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Review

Evolutionary perspectives on ageing

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ABSTRACT

From an evolutionary perspective, ageing is a decrease in fitness with chronological age – expressed by an increase in mortality risk and/or decline in reproductive success and mediated by deterioration of functional performance. While this makes ageing intuitively paradoxical – detrimental to individual fitness – evolutionary theory offers answers as to why ageing has evolved. In this review, I first briefly examine the classic evolutionary theories of ageing and their empirical tests, and highlight recent findings that have advanced our understanding of the evolution of ageing (condition-dependent survival, positive pleiotropy). I then provide an overview of recent theoretical extensions and modifications that accommodate those new discoveries. I discuss the role of indeterminate (asymptotic) growth for lifetime increases in fecundity and ageing trajectories. I outline alternative views that challenge a universal existence of senescence – namely the lack of a germ-soma distinction and the ability of tissue replacement and retrogression to younger developmental stages in modular organisms. I argue that rejuvenation at the organismal level is plausible, but includes a return to a simple developmental stage. This may exempt a particular genotype from somatic defects but, correspondingly, removes any information acquired during development. A resolution of the question of whether a rejuvenated individual is the same entity is central to the recognition of whether current evolutionary theories of ageing, with their extensions and modifications, can explain the patterns of ageing across the Tree of Life.

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1. Introduction

Understanding the process of ageing represents one of the key challenges in current basic and applied biological research [1–10]. One reason for this is that ageing is a complicated and multifarious process. The inherent complexity of ageing may be better compre-

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hended when considered from an evolutionary perspective. While biogerontological research is centred on functional understanding of the ageing process and seeks potential ways to mitigate its pathologies [3–6], an evolutionary outlook considers functional declines as a consequence rather than cause of ageing. Evolutionary considerations of ageing aim to understand why ageing has evolved, why it is not eliminated by natural selection, and why ageing patterns vary among individuals, populations and species [7–10].

Evolutionary theories of ageing consider the emergence and persistence of functional declines within the broader concepts of evolutionary biology. I believe that such understanding provides critical insights into the practical aspects of biogerontological research, similarly to the advances gained from an evolutionary perspective on disease and medicine [11]. Theodosius Dobzhansky's assertion that "nothing in biology makes sense except in the light of evolution" [12] has particular relevance in understanding ageing. The prevalence of ageing is inherently puzzling because, in a strikingly wasteful manner, it leads to the destruction of individuals who have successfully managed to develop a complex body from a single cell only to subsequently fail in the seemingly simple task of maintaining what has already been formed [13]. Evolutionary theory offers a satisfactory answer to this puzzle, showing us why this is necessary for some organisms while others may perhaps escape the ageing process. In this review, I treat ageing as an intriguing evolutionary question and demonstrate that, despite major progress in our insights to the origin, diversity and pervasiveness of ageing in recent decades, our understanding of its foundations and variation across the Tree of Life is far from complete.

I first review the classic evolutionary theories of ageing and their empirical tests. By highlighting challenging discoveries, I then demonstrate that many recent challenges can be accommodated within new developments of the classic theories that are their logical extensions. I then summarize more problematic points posed to the current paradigm and provide a summary of alternative views developed primarily by insights from non-model taxa. Finally, I aim to provide a balanced view on whether ageing is indeed universal to all organisms.

2. Classic theories on the evolution of ageing

2.1. Theoretical underpinnings of the classic evolutionary theories of ageing

The classic evolutionary theories of ageing (CETA) hinge on a simple argument explaining how ageing evolved and is maintained. This argument recognizes that all organisms inevitably die from extrinsic sources, be it accident, predation, disease outbreak or bouts of exceptionally harsh conditions. Hence, no individual is immortal, irrespective of the existence of ageing. From an evolutionary viewpoint, this makes later periods of life less important as progressively fewer individuals escape the extrinsic sources of mortality to experience the effects of natural selection [14,15].

This paradigm of the evolution of ageing is rooted in Weismann's recognition of the fundamental difference between immortal germ line and mortal soma [16]. Later explicit acknowledgment of the genocentric view of evolution [17,18] underscored the fact that the body constitutes a mere envelope – a vehicle – to pass on genes (genetic information), which are the ultimate subjects of selection, through evolutionary time. The germ cells contain the complete genetic information to build a soma and pass it from generation to generation, protected from somatic mutations and other modifications of the information content. Rare mutations within the germ line, along with recombination between parental genetic information, ensure that evolution can act on the resulting variation. This process enables organisms to adapt to changing environments and evolve novel variation. In contrast, somatic cells, less protected from the impacts of the external environment, accumulate errors over an individual's lifespan [1–3,7].

Traditionally, three main classic evolutionary theories of ageing (CETA) are recognized [13–15]. All of them, sometimes implicitly rather than explicitly, are centred on the distinction between the germ line and soma and use genocentric reasoning. The three theories provide complementary explanations rather than exclusive treatments of the same problem, and all ascribe ageing to an inevitability of extrinsic mortality that erodes the strength of natural selection later in life.

2.2. Mutation accumulation, Antagonistic pleiotropy, Disposable soma

The Mutation Accumulation theory (MA) [14] proposes that mutations with harmful effects on individual condition can accumulate in the genome when they are expressed late enough in life, when most individuals have perished from external sources anyway. Thus, MA suggests the existence of a 'selection shadow', resulting in the maladaptive accumulation of senescent changes to organismal performance because natural selection is not effective in purging those mutations from the population. Under MA, a widespread expression of senescent declines should only be manifested when the sources of external mortality are considerably reduced and extended individual survival is achieved. In the real world, removal of extrinsic mortality can be accomplished by keeping animals in protected conditions in captivity or through dramatic reduction of externally inflicted mortality via sanitation, medical care, vaccination and other improvements of life conditions, such as those experienced by many modern day humans. In the absence of protection from extrinsic mortality, senescence is rarely experienced.

The Antagonistic Pleiotropy theory (AP) proposes that ageing can evolve via natural selection rather than being a non-adaptive side effect. Williams [13] suggested that alleles with age-specific pleiotropic effects, that increase early-life fitness at the expense of late-life detrimental effects, could spread across populations. Given that early-life events are under stronger selection (because more individuals are alive before succumbing to extrinsic mortality), such pleiotropic alleles can readily experience positive selection. Hence, AP suggests a clear trade-off between current and future fitness reward and predicts more dynamic evolution of longevity than MA in respect to changing external conditions and the strength of extrinsic mortality.

Both MA and AP assume that higher extrinsic mortality leaves fewer individuals to survive to reproduction at later ages, making selection for longevity relatively irrelevant [1–3]. As a consequence, a higher intrinsic mortality (i.e. more rapid functional senescence) evolves, either simply by a random deterioration (genetic drift) when a "quality check" has little power (MA), or via selection for reallocating resources to early life at the expense of self-maintenance over the long term (AP). In both cases, it leaves populations of ageing individuals with intrinsically limited lifespans even with a contemporary reduction of external sources of mortality (e.g. in captivity). The nature of their evolution (drift or natural selection) implies, however, different predictions related to the association between early-life fitness and the rate of ageing [19]; no correlation for MA but a positive relationship under AP. Further, given the early life benefit of pleiotropically acting alleles, senescent deterioration of vital functions is expected to be commonly detected in natural populations under AP but not under the predictions of MA.

Kirkwood's Disposable Soma theory (DS) [15,20,21] explicitly highlights the distinction between the germ line (sperm, eggs and