pointing to an additional area warranting
685 exploration because of its potential impact on the pool and cycling of environmental nutrients.
686 Host survival can be prolonged or shortened depending on a combination of pathogen traits (e.g.,
687 biotrophic vs necrotrophic) and nutrient availability (section 3.1), suggesting the potential for
688 higher-order interactions to modify the resulting dynamics. Addition of nutrient dependence to
689 the model rates could change whether, and the conditions under which, oscillatory behaviors
690 occur.

691 While the host-pathogen biology reviewed here suggests many opportunities for expanding
692 the DND model, the DND model points to interesting possibilities for examining dynamics in
693 empirical systems, as well. For example, the large cycles of infection prevalence predicted at
694 high nutrients raise the question of whether, like the paradox of enrichment (Rosenzweig 1971),
695 high resource availability could potentially lead to stochastic extinction of the pathogen because
696 cycles, including extremely low numbers of infected hosts, could potentially reach zero. If so, the
697 system would be fundamentally rewired to exclude the pathogen, leaving only an autotroph and
698 its nutrient resources, thus inducing stability. Typically, host resources are assumed to either
699 benefit or hinder pathogens (section 3.3), depending on the relative impacts of host resources on
700 host growth, density, and immunity, as well as pathogen spread (section 3). The cycles of
701 infection prevalence that arise at elevated nutrient availability when we include nutrient
702 recycling (section 2) suggest that there may be situations where lower levels of nutrient
703 availability could increase prevalence, but higher nutrient availability could ultimately cause
704 pathogen loss from the system via stochastic extinction. Empirical tests of these predictions
705 would advance our understanding of the ways in which disease and ecosystems will respond to
706 ongoing environmental change.

707 Although some of the DND model dynamics play out over very long time periods, we lack
708 long timescales of field data to quantify the linkages and importance of disease and nutrient
709 cycling. Long-term data will be particularly important for understanding the role of DND in
710 long-lived hosts (Borer et al 2021). Multi-year datasets to understand the mechanisms underlying
711 these processes also will fill a critical gap, particularly in seasonal systems, where nutrients
712 pulsed into systems early in a season stimulate autotroph growth and pathogen build-up, with
713 increasing importance through a season (Kagami et al. 2007). For all ecosystems, however, long

714 term sampling will be important for understanding the role of DND in because of directional
715 global changes, especially long-term increases in background nutrient inputs to ecosystems
716 (Ackerman et al. 2019).

717

718 **6. Conclusions**

719 Disease and ecosystem nutrient dynamics are clearly linked in biological systems via
720 multiple pathways. Although most work to date has treated these relationships as unidirectional
721 processes in which nutrients impact disease dynamics or infection alters nutrient dynamics,
722 integration of these approaches to explicitly include the bidirectionality and simultaneity of
723 disease-nutrient links demonstrates the potential for feedback loops and emergent dynamics. By
724 distinguishing the broad suite of processes that link disease with ecosystem nutrients and
725 capturing the dynamic effects of biological differences between free-living consumers and
726 pathogens, the DND framework bridges disease and ecosystem ecology and opens new areas of
727 inquiry for both.

728 The directions suggested by the biology and ecology of hosts and pathogens provide a
729 rich area for both theoretical and empirical exploration of disease-mediated nutrient dynamics.
730 Many potential elaborations on the structure of DND models to better reflect real systems are
731 likely to have dynamic consequences. For example, variation in heterogeneity – e.g., among host
732 and pathogen individuals and species, among spatially distinct host populations, and among
733 species in host communities – and nutrient-dependent rates – e.g., transmission, mortality – are
734 likely to be fruitful areas of inquiry for reconciling apparent differences between modeled and
735 observed dynamics. Additional work to generate a more mathematically robust identification of
736 the necessary and sufficient conditions for sudden shifts in stability and analytically verifying the

737 existence of stable limit cycles will deepen our understanding of the mechanisms underpinning
738 state transitions in DND models. Investigation of model dynamics across parameter ranges
739 reflecting autotrophic hosts from phytoplankton to trees and the equally wide range of pathogen
740 traits will advance understanding of the importance of absolute and relative parameter values.
741 This type of biologically motivated sensitivity analysis could identify the systems and conditions
742 most likely to exhibit the predicted variation in host, pathogen, and nutrient dynamics. Long-
743 term field sampling and experimental work aimed specifically at creating the conditions to test
744 model dynamics also promise to advance knowledge spanning disease and ecosystem ecology.

745

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749

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1113

1114 **Table 1:** Disease-mediated nutrient dynamics model with modifications including population
 1115 growth with contributions from infected autotrophic hosts and the decoupled growth model.
 1116 Parameter values are based on a deciduous forest, see (Borer et al. 2021) for details of parameter
 1117 estimation.
 1118

Model Equations			
$\frac{dS}{dt} = r \left[1 - \frac{S+I}{\min\left\{\frac{Kr}{r-\delta}, \frac{N_S(S+I)}{qS}\right\}} \right] S - \beta SI - \delta S$			Susceptible host density (g C/m ²)
$\frac{dI}{dt} = \beta SI - [\delta + v]I$			Infected host density (g C/m ²)
$\frac{dN_S}{dt} = u(N_E)S - \frac{N_S}{S}\beta SI - \delta N_S$			Nutrients in Susceptible hosts (g N/m ²)
$\frac{dN_I}{dt} = u(N_E)I + \frac{N_S}{S}\beta SI - [\delta + v]N_I$			Nutrients in Infected hosts (g N/m ²)
$\frac{dN_E}{dt} = -u(N_E)S - u(N_E)I + \delta[N_S + N_I] + vN_I$			Nutrients in environment (g N/m ²)
Stoichiometric Quota Expressions			
$Q_s = \frac{N_S}{S}$			Susceptible host N:C ratio (g N/ g C)
$Q_I = \frac{N_I}{I}$			Infected host N:C ratio (g N/ g C)
Parameter/Function	Meaning		Value
r	maximum growth rate		0.0754/year
K	C dependent carrying capacity		22 Kg C/m ²
q	minimum host N:C ratio		1/439 g N/ g C
δ	C natural death rate		0.0412/year
v	C diseased induced death rate		0.01/year
β	transmission rate		1.2×10^{-5} m ² /g C/year
α	maximum N:C uptake rate		3.8147×10^{-4} g N/g C/year
κ	N:C uptake half saturation constant		0.009 g N/m ²
$u(N_E)$	Nutrient uptake function		$\frac{\alpha N_E}{\kappa + N_E}$
σ	reduction in growth rate for Infected		0-1
Decoupled growth model: replace S equation in system above			
$\frac{dS}{dt} = r \left[1 - \frac{S+I}{\frac{Kr}{r-\delta}} \right] S - \beta SI - \delta S$			
Model Equations with Infected growth: replace S equation in system above			
$\frac{dS}{dt} = r \left[1 - \frac{S+I}{\min\left\{\frac{Kr}{r-\delta}, \frac{N_S(S+I)}{qS}\right\}} \right] S + \sigma r \left[1 - \frac{S+I}{\min\left\{\frac{Kr}{r-\delta}, \frac{N_I(S+I)}{qI}\right\}} \right] I - \beta SI - \delta S$			
Decoupled growth model with Infected growth: replace S equation in system above			
$\frac{dS}{dt} = r \left[1 - \frac{S+I}{\frac{Kr}{r-\delta}} \right] S + \sigma r \left[1 - \frac{S+I}{\frac{Kr}{r-\delta}} \right] I - \beta SI - \delta S$			

1119 **Figure Legends**

1120

1121 **Figure 1.** Disease-mediated nutrient dynamics place host-pathogen interactions into an
1122 ecosystem context. Environmental nutrients modify, and are modified by, infection. These
1123 processes include how uptake of environmental nutrients (purple arrow, 1) shapes pathogen
1124 prevalence and disease severity via individual host and pathogen phenotypes, population
1125 attributes, and community properties (inner boxes). Infection-induced changes to hosts at any of
1126 these scales can alter subsequent nutrient uptake (green arrow, 1). Infection also alters host
1127 physiology or mortality, modifying populations and communities, feeding back to change the
1128 quantity and nutrient content of necromass (dead host and pathogen biomass) that is recycled via
1129 decomposition (green arrow, 2). Jointly, these disease-nutrient relationships create the potential
1130 for both positive and negative feedbacks.

1131

1132 **Figure 2.** The disease-mediated nutrient dynamics model (A) includes nutrient-dependent
1133 growth rate (dashed arrow) for susceptible (S) and infected (I) autotroph hosts. The model tracks
1134 infection-dependent nutrient flux among susceptible hosts (N_S), infected hosts (N_I), and the
1135 abiotic environment (N_E). Removing the nutrient-dependent growth link breaks the feedback
1136 loop, producing a decoupled growth model, in which growth and infection are independent of
1137 environmental nutrients. The DND model describes a disease-nutrient feedback loop (B). Abiotic
1138 nutrient availability impacts autotroph host growth rate and susceptible host density, with
1139 outcomes for pathogen spread and prevalence (purple arrows). Disease-induced changes in
1140 growth rate and mortality alter host density and the rate of nutrient return to the environmental

1141 nutrient pool (green arrows). Removing the nutrient-dependence of growth (dashed arrows)
1142 breaks the feedback loop (decoupled growth model).

1143

1144 **Figure 3.** Infection prevalence (A), nutrient content (B), growth rate (C), and elemental
1145 stoichiometry (D) dynamics resulting from the disease-mediated nutrient dynamics (DND)
1146 model (solid lines) and the decoupled growth model (dashed lines) under nutrient conditions
1147 with $N = 80 \text{ g N/m}^2$. Time zero on these graphs is the moment of pathogen introduction;
1148 simulations began 1,000 years prior to the pathogen introduction to allow for a steady state to be
1149 reached.

1150

1151 **Figure 4.** Ecosystem nutrients impact pathogen basic reproductive number (R_0 , thin lines) and
1152 host *per capita* growth rate (bold lines) at low (A) and high (B) transmission rates when
1153 autotroph growth depends on recycled nutrients (DND model) or not (decoupled growth model).
1154 Blue dashed lines indicate nutrient conditions below which infection is not sustained ($R_0=1$).
1155 Infection prevalence is impacted differently by nutrient availability at low (C) and high (D)
1156 transmission rates. Like prevalence, nutrients also impact host dynamics differently at low (E)
1157 and high (F) transmission rates. Shaded regions indicate magnitude of cycles. Based on
1158 numerically observed cycles, the DND model appeared to have sustained cycles after 1 million
1159 years using Matlab's Runga Kutta ode45 solver.

1160

1161 **Figure 5.** Pathogen transmission rate impacts (A) host stoichiometric quota and (B) nutrient
1162 pools in susceptible and infected autotrophs (NS, NI) and the abiotic environment (NE).
1163 Feedbacks between disease and nutrients in the DND model cause cycling with increasing

1164 transmission; shaded regions show cycle minima and maxima, and solid lines indicate the mean.
1165 Without a feedback (decoupled growth model), equilibria are stable for all transmission rates.
1166 Nutrient conditions are set at 60gN/m².

1167

1168 **Figure 6.** Incorporating infected population growth without vertical transmission (infected
1169 producing susceptible) influences (A) prevalence and (B) host density. Shaded regions show
1170 cycle minima and maxima, and solid lines indicate the mean. The basic DND model dynamics
1171 occur where the x-axis is zero. Predictions were obtained after running simulations for 100,000
1172 years using Matlab's Runga Kutta solver ode45.

1173

1174 **Figure 7.** Marine systems provide one case study of how environmental nutrients modify, and
1175 are modified by, infection. In this case study, these processes include marine phytoplankton
1176 uptake of environmental nutrients (purple arrow, 1) that determines infection and disease
1177 manifestation in individuals and alters phytoplankton community composition (inner boxes).
1178 These infection-induced changes to phytoplankton individuals and communities alter nutrient
1179 uptake from the environment (green arrow, 1). Infection feeds back to increase both cell leakage
1180 and lysis, releasing dissolved organic carbon (DOC) and other dissolved organic matter (DOM),
1181 often containing nitrogen and other elements, and speeding the recycling rate of carbon and
1182 nutrients (green arrow, 2).