Death happy: Adaptive ageing and its evolution by kin selection in organisms with colonial ecology

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Standard evolutionary theory, supported by mathematical modelling of outbred, dispersed populations predicts that ageing is not an adaptation. We recently argued that in clonal, viscous populations, programmed organismal death could promote fitness through social benefits and has, in some organisms (e.g. *Caenorhabditis elegans*), evolved to shorten lifespan. Here we review previous adaptive death theory, including consumer sacrifice, biomass sacrifice, and defensive sacrifice types of altruistic adaptive death. In addition we discuss possible adaptive death in semelparous fish, coevolution of reproductive and adaptive death, and adaptive reproductive senescence in *C. elegans*. We also describe findings from recent tests for the existence of adaptive death in *C. elegans* using computer modelling. Such models have provided new insights into how trade-offs between fitness at the individual and colony levels mean that senescent changes can be selected traits. Exploring further the relationship between adaptive death and social interactions, we consider examples where adaptive death results more from action of kin than from self-destructive mechanisms and, to describe this, introduce the term adaptive killing of kin.

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1. Introduction

Is ageing an adaptation? An early idea was that ageing benefits the species by removing worn out, old individuals, thereby increasing resource availability for those still able to reproduce. However, today the consensus among evolutionary biologists is that senescence is not adaptive [1], but rather has evolved as the result of the decline in purifying selection with increasing age. Two plausible theories describe how this happens. First, the mutation accumulation theory reasons that the age decline in selection leads to accumulation of mutations with little effect on fitness earlier in life, but with detrimental effects in old age [2]. Second, the antagonistic pleiotropy theory proposes that ageing evolves due to positive selection for gene variants with pleiotropic effects at different ages: promoting early-life fitness and but also with late-life detrimental effects [3]. Importantly, neither theory predicts that ageing per se evolves because it provides a selective advantage.

From a population genetic perspective, the impact of the decline in the force of natural selection on the evolution of ageing has been described mathematically [4, 5]. Notably, these analyses were based on idealized Wright-Fisher populations (i.e. dispersed and out-crossing), and did not include social and ecological factors, including spatial structure, access to resources or dispersal. Also, the theory considers only the effect of natural selection, and does not account for other evolutionary mechanisms such as genetic drift, gene flow and mutation bias.

The mutation accumulation and antagonistic pleiotropy theories predict that ageing should be a result of action of many genes with small effects. However, the existence of mutations that dramatically extend lifespan in the nematode *C. elegans* presented a challenge to the classic evolutionary theory. For example mutation of the *daf-2* insulin/IGF-1 receptor gene can more than double *C. elegans* lifespan [6]. This led to renewed speculation about possible adaptive benefits of ageing [7]. For example, it was suggested that the wild-type *daf-2* allele "may have been selected because of its effects on aging if, for example, species whose members have short life spans prospered from increased genetic diversity or decreased competition between parents and offspring" [8].

2. Can organismal death ever promote fitness?

Could the existence of mutations that dramatically increase lifespan really reflect the presence of programmed ageing? To avoid the ambiguity in meaning of both the terms "ageing" and "programmed" [9], we will use instead the term "adaptive death" to emphasize the proposal that death itself increases inclusive fitness for the focal individual [7].

In classical evolutionary theory, kin selection was incorporated into the model of age-structured populations [10], though it was not actually explored as a possible mechanism that could affect ageing. However, it was concluded that altruistic behaviour is more likely to evolve where social interactions involve a donor of low reproductive value and a recipient with a high one. Thus, where resources are limited (as they often are in nature), altruistic behaviour can evolve by kin selection to transfer resources from parents to offspring. Where social interactions occur, the fitness of an individual includes not only the number of its progeny, but also the reproductive success of its closer relatives or kin (inclusive fitness). In principle, adaptive death could occur as an extreme form of altruism where parents sacrifice themselves to increase their inclusive fitness.

But is adaptive death a real thing? The behaviour of several computer models provided evidence that ageing can evolve through kin selection when dispersal is limited and reproductive capacity declines with age [11-13]. Thus, while classical theory predicts that ageing per se is a non-adaptive by-product of evolution, it does not rule out the possibility that ageing can be adaptive in some ecological conditions.

We recently elaborated a theory of adaptive death based on evolutionary theory, earlier discussion of the topic [14] and a survey of organisms in which adaptive death may occur. This has been described elsewhere [7, 9] but, briefly, it argues that conditions exist for the emergence of adaptive death where closely related individuals dwell in discrete, viscous (non-dispersed) populations or colonies, such that colony level (or inter-demic) selection can occur. Importantly, in viscous populations greater levels of altruism can evolve due to reduced risk of exploitation by non-altruist, non-kin cheaters. Adaptive death is expected to occur particularly in microorganisms that exist as clonal colonies, such as bacteria, yeast and some small metazoans such as free-living nematodes. We previously discussed in detail evidence for adaptive death in *Saccharomyces cerevisiae* and *C. elegans* [7, 9]. In this essay we explore further the biology of adaptive death in a wider range of highly social organisms, and develop further the adaptive death theory.

3. Stress, damage and adaptive death

Fitness benefits from adaptive death are only expected to exist where a significant proportion of a population is older [15]. The proportion of old individuals is predicted to be low in exponentially growing populations, but to increase in populations whose growth rate has stalled, e.g. due to nutrient depletion, pathogens or other insults. Thus, an expectation is that adaptive death is more beneficial in stressed populations. Molecular damage accumulation can contribute to ageing, including reproductive decline [16]. Thus, stressful conditions may both accelerate ageing and increase the benefits of adaptive death in a colonial milieu (figure 1*a*).

4. Adaptive death in colonial unicells

Many unicellular organisms grow as clonal colonies in which individual cells have limited motility. In some species colonies exhibit properties of primitive multicellular organisms, where some cells differentiate and perform altruistic functions. For example, *S. cerevisiae* exhibit programmed cell death driven by molecular mechanisms similar to those in multicellular organisms [17]. As nutrient availability declines in ageing yeast colonies, some cells in the centre of the colony undergo programmed cell death triggered by an ammonia signalling gradient, apparently to supply nutrients to the younger kin growing at the colony's edge [18, 19]. We have defined this form of adaptive death as *biomass sacrifice* since the altruists feed their kin with their own biomass [7]. However, it is also possible in principle that *consumer sacrifice* is operative, where older individuals die in order to cease consumption, thereby increasing nutrient availability for kin. Consumer sacrifice is similar to the adaptive value attributed to ageing by August Weismann in the 19th century [1].

Adaptive death occurs in a number of bacterial species, for example in the social predator *Myxococcus xantis* [20]. Upon nutrient depletion these bacteria aggregate to form a fruiting body, which can contain up to 100,000 cells. During fruiting body development ~80% of cells die due to altruistic lysis, providing nutrients by biomass sacrifice to the remaining 20% of cells, which develop into spores [21].

In *Escherichia coli* toxin-antitoxin systems can be activated in response to nutrient depletion or other stressors, and promote biomass sacrifice adaptive death that increases colony fitness [22]. Toxin-antitoxin systems are also employed by many bacterial species to prevent the spread of bacteriophage infection [23], an example of *defensive sacrifice* [7, 9]; thus, adaptive death benefits do not only stem from nutritional improvements.

Quorum sensing communication can also coordinate adaptive death in bacterial populations and biofilms. In this context, action of various signalling molecules have been described, including extracellular death factors in *E. coli* [24]; Spo0A-P factor during sporulation in *B. subtilis* [25]; and competence-stimulating peptide in *Streptococcus pneumoniae* [26].

(a) Adaptive altruistic death and adaptive killing of kin: two ends of a spectrum

The behaviour of the bacterium *Bacillus subtilis* provides an interesting perspective on the adaptive death concept. Something akin to adaptive death occurs as a mechanism by which *B. subtilis* delays the resource-intensive process of sporulation in biofilms facing a lack of nutrients [27]. In response to starvation, *B. subtilis* adopts a bet-hedging strategy in which cells differentiate

in one of two ways: either they sporulate or they continue growth by subsisting on alternative metabolites [28]. Interestingly, sporulating cells can produce so-called cannibalism toxins that kill their non-sporulating kin, thereby providing sporulators with nutrients that enable them either to survive until conditions improve or commit to spore maturation if they worsen.

Here the *B. subtilis* cells that die likely experience inclusive fitness benefits through adaptive death. However, this differs from adaptive death as previously conceived [7], where individuals undergo programmed death altruistically to benefit their kin. By contrast, in *B. subtilis* the dying cell is not so much an altruist as a victim of fratricide (or cannibalism). In *The Voyage of the Beagle* Darwin describes how, in times of food scarcity, Fuegian indians would sometimes hunt down and eat their elderly relatives (usually women) [29], an example of senicide. This we would describe as predation (or murder) rather than adaptive altruistic death. Yet by being murdered and eaten the unfortunate old women could have experienced inclusive fitness benefits, as shown in spider matriphagy [30].

Here one can, in theoretical terms, broadly define a continuum of adaptive death between two extreme cases. At one extreme, an individual within a community self-destructs to benefit their kin (adaptive altruistic death); at the other, an individual is killed by their kin for their benefit. In both cases death increases the inclusive fitness of the deceased. As one moves along the continuum from suicide to killing (figure 1*b*), the agency (i.e. active role) of the one that dies decreases, and the agency of the beneficiaries increases, initially through harmful social cues and signals, and eventually through weapons (e.g. toxins). According to this account, to be defined as such, adaptive altruistic death must entail a substantial element of agency by the one that dies, i.e. it must result predominantly from self destruction. By contrast in *adaptive killing of kin* death is largely caused by action of others (figure 1*b*). But both are forms of adaptive death.

Distinguishing adaptive death from adaptive killing of kin is easy in some cases but difficult in others. For example, in *S. cerevisiae* the ammonia gradient that provides a social cue for the decision by individual cells to undergo adaptive death is a property of the overall colony [19]. This appears not to be a case of one group of cells killing another, and so may be defined as altruistic, adaptive death. By contrast, in *B. subtilis* the toxins produced by sporulating cells (SKF peptide and SdpC protein) appear to be relatively non-specific in their action, inducing leakage in cellular membranes. SdpC also acts as a signaling protein increasing susceptibility of non-sporulating cells to toxins [27]. Resistance to toxin in sporulating cells is, at least partly, determined by pumping toxins outside the cell [31, 32]. Thus, this appears to be an example of adaptive killing of kin, though it remains possible that death in the victims involves as yet undiscovered mechanisms of self destruction.

(b) Enhancing adaptability of kin: heterogenetic adaptive death

A different mode of adaptive death is seen among *Streptococcus* and *Vibrio* species [33]. For example, during exponential growth in *S. pneumoniae* accumulation of competence-stimulating peptide causes a sub-population of cells to acquire competence (ability to uptake exogenous DNA), and also causes them to produce bacteriocins that selectively kill non-competent cells [26]. The death of the latter provides competent kin with new genetic material (new due to earlier mutagenesis) which is thought to improve survival in a changing environment [34]. Given the type of benefit provided by such adaptive killing of kin, it may be described as *heterogenetic* adaptive death. Heterogenic adaptive death also appears to occur in other bacterial species capable of natural competence [28, 35].

5. Adaptive death in colonial metazoans

Adaptive death appears to be relatively common in colonial unicellular organisms, but does it occur in colonial metazoans? We recently argued that *C. elegans* can be considered to be a colonial organism in which adaptive death could evolve due to benefits from consumer sacrifice [7]. In its boom-and-bust ecology (figure 1*c*), a food source (typically a rotten plant stem) is encountered by dauer larva propagules (typically 3-7)[36]. These then develop into adults, reproduce and generate new dauers for dispersal and further colony establishment [37]. *C. elegans* populations are largely clonal, consisting of self-fertilizing hermaphrodites. An important detail is that *C. elegans* hermaphrodites are protandrous (generating first sperm and then oocytes); the resulting limitation to sperm number leads to cessation of reproduction only 2-3 days after sexual maturity, potentially favouring adaptive death [7].

To probe further the plausibility of adaptive death in *C. elegans*, we recently created a computer model based on an approximation of *C. elegans* life history [38]. This involved colonization and consumption of a food patch, with colony fitness measured as dauer yield. The behaviour of simulations showed that, as predicted, colony fitness was increased by shorter life when reproductive span was short (c.f. protandry). Moreover, shorter life was more beneficial when adult food consumption was higher, i.e. death is more adaptive when parents are greedier [38].

(a) Minimising futile food consumption to maximise *C. elegans* colony fitness

C. elegans adults show a marked age decline in feeding rate that begins within days of sexual maturation [39]. In model simulations a sharp age decline in feeding rate increased colony fitness

[38]. This suggests that the age decline in feeding rate in *C. elegans* may represent adaptive ageing, i.e. a functional decline that may decrease individual fitness but enhances colony fitness by increasing food availability for kin. This illustrates how consumer sacrifice can be effected by adaptive behavioural ageing as well as adaptive death.

This and other properties of the model draw attention to a previously little-considered feature of *C. elegans* ecology: how colony fitness is increased by maximizing efficiency of conversion of food into dauers. This requires minimization of non-productive food consumption, which can be achieved in a variety of ways: not only by killing off post-reproductive adults, but also by tuning down their food consumption, and by optimising population structure to avoid an excess of larvae that will starve before reaching the dauer stage. Thus, optimization of fitness involves trade-offs between individual fitness and colony fitness: if the latter is increased by reducing the former, then reduced individual fitness may be favoured by natural selection (figure 1*c*). Our modelling supports the view that the consumer sacrifice type of adaptive death at least partly accounts for the very short lifespan of *C. elegans*.

(b) Programmed reproductive senescence in *Caenorhabditis* to increase colony fitness?

One unexpected behaviour of the model was that where fecundity was high, shorter reproductive span could increase colony fitness, apparently because it results in a population structure that reduces futile food consumption [38]. This could explain paradoxical findings reported more than a decade ago, as follows. In selfing hermaphrodites, reproduction ceases after only 2-3 days due to sperm depletion, but mating with males can provide sufficient sperm to sustain reproduction for many more days. One would therefore expect that mating would result in sustained progeny production rate, but in fact it shows a rapid age decline [40] (figure 1*d*). This is also true for females from the gonochoristic species *C. remanei* [41]. This reproductive decline in *C. elegans* is accompanied by visible deterioration and atrophy of the gonad [40, 42]. Thus natural selection appears to have favoured early reproductive senescence; as put by Hughes *et al*. (2007): "*C. elegans* is not engineered to generate the maximum possible number of progeny. We speculate that there is an optimal number of F1 progeny, and reproductive aging contributes to the ability of the animal to generate the optimal progeny number" [40]. Consistent with this, the behaviour of our *in silico* model suggests that early reproductive senescence shapes colony population structure in order to minimize futile food consumption, thereby maximising colony fitness.

6. Does adaptive death occur in semelparous fish?

Is adaptive death restricted to colonial microorganisms or could it evolve in more complex taxa, such as vertebrates? It has been suggested that adaptive death occurs in some species of salmon [43, 44]. Rapid death following reproduction is well documented as part of the semelparous life history of Pacific salmon such as *Oncorhynchus nerka*, and is usually interpreted as a non-adaptive by-product of massive reproductive effort (reproductive death) [45]. However, it has also been suggested that the presence of large numbers of decomposing salmon carcasses in the nutrient-poor upper reaches of rivers and streams provides nutrients, particularly nitrogen and phosphorus, that can support growth of phytoplankton and zooplankton upon which salmon fry feed [44](figure 2*a*). Salmon fry (*Oncorhynchus spp.*) have also been observed to directly consume adult carcass biomass [46]. Notably, the death rate from starvation in salmon fry can reach 90%, underscoring the potential benefits from carcasses [47]. Thus, adult salmon could exhibit biomass sacrifice adaptive death.

The possibility of adaptive death in semelparous fish was proposed by ichthyologists but appears to have passed below the radar of evolutionary biologists. Is adaptive death in salmon plausible from an evolutionary perspective? We have argued that adaptive death can evolve in clonal, viscous populations, but salmon are dioecious and do not exist as clonal populations. However, at the time of putative adaptive death, large numbers of closely related individuals do form relatively viscous populations.

Most salmon hatch and develop in streams, and then migrate to the sea where they hunt and grow for up to several years before returning to fresh water to spawn [48]. Salmon are famous for their ability to return to the streams of their birth, apparently using magnetoreceptive navigation to locate their home coastal area, and then olfactory cues to locate their natal stream [49]. The proportion of returning adults that correctly locate the stream of their birth varies between species, from 73 to 98% [50, 51]. The rates of successful homing and selection on the one side (isolation) and straying (homogenisation) determine genetic structure and evolution of salmon. The fact that the strays are less fit at their new locations is further evidence of reproductive isolation [52]. The presence in different rivers of distinct salmon sub-populations with unique local adaptations is taken into account in salmon conservation strategy [53]. For example, steelhead salmon from the Columbia River in Oregon are resistant to the cnidarian parasite *Ceratonova shasta*, whereas steelhead from the Siletz River experience up to 98% mortality from the parasite [54]. Relative gene diversity within populations between years is almost negligible (0.03-0.2%) consistent with enduring existence of distinct local populations [55]. Moreover, there is evidence that even local salmon populations are not panmictic (all can mate with all), but exist as metapopulations with hierarchical structure [56, 57]. Taken together, these findings imply that adult Pacific salmon

returning to spawn in their streams of origin are highly related to one another. Moreover, the decreasing volume of streams as salmon swim up river cause populations to become increasingly viscous. We suggest that high relatedness and population viscosity in spawning salmon populations help ensure that benefits from parental biomass sacrifice are received by kin (figure 2*a*). This might also apply to lampreys where, again, death occurs immediately after spawning and carcasses bring nutrients to the streams in which they have spawned [58].

7. Co-evolution of reproductive death and adaptive death: double death.

Recent work from our research group suggests that, like salmon, *C. elegans* hermaphrodites exhibit semelparous reproductive death as well as adaptive death [7, 38](C.C. Kern, D. Gems, in preparation). This could either be a remarkable coincidence, or it could reflect co-evolution of the two traits. One possibility is that adaptive death more readily evolves in organisms that undergo reproductive death [7]. According to Hamilton's rule an altruistic behaviour can be favoured by natural selection when $rB>C$, where r is the relatedness, B is the benefit to the recipient, and C is the cost to the donor [59]. After reproduction in semelparous organisms, their individual fitness becomes very small or negligible, and therefore so does C. This means that adaptive death involves little or no individual fitness cost, but only potential inclusive fitness benefits. In the case of Pacific salmon this suggests that their ancestors will have reproduced and then swum away to gradually die down river, but natural selection favoured demes where death occurred rapidly at the site of spawning (figure 2*b*). These arguments predict that adaptive death is likely to occur in other semelparous organisms that are non-clonal and non-colonial, and that other examples of such *double death* (reproductive and adaptive death) exist in nature.

8. Adaptive death: theoretical approaches

The existence of adaptive death in clonal populations can be explained by kin selection since clonal colonies can be considered as "super-organisms". Here, because relatedness $r = 1$, Hamilton's rule $rB > C$ becomes $B > C$ and is more easily satisfied, and the cost C of the focal individual's death is outweighed by the benefit B of many kin. As shown by Travis (2004) using computer models, this can also be true in spatially and age-structured populations (individuals were stratified by age as their reproductive value decreased with age)[13]. The main condition for kin selection to operate in Travis's model was low dispersal as a way for a focal individual to pass resources (a cell in the grid) to their relatives.

An alternative and complementary approach to kin selection to address the mechanisms of natural selection is multilevel selection (MLS). Despite being rejected conventionally in the 1970-80s, this approach is developing and has received more support recently [60]. The condition for multilevel selection can be deduced from the Price equation as $\hat{w}\Delta\hat{g} = cov(W, G) + cov(\Delta W, \Delta G) > 0$, where $\hat{w}$ is the mean fitness of the parent population (here the circumflex [or hat] denotes the mean), $\Delta\hat{g}$ is the change in mean gene frequency between offspring and parental populations, cov(W, G) is the term in the equation responsible for between-group selection, and cov(ΔW, ΔG) is the term in the equation responsible for within-group selection. The mathematical equivalency of multilevel selection and kin selection was recently demonstrated [61].

The two approaches are complementary in the sense that kin selection is more convenient when trying to establish a phenotype optimum, whereas multilevel selection shows more clearly the strength of selection [62]. The MLS approach can be difficult to interpret as the benefit at the group level can be diminished by within-group selection. This is why group adaptation is more difficult to prove and one of the requirements for its justification is the absence of selection within the group [63]. Where it is possible is in clonal populations e.g. colonies of bacteria, yeast and even multicellular *C. elegans*, as described in this review.

Most biological populations are structured by class (e.g. sex, age, caste), so the fitness of a social group can be changed due to rearrangement of classes which is not relevant for genes and natural selection. A recent approach makes it possible to account for the difference in social group members' reproductive quality and considers covariates between classes of different groups rather than groups themselves [64]. While considering adaptive death in clonal populations is relatively straightforward, application of MLS to populations with class structure and lower level of relatedness might be useful. Gardner (2015) also makes a distinction between 'aggregate' (where group value is the mean of individuals' values in the group) and 'emergent' (where group values, such as sex ratio, or food source size in the case of *C. elegans*, are difficult to define at the individual level) features of groups, and argues that the genetic theory of multilevel selection can account for 'emergent' traits [64].

Kin selection and multilevel selection are complementary approaches. However, kin selection is currently the better developed and more widely used approach for which the focal individuals can be defined. It is more general than multilevel selection as it does not need group selection to operate; also, when groups exist they can overlap in the kin selection approach, which is not true for multilevel selection. Both approaches could potentially be applied to establish whether adaptive death is able to evolve in non-clonal populations where there is a population structure, lack of resources and opportunities for transfer of resources from parents to their kin.

9. Conclusions

This essay illustrates how high levels of social interaction can lead to suicide and murder that promotes inclusive fitness. Adaptive death is a form of extreme altruism that currently appears to be largely restricted to organisms showing a level of relatedness, social organization and physical association so high that they possess features of higher order individuals: forming colonies of unicells with some metazoan features, or colonies of metazoans with some super-organismal features. It is part of a broader phenomenon, where colony level fitness is enhanced by loss of fitness at the individual level. The apparent presence of adaptive death in semelparous fish suggests that there may be other interesting examples of such socially-mediated, indirect fitness benefits of ageing or death in the animal and plant kingdoms.

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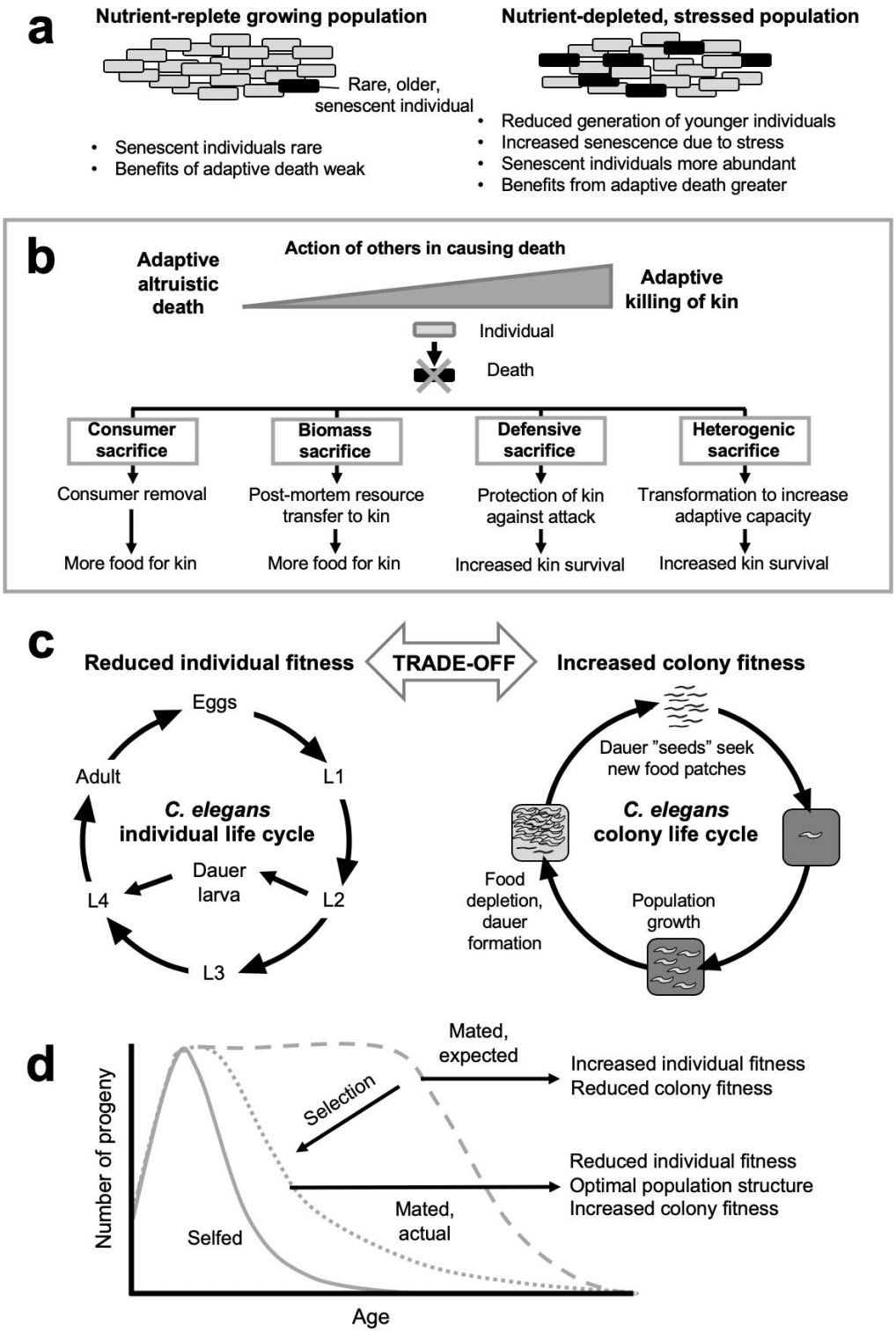

Figure 1. How reduction of individual fitness can increase colony level fitness
(*a*) The fitness benefits of adaptive death are expected to be greater in populations with less reproduction and more stress.
(*b*) Adaptive altruistic death and adaptive killing of kin. Adaptive death can be triggered by social interactions with kin. There exists a graded spectrum between adaptive altruistic death resulting from self-destruction, and adaptive killing of kin caused entirely by others. This spectrum ranges from subtle social cues to trigger death, through cannibalism toxins, to murder and predation.
(*c*) Trade-offs between fitness traits at the level of the individual and the colony: reduction of individual fitness traits (including adaptive death) can increase colony fitness, particularly by reducing futile food consumption (that which does not increase dauer yield) [38].
(*d*) Hypothesis: selection to minimize futile food consumption could explain how mechanistically programmed reproductive senescence in *Caenorhabditis* promotes colony fitness (see main text for explanation).

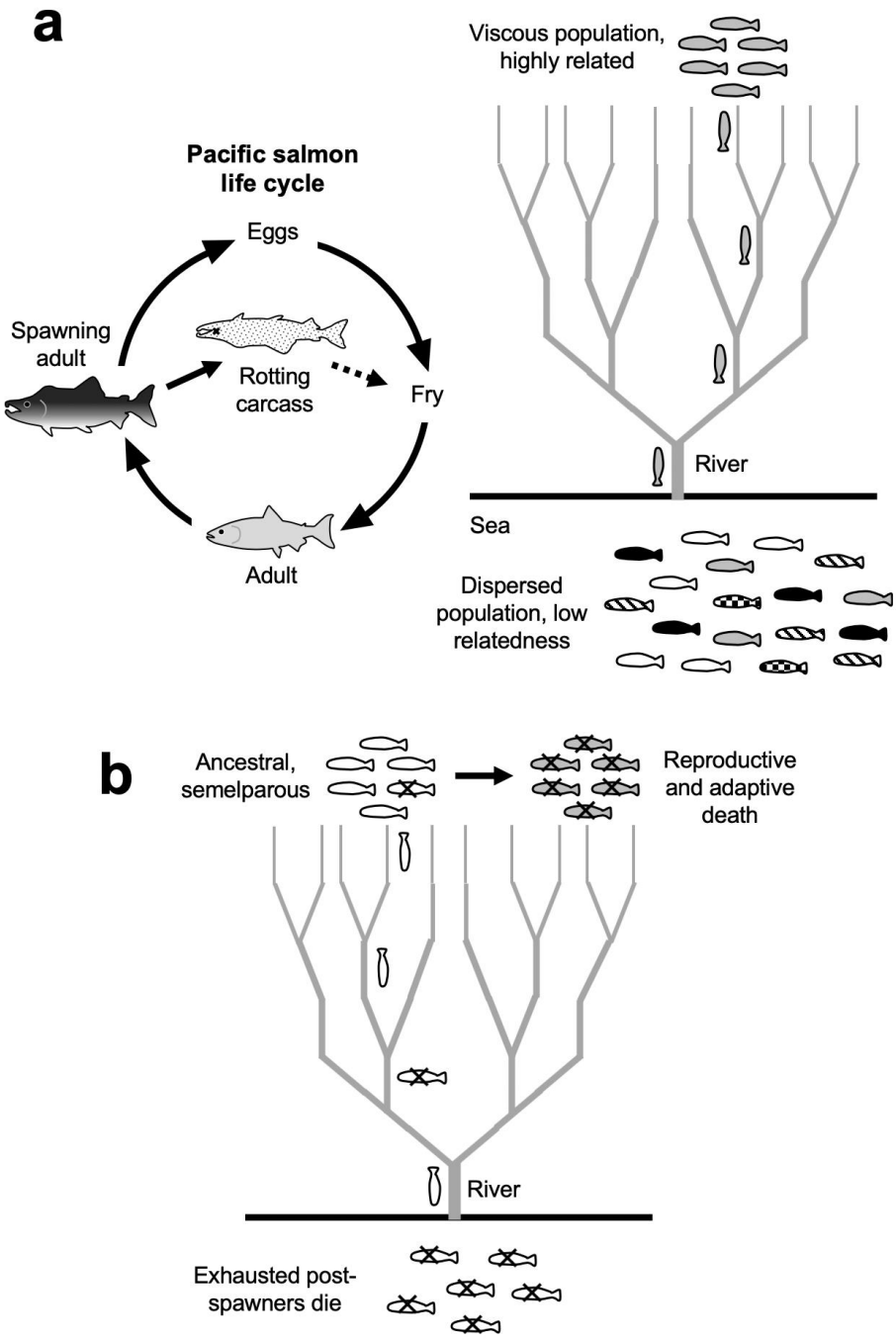

Figure 2. Hypothesis: Evolution of adaptive death in Pacific salmon.
(*a*) Left: Pacific salmon life cycle, including hypothetical biomass sacrifice adaptive death [44]. Dotted arrow: post-mortem resource transfer from post-spawning adult to fry (juvenile fish). Right: How Pacific salmon could meet the conditions for evolution of adaptive death: viscous populations of closely related individuals. Thanks to their homing capacity, closely-related groups of salmon leave the dispersed, outbred population in the sea, and concentrate within their natal streams.
(*b*) The double death hypothesis: adaptive death co-evolved with semelparous reproductive death. The figure describes a hypothesis about how adaptive death evolved in Pacific salmon from a purely semelparous ancestor. In the ancestral species (as in modern Atlantic salmon, *Salmo salar*), exhausted post-spawning adults swim or are carried down-river with only a small chance of further reproduction. Evolution of mechanisms to cause rapid death after spawning increases fitness via biomass sacrifice adaptive death (nutrients from decomposing carcasses increase survival of fry). Given the low fitness of post-spawning adults, evolution of adaptive death involves only a small loss of individual fitness.