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Solitary living species age too, and fast!

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Lidsky and Andino claim the pathogen control hypothesis (PCH) has more explanatory power than current theories on the evolution of ageing [1]. The PCH, based on kin selection, proposes that selection for shorter lifespans is adaptive because it protects close kin (carrying the same alleles for shorter lifespans) from fitness-reducing infections [1].

While host-pathogen interactions might influence the evolution of lifespan and senescence, it cannot replace other theories. It lacks generality, relies on assumptions that do not hold across senescing species, and does not consider four important aspects of evolutionary and ecological theory. First, it assumes population viscosity, ignoring numerous species of strong dispersal capacity and low viscosity. Second, it has weak explanatory power for species **with solitary living** (see Glossary) social organization, i.e. the majority. Third, it overlooks pathogen fitness and evolution. Finally, it assumes no mortality in young years, while differential mortality risks among age classes are well-known to affect ageing differently among age classes, likely shaping lifespan evolution. Finally, it does not consider other important selection agents in the evolution of lifespan.

The claim that the PCH explains shorter lifespans and the "absence of mutant variants that do not age" needs to be rejected. There is a simple way to test this: do species where kin selection plays no role have longer lifespans and mutant variants that do not age? Unlikely.

Most animal species are solitary, making indirect fitness benefits due to a reduced spread of infectious diseases by early death very low. If kin selection has indirect fitness benefits due to shorter lifespans, this effect should be stronger in social species. Empirical data show the opposite: social species tend to live longer, not shorter [2].

Similarly, the PCH does not account for differences in selection pressure related to age (age-dependent mortality) or between short- and long-lived species [3]. The present formulation of the PCH only considers old-age mortality as an influence on senescence evolution (see Box Figure IA in [1]). Refining the PCH to account for **age-dependent mortality** across the lifespan may yield better insights into the potential role of parasites in shaping longevity. At present, the PCH only considers the higher mortality of older individuals, putatively promoting the survival of younger kin because these have higher short-term fitness perspectives. The way pathogen and kin selection interact to affect the evolution of ageing largely depends on how age-dependent mortality modulates population (demography) and individual (survival) fitness. In long-lived species, individual fitness depends more on a decrease in longevity than in short-lived species [3], and so selection pressure against pathogen virulence decreasing adult survival (rather than fecundity as stated by the PCH) should be stronger in the former. If environmental-driven mortality is enhanced by pathogen virulence, the most parsimonious hypothesis for the evolution of ageing is not differential mortality of long-lived individuals, but selection of stronger immune resistance (**see box 1**). The way individuals are affected by given pathogens depends on pathogen virulence, ranging from high to mild. This has led to coevolution of virulence and host immune responses with effects on host (ageing) phenotype and fitness [4]. The PCH only considers chronic sterilizing pathogens (a restricted set of organisms). Lindsley and Andino should clarify how pathogen-inflicted mortality is likely to modify the PCH predictions on ageing evolution, as well as how chronic infections will affect differently individual fitness in long- and short-lived species, in which transient

impaired annual fertility unequally determines lifespan reproductive success. Overall, the PCH considers the perspective of the host in terms of the evolution of resistance to pathogens, but so far fails to integrate pathogen fitness and virulence evolution.

The example of eusocial ants as a support for the PCH [1] is questionable. In some species, Queens live ten times longer than workers [7]. If an immunological basis is possible, it is not fully demonstrated. Some studies have found decreased expression of immunological markers in queens [8], while others have shown elevated expression of immune genes [9] or the existence of pathogen-independent age-related pathways [10]. Thus, the PCH prediction of short-lived workers dying faster from pathogens to protect breeding Queens is not always upheld. Further, Queen phenotypes have been demonstrated to be the consequence of specific interactions with the workers and larvae, rather than due to pathogen-related selection [11]. Social immunity (disease-shielding provided by colony organization) in fact protects Queens, but also short-lived nurse workers and eggs [12]. Current studies on Queen longevity provide more support for an indirect effect of eusociality on immunity, rather than an immune root of eusocial evolution and differential caste longevity.

We agree with Lidsky and Andino that infectious diseases are an important selective agent shaping the evolution of lifespan, but disagree that the PCH can explain more than previous theories. The explanatory power of the PCH is restricted to “viscous populations” where neighbors are close kin due to reduced dispersal. In nature, this is the case in relatively few animal species, most species being solitary living. We consider that Lidsky and Andino are overgeneralizing a mechanism that only operates under a restrictive range of conditions and is unlikely

universal in nature. More caution and consideration of other selective agents is needed to understand the evolution of ageing.

Box 1: A counter-example of the PCH: the canine distemper virus and ageing in wolves

Pathogens can alter survival indirectly *via* selection for stronger immune systems and the resulting trade-off with other energy-consuming traits (life history trade-offs). In wild wolves (*Canis lupus*) differences in fur coloration (black versus grey) are due to a specific gene (K and B alleles of the CBD103 locus). The K allele provides disease resistance against the canine distemper virus (CDV), but at the cost of lower reproductive fitness [5]. Interestingly, as predicted by the PCH, KK homozygotes suffer from lower survival rates, while KB heterozygotes have higher reproductive success under high pathogen pressure. Yet, KB individuals have similar survival rates than BB (*non-resistant*) individuals [6]. While allele frequency varies with CDV, the persistence of the K allele thanks to heterozygosis has no apparent impact on ageing rates, likely because selection processes (e.g. sexual selection) other than PCH act on a large number of pathways with pleiotropic effects on ageing.

GLOSSARY

Age-dependent mortality: differences in the rate of mortality of individuals that belong to different age-classes within a given population. This is mostly attributed to genetically induced changes in longevity. Environmental factors act on longevity by affecting the age-independent component of the mortality rate.

Social organization: describes the social composition of groups (e.g. adults, juveniles, kin, non-kin)

Solitary living: is said of species where both adult males and females sleep and forage alone and only meet for courtship and mating.

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