Evolution and Maintenance of Mutualism between Tubeworms and Sulfur-Oxidizing Bacteria

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ABSTRACT: Tubeworms and sulfur-oxidizing bacteria mutualism, an essential part of the chemosynthetic ecosystem in the deep sea, has several puzzling features. After acquiring sulfur-oxidizing bacteria from the environment, tubeworms become fully dependent on their symbiont bacteria for nutrient intake. Once ingested by the tubeworm larva, no additional symbionts join from the environment, and no symbionts are released until the host tubeworm dies. Despite this very narrow window to acquire symbionts, some tubeworm species can live for >200 years. Such a restricted release of symbionts could lead to a shortage of symbiont bacteria in the environment without which tubeworms could not survive. In our study, we examine the conditions under which this mutualism can persist and whether the host mortality rate evolves toward a low value using a mathematical model for the tubeworm–symbiont bacteria system. Our model reveals that mutualism can persist only when the host mortality rate is within an intermediate range. With cohabitation of multiple symbionts strains in the same host, host mortality rate evolves toward a low value without driving either host or symbiont to extinction when competition among symbionts is weak and their growth within a host is slow. We also find the parameter conditions that lead to unlimited evolutionary escalation of host mortality rate toward coextinction of both tubeworms and symbionts populations (evolutionary double suicide). The generality of this evolutionary fragility in obligate mutualistic systems as well as the contrasting evolutionary robustness in host-parasite systems are discussed.

Keywords: mutualism, adaptive dynamics, evolutionary suicide, coextinction, tubeworm, symbiont.

Introduction

Many animals living in the deep-sea environment have a mutualistic relationship with chemosynthetic bacteria and often depend on these symbionts for nutrient acquisition. A majority of these environments are dominated by siboglinid annelid tubeworms that have a mutualistic relationship with sulfur-oxidizing bacteria. Tubeworms act as foundation species by creating bush-like aggregations that provide living space for other species (Corliss et al. 1979; Paull et al. 1984; Gibson et al. 2010). Sulfur-oxidizing bacteria provide nutrients for their host tubeworm produced by chemosynthesis reactions (Felbeck and Jarchow 1998; Bright et al. 2000). In return, within the trophosome—a specialized organ within the host (Cavanaugh et al. 1981; Tyler and Young 1999)—symbiont bacteria enjoy an environment without competition from free-living bacteria. Tubeworms have two distinct life stages: free-living larva and sessile adult. The free-living larva metamorphoses into a tubelike adult after ingesting symbionts from the environment by horizontal transmission (Nussbaumer et al. 2006), after which it loses its digestive organ. After a larva captures the symbionts, it becomes a sessile adult, which neither ingests more symbionts nor releases them until it dies (reported for *Riftia pachyptila* by Klose et al. [2015]; see also Bright and Lallie [2010], which mentions no evidence of symbiont release). Symbiont species that dominate in the bacterial fauna inside tubeworms are rare in the bacteria fauna outside tubeworms (Patra et al. 2016). This suggests that the free-living symbionts would be competitively inferior to nonsymbiotic bacteria in the environment outside tubeworms. Thus, the symbionts have a limited opportunity to be released into the environment, and the population of symbionts in the environment declines if host tubeworms are long-lived. The infrequent release of symbionts therefore could be expected to threaten both the symbiont population and the tubeworm population: without the release from tubeworms symbionts cannot maintain themselves, and the tubeworms cannot live without the symbionts. Nevertheless, some tubeworm species found in cold seeps, such as *Lamellibrachia luymesi*, live surprisingly long—some individuals are reported to live >200 years (Gibson et al. 2010)—while maintaining a mutualistic relationship with their symbionts.

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In the present article, we focus on three puzzling features that appear to make the secure maintenance of this symbiotic system difficult: (1) symbionts are released only when their host dies, (2) symbionts are captured only by horizontal transmission, and (3) the longevity of host lives could be very long (>200 years). Our strategy to understand these phenomena is to study the ecological conditions under which the mutualistic system persists and to examine the evolution of a life-history parameter, the host's mortality, that plays a key role in this mutualistic system. Living long might be good for an adult tubeworm because it allows the tubeworm to produce many pelagic larvae during its lifetime (tubeworms are iteroparous); however, the number of symbionts released after the tubeworm's death might not linearly increase with its age. Therefore, a long-lived tubeworm implies a shortage of availability of symbiont bacteria to be ingested by newborn tubeworms, which might disrupt the mutualistic system. Given that the death of host tubeworms by external factors, such as predation, is rare (Micheli et al. 2002) and that the tubeworm could immediately die of starvation if its symbionts stopped supplying nutrients, we speculate that host death is mostly controlled by the symbionts. Additionally, some symbiont bacteria have a gene related to virulence for eukaryotes (Perez and Juniper 2016). Thus, it is natural to presume that a symbiont can influence how long its hosts live. It appears to be to the symbiont's advantage to kill its host earlier by not supplying them with nutrients rather than to remain captured inside its body for quite a long time; therefore, we question why symbionts accept their fate of being held captive for a long time yet remain cooperative with their hosts.

As far as we know, there have been no studies that focus on this paradox of the stability of this mutualistic system and the long life of tubeworms. Many studies that have discussed tubeworm longevity have attempted to understand it as the tubeworm's life-history strategy against the physical environment (Tyler and Young 1999; Cordes et al. 2005; Durkin et al. 2017); however, no study has focused on it as a symbiont's strategy or has investigated why such longevity could evolve under this paradoxical situation. In this study, we consider the population dynamics of this mutualistic system by focusing on the life cycle and evolutionary dynamics of the symbiont's strategy by using the adaptive dynamics theory (Metz et al. 1995). Using this model, we demonstrate how this mutualism can be maintained and how the host's long life is evolutionarily maintained.

Although the present article is motivated by an interesting mutualism of deep-sea tubeworms and sulfur-oxidizing bacteria, the results have general implications for other host-symbiont relationships. We will show in the present article that evolutionary double suicide—that is, evolution-driven coextinction of both host and symbiont species—occurs robustly in our model, we will discuss that this fragility is an inherent nature of obligate mutualistic systems, and we will contrast it with the evolutionary robustness of host-parasite systems.

Model

Ecological Dynamics

To study the conditions under which mutualism persists between tubeworms and sulfur-oxidizing bacteria, we derive a population dynamical model of tubeworms and bacteria based on their life cycles, as depicted in figure 1. This model contains three variables—the density of free-living tubeworm larvae L, that of adult tubeworms T, and that of free-living symbionts S—that change with time as

$$\begin{aligned} \frac{dL}{dt} &= -\beta LS + \lambda T - dL, \\ \frac{dT}{dt} &= \beta LS\rho(T) - (u + x)T, \\ \frac{dS}{dt} &= Q(x)(u + x)T - \delta S. \end{aligned} \tag{1}$$

Here, β is the rate at which a free-living tubeworm larva ingests a symbiont bacterium and metamorphoses into a sessile adult tubeworm. Among βLS, the newly metamorphosed sessile tubeworms per unit time interval, only a fraction $\rho(T)$ are presumed to establish themselves in the competition between tubeworms. This probability $\rho(T)$ of successful colonization is assumed to monotonically decrease with tubeworm density T from $\rho(0) = 1$ at $T = 0$.

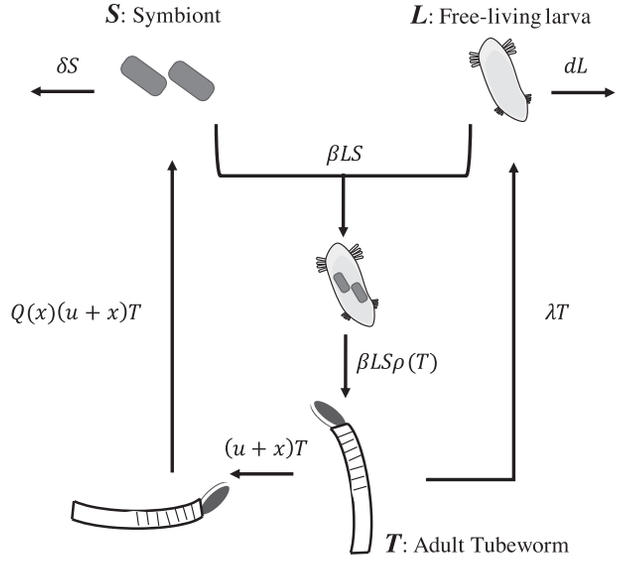

Figure 1: Schematic diagram of the life cycle of a tubeworm and symbiont bacteria.

An adult tubeworm releases its offspring larva at the rate λ and dies with the mortality rate $u + x$, where u is the basal mortality and x is symbiont-controlled mortality. When a host dies, a fixed number $Q(x)$ of symbionts are released into the environment per dead tubeworm, which depends on symbiont trait x, as explained later. Free-living symbionts and larval tubeworms die at rates δ and d, respectively; δ may include the growth rate of free-living symbionts in the environment ($\delta = \mu_F - r_F$), but we assume that the mortality rate μ_F of the free-living symbiont exceeds its growth rate r_F in the environment (i.e., $\delta > 0$). Throughout the article, we assume that $\lambda > u + x$, implying that the expected total number of larvae produced from an adult tubeworm until it dies is greater than 1 ($\lambda/(u + x) > 1$).

Now we describe how the number $Q(x)$ of symbionts released at host death depends on the symbiont trait x, the symbiont-controlled host mortality. This is given by specifying assumptions on the life history of symbionts from their ingestion by host larvae to their release at the host death. We first assume that a fixed duration τ is needed before the ingested symbiont population reaches a carrying capacity $K(x)$ within a host (which also gives the number of symbionts released when a host dies at an age older than τ)—we call this the transient period for symbiont growth in the host. The carrying capacity $K(x)$ also depends on the symbiont trait x, which will be explained later. Although a logistic growth curve with the inflection point at τ would be more realistic, we approximate it as a step function with the transition at time τ (fig. 2*a*). By assumption, the host survivorship curve is exponential with parameter $u + x$; hence, the probability that the host dies within the time interval $(s, s + ds)$ is given by $(u + x)e^{-(u+x)s}ds$ (fig. 2*b*). With these assumptions, the expected total number Q of released symbionts per dead tubeworm is

$$Q(x) = \int_{\tau}^{\infty} K(x)(u + x)e^{-(u+x)s}ds = K(x)e^{-(u+x)\tau}. \quad (2)$$

Note that this model adopts a simplifying assumption that all tubeworms release the average number of symbionts when they die. More accurate formulations—a stage-structured model in which adult tubeworms are divided into immature and matured stages and an immature sessile tubeworm cannot release symbionts if it dies (app. A; apps. A–D are available online in the supplemental PDF) and an age-structured model in which the within-host symbiont density is formulated as a function of the age of an adult tubeworm (app. B)—give essentially the same results as this average model.

Because the host tubeworm completely depends on the nutrient supply of their symbionts' chemosynthetic product, their survivorship (and mortality) should strongly depend on how symbionts allocate their product for their host. The additional mortality x would increase if symbionts allocate fewer nutrients to their host. This may also affect the number $K(x)$ of symbionts that are released into the environment when a "matured" host (of age greater than τ) dies. Considering this potential trade-off, we expect that the carrying capacity of symbionts inside the host, $K(x)$, is a nondecreasing function of x. For simplicity, we assume either that it is constant ($K(x) = K_0$ if such trade-off is negligible) or that it is a linearly increasing function of x (e.g., $K(x) = kx$ with a positive constant k if such a trade-off is vital).

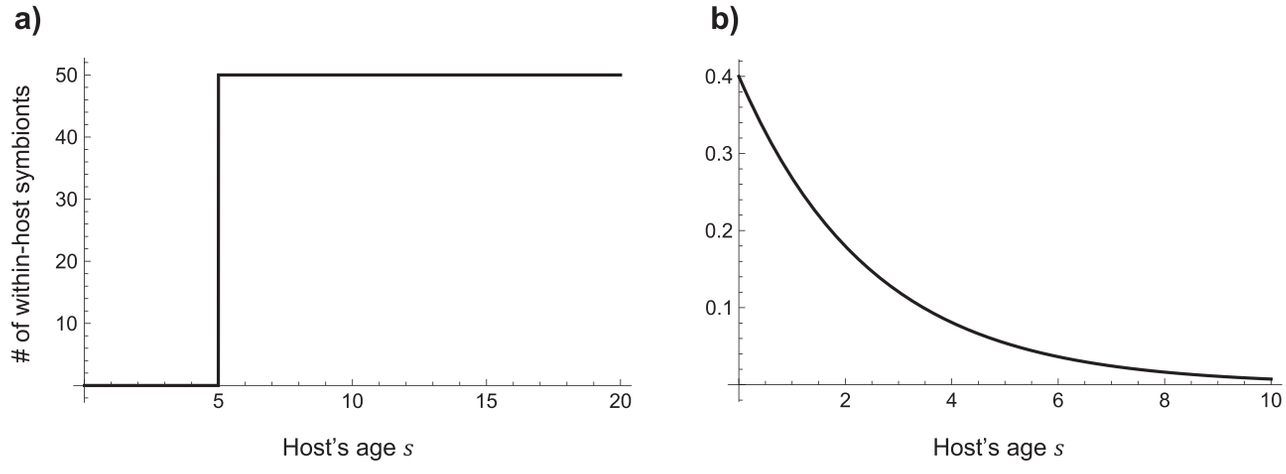

Figure 2: Hypothetical growth of symbiont in a tubeworm (*a*) and the corresponding distribution of the lifetime of the tubeworm (*b*). Our model assumes that a certain time ($\tau = 5$) is essential for symbionts to proliferate in a host up to the within-host carrying capacity.

For mathematical simplicity, we neglected the term $-\beta LS$, representing the loss of free-living symbionts resulting from their capture by tubeworm larvae, in the time change of S because it would be sufficiently smaller than the term $-\delta S$ by the natural death of free-living symbionts, as the life span of the free-living bacteria will be much shorter than half the time for the ingestion process.

Evolutionary Dynamics

To examine the evolutionarily stable host mortality rate controlled by symbionts, we examine whether a mutant strain of symbiont bacteria that adjusts the additional mortality of host to y can invade the resident symbiont population that adjusts it to x. We here assume that each larval tubeworm captures n free-living symbionts when it will be a metamorphosed adult, where n is assumed to follow a Poisson distribution with mean ν (>0) and the number $l\,(l = 0, 1, \ldots, n)$ of mutants among n symbionts captured by a larval tubeworm is assumed to follow a binomial distribution with parameters n and p, where p is the fraction of mutants in the free-living symbionts in the water column (fig. 3*a*). If a larval tubeworm captures l mutants and $n - l$ residents, the probability that the mutant eventually occupies the host is assumed to be

$$\Pi_l^n(y,x) = \frac{lg(y)}{(n-l)g(x) + lg(y)}, \tag{3}$$

where $g(x)$ is the competitive ability of a symbiont that adjusts host mortality rate to x; that is, the fixation probability of a mutant symbiont strain within a host is determined by the ratio of the whole mutant's competitive ability $lg(y)$ relative to that of the residents $(n - l)g(x)$. We assume that $g(x)$ is an increasing function of x because a symbiont with a larger x behaves more selfishly and uses its chemosynthetic products for competition within the host (fig. 3*b*).

The combined ecological dynamics for the densities S_r and S_m of free-living resident and mutant symbionts; the densities T_r and T_m of sessile tubeworms, the bodies of which are dominated by resident and mutant symbionts; and the density L of larval tubeworms are

$$\begin{aligned}
\frac{dL}{dt} &= -\beta LS + \lambda T - dL,\\
\frac{dT_r}{dt} &= \beta LS\rho(T)\sum_{n=1}^{\infty}\sum_{l=0}^{n} P_l^n(1 - \Pi_l^n(y,x)) - (u+x)T_r,\\
\frac{dT_m}{dt} &= \beta LS\rho(T)\sum_{n=1}^{\infty}\sum_{l=0}^{n} P_l^n\Pi_l^n(y,x) - (u+y)T_m,\\
\frac{dS_r}{dt} &= Q(x)(u+x)T_r - \delta S_r,\\
\frac{dS_m}{dt} &= Q(y)(u+y)T_m - \delta S_m,
\end{aligned} \tag{4}$$

where $S = S_r + S_m$ is the total density of free-living symbionts, $T = T_r + T_m$ is the total density of adult tubeworms, and

$$P_l^n = \frac{\pi_n}{1-\pi_0}\binom{n}{l}p^l(1-p)^{n-l},\ (n = 0, 1, \ldots; l = 0, 1, \ldots, n), \tag{5}$$

is the probability that a tubeworm larva captures n symbionts, of which l are mutants and $n - l$ are residents,

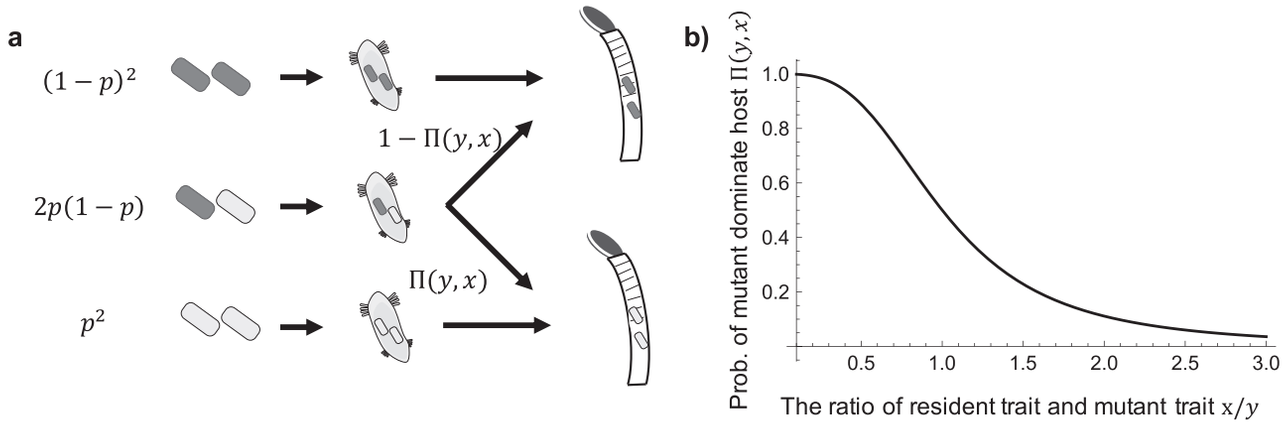

Figure 3: *a*, Schematic diagram of the coinfection of different strains of symbiont and their fixation in the tubeworms when the number of symbionts captured by a larva is two. *b*, Within-host competency and ratio of the resident-controlled host mortality rate to that of the mutant-controlled one when $g(x) = ax^m$, where a and m are positive constants. If two strains having different traits are captured by the same host larva, competition between them in the metamorphosed adult tubeworm leads to the fixation of only one of the strains. The probability that a mutant strain will eventually dominate in a host is $\Pi_1^2(y,x) = 1/(1 + (x/y)^m)$. A symbiont strain with a higher symbiont-controlled host mortality rate is more likely to dominate in a host. $n = 2$ and $m = 3$.

where $p = S_m/(S_r + S_m)$ is the frequency of mutants in the free-living symbiont bacteria in the environment and

$$\pi_n = \frac{\nu^n}{n!}e^{-\nu}, \quad (n = 0, 1, \dots), \tag{6}$$

is the probability that a larva captures n symbionts, where n is Poisson distributed with mean ν (>0). Given that n (≥1) symbionts are captured by a larva with probability $\pi_n/(1 - \pi_0)$, l are the mutants and $n - l$ are the residents with probability $[n!/l!(n - l)!]p^l(1 - p)^{n-l}$.

If all symbionts are the residents ($p = 0$), we see that $P_0^n = \pi_n/(1 - \pi_0)$ and $P_l^n = 0$ for all $l \geq 1$, which yields $\sum_{n=1}^{\infty}\sum_{l=0}^{n}P_l^n(1-\Pi_l^n(y,x)) = \sum_{n=1}^{\infty}\pi_n/(1-\pi_0) = 1$; hence, system (4) reduces to system (1) with $T = T_r$ and $S = S_r$.

Parameters

We scale rate parameters in units of years. In figures 4–6, the mortality rate of the free-living larvae is set to $d = 12$ per year (as the mean life span of a free-living larva is reported to be about 1 month; Marsh et al. 2001), the mortality rate of the free-living symbionts is set to $\delta = 16$ per year, and the baseline mortality rate of adult tubeworms is set to $u = 0.001$ per year. The capturing rate of symbionts by host larva is set to $\beta = 32$ (with which the mean waiting time until a free-living symbiont is captured by a host larva $1/\beta L^*$ is about 6 days at demographic equilibrium). Larval production rate is set to $\lambda = 2.4$ (the total lifetime production of free-living larvae into the local habitat $\lambda/(u + x)$ is about 220 per 10 cm^3 after dilution to surrounding area if $x = 0.01$). The maximum density of adult tubeworm T_0 is set to 10 per 10 cm^2 (with which tubeworms occupy all of the local habitats). As for symbiont density K within a host, the linear coefficient of trade-off ($K = kx$) is set to $k = 2{,}000$. For the case of no trade-off, $K = K_0 = 200$, with which the expected number of symbionts released from a dead host into the local habitat $Q(x) = K_0e^{-(u+x)\tau}$ is about 120 per 10 cm^3 if $(u + x)\tau = 0.5$.

Results

Persistence of Host-Symbiont Populations

We analyze the equilibrium states of the population dynamics in system (1). According to the analysis in appendix C, we see that the extinction equilibrium, $L^* = T^* = S^* = 0$, is always locally stable, implying that if the densities of hosts and symbionts are initially small, the populations cannot maintain themselves irrespective of their life-history parameters. This also implies that if there is a stable internal equilibrium where both tubeworm

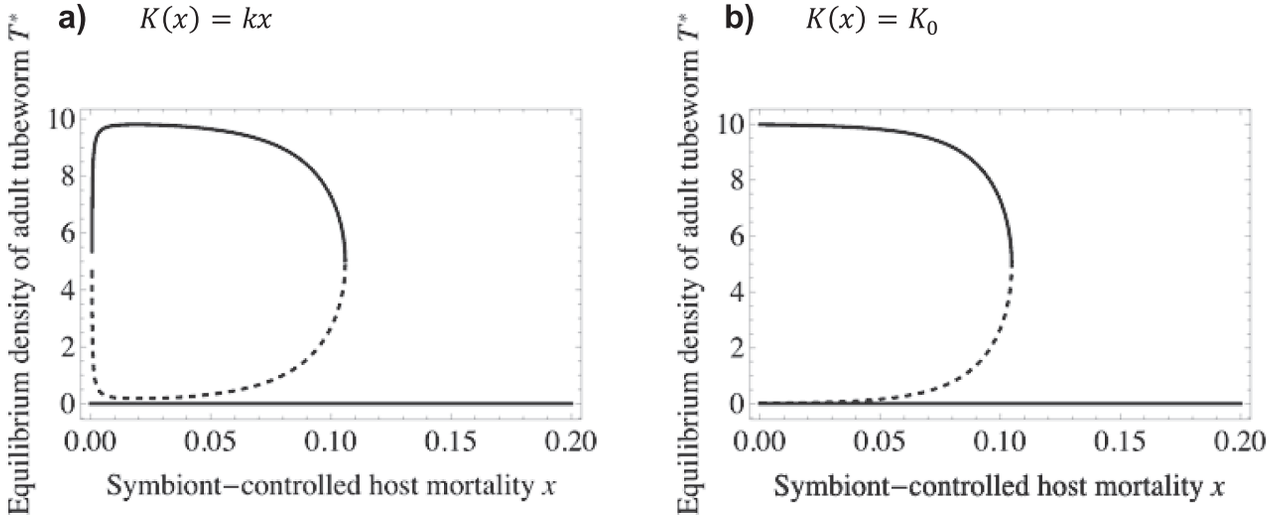

Figure 4: *a*, Equilibrium density of free-living symbiont T^* plotted against the symbiont-controlled host mortality rate x with $\rho(T) = 1 - T/T_0$ and $K(x) = kx$, obtained by equating the right-hand sides of system (1) to zero. The internal equilibrium density of symbionts, T^*, is obtained from the solution to a quadratic equation (eq. [C4] with $\rho = 1 - T/T_0$). For a range of intermediate x, the quadratic equation has two positive roots, corresponding to a pair of stable (solid line) and unstable (dashed line) equilibria. The pair of positive stable equilibrium and unstable equilibrium collides with each other and disappear at a high and a low threshold value of the symbiont-controlled host mortality rate x. The other parameters are $\beta = 32$, $\delta = 16$, $T_0 = 10$, $x = 2.4$, $d = 12$, $k = 2{,}000$, $\tau = 50$, and $u = 0.001$. *b*, Same diagram as in *a* for the case that there is no trade-off between within-host carrying capacity of symbionts and symbiont-induced host morality: $K(x) = K_0$. The other parameters are $\beta = 32$, $\delta = 16$, $T_0 = 10$, $\lambda = 2.4$, $d = 12$, $\tau = 50$, $u = 0.001$, and $K_0 = 200$.

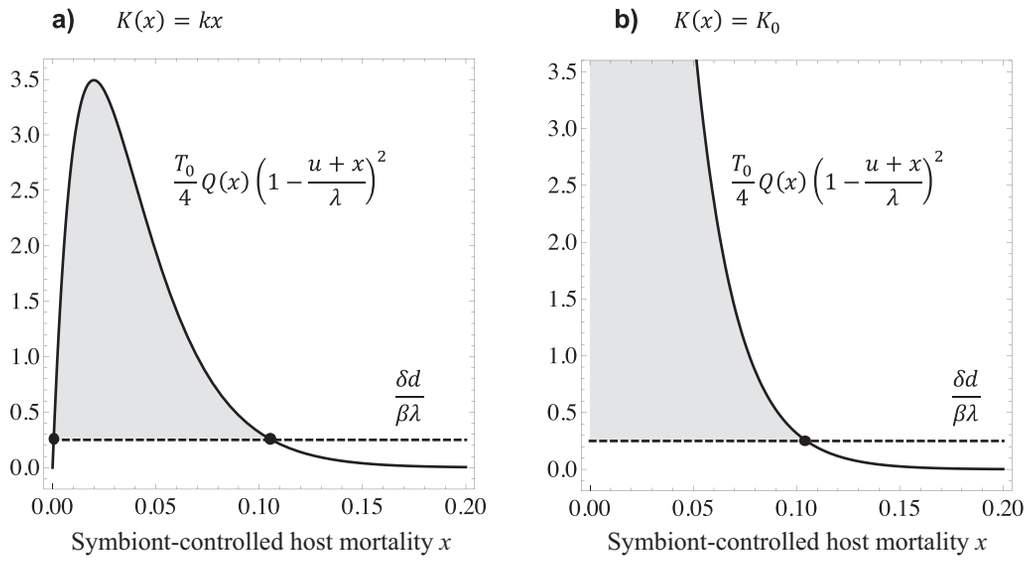

Figure 5: Parameter dependency of the demographic behavior of our mutualistic system against the symbiont-controlled host mortality x. The solid line shows the left-hand side of equation (7), $T_0Q(x)(1-(u+x)/\lambda)^2/4$, and the dashed line shows the right-hand side of equation (7), $\delta d/\beta\lambda$. *a*, $Q(x) = kxe^{-(u+x)\tau}$; *b*, $Q(x) = K_0e^{-(u+x)\tau}$. The saddle-node bifurcation occurs at the intersection point of the solid line and the dashed line, and the positive stable equilibrium can exist only when the solid line is above the dashed line. Parameters are $\beta = 32$, $\delta = 16$, $T_0 = 10$, $\lambda = 2.4$, $d = 12$, $\tau = 50$, $u = 0.001$, and $k = 2{,}000$ for *a* and $\beta = 32$, $\delta = 16$, $T_0 = 10$, $\lambda = 2.4$, $d = 12$, $\tau = 50$, $u = 0.001$, and $K_0 = 200$ for *b*.

and symbionts are stably maintained with positive densities, L^*, T^*, and S^*, our system (1) should show bistability. Because bistability is inherent in our symbiotic system whenever an internal equilibrium is locally stable, it is critical to find a saddle-node bifurcation point at which a pair of stable and unstable internal equilibria emerge. We see in appendix C that a saddle-node (blue-sky) bifurcation occurs at the set of parameters satisfying

$$\frac{T_0}{4}Q(x)\left(1-\frac{u+x}{\lambda}\right)^2 = \frac{\delta d}{\beta\lambda}, \tag{7}$$

and that there is bistability of internal and extinction equilibria if x lies in the range in between two saddle-node bifurcation points defined by equation (7) (figs. 4, 5), where a specific form of colonizing probability $\rho(T) = 1 - T/T_0$ is assumed.

We now highlight the results for the case where host mortality x induced by symbionts and their within-host carrying capacity $K(x)$ are proportional ($K(x) = kx$) and for the case where they are mutually independent ($K(x) = K_0$).

Linear Trade-Off: $K(x) = kx$. With a linear relationship between within-host carrying capacity and symbiont-induced host mortality, $K(x) = kx$, the number of symbionts released at host death, $Q(x) = kxe^{-(u+x)\tau}$, is a one-humped function of x. Under this trade-off, there are two thresholds in the symbiont-induced host mortality rate x, such that the mutualistic system cannot maintain itself if the symbiont-induced host mortality is lower than a threshold or higher than another threshold (fig. 4*a*). As shown in figure 4*a*, for a range of intermediate symbiont-induced host mortalities x the population dynamics have two stable equilibria: $T^* = 0$ and a positive equilibrium (solid lines) as well as an unstable equilibrium in between them (dashed line). Because the tubeworm and the symbionts are mutually indispensable partners, a sufficient density of either species is necessary to maintain the other; therefore, even in the parameter region in which a stable positive equilibrium exists, both populations would become extinct if the initial density of either species is not sufficiently large. The range of host mortality for stable maintenance of this mutualistic system is widened with increasing capturing rate of symbionts by host larvae (β), production rate of host larvae (λ), and within-host carrying capacity of symbionts (K), while it is narrowed with increasing mortality rates of free-living symbionts (δ) and host larva (d), competition coefficient in the colonization of the sessile host ($1/T_0$), and the transient period (τ) until the within-host density of symbionts reaches its carrying capacity (fig. 5*a*).

The reason why both tubeworms and symbiont bacteria become extinct when the hosts live too long (i.e., when the host mortality rate falls below the lower threshold level) is that because of the trade-off, the number of symbionts

released at the death of a long-lived host is too small to sustain this deep-water ecosystem. If a symbiont that induces a low host mortality rate tends to allocate more resources for its host rather than for its own growth, we expect to have a positive trade-off between x and $K(x)$. Our result shows that such generous symbionts could drive the populations of both hosts and symbionts to become extinct, as a result of the shortage of symbiont supply.

No Trade-Off: $K(x) = K_0$. In contrast, if K is independent of x, $K(x) = K_0$, the lower threshold host mortality for the maintenance of the mutualistic system disappears. Indeed, with a constant K, a smaller x always makes the maintenance of the mutualistic system easier (figs. 4*b*, 5*b*). Even without a trade-off between x and K, the host's long life leads to the shortage of symbiont release, limiting the free-living symbiont density in the environment. However, as for tubeworms, this shortage of symbiotic partners necessary for their recruitment can be well balanced with the low mortality of the adults (the low-risk, low-return strategy pays if within-host carrying capacity remains high for a symbiont strain that induces a low host mortality rate).

Evolution of Symbiont-Controlled Host Mortality Rate

The invasibility of a mutant with symbiont-induced host mortality y in the population of resident with symbiont-induced mortality x is examined with the dynamics for mutant-carrying tubeworm T_m and free-living mutant symbiont S_m when the mutant is rare so that the terms with p^2 or higher can be ignored in system (4):

$$\begin{aligned}\frac{dS_m}{dt} &= Q(y)(u+y)T_m - \delta S_m,\\ \frac{dT_m}{dt} &= \beta L^* \rho(T_r^*)\sum_{n=1}^{\infty}\frac{\pi_n}{1-\pi_0}n\frac{g(y)}{(n-1)g(x)+g(y)}S_m\\ &\quad -(u+y)T_m,\end{aligned} \tag{8}$$

(see app. D for derivation), where L^* and T_r^* are the equilibrium larval density and adult tubeworm density in the resident population. The invasion fitness $s(y,x)$ of a mutant with trait y in the population of a resident with trait x is then defined as the dominant eigenvalue of the next generation matrix calculated from equation (8) in appendix D:

$$s(y,x) = \sqrt{\sum_{n=1}^{\infty}\frac{\pi_n}{1-\pi_0}n\frac{g(y)}{(n-1)g(x)+g(y)}\frac{Q(y)}{Q(x)}}. \tag{9}$$

It will then be shown in appendix D that the continuously stable (i.e., both convergence and evolutionarily stable) symbiont-induced mortality x^* maximizes the weighted product of the within-host competency $g(x)$ and the expected number of symbionts $Q(x)$ released at the death of a host:

$$g(x)^{(1-E_+[1/n])}Q(x) \to \text{maximum}, \tag{10}$$

where

$$E_+\left[\frac{1}{n}\right] = \sum_{n=1}^{\infty}\frac{1}{n}\pi_n \Big/ \sum_{n=1}^{\infty}\pi_n = \sum_{n=1}^{\infty}\frac{\nu^n}{n\cdot n!}\Big/\sum_{n=1}^{\infty}\frac{\nu^n}{n!} \tag{11}$$

is the expectation of the reciprocal of the number n of symbionts captured by a larva averaged over all positive n's.

Here, we examine the evolution of symbiont-controlled host mortality rate by assuming that within-host competitive ability of symbiont is a geometrically increasing function of the symbiont-controlled host mortality rate x:

$$g(x) = ax^m, \tag{12}$$

where a is a positive constant and m (>0) is a parameter that measures the shape of the trade-off—the competitive ability increases acceleratingly with x if $m > 1$, while it shows a diminishing return if $0 < m < 1$. We then examine, as before, two contrasting trade-off shapes between the within-host carrying capacity of symbionts and the symbiont-controlled host mortality rate, K and x: a linear trade-off, $K(x) = kx$, and no trade-off, $K(x) = K_0$.

The Case $K(x) = kx$. We here assume that the number of symbionts released at the death of a host is a linearly increasing function of symbiont-induced host mortality rate x: $K(x) = kx$, where k is a positive constant. With this assumption, $Q(x) = kxe^{-(u+x)\tau}$, and the evolutionarily singular strategy x^* that maximizes $g(x)^{(1-E_+[1/n])}Q(x)$ is

$$x^* = \left(1 - E_+\left[\frac{1}{n}\right]\right)\frac{m}{\tau} + \frac{1}{\tau}. \tag{13}$$

The condition for the evolutionarily and convergence stability is always satisfied:

$$\frac{d^2}{dx^2}\left\{\log(g(x)^{(1-E_+[1/n])}Q(x))\right\}\Big|_{x=x^*} = -\left(1 - E_+\left[\frac{1}{n}\right]\right)\frac{m}{x^{*2}} - \frac{1}{x^{*2}} < 0.$$

Thus, the evolutionarily singular strategy is always evolutionarily and convergence stable (i.e., it is a continuously stable strategy [CSS]; Christiansen 1991) .

Figure 6*a1* and figure 6*a2* show the CSS symbiont-controlled host mortality rate x^* as a function of the transient period τ for symbiont growth in a host (solid line) as well as the range of host mortality rate that allows the stable maintenance of mutualistic species (shaded region). It

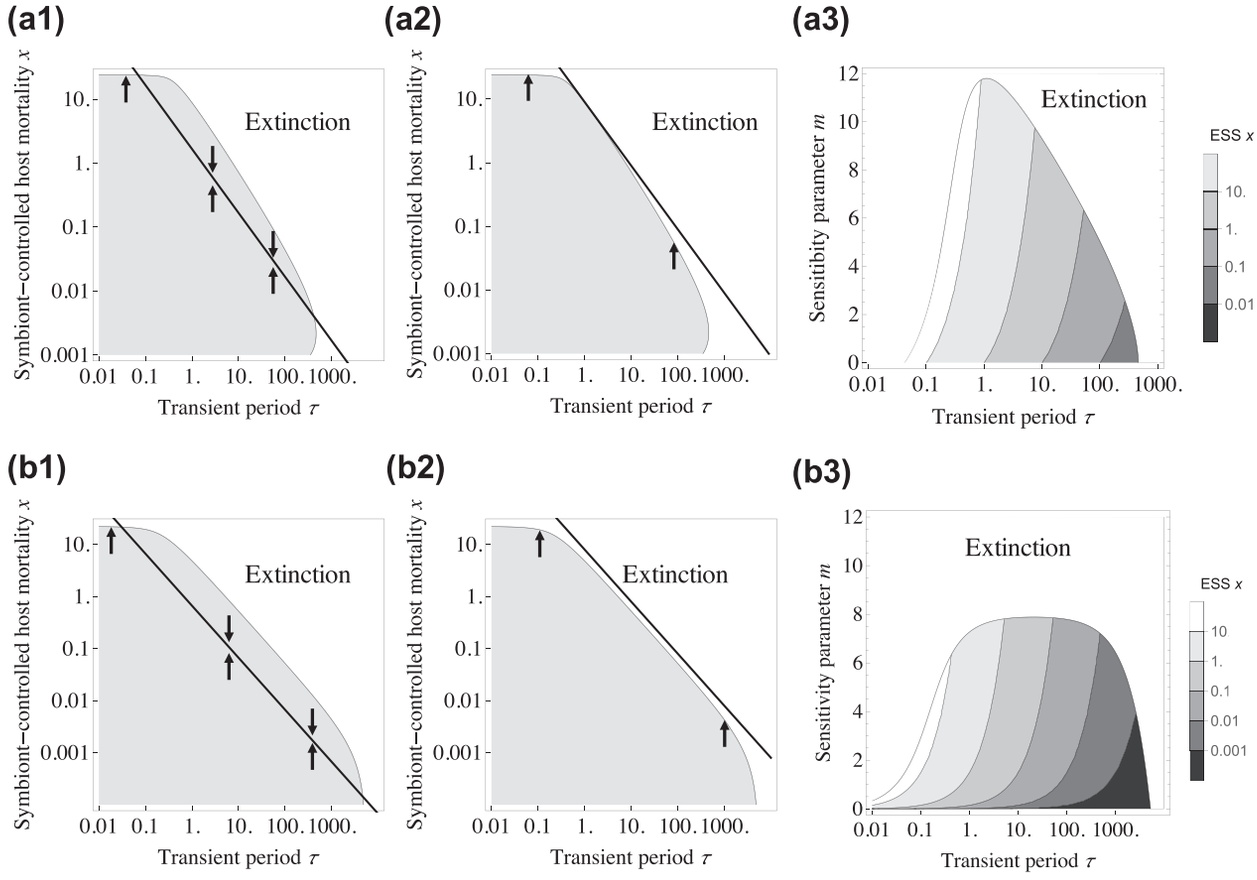

Figure 6: *a1*, *a2*, Parameter dependency of the continuously stable strategy (CSS) symbiont-controlled host mortality rate (solid line) and the range of host mortality rate for stable maintenance (gray area) on the transient period τ for different values of sensitivity parameter m of the relationship $g(x) = ax^m$ between competitive ability and symbiont-controlled host mortality rate x. For *a1*, $m = 1$; for *a2*, $m = 12$. $\rho(T) = 1 - T/T_0$ and $K(x) = kx$. If the CSS value is within the gray area, this mutualism can be evolutionarily maintained. However, for the ranges of transient period τ in which the solid curve is outside the shaded area, the symbiont-controlled host mortality evolves to the edge of the shaded area and drives the populations to extinction (evolutionary suicide). Parameters: $\beta = 32$, $\delta = 16$, $T_0 = 10$, $\lambda = 2.4$, $d = 12$, $k = 2{,}000$, $u = 0.001$, $n = 3$; a can be arbitrary (it does not affect the result). *a3*, Contour of the CSS symbiont-controlled host mortality rate plotted against the transient period τ for sufficient symbiont growth within host and the sensitivity parameter m of the competitive ability function described above. The contour is plotted with a log scale. In the white region, the symbiont-controlled host mortality rate evolves toward the edge of the host-symbiont persistence (evolutionary suicide). *b1–b3*, Same diagrams as in *a1–a3* but with $K(x) = K_0$, with $K_0 = 200$. The sensitivity parameter m of the competitive ability on symbiont-controlled host mortality rate is $m = 1$ in *b1* and $m = 12$ in *b2*. *b3*, Contour of the CSS symbiont-controlled host mortality rate plotted against the transient period τ and the sensitivity parameter m. The contour is plotted with a log scale.

shows that when the transient period τ is either too large (i.e., if it takes a long time before symbionts reach the carrying capacity in the host) or too small (i.e., symbionts reach the carrying capacity soon), the CSS symbiont-controlled host mortality rate is placed outside the range of host mortality rate for stable persistence of both populations (fig. 6*a1*–6*a3*). In these cases, the evolution of symbiont-controlled host mortality rate leads to the extinction of both species (evolutionary suicide; Abrams et al. 1993; Matsuda and Abrams 1994; Haraguchi and Sasaki 1996; Gyllenberg and Parvinen 2001; Ferrière et al. 2002; Jones et al. 2009).

When the sensitivity parameter m is large, the CSS symbiont-controlled host mortality rate becomes large (see the contours in fig. 6*a3*) and the range where the population can be maintained becomes narrower (from fig. 6*a1* to 6*a2*; more easily seen in fig. 6*a3*). If m is too large, the population cannot be maintained regardless of the value of the transient period τ (fig. 6*a3*).

Figure 6*a3* shows that there are two threshold values of the transient period τ and a single threshold value of the sensitivity (shape) parameter m for the host and symbiont populations to be stably maintained. Figure 6*a3* also show

that within the region for population maintenance, the CSS symbiont-controlled host mortality rate x^* is high toward the top left direction (short τ and large m) and low toward bottom right direction (long τ and small m). Therefore, for a long-lived tubeworm (a small x^*) in the tubeworm–sulfur-fixing bacteria symbiotic system to be maintained demographically and evolutionarily, the growth of the symbiont should be sufficiently slow (a sufficiently long τ) and the trade-off between competitive ability and symbiont-controlled host mortality rate should be sufficiently weak (a small m), although a too long τ leads to evolutionary double suicide. The tendency that a longer transient period τ and an insensitive trade-off between host mortality rate and competitive ability promote the evolution of low host mortality rate is found to be robust for the changes in the trade-off function forms (see below).

The Case $K(x) = K_0$. We here assume that the number of symbionts released at the death of a host is independent of the symbiont's trait x: $K(x) = K_0$ (constant). With this assumption, $Q(x) = K_0 e^{-(u+x)\tau}$, the evolutionarily singular strategy x^* that maximizes $g(x)^{(1-E_+[1/n])}Q(x)$ is

$$x^* = \left(1 - E_+\left[\frac{1}{n}\right]\right)\frac{m}{\tau}. \tag{14}$$

As before, the condition for its evolutionary stability and convergence stability is always satisfied:

$$\frac{d^2}{dx^2}\left\{\log(g(x)^{(1-E_+[1/n])}Q(x))\right\}\Big|_{x=x^*} = -\left(1 - E_+\left[\frac{1}{n}\right]\right)\frac{m}{x^{*2}} < 0.$$

Thus, the evolutionarily singular point is always both evolutionarily and convergence stable.

Comparing this result (eq. [14]) with the case of a linear trade-off between K and x (eq. [13]), we see that the evolved mortality is lower by $1/\tau$. Figure 6*b3* shows the contour of the evolved symbiont-controlled host mortality rate (eq. [14]) plotted against τ and m. Comparing to the case where K is linearly increasing with x (fig. 6*a*), the range of stable maintenance of the population is widened for small m. This is because the CSS host mortality rate (eq. [14]) is decreased by $1/\tau$ from when the carrying capacity K is linearly increasing with x, and hence x^* is easier to stay in the shaded region for the stable maintenance of symbiont and host populations (fig. 6*b1*–6*b3*). In the same vein, the persistence of the population becomes easier when m is increased than in the case of linearly increasing $K(x)$.

The reason why the additional term, $1/\tau$, is missing in the CSS host mortality rate is that the number of released symbionts from a dead host, $Q(x)$, is maximized at $x = 1/\tau$ if the carrying capacity within a host increases linearly with x, while in the absence of x dependency in K, $Q(x)$ is maximized at $x = 0$.

That $Q(x) = K_0 e^{-(u+x)\tau}$ is maximized at $x = 0$ indicates that the CSS host mortality rate becomes zero if the symbiont-controlled host mortality rate does not affect the competitive ability of the symbiont strain (i.e., if $g(x) = \text{const}$ or, in the case of $g(x) = ax^m$, if $m = 0$); see eq. [14] with $m = 0$. However, when $K(x)$ is an increasing function of x, as discussed in this section, the CSS x^* remains finite even without the trade-off between competitive ability and the symbiont-controlled host mortality rate ($g(x) = \text{const}$). We confirmed the robustness, for different functional forms of $g(x)$, of our result that the CSS host mortality rate becomes higher and the stable maintenance becomes hard when the competitive ability is sensitive to increasing the host mortality rate (see the "Supplementary Information 1" section in the supplemental PDF).

Evolution of Host-Controlled Host Mortality Rate

So far, we have discussed the evolution of host mortality rate when it is controlled by symbionts. We here examine the case where the host mortality rate is controlled by the host itself. As the simplest case, we first assume that the host-controlled host mortality rate mortality x_H is independent of other life-history parameters of the host, such as its fecundity. If this is the case, it is obvious that smaller mortality is always selected for. As both symbiont and host populations become extinct as the host mortality rate becomes too low, we conclude that if the host mortality rate is controlled by itself, if there is no trade-off between mortality and another life-history trait, and if there is a linear trade-off between K and x ($K(x) = kx$), the evolution of host-controlled host mortality always leads to the extinction of both symbiont and host populations.

Next, we examine a more interesting case in which lower mortality of the host is compensated by a lower fecundity λ, as observed in *Drosophila* (Djawdan et al. 1996): $\lambda = \lambda(x_H)$. If this is the case, the invasion fitness of a mutant host with mortality y_H in the population of residents with mortality x_H is

$$s_H(y_H, x_H) = \sqrt{\frac{\lambda(y_H)\beta S^* \rho(T_r^*)}{(u + y_H)(\beta S^* + d)}} = \sqrt{\frac{\lambda(y_H)}{(u + y_H)}\frac{u + x_H}{\lambda(x_H)}}, \tag{15}$$

where S^* and T_r^* are the equilibrium densities of free-living symbionts and adult tubeworms in the population of the symbiont and the resident host. From this invasion

fitness, we see that the evolution of the host-controlled mortality x_H maximizes the Fisher's reproductive value $\lambda(x_H)/(u + x_H)$ of the host. The evolutionary consequence then depends on the shape of a trade-off between fecundity and mortality (i.e., between fecundity and mean lifetime). If host fecundity $\lambda(x_H)$ increases faster than linearly with x_H, then x_H always evolves to infinity, and hence neither the host nor the symbiont population can be maintained. In contrast, if it increases slower than linearly, an intermediate evolutionarily singular host-controlled mortality x_H^* exists that satisfies $\lambda'(x_H^*) = \lambda(x_H^*)/(u + x_H^*)$. Therefore, when this intermediate evolved mortality falls into the range where the population can be maintained, the mutualism can be evolutionarily maintained with the evolution of host-controlled mortality.

It is, however, difficult to assume that symbionts will never affect the host mortality rate. The component of mortality that is controlled by symbionts is free from the fecundity-mortality trade-off of the host, and hence if both host-controlled and symbiont-controlled host mortality rates evolve simultaneously, they should evolve independently, although the persistence of the population is affected by either of the traits. We discuss these points later.

Discussion

We modeled the demography and evolution of the mutualistic system between the tubeworm and sulfur-oxidizing bacterium to answer the following questions. First, how can this mutualism be maintained with long-lived hosts such as *Lamellibrachia luymesi*, which lives more than 200 years, despite the fact that symbiont bacteria can be released into the environment only when the host dies? Second, how and why do such long-lived hosts evolve? Under the presence of a positive trade-off between symbiont-controlled mortality and the symbiont burden in the host, our model revealed the following results: (1) this mutualism can be stably maintained when the host mortality rate is intermediate (fig. 4*a*) or smaller than a threshold (fig. 4*b*) and (2) a long-living host can evolve and be maintained when the transient period (τ) for symbionts to sufficiently proliferate inside it is long and the sensitivity of the symbiont-controlled host mortality rate for the competitive ability (i.e., m) is weak (fig. 6). If there is no trade-off between symbiont-controlled mortality and the symbiont burden in the host, the mutualistic system can more easily be maintained in broader parameter space and for long-lived hosts, but the aforementioned parameter dependence remains similar (compare fig. 6*a3* with fig. 6*b3*), as a long transient period is necessary to reward symbionts for delaying host mortality and a weak within-host competition is necessary for symbionts not evolving toward extinction. Moreover, these tendencies are insensitive to the specific $K - x$ trade-off shape.

Demographic Stability of Tubeworm–Sulfur-Fixing Bacteria Mutualistic System

We have stated that the high capturing rate of symbionts by host larvae (β), the high rate of larval production (λ), and the large within-host carrying capacity (K) of symbionts widen the range of host mortality rate within which the stability of the mutualistic species and their relationship is maintained. Conversely, the high mortality of free-living symbionts (δ) and host larva (d), the greater competition (i.e., smaller T_0) in the colonization of the sessile host, and a longer time (τ) until the within-host density of symbionts reaches its carrying capacity narrow the range of host mortality rate necessary to stabilize the mutualistic relationship and species (fig. 5). Little is known of the values of these parameters in nature, but tubeworms are believed to produce a large number of larvae, suggesting that λ would be large (Tyler and Young 1999). The number of symbionts released after the death of an adult tubeworm is also known to be very large ($\sim 7.0 \times 10^5$; Klose et al. 2015), suggesting a large value of within-host carrying capacity K of symbionts. The life span of a larva is about 1 month (Marsh et al. 2001), suggesting a per-year mortality of larva d is around 12. These facts suggest that the range of host mortality rates necessary to maintain this mutualism may not be too narrow.

If the within-host carrying capacity is an increasing function of a symbiont's strategy of controlling the host mortality rate—for example, if it is a linearly increasing function ($K(x) = kx$)—the corruption of mutualism occurs when either the host mortality rate is too high or too low. In contrast, if the within-host carrying capacity of the symbiont is independent of the symbiont's strategy of controlling host mortality rate ($K(x) = K_0$), the corruption of mutualism occurs only when the host mortality rate is too high. The reason why the lower threshold of the symbiont-induced host mortality disappears in this case is that a shortage of free-living symbiotic partners necessary for the recruitment of tubeworms can be well balanced with the low mortality of the adults (low-risk, low-return strategy pays if the within-host carrying capacity remains high for a symbiont strain that induces a low host mortality rate).

Because the symbiont that makes their host live longer should allocate their chemosynthetic products more to their host than to their own, we expect a positive trade-off between their within-host carrying capacity and the host mortality rate they induce. Therefore, we expect that the host mortality should not be too low (i.e., it should be intermediate) for stable maintenance of this mutualistic system.

When the host mortality rate is greater than but close to the lower threshold for stable maintenance of the system, a pair of stable and unstable internal equilibria are close to each other, and either demographic or environmental stochasticity can easily drive the population into the basin of attraction toward population extinction (note that the extinction equilibrium is always locally stable in our model, as in other models of obligate mutualisms). Conversely, as the densities are far distant between extinction equilibrium and unstable internal equilibrium even near the threshold point, once the population is dragged near extinction, the mutualistic system can hardly escape extinction.

Evolution of Symbiont-Controlled Host Mortality

Our model reveals that when the host mortality rate is controlled by symbionts, a long-living host evolves if the time τ for symbionts to reach within-host carrying capacity is large or if the sensitivity m of within-host competency to the symbiont-induced host mortality rate x is small. Conventional wisdom relates a great diversity in the growth rate and the longevity of tubeworms to the stability of vent flow habitats: long-lived tubeworm species, such as *L. luymesi*, grow very slowly; in contrast, relatively short-lived tubeworm species are known to grow very fast (Fisher et al. 1988). This marked difference in a tubeworm's lifetime has been ascribed to the difference in habitat stability (Gibson et al. 2010; Durkin et al. 2017) as follows: species living in relatively unstable hydrothermal vents grow extremely fast and are relatively short-lived (Fisher et al. 1988; Hessler et al. 1988), while species living in relatively stable cold seeps grow extremely slowly and are relatively long-lived (Fisher et al. 1997; Julian et al. 1999). A similar relationship between habitat stability and growth rate/longevity is found in the different morphotypes of a single tubeworm species, *Redgeia piscesae* (Urcuyo et al. 2007).

Our model argues that the longevity of tubeworms should be shorter in the vent with higher sulfidic inflow. In the tubeworms living in a higher vent flow environment, the endosymbiont could grow up earlier by enjoying higher sulfidic inflow. Our model then suggests that evolution leads to higher symbiont-controlled host mortality. Our hypothesis would be supported if comparable data on the symbiont-genome among different host species living in different sulfidic flow environments would show greater numbers or higher expression of virulence-related genes in the habitats with a shorter lifetime of tubeworm and rapid symbiont growth.

We have assumed that the mortality of tubeworms can largely be affected by their endosymbionts. Genetic variations in the endosymbionts of the above-mentioned tubeworm species *R. piscesae* are found between individuals living in different environments as well as between individuals in the same aggregation (Chao et al. 2007). In addition, a relatively high dN/dS ratio (the ratio of nonsynonymous to synonymous substitutions) is observed in the endosymbiont genes related to the virulence factors against eukaryotic cells or competing bacteria when compared between endosymbionts of the tubeworms in different habitats (Perez and Juniper 2016), suggesting adaptive diversification of these virulence-related genes in varying environments.

The symbiont-controlled host mortality x should evolve to zero or a physiological minimum if there is no trade-off with the within-host carrying capacity K or with the competitive ability in a host. An intermediate host mortality rate evolves with either of the trade-offs. For example, with only the trade-off between host mortality x and competency (i.e., if the within-host carrying capacity K is independent of x—the case we discussed in "The Case $K(x) = K_0$"—and would be expected if a host species has a specialized organ to house symbionts), the evolved symbiont-controlled host mortality rate is determined by the balance between two contrasting factors: a stronger competency by having a larger x and a lower risk of dying before filling the host by having a lower x. In contrast, with only the trade-off between host mortality x and within-host carrying capacity K, the evolved symbiont-controlled host mortality rate is determined again by the balance between two factors, but the factor favoring a larger x is the larger number $K(x)$ of released symbionts. If there are two trade-offs, the evolved host mortality is larger than when there is only one of the trade-offs. Indeed, the ESS host mortality under the two trade-offs is given simply by the sum of the ESS mortalities under each trade-off (eq. [13]).

Although we assume that the main factor that determines the host mortality rate could be the effect of symbiont-controlled mortality, we cannot neglect the effect of host evolution. If there is a trade-off between host-controlled host mortality x_H and host fecundity, $\lambda = \lambda(x_H)$ and if the shape of the trade-off function $\lambda(x_H)$ increases slower than linearly with x_H, an intermediate evolutionarily stable host-controlled mortality exists because the evolution simply maximizes $\lambda(x_H)/(u + x_H)$. In general, the host mortality rate can be made up of the sum of the symbiont-controlled and the host-controlled components of mortality. If we consider the evolution of symbiont-controlled host mortality, we can set the host-controlled component of host mortality as the base mortality $u = x_H$. As we have shown, the evolution of symbiont-controlled host mortality is independent of the base mortality (eq. [10]), and hence our results under the assumption of symbiont-controlled host mortality do

not change if there is a host-controlled component of mortality.

Evolutionary Double Suicide

Our analysis shows that the evolution of symbiont-controlled host mortality rate can lead to the extinction of both host and symbiont species (evolutionary double suicide) by driving the host mortality rate toward outside its range for stable maintenance of a mutualistic system. Such evolutionary double suicide occurs when symbionts grow either too quickly or too slowly (i.e., a transient period [τ] is too short or too long to fill the tubeworm) or when their competition within a host is too strong (i.e., m is too large). The evolutionary double suicide by the escalation of host mortality rate that occurs when the sensitivity m of competency is high or the transited period τ is short can easily be understood: a strong competition within a host favors a higher host mortality rate in the evolution of symbionts, and a higher host mortality rate results in a stronger competency in a host because they are positively correlated in our model. This positive feedback makes it difficult to stop the evolutionary escalation of host mortality rate before it dooms the mutualistic system to extinction. A short transient period to fill the host also promotes the evolution toward a high host mortality rate because it costs little to the symbionts to kill the host early. Therefore, the evolutionarily stable (ESS) host mortality rate for short τ becomes too high and exceeds its threshold for extinction. The evolutionary double suicide that occurs for long τ requires more subtle interpretation. The evolutionarily stable symbiont-controlled host mortality is independent of the equilibrium density of either tubeworms or symbionts, whereas as τ is increased, the range of host mortality rate required for the demographic maintenance of both species becomes too narrow to hold the ESS host mortality rate in it (fig. 6). In other words, this ESS mortality is much less sensitive to the transient period than to the thresholds for demographic maintenance, and it ultimately finds itself outside the range of demographic maintenance over a long enough transient period.

As stated, for the evolutionary maintenance of mutualism, the parameter τ and the efficiency of increasing the host mortality rate for competitive ability (i.e., m) must lie within an intermediate range. Any environmental disturbance of some of these parameters can collapse the mutualism between the two species. This is particularly pronounced when a long-living host is evolutionarily stable. For such a low host mortality rate to be evolutionarily stable, τ must be sufficiently large, which makes the range of host mortality rate that leads to a stable demographic equilibrium narrower than when τ is intermediate or small. Furthermore, for a sufficiently large τ, the population can never be stable regardless of the value of host mortality rate; therefore, a mutualistic system with a long-living host is hard to exist. When it does exist, it is fragile against disturbances to the parameters of stability. This might agree with the fact that known long-living tubeworms, such as *L. luymesi*, live in cold seeps, which is considered to be a very stable environment (Gage and Tyler 1992).

General Implication for Other Host-Symbiont Relationships

Although the present article is motivated by an interesting mutualism of deep-sea tubeworms and a sulfur-oxidizing bacterium, the results have general implications for other host-symbiont relationships.

We have shown that evolutionary double suicide (the evolution-driven extinction of both host and symbiont species) occurs robustly in our model. This is not limited to our model but should apply to other obligate mutualisms with horizontally transmitted symbionts, as it is an inherent outcome of coexistence-coextinction bistability in their ecological dynamics (see fig. 4). As both host and symbiont species rely on the other partner species to grow efficiently, the populations cannot recover once they fall off to low densities. Extinction is thus inevitable in such mutualisms if populations are put at low densities: with a low density of host or symbiont species, the chance becomes remote for finding the partner, and the scarcity in symbiotic service further reduces its growth potential (we call this a low-density spiral). In such circumstances, a coexistence equilibrium where either species is densely populated can disappear out of the clear blue sky (saddle-node bifurcation) when a parameter is continuously varied by an evolutionary change in a trait of one of the species, leading to a dive into coextinction. As Gyllenberg and Parvinen (2001) have shown, that population dynamics show a discontinuous transition to extinction is a prerequisite of evolutionary suicide because if the transition to extinction takes place continuously, a mutant that increases its growth can always invade in the environment where the population is doomed to extinction with the resident, pushing the state back to the region for its existence. This is the reason why we expect evolutionary double suicide to robustly take place in obligate mutualisms with the horizontal acquisition of symbionts

This ecological fragility (tendency to coextinction out of the clear blue sky) and evolutionary fragility (inclination toward evolutionary suicide) should be relaxed if either the degree of interdependency between partner species or the way hosts acquire symbionts is changed. The ecological fragility is less likely if mutualistic interaction is facultative, that is, when either free-living host or free-living symbiont can maintain themselves. In such a case,

the populations can get out of the low-density spiral by a gradual increase of one of the species, which then recovers the other and reinforces itself with highly profitable mutualistic service. Likewise, the mutualistic system should be free from the low-density spiral if symbionts are vertically transmitted from a host to its offspring. Without the ecological fragility of blue-sky coextinction, evolutionary suicide will not take place either.

Such evolutionary double suicide is hardly expected in host-parasite relationships. If a symbiont species is parasitic, its evolutionary escalation for host exploitation is expected to stop before endangering the persistence of the host population. For example, in host-parasite dynamics with a free-living parasite stage, parasite virulence evolves to maximize its basic reproductive ratio R_0 in a stable population or to maximize its intrinsic growth rate in a growing population (Bonhoeffer et al. 1996); in either case, an evolutionary change makes the persistence of parasite easier. The R_0 maximization principle holds in many models of unstructured host populations for the evolution of virulence (May and Anderson 1983; Bull 1994; for a review, see Lion and Metz 2018) where coextinction due to the evolution of virulence hardly occurs. The evolution of virulence may result in coextinction of host and parasite if the transmission is perfectly frequency dependent, as in idealized vector-borne disease (Boots and Sasaki 2003) or if dispersal and transmission are limited between neighbors in spatially explicit models (Haraguchi and Sasaki 2000; Boots and Sasaki 2002), but the conditions are more restrictive than in the case of obligate mutualisms (i.e., ecological rescue by negative feedback in the host-parasite system should efficiently be masked by frequency-dependent transmission or viscosity in dispersal/transmission).

Ecological and evolutionary instability of mutualisms has already been highlighted in the literature (Ferrière et al. 2002; Sachs and Simms 2006; Rezende et al. 2007; Jones et al. 2009; Colwell et al. 2012). The dynamical vulnerability of obligate mutualisms has been shown in Lotka-Volterra models since the 1970s (May 1976; Briand and Yodzis 1982). Mutualisms are also thought to be evolutionarily vulnerable to exploiters. Two pathways to mutualism breakdown proposed so far are (i) continued invasions of cheater mutants inside mutualism (Trivers 1971; May 1976; Axelrod and Hamilton 1981) giving rise to the erosion of mutualistic services (Ferrière et al. 2002) and (ii) exposure to parasites of mutualisms (a species outside mutualism exploits symbiotic services without reciprocation; Yu 2001; Sachs and Simms 2006; Jones et al. 2009). Our study focuses on yet another pathway to mutualism breakdown—evolutionary double suicide triggered by an evolutionary shift in a life-history parameter, in our case, the timing of host killing—and gives theoretical insight into conceptual and simulation models for coextinction in mutualistic communities (Rezende et al. 2007; Colwell et al. 2012), which have shown that extinction of one species in a mutualistic network tends to set off a chain reaction coextinction of other species.

In our deep-sea mutualism, symbionts are released only at the very end of its symbiosis, that is, at the time the host dies. The later the symbionts kill the host, the greater the number of symbionts released at the host's death should be. In our model, symbiont-induced host mortality is adjusted to make the mean host lifetime sufficiently greater than the time at which symbiont density is sufficiently increased in a host. Even if a symbiont can freely choose the timing to kill the host, the optimum timing should be around the time when the increase in within-host density is saturating. Such delayed host killings are widely observed in pathogens. A pathogen's incentive for keeping its host alive becomes less and less important as the host approaches its end—game-theoretical and dynamic optimization analysis indeed reveals that pathogens should become more virulent toward the end of host life (Axelrod and Hamilton 1981; Bremermann and Pickering 1983; Sasaki and Iwasa 1991). Long latency in human immunodeficiency virus (HIV) infection, for example, can be interpreted as a viral strategy to take advantage of asymptomatic infections before the onset of immunodeficiency; when combined with the saturated transmission rates with viral set points of HIV (Fraser et al. 2007), earlier explosive growth should only reduce its R_0 (Sasaki and Iwasa 1991). Adaptive significance of delayed killing in male killer cytoplasmic agents (late male killers in mosquitoes) can also be ascribed, as in our system, to sufficient accumulation of symbionts for their horizontal transmission (Hurst 1991).

Future Perspective

Our model has clarified that extremely indispensable mutualism between tubeworms and symbiont bacteria can be maintained if the host mortality rate lies within an intermediate range. It also revealed that within a specific range of parameters for within-host growth and the strength of intrahost symbiont competition, the host mortality rate is evolutionarily stably maintained within this range. However, we have also revealed that within some ranges, the change in parameters resulting from environmental disturbances can easily cause the mutualistic system to collapse. One of the reasons for this vulnerability is that the release of symbionts is limited to host death, although tubeworms need a sufficient number of free-living symbionts to maintain their population. Why symbionts are not continuously released by tubeworms during their lifetime, as they do for their own larva, is an important question to be studied. A suggested mechanism by which symbionts are completely trapped by the host tubeworms until

their death is the host's immunological defense against symbionts trying to escape the trophosome, which is thought to prevent the early release of symbionts (Nyholm et al. 2012). Several questions then arise: whether a continuous release of symbionts results in a more persistent mutualism, why tubeworms enclose their symbionts until their death at the expense of immunological defenses, and why tubeworms and symbionts have coevolved toward such strongly indispensable mutualistic dependency. In our future work, we will focus on the evolution of the symbiont-enclosing strategy and the emergence of a strong mutual dependency on mutualism between the two species.

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Statement of Authorship

M.S and A.S designed the study and wrote the manuscript.

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