Saved by the Symbiont: Environmental Stress Intensity and Endosymbiont-Mediated Stress Response Determine Evolved Host Complexity

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Abstract

Understanding how stress responses affect the trajectory of host–symbiont coevolution is central to predicting and managing species outcomes in the face of disturbances to ecosystems. Critically, it remains an open question how exactly we expect stressors to influence the coevolutionary dynamics of symbioses (on either end of the parasitism–mutualism continuum). In this work, we use *in silico* experiments to examine how stressor frequency and intensity affect host and symbiont diversity and complexity when mutualists defend their hosts from stress and parasites render their hosts stress-susceptible. We find that stress-protective mutualism can enable host survival where it would otherwise be impossible, but can also constrain host diversification and complexification. These effects are particularly strong when stressors are more destructive (and thus protection is more crucial). Meanwhile, stress-exploitative parasitism can increase the evolution of complex traits (to escape trait-matching parasites). Hosts cycle between complex, parasite-resistant and simpler, parasite-vulnerable states over time. These results better enable us to predict (and, potentially, influence) how natural symbioses respond to environmental instabilities such as climate change.

Code available at: `https://doi.org/10.5281/zenodo.19477499`

Introduction

Changing climates and human activities transform natural habitats, causing conditions to repeatedly leave the range that local organisms are adapted to, creating stress. These organisms are part of complex webs of interdependent biota, many of which are engaged in *symbiosis* (Lafferty et al., 2006). Symbiosis occurs when organisms of one species engage in relationships with another species that are sustained over evolutionary time. Here, we will focus specifically on relationships between hosts and the endosymbionts living *within* them. Symbiotic interactions can range from mutually beneficial (mutualism) to antagonistic (parasitism). How these interactions influence evolution remains to be fully understood (McFall-Ngai, 2024), particularly in the presence of repeated environmental stress. In this work, we investigate how symbioses that either improve or worsen host stress responses affect evolved host complexity.

Environmental stressors come in many forms with wide ranging effects. In the context of evolution, stressors are environmental conditions that reduce fitness. Such stressors can be biotic (pathogen presence, food availability, *etc*.) or abiotic (temperature, salinity, *etc*.) (Schulte, 2014). Symbionts can affect their host's stress responses in both beneficial and deleterious ways (Apprill, 2020; Bénard et al., 2020). For example, there are symbionts that directly help their hosts respond to stressors by producing microbial substances and toxins (Haine, 2008), participating in pesticide resistance (Sato et al., 2021; Gundel et al., 2012), or improving heat tolerance (Richier et al., 2005). However, some symbionts which are beneficial under non-stressful conditions have stress responses that harm their hosts (Vidal-Dupiol et al., 2009). Stress can also lead to the loss of beneficial symbionts and subsequent colonization by less helpful organisms (Fan et al., 2013).

While parasites can significantly reduce a host's fitness in the short term, they can also have a powerful impact on the population's evolutionary trajectory. For example, parasites have been shown to alter the ability of hosts to fully occupy their theoretical niche (Ricklefs, 2010), influence interhost competition and reproductive capability (Buckling and Rainey, 2002; Joy, 2013), and promote the evolution of complex features (Zaman et al., 2014). Parasites have also been shown to drive host diversification (Vanhove et al., 2025). Specifically, Karvonen and Seehausen (2012) suggest that parasitism can act as a driver of divergent host selection when host immunity to parasitism is variable and parasitism is sufficiently harmful. Further, in trait matching experiments, Yoder and Nuismer (2010) found that parasite-afflicted hosts were more diverse than hosts evolved in the absence of parasites. However, this effect is not universal; parasites can suppress host diversity in structured populations by reducing host competition (Brockhurst et al., 2004).

In the mutualist case, simulations have shown that phenotype matching—the requirement that hosts and symbionts share traits—can constrain host diversification (Yoder and

Nuismer, 2010). This finding is consistent with results from a wet lab experiment on a non-endosymbiotic cross-feeding mutualism, in which Pauli et al. (2022) found that the mutualism constrained the partners' ability to evolve resistance to an antibiotic relative to controls where no mutualism was present. In contrast, phylogenetic analyses of plants with extrafloral nectaries have found that mutualism is associated with higher rates of host diversification (Weber and Agrawal, 2014). Joy (2013) also provides evidence that mutualism promotes host diversification; specifically, they found that mutualism between midges and fungi enabled midge diversification and expansion of the niche in which midges could survive (compared to non-fungal-partnered midges). This symbiont-mediated range expansion has been observed in other species too (Fowler et al., 2023), and is effectively equivalent to the scenario of mutualists protecting hosts from stressful environments (since regions outside the host's normal niche are, by definition, stressful). This effect could promote the evolution of diverse and complex traits by enabling hosts to better survive. However, given the prior results showing that mutualism can constrain evolution, it is not obvious what outcome to expect from these scenarios in practice.

Here, we investigate how stress-exploitative parasites and stress-protective mutualists impact host evolution under varying levels of periodic environmental stress. What patterns from the literature (promotion or constraint of host complexity by mutualism *vs.* parasitism) occur when the effect of symbiosis is periodic rather than constant? Do stress intensity and frequency affect evolved host complexity? We find that mutualism constrains and parasitism promotes the evolution of complex traits. However, host–symbiont dynamics over time can be complex. Under mutualism, hosts are faced with a trade-off between getting more resources from performing complex tasks and getting stress protection from simple mutualists. In the parasite case, hosts cycle between performing more complex tasks (which are less susceptible to parasitism) and performing more simple tasks (which are more susceptible to parasitism), switching between these states often.

Methods

Study system

We conducted experiments using the agent-based symbiosis modeling software Symbulation (Vostinar, 2021; Vostinar and Ofria, 2019). In our experiments, we coevolved populations of digital hosts and endosymbionts that lived inside of hosts. Hosts competed for limited space in a well-mixed environment with a maximum carrying capacity of 10,000 hosts. Each host could contain up to one symbiont at a time, and as such, symbionts competed for limited space inside the host population. We initialized all experiments with full host populations, each infected with an initial symbiont.

Tasks Each organism comprised a linear sequence of assembly-like instructions (its genome) and a virtual CPU capable of executing those instructions (Moreno et al., 2021). Organisms gained resources by performing one- and two-input Boolean logic tasks (Table 1). These logic tasks are commonly used in digital evolution studies because their relative complexity can be quantified based on the number of NAND gates required to complete them (Zaman et al., 2014; Lenski et al., 2003). Genomes were fixed at 100 instructions and they mutated upon reproduction; per-instruction and per-argument substitution rates were set to ≈ 0.0396 through preliminary parameterization (the precise per-bit mutation rate was set to 0.00495). We initialized organisms with genomes capable of performing the NAND task and reproducing. All other instructions in the starting genome were "no-operation" instructions. All other task completions observed in our results arose through evolution. Organisms received resources for each task completion, with the precise amount varying based on experimental conditions as detailed below. Each timestep, organisms received 16 CPU cycles, enabling them to execute 16 instructions in their genomes.

In addition to providing resources, task completion patterns were also used as the basis for symbiotic interaction, modeling trait matching observed in organic symbioses (*e.g.*, between flower tube length and fly tongue length (Anderson and Johnson, 2008)). We limited organisms to performing one type of task during their lifetime; however, they could perform that task repeatedly to gain more resources. Hosts and symbionts engaged in symbiosis by "task matching" with one another.

Reproduction Hosts reproduced once they accrued sufficient resources and executed a reproduce instruction. A host's offspring was then placed into a random position in the world, overwriting any previous occupants. Symbiont offspring transmitted via two mechanisms: if a symbiont accrued sufficient resources and executed a reproduce instruction, the offspring transmitted via *horizontal transmission*, in which a symbiont offspring attempted to infect a random other host. If a symbiont was mutualistic, it could also transmit its offspring via *vertical transmission*, in which a symbiont offspring transmitted into the offspring of the parent symbiont's host. If a symbiont attempted to horizontally transmit into a host that already had a symbiont, it could succeed (killing the previous symbiont in the process) *only* if the invading symbiont matched tasks with the host and the preexisting symbiont did not.

Environmental stress events We modeled environmental stress as a sequence of periodic stress events. During each stress event, hosts had a configurable chance of dying that was modified by the properties of their symbiont. Without a symbiont or with a symbiont that did not match the

host's tasks, hosts died with the set base death chance. Hosts with task-matching symbionts died according to a symbiont-conferred death chance. Mutualists decreased and parasites increased their host's chance of death, respectively.

Experimental design

Mutualism experiment To determine the effect of mutualists on host evolution under stressful conditions, we ran a condition with symbionts restricted to mutualistic behavior and varied frequency and intensity of stress events. When the mutualists task-matched with their hosts, they conferred full immunity from stressors (*i.e.*, stress events had no effect on hosts with matching mutualists). We ran experiments with base death chance: 0, 50, and 100%. Stress events occurred every 25 or 200 timesteps (for a total of 4,000 and 500 stress events during an experiment, respectively).

In this experiment, symbionts were always able to vertically transmit when their hosts reproduced. Symbionts were also capable of horizontal transmission if they accrued sufficient resources. Hosts needed 100 resources to reproduce, and symbionts needed 0 resources to reproduce vertically and 50 to reproduce horizontally. Organisms received more resources for completing more complex tasks (see Table 1).

Task	Resources rewarded	Complexity
NOT, NAND	1	1
AND, ORN	2	2
OR, ANDN	4	3
NOR, XOR	8	4
EQU	16	5

Table 1: Task completion resource rewards in the mutualism experiment, which scale with task complexity (*i.e.*, the number of NAND gates required to implement).

Parasitism experiment To determine the effect of parasitism in varying environmental stress levels, we ran experiments with variable stress frequency and with variable levels of parasite-induced stress susceptibility. When a parasite's completed task matched that of its host during a stress event, the parasite's induced death chance specified the stress event's probability of killing the host. Host death probability during stress events was otherwise 0%. A parasite that caused the death of its host during a stress event reproduced horizontally 8 times for no resource cost and with no minimum cycle requirement; thus, parasites benefited from harming their host. We ran experiments with parasite-induced death chances of 0, 50, and 100%. Stress events occurred every 25 or 200 timesteps (totaling 4,000 and 500 stress events over the course of the experiment, respectively).

Hosts never vertically transmitted their parasite; parasites exclusively horizontally transmitted. Hosts required 10 resources to reproduce, and parasites required 5. In addition to the minimum reproduction requirements, hosts and symbionts were required to complete 400 and 25 CPU cycles before they could reproduce, respectively, which modeled a need to reach maturity before reproducing. These minimum cycle requirements were necessary in this experiment to prevent hosts from evolving to skip the majority of their genome early in the experiment, which caused them to be unable to evolve to complete any other tasks later. All completed tasks were rewarded with 5 resources. We chose not to provide a resource-based incentive to evolve more complex tasks because we were interested in determining whether parasites promote the evolution of complex features (as has been observed under other conditions). Creating a resource-based pressure to complete complex tasks would potentially obscure this effect, making it unclear if it was driven by the reward or the parasites. In contrast, in the mutualism experiments we needed the ability to detect constraint, which required a control condition where complex tasks would evolve.

Statistical analyses

Differences between conditions were analyzed with Fisher's exact test and Bonferroni corrections for multiple comparisons.

Code and data availability

This work was conducted using Symbulation, which is open source and freely available along with its core dependency Empirical (Vostinar, 2021; Vostinar et al., 2024). Supplementary material for this work can be found here: https://doi.org/10.5281/zenodo.19477499.

Results

Stress mutualism constrains evolution of complex host tasks

To determine the effect of stress-protective mutualism on host task evolution, we ran experiments under conditions where hosts evolved complex tasks when not faced with stress. We exposed hosts in these experiments to mild and intense environmental stress at infrequent and frequent levels (mutualism experiment; see Figure 1a). We find that when stress is frequent and at least mildly harmful, hosts with stress-protective mutualists evolve complex tasks (ANDN, NOR, XOR, EQU) significantly less frequently than when there is no environmental stress (stress frequency 25, base death chance 50 and 100%, $p < 0.05$, Fisher's exact test, corrected). Hosts also evolve complex tasks (ANDN, NOR, XOR, EQU) in significantly fewer replicates when stress is infrequent but very harmful than when there was no effect of environmental stress (stress frequency 200, base death chance 100%, $p < 0.05$, Fisher's exact test, corrected).

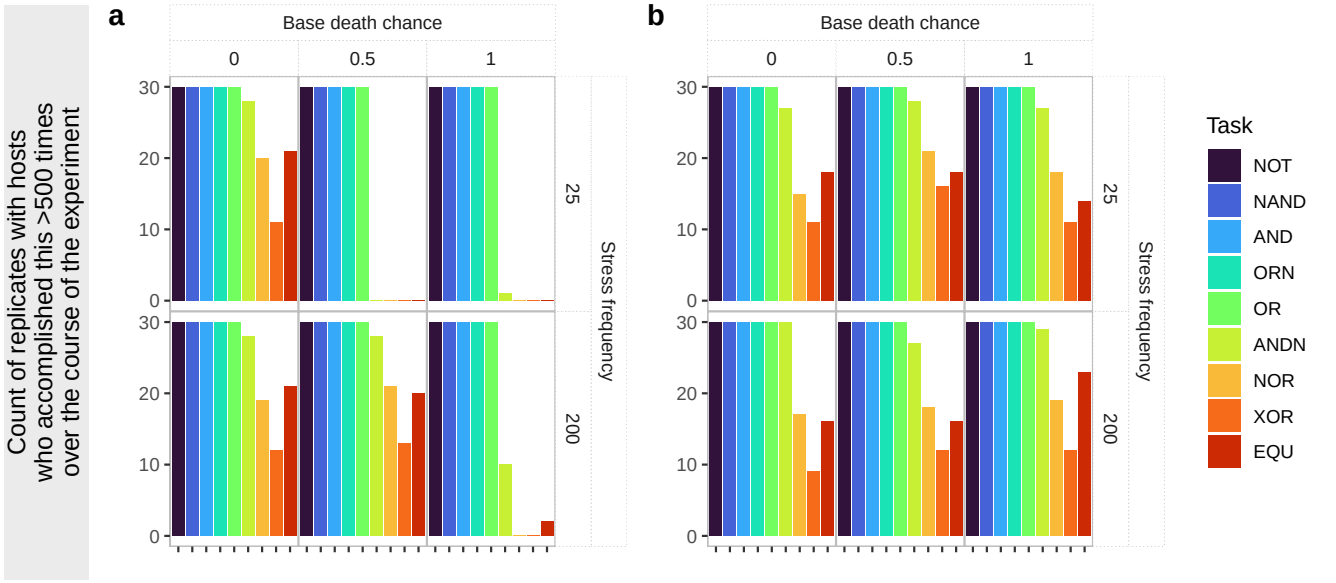

Figure 1: **Mutualism experiment task completion.** Count of replicates wherein hosts accomplished a task at least 500 times over the 100,000 timesteps of the experiment for the mutualism experiment (a) and the mutualism bottleneck control (b). Task-matching mutualists reduce host death chance to 0% (from base death chance).

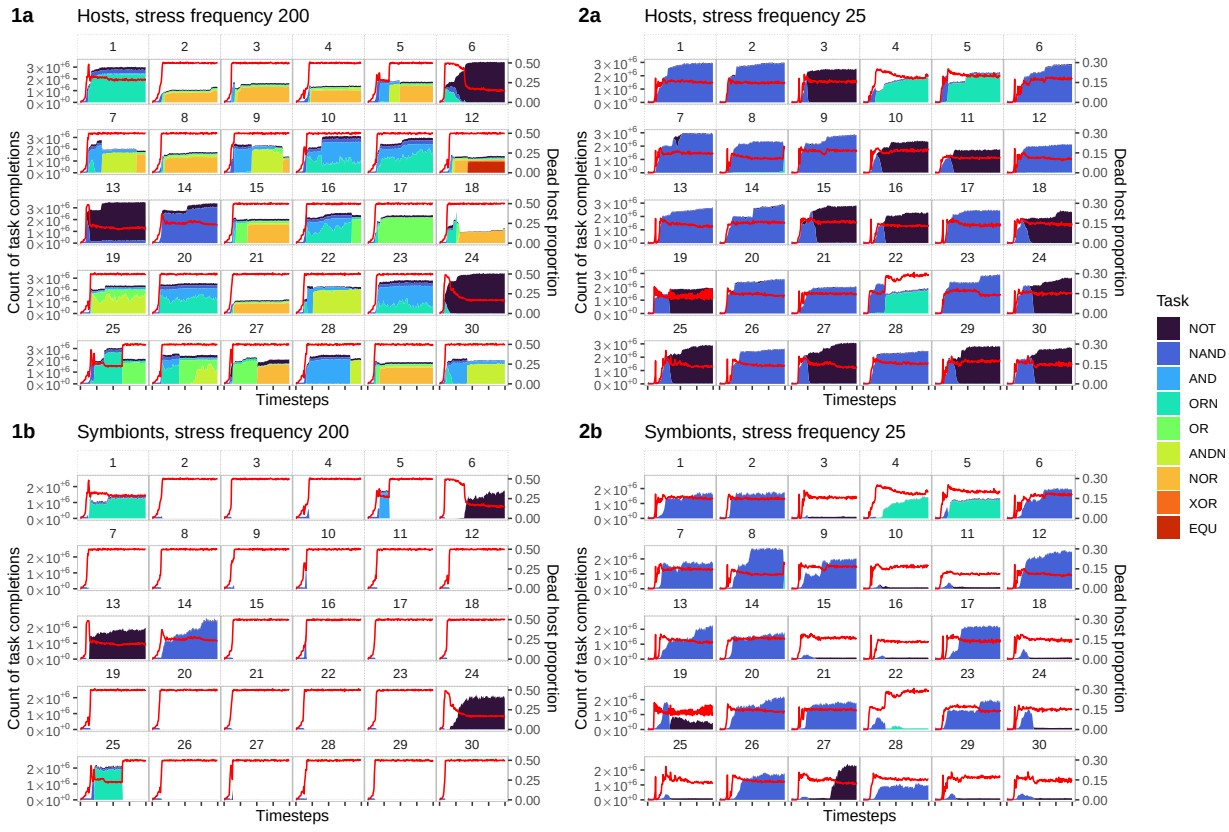

Figure 2: **Host and mutualist task completion over time for each replicate at two stress frequencies.** Pooled counts of tasks completed by hosts (a) and symbionts (b) over time in the mutualism experiment when base death chance was set to 50% and stress frequency was set to 1) 200 and 2) 25.

To determine whether host task evolution is a product of mutualist influence or simply bottleneck dynamics resulting from stress events, we compared to a control in which hosts were killed randomly at similar rates but without consideration of their completed tasks. Specifically, we paired each control replicate with a mutualism experiment replicate, calculated how many hosts survived each stress event in the experimental replicate, and removed hosts in the control to match the experimental host population count at each stress event. Hosts were randomly selected for removal in the control; host task completion and symbiont task matches did not influence host survival in control runs.

Under these control conditions, hosts consistently evolve complex tasks, presumably due to the higher resource rewards (see Figure 1b). Even with similar rates of host death, hosts evolve complex tasks (ANDN, NOR, XOR, EQU) in significantly fewer replicates in the stress experiment than in the bottleneck control when stress is frequent and mild or intense (stress frequency 25, base death chance 50 and 100%) or infrequent and intense (stress frequency 200, base death chance 100%). These results suggest that the constraint of host tasks is produced by coevolutionary pressure from mutualists.

Stress-protective mutualisms do not always yield constraint, however. When stress is infrequent and mild (stress frequency 200, base death chance 50%), hosts evolve complex tasks (ANDN, NOR, XOR, EQU) significantly more frequently than when stress is infrequent and severe (stress frequency 200, base death chance 100%, $p < 0.05$, Fisher's exact test, corrected). Hosts in this mild, infrequent stress case do not evolve complex tasks significantly more or less often than in the paired control. Stress may not produce sufficient pressure at these levels for the pressure to retain stress-protective mutualists to constrain host complexification.

Overall, these results indicate that trait-matching mutualism can constrain the evolution of host complexity when mutualist success strongly impacts host survival of stress events. This effect disappears when stress is less intense. However, we also tested host survival of environmental stress when unprotected by mutualists, and found that hosts rapidly go extinct (mutualist settings no-symbiont control; see supplemental material, Figure S1). Thus, mutualism is a double-edged sword: mutualists promote host survival by preventing them from going extinct, but it comes with a cost of lowered adaptability.

Less intense stress unlinks host–mutualist tasks performed over time

Do host and mutualist tasks evolve in response to one another? To investigate this question, we examined per-replicate task completion counts for both hosts and mutualists.

As demonstrated in the previous section, hosts exposed to frequent stress events are more constrained than hosts exposed to stress more rarely. When stress occurs every 25 timesteps, hosts primarily complete NOT, NAND, or ORN; mutualists complete the same tasks. The proportion of hosts killed during stress events spikes initially alongside host task evolution but mostly plateaus afterwards. Hosts evolve ORN in three replicates (4, 5, and 22). When hosts first switch to ORN, the proportion of dead hosts spikes beyond the level observed in other cases. In two of the three replicates (4 and 5) mutualists also gain ORN, and the dead host proportion subsequently declines. In replicate 22, mutualists also gain ORN, but perform it at lower levels; dead host proportion remains rather high (0.3), but does not reach the maximum possible (0.5). In these cases, hosts evolve to complete a higher-reward task rather than "play it safe" with their mutualists; mutualists in these cases evolutionarily track their hosts. This dynamic suggests that evolutionary pressure in these relationships may be bidirectional—sometimes host task evolution is guided by mutualists, other times, mutualist task evolution is guided by hosts.

Hosts exposed to stress at less frequent intervals evolve a more diverse and complex array of tasks across replicates (Figure 2-1a). In most cases, hosts rapidly evolve complex tasks and are killed during stress events at the rate specified by base death chance (50%). Mutualists in these cases accomplish few tasks (though more than zero). This behavior may occur because the resource benefit of more complex tasks outweighs the protection offered by mutualists when stress is infrequent. As hosts evolve complexity, this dynamic may create an uncrossable fitness valley for mutualists because evolving more complex tasks is only useful if there are hosts performing that task (either so the host can be infected through preferential ousting or protected during stress). Thus, if hosts get too "far ahead," mutualists may become evolutionarily unlinked from them.

In other cases, however, host and mutualist task matching remains meaningful over time. In replicate 6, for example, hosts initially perform a mix of NOT, NAND, and ORN and are killed in high proportions during stress. Midway through the experiment, however, hosts evolve to primarily perform NOT tasks. At this point, symbiont NOT task completion surges, and the proportion of hosts killed during stress drops. Likewise, replicates 1, 13, 14, and 24 display high initial rates of host death as host task completion rises before hosts settle into relatively simple tasks. Mutualists then begin completing the same simple task as their hosts more frequently, leading a lower proportion of hosts to die.

We also observed evolutionary disengagement and engagement between hosts and mutualists in a single run. For example, in replicates 5 and 25, hosts initially complete tasks also completed by their mutualists, resulting in relatively low proportions of dead hosts. In replicate 5, hosts then switch from completing AND (which yields 2 resources) to ANDN (which yields 4) and lose their mu-

tualists, which complete few tasks once hosts switch. At the same time, the proportion of hosts killed during stress increases to nearly the maximum possible expected rate (50%). A similar pattern occurs in replicate 25 when hosts switch from completing primarily ORN to OR: hosts switch to a task that provides more resources but become unlinked from their mutualists, which causes an increase in the proportion of hosts killed from stress.

Altogether, these results suggest that host evolution sometimes prioritizes retention of stress-protective symbionts over maximizing reproductive speed (accruing points as quickly as possible), particularly when stress is intense and the protection offered by the mutualist is significant. Critically, however, hosts can leave behind their symbionts if the resource reward of their newly-discovered task is sufficiently high. In some cases, mutualists manage to follow their hosts to the new task, but not always. Hosts that remain simple also complete far more tasks, potentially "making up" for their lower resource reward. Further unpacking these complex dynamics and the trade-offs between investment in defense and reproduction is an avenue for further work.

Stress parasitism promotes the evolution of complex host tasks

We also investigated the role of stress-exploitative parasitism on the evolution of complex host tasks by running experiments where task-matching parasites worsen host stress survival probability (parasitism experiment; Figure 3a). We find that hosts evolve more complex tasks when subjected to more extreme stress events and parasites that more strongly impede host stress responses. When stress is most frequent and parasites are either slightly or severely harmful (stress frequency 25, parasite death chance 50 and 100%), hosts evolve complex tasks (EQU, XOR, NOR) significantly more frequently than when parasites do not affect host survival (parasite death chance 0%, $p < 0.001$, Fisher's exact test, corrected). When stress is less frequent and parasites are maximally harmful, hosts evolve some complex tasks (NOR) significantly more frequently than when parasites do not affect host survival (parasite death chance 0%, $p < 0.001$, Fisher's exact test, corrected).

To determine whether these trends were due to the higher death rates experienced by higher-stress condition hosts, we again ran bottleneck controls where hosts were randomly killed at similar rates (rather than being killed based on parasite task-matching status; parasitism bottleneck control; Figure 3b). We find that without parasite pressure, hosts evolve some complex tasks (up to ANDN in several replicates across conditions), but fail to evolve the most complex tasks (NOR, XOR, EQU) in any replicate. Hosts evolve complex tasks significantly more frequently than in the bottleneck control when stress is infrequent and parasites are very dangerous (stress frequency 200, parasite death chance 100%; NOR, $p < 0.001$, Fisher's exact test, corrected), when stress is frequent and parasites are somewhat dangerous (stress frequency 25, parasite death chance 50%; ANDN, NOR, XOR, EQU, $p < 0.001$, Fisher's exact test, corrected), and when stress is frequent and parasites are very dangerous (stress frequency 25, parasite death chance 100%; ANDN, NOR, XOR, EQU, $p < 0.001$, Fisher's exact test, corrected). These comparisons show that the observed evolution of host complexity under high-stress conditions is driven by task-matching stress-exploitative parasites.

If stress pressure is too weak, however, we fail to see significant changes in evolved host complexity compared to the control. When stress is infrequent and parasites are mild, hosts fail to evolve complex tasks in significantly more replicates than in the control (stress frequency 200, parasite death chance 50%). While we do see significantly more evolution of some complex tasks (NOR) when stress is infrequent and parasites are dangerous than in the control, we do not observe significantly higher rates of the most complex tasks (XOR, EQU).

Taken together, these results indicate that parasite pressure promotes the evolution of more complex tasks in hosts. When hosts are killed randomly, or stress is mild, hosts fail to evolve the most complex tasks. When stress is more threatening (because it occurs more frequently or parasites are more dangerous), hosts evolve the most complex tasks. We suggest stress frequency and severity combine to increase the selective pressure on hosts to escape parasites; consequently, hosts evolve higher complexity under more intense stress.

Complex host tasks sustained by parasite pressure

How does host task evolution respond to parasite pressure over time? Hosts that evolve complex tasks escape parasites, resulting in diminished host death rate and simplified symbionts; this behavior is intensified under more stressful conditions.

When stress events occur every 200 timesteps, hosts and symbionts principally evolve and execute more simple tasks (NOT, NAND, AND, ORN; Figure 4-1a,b). In some cases, spikes in host completion of tasks more complex than NOT (NAND, AND, ORN) occur at the same time as sharp drops in the proportion of hosts killed during stress events (a good example of this appears halfway through replicate 13). Parasites in these cases do not complete equivalent proportions of the tasks more complex than NAND.

When stress events occur every 25 timesteps, hosts evolve the most complex tasks far more frequently; parasites still principally complete simpler tasks, but achieve higher complexity than when stress occurs less frequently. Generally, we can categorize host–parasite interactions into two patterns. In the first observed pattern, hosts and parasites both complete a small and evenly distributed number of the less complex tasks (with parasites completing simpler tasks than hosts), resulting in a higher proportion of hosts

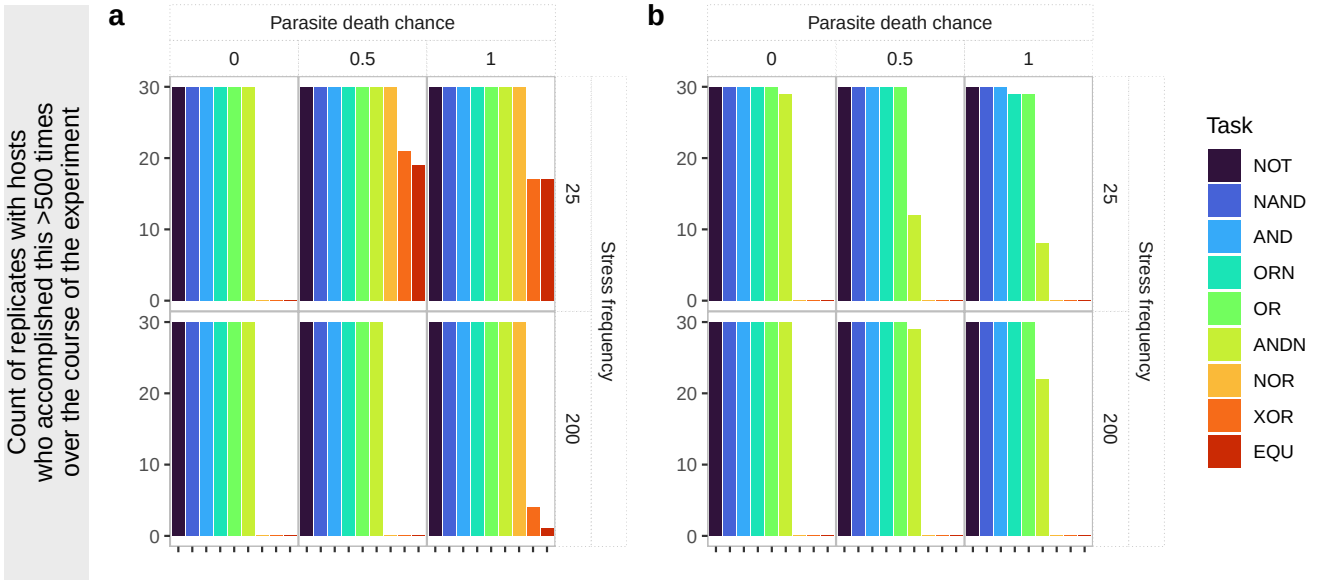

Figure 3: **Parasitism experiment task completion.** Count of replicates in the parasitism experiment (a) and the parasitism bottleneck control (b) in which hosts accomplished a task at least 500 times over the 100,000 timesteps of the experiment. Task-matching parasites increase host death chance to the parasite death chance value (from 0%).

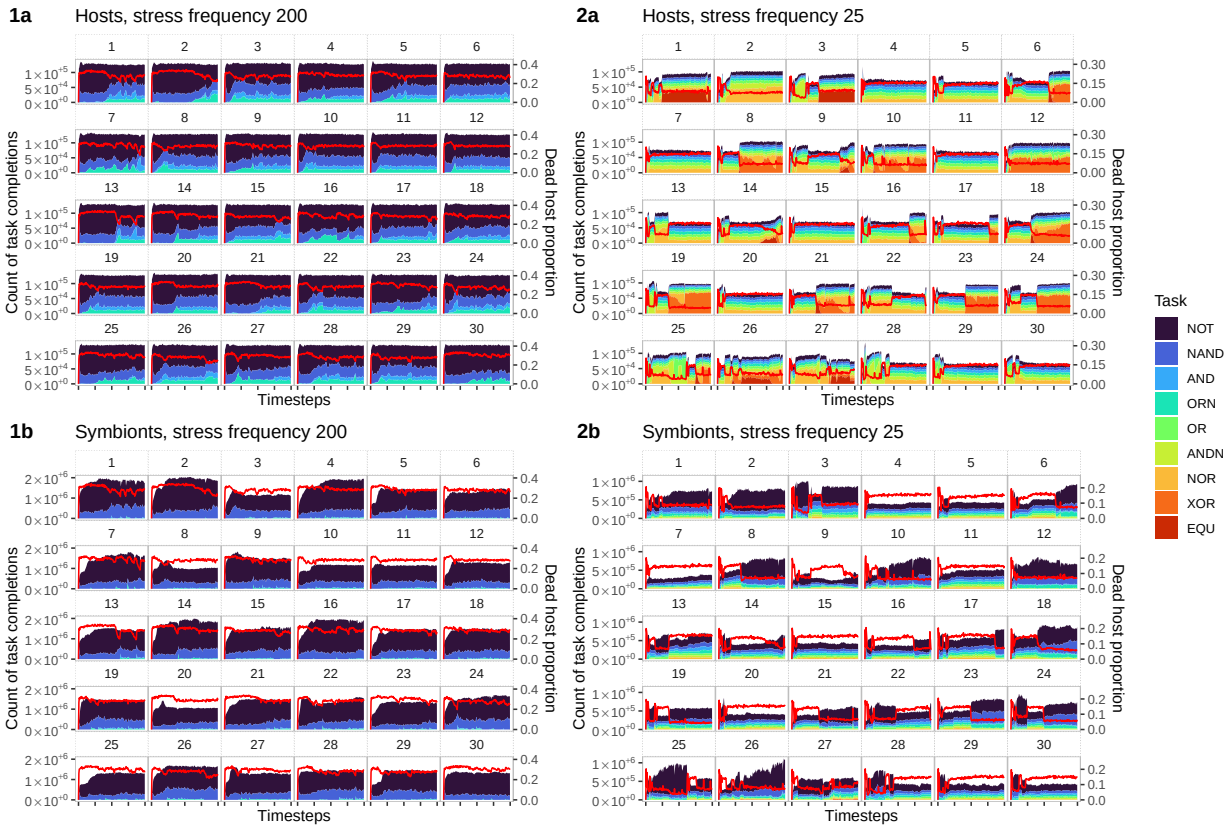

Figure 4: **Host and parasite task completion over time for each replicate at two stress frequencies.** Pooled counts of tasks completed by hosts (a) and symbionts (b) over time in the parasitism experiment when parasite death chance was set to 50% and stress frequency was set to 1) 200 and 2) 25.

killed during each stress event. In the second observed pattern, hosts complete more complex tasks(XOR, EQU particularly), while parasites switch to completing predominantly NOT and NAND (the simplest tasks); this yields a much lower proportion of hosts killed during stress events. Over the course of a run, several replicates switch between these states.

In replicate 23, for example, both hosts and parasites complete a mix of semi-complex tasks for the first half of the experiment before hosts dramatically increase their XOR task completion count and parasites switch to primarily completing NOT and NAND. Concurrently, the proportion of hosts killed by parasites during stress events drops dramatically, and with it the number of free offspring parasites produced through killing hosts. In this case, hosts successfully evolve a task the parasites lack, and escape them. The parasites, in response, fall back to easier tasks and "give up" on tracking (and killing) hosts.

These results imply that the persistence of complex host tasks is driven by parasite pressure. In replicate 10, hosts evolve XOR early, and experience a lower kill rate. However, hosts start to lose XOR several times, which are accompanied by spikes in proportion of hosts killed and then a return of XORs completed. This cycle occurs several times. These results hint at a feedback mechanism between which tasks hosts complete and the rate at which they are killed during stress, thus driving host task evolution.

Discussion and Conclusion

We have demonstrated that stress parasitism can promote host complexification, and stress mutualism can suppress it, with the caveat that mutualism also enables host populations to survive where they otherwise would not. More intense stress strengthens these patterns.

When provided with a reward for performing more complex tasks, hosts under minimal stress successfully do so. However, when stress is more intense, hosts remain simpler. Over time, hosts under less intense stress often rapidly abandon simple tasks for more complex high-reward tasks, trading a loss of mutualist protection with (presumably) a higher reproductive rate. Hosts facing more stress are generally unable to make this trade. These results suggest that ecological systems with previously stable mutualisms may evolve more slowly in response to environmental stress caused by climate change and that increasing severity and frequency of stress events will further exacerbate this effect. However, if the severity of the stress events can be lowered, the host populations may be able to evolve away from dependency on their mutualists, enabling their long term survival. Thus, any amount of mitigation of climate stress events could be helpful in enabling key host species survival through the climate crisis.

When stress is sufficiently strong, parasites drive hosts to perform more complex tasks, even in the absence of an explicit reward for doing so (other than escaping the parasites). When stress is infrequent, spikes in host performance of more complex tasks are accompanied by a slight decrease in proportion of hosts killed, and parasites do not meaningfully respond. When stress is much more frequent, however, host–parasite dynamics switch between two states (often within a single replicate). First, hosts switch between states of high-complexity task completion, during which parasites "give up" on matching with hosts and instead complete a higher volume of simple tasks; the proportion of hosts killed is lower in this state. Second, hosts will sometimes complete a more even spread of middling and low complexity tasks, and so will parasites; a higher proportion of hosts are killed under this behavior pattern.

Notably, even when either populations of hosts or parasites achieve more complex tasks, they often retain subpopulations capable of lower-order tasks. Other digital evolution systems observed the evolution of generalist parasites which completed multiple tasks; their population genetic memory barred hosts from back-evolving to simpler tasks (Zaman et al., 2014). Under the conditions we tested, however, organisms were constrained to a single task type over their lifetime. As a result, memory of previously-evolved tasks is retained at a population level instead of an individual level.

While we have already demonstrated strong effects of symbionts and stress events on host evolution, there are many other ways in which symbiotic coevolution could impact both host and symbiont evolutionary trajectories. For instance, symbionts that improve host fitness when faced with stressors are often more costly or less capable (than stress-susceptible symbionts) when not faced with stress (Dunbar et al., 2007; Tortorelli et al., 2025; Bonforti et al., 2025; Baker et al., 2004; Bénard et al., 2020). Population dynamics of competing, differentially stress-protective or -exploitive symbionts are not yet fully understood. We have established a foundation upon which all of these dynamics can be further explored in future work.

Overall, these results suggest that host evolutionary trajectory and diversity is highly sensitive to host–symbiont dynamics activated by stressors. As ecosystems are increasingly perturbed by changing climates, understanding and predicting evolutionary responses to stress is crucial.

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