Experimental Gerontology xxx (2015) xxx–xxx

Contents lists available at ScienceDirect

Experimental Gerontology

journal homepage: www.elsevier.com/locate/expgero

A theoretical model of the evolution of actuarial senescence under environmental stress

H. Watson [a], A.A. Cohen [b], C. Isaksson [a,*]

[a] Evolutionary Ecology Unit, Biology Department, Ecology Building, Lund University, SE-22362 Lund, Sweden
[b] Groupe de recherche PRIMUS, Department of Family Medicine, University of Sherbrooke, Sherbrooke, Québec J1J 3H5, Canada

ARTICLE INFO

Article history:
Received 19 May 2015
Received in revised form 17 August 2015
Accepted 18 August 2015
Available online xxxx

Section Editor: Chennai Guest Editor

Keywords:
Ageing
Senescence
Urban
Birds
Environmental stress
Evolutionary model

ABSTRACT

Free-living organisms are exposed to a wide range of stressors, all of which can disrupt components of stress-related and detoxification physiology. The subsequent accumulation of somatic damage is widely believed to play a major role in the evolution of senescence. Organisms have evolved sophisticated physiological regulatory mechanisms to maintain homeostasis in response to environmental perturbations, but these systems are likely to be constrained in their ability to optimise robustness to multiple stressors due to functional correlations among related traits. While evolutionary change can accelerate due to human ecological impacts, it remains to be understood how exposure to multiple environmental stressors could affect senescence rates and subsequently population dynamics and fitness. We used a theoretical evolutionary framework to quantify the potential consequences for the evolution of actuarial senescence in response to exposure to simultaneous physiological stressors – one versus multiple and additive versus synergistic – in a hypothetical population of avian "urban adapters". In a model in which multiple stressors have additive effects on physiology, species may retain greater capacity to recover, or respond adaptively, to environmental challenges. However, in the presence of high synergy, physiological dysregulation suddenly occurs, leading to a rapid increase in age-dependent mortality and subsequent population collapse. Our results suggest that, if the synergistic model is correct, population crashes in environmentally-stressed species could happen quickly and with little warning, as physiological thresholds of stress resistance are overcome.

1. Introduction

There is now no doubt that senescent declines in fitness occur widely in free-living animals and, in recent years, a large body of empirical evidence for ageing in the wild has accumulated (e.g. Bouwhuis et al., 2010; Jones et al., 2008; Nussey et al., 2013, Nussey et al., 2007). Senescence is defined as a progressive deterioration in physiological function, widely believed to be driven by the accumulation of somatic damage and/or mutations (Beckman and Ames, 1998; Harman, 1956; Kirkwood and Holliday, 1979). Understanding how senescence – a disadvantageous trait – has evolved, has long been of interest to evolutionary biologists and the foundations of the modern evolutionary theory of ageing were laid in the mid-20th century by the likes of Haldane (1941), Medawar (1952), Williams (1957), Hamilton (1966) and Kirkwood (1977). The various non-mutually-exclusive theories do not clearly identify a proximate cause for ageing; however, given the role of physiological deterioration, factors influencing physiological defence and repair mechanisms are expected to be important in the evolution of senescence rates.

Senescence is most often studied in model organisms under controlled laboratory conditions (e.g. Klass and Hirsh, 1976; Maynard-Smith, 1958; Tatar et al., 1997a). However, free-living non-model organisms are exposed to a wide range of environmental stressors that pose a major challenge to physiological systems; these stressors can affect different components of stress-related and detoxification physiology, resulting in damage to DNA, lipids and proteins (Isaksson, 2010; Li et al., 2003). It is clear that environmental effects are particularly important in modulating expression of age-dependent mortality rates; the effects of temperature (e.g. Maynard-Smith, 1958; Pletcher and Neuhauser, 2000) and heat shock (Tatar et al., 1997b) on senescence rates in laboratory animals are well documented, while, more recently, environmental cues have also been shown to be important in influencing ageing rates in natural populations (e.g. Kawasaki et al., 2008; Mangel, 2008; Nussey et al., 2007). It is clear that senescence rates could have significant impacts on population dynamics, yet the evolutionary and ecological importance of environmentally-induced changes in senescence in wild populations is poorly understood, particularly at microevolutionary scales.

While there is evidence that evolutionary change can accelerate due to human ecological impacts (e.g. Palumbi, 2001; Yeh, 2004), it remains to be understood whether multiple anthropogenic-induced environmental stressors challenge the individual to such a degree that the

* Corresponding author.
E-mail address: caroline.isaksson@biol.lu.se (C. Isaksson).

http://dx.doi.org/10.1016/j.exger.2015.08.009

rate and pattern of senescence, which would ultimately affect population demography and fitness, are altered. Most theoretical studies of senescence have only considered the effects of a single extrinsic stressor and consequences for life-history evolution at the individual level. Indeed, early theoretical models of ageing did not allow for any environmental or individual variation (see Mangel, 2001). State-dependent life-history theory has proved a useful framework for investigating optimal strategies in relation to the evolution of senescence, though studies have thus far only considered a single stressor at a time (Mangel and Munch, 2005; Mangel, 2008, 2001; McNamara et al., 2004; Shanley and Kirkwood, 2000). However, environmental stressors rarely act in isolation and the potential for additive or synergistic effects on individual physiology, and thereby possibly the rate of senescence, needs to be taken seriously. For example, some hunter-gatherer populations show high levels of certain risk factors for cardiovascular disease, but no actual disease (Vasunilashorn et al., 2010). The modern heart-disease epidemic thus appears to result from synergistic effects of multiple lifestyle/physiological differences relative to our ancestral state. Physiological systems often show coordinated responses to changing conditions, and there is reason to expect that overall physiological regulatory mechanisms have evolved substantial robustness to normal environmental variation (Cohen et al., 2012). However, in theory, highly-optimised complex systems, such as organismal physiology, are constrained in their ability to optimise robustness to potential stressors, due to functional correlations among related traits (Kriete, 2013). This suggests that multiple simultaneous stressors might be tolerated only up to a point, beyond which an organism's tolerance is overcome and physiological condition degrades rapidly. This theoretical perspective supports a synergistic model of how stressors interact. While other authors have considered multiple mechanisms underlying senescence, including feedback loops among them (e.g. Kowald and Kirkwood, 1996), models have not been used to infer demographic consequences.

Here, we employed evolutionary simulations to investigate the potential evolutionary consequences of exposure to single and multiple external anthropogenic stressors, mediated by a trait conferring stress resistance, in a hypothetical wild animal population. To make the paper and the model biologically relevant and timely, we have envisaged the hypothetical population to be a free-living avian “urban adapter" species. While the urban environment may remove many of the ecological constraints that exist elsewhere (e.g. reduced climatic stress in temperate regions and year-round high food-availability), towns and cities present a wide range of novel environmental stressors arising from, for example, air, artificial light, water and noise pollution (McKinney, 2002; Shochat et al., 2006). Marked changes in behaviour, physiology and morphology have been widely demonstrated among organisms inhabiting urban environments (Giraudeau and McGraw, 2014; Isaksson et al., 2005; Kempenaers et al., 2010; Liker et al., 2008; Slabbekoorn and Peet, 2003) and could underlie significant evolutionary change (e.g. Yeh, 2004). Urban adapter species are considered to be those that can persist in the urban environment, even reaching very high densities in towns and cities, but typically perform less well than their rural conspecifics (Blair, 1996; McKinney, 2002; Shochat et al., 2006). Many resident (i.e. non-migratory) passerine species fall into the group of urban adapters (e.g. great tit *Parus major*, common blackbird *Turdus merula*, and American robin *Turdus migratorius*) and therefore offer a suitable model system for investigating the potential for evolutionary change associated with urbanisation in free-living populations. Specifically, we modelled the potential effects of exposure to stressors – single versus multiple, and in the presence of multiple stressors, whether stressors act additively versus synergistically – on the evolution of actuarial senescence, i.e. the change in the likelihood of mortality with age. Although detailed studies have now demonstrated age-related declines in organismal function in the wild (Bize et al., 2008; Broggi et al., 2010; Hruska et al., 2010), actuarial senescence continues to be a useful tool for comparing ageing rates among individuals and populations, since it directly influences population dynamics and ultimately evolution.

2. The evolutionary model framework

2.1. Overview

We simulated the evolution of stress resistance and its impacts on actuarial senescence in a closed population of 1000 individuals over 500 discrete generations subject to gradually increasing environmental stress. The hypothetical population is that of a passerine bird inhabiting an urban environment, though the results can be construed more broadly. Three core scenarios of stress exposure were simulated, involving (i) a single extrinsic stressor, (ii) two extrinsic stressors acting additively, and (iii) two extrinsic stressors acting synergistically. Parameters that were allowed to vary within models were extrinsic stress level(s), stress resistance, age-dependent mortality, lifespan and lifetime reproductive success. The model does not take into account density-dependence mechanisms, population dynamics, emigration and immigration and assumes no effects on age-independent (baseline) mortality. All data simulations were performed in R 3.1.3. The R code is available in the supplementary material (Appendix A).

2.2. Modelling extrinsic stress

Extrinsic stress levels were modelled supposing exposure to one (scenario i) or two (scenarios ii and iii) environmental stressor(s). The levels of stressors to which each individual was subject were generated by randomly sampling from a normal distribution around a variable mean (see below) and with a standard deviation fixed at 10% of the mean. Variation in extrinsic stress among individuals within the population reflects the fact that stressors are typically heterogeneous across an environment. In the single-stressor model, total extrinsic stress exposure was simply the value of a single extrinsic stressor; in the additive and synergistic models, total extrinsic stress exposure was calculated as the sum or product, respectively, of two extrinsic stressors. These different ways of calculating an individual's stress exposure reflect different hypotheses for the physiological impact of exposure to multiple simultaneous stressors. In the first generation, mean levels of each stressor were assigned an arbitrary value of 1 (scenarios i and iii) or 0.5 (scenario ii), thus the mean total stress exposure was 1 across all three scenarios. We modelled different rates of increasing environmental stress, reflecting the increasing extrinsic pressures that many urban populations are experiencing due to human population growth and associated urban development. The rate of increase of environmental stress was constant within simulations, but was allowed to vary between simulations (see Table 1). As a control, scenarios were also modelled in the absence of any change (with the exception of stochastic variation) in extrinsic stress exposure.

2.3. Modelling age-dependent mortality

Stress exposure acted upon age-dependent mortality (b) and subsequently age at death (see Section 2.4) according to the exponential function:

$$b = c * e(S_{total} * 1/SR) \quad (1)$$

where S_{total} is the total extrinsic stress exposure, SR is an individual's level of stress resistance (see Section 2.5) and c is a constant fixed at 0.01 across all simulations (see Fig. 1). As stress resistance increases, the same value of extrinsic stress results in a lower value of b, and thus a lower mortality rate. b was constrained at the lower limit, such that it could never fall below the mean of the first generation; i.e. a population cannot end up better than where it started, when both extrinsic stress and stress resistance were low. Essentially, we were constraining

Please cite this article as: Watson, H., et al., A theoretical model of the evolution of actuarial senescence under environmental stress, Exp. Gerontol. (2015), http://dx.doi.org/10.1016/j.exger.2015.08.009