Why and how do termite kings and queens live so long?

Eisuke Tasaki*, Mamoru Takata, Kenji Matsuura

ORCID: ET, 0000-0002-6514-5134; MT, 0000-0002-8181-9987; KM, 0000-0002-9099-6694

Laboratory of Insect Ecology, Graduate School of Agriculture, Kyoto University

*Corresponding author: tasaki.eisuke.4r@kyoto-u.ac.jp

Summary

Lifespan varies greatly across the tree of life. Of the various explanations for this phenomenon, those that involve trade-offs between reproduction and longevity have gained considerable support. There is an important exception: reproductives (queens and in termites, also kings) of social insects exhibit both high reproductive outputs and extraordinarily long lives. As both the ultimate and proximate mechanisms underlying the absence of the fecundity/longevity trade-off could shed light on the unexpected dynamics and molecular mechanisms of extended longevity, reproductives of social insects have attracted much attention in the field of ageing research. Here, we highlight current ecological and physiological studies on ageing and discuss the various possible evolutionary and molecular explanations of the extended lifespans of termite reproductives. We integrate these findings into a coherent framework revealing the evolution of longevity in these reproductives. Studies on termites may explain why and how ageing is shaped by natural selection.

Keywords

ageing, evolution, longevity, homeostasis, hypoxia, social insects

1. Introduction

Termites are one of the most abundant terrestrial animals on earth [1,2]. A key factor in terms of their ecological success is the reproductive division of labour that is a central feature of eusociality. In a colony, a limited number of male and female reproductives (kings and queens) produce all the offspring and a large number of non-reproductive individuals (workers and soldiers; but workers can become at least neotenic reproductives in most lower termites [3–5]) perform most of foraging, nest-building, brood care and nest-guarding [6,7]. Intriguingly, reproductives and non-reproductives exhibit up to a 100-fold difference in lifespans, and even more compelling is the fact that the reproductives live for an order of magnitude longer than solitary insects [8]. These features are also found in social Hymenoptera such as ants and honeybees, but the eusociality of termites has evolved independent of social Hymenoptera [9,10]. Therefore, unlike social Hymenoptera, termites have hemimetabolous development, bisexual societies and diplodiploid sex determination system [7]. Most notable is the continuous presence of sperm-producing kings in termites, which is a universal characteristic among termites, but completely absent in social Hymenoptera [1,7]. These differences result in an interesting feature in which only the queens are long-lived in social Hymenoptera, while in termites, not only the queens but also the kings exhibit both high reproductive outputs and extraordinarily long lives [5,7,11–16]. Although longevity is negatively correlated with reproduction in most organisms [17–19], the trade-off between fertility and longevity is apparently absent in termites reproductives [20]. Both the ultimate and proximate mechanisms underlying this unique characteristic

could shed light on the unexpected dynamics and molecular mechanisms of extended longevity.

The aim of this review is to summarise recent ecological and physiological studies of termite ageing and longevity. First, we describe the evolutionary and ecological determinants of the long lifespans of termite reproductives. Then, we discuss the physiological and molecular mechanisms in play. Finally, we integrate these findings into a coherent framework explaining the evolution of longevity in such reproductives.

2. Evolutionary and ecological determinants of long lifespans

To explain why termite reproductives exhibit extended lifespans, it is necessary to understand the evolutionary aspects of longevity. The key idea underpinning the evolution of the lifespan is the "selection shadow"; the strength of natural selection declines after sexual maturation and with progressing age [17,21–25]. The selection shadow is shaped by age-independent random extrinsic mortality, which is in turn affected by external factors such as predation and early-life reproductive investment. But note exceptions if extrinsic mortality is condition-dependent not random [26]. High extrinsic mortality reduces the probability of later-life reproduction, reducing selective forces at old age. Such decreases allow alleles with late acting deleterious effects to accumulate over generations (the mutation accumulation theory) [27]. Moreover, genes associated with beneficial early-life effects would be favoured by selection even if they were deleterious in later life (the antagonistic pleiotropy theory) [21].

The deleterious effects cause physiological deterioration (ageing, also termed senescence) of an organism as age advances, reducing reproductive capacity and increasing the risk of death [28].

(a) Low extrinsic mortality

A major reason why termite reproductives live so long is that they experience low extrinsic mortality after the colony is established. The evolution of a reproductive division of labour is associated with strong social-level defence by non-reproductives [29], including nest defence, social immunity and self-sacrificial behaviours. These reduce the risks of predation, disease, starvation and desiccation; greatly limiting the extrinsic mortality of reproductives (Figure 1; Table 1).

In general, termite reproductives live deep inside the nest of central area (e.g. "royal chamber" in a few species) of an enclosed and protected mound or nest (subterranean, epigeal, or arboreal) safe from predation, thermal stress and desiccation, where tens of thousands of non-reproductives engage in social labour [30]. The most elaborate royal chamber is that of *Macrotermes* termites. It resembles a thick-walled protective bunker located in the most sheltered central part of the nest, often immediately below ground level [30–32]. The subterranean *Reticulitermes* termites also have royal chambers that are generally located deep inside the wood of the central areas [33,34]. Termite nests are multi-layered structures; many chambers are connected vertically and horizontally via very small openings. Thus, predators

such as ants are required to break the multi-layered defence mounted by non-reproductives to access the royal chamber.

The social behaviour and group living of termites reduce the risks of disease and starvation in reproductives. Termites limit the spread of infection within the nest; the antennae recognize pathogenic conidia [35], followed by initiation and maintenance of allogrooming (social grooming) of contaminated body parts [36–38]. The additive effects of individual immune responses and their social amplification are also important in terms of anti-pathogen defence. Grouped termites cope better with disease than do isolated individuals [36,39], suggesting that resistance is socially enhanced [40,41]. Group foraging may reduce the risk of starvation in reproductives; especially in some foraging termite species, non-reproductives forage actively, discover new food sources and store foods [42–46]. If termite non-reproductives have residual nutrients to be shared via trophallaxis, the reproductives are prioritised; the reproductives thus survive for longer even within a starved colony [47].

In non-reproductive workers, there tend to be differences in the maximum lifespan among termite species [5], which may be dependent on their lifestyles. Termites can be divided into wood-dwelling and foraging termites (also called one-piece type and separate type, respectively) [3,48]. The wood-dwelling termites utilise a single piece of wood both as food source and shelter and produce simple and small colonies with totipotent workers that can become reproductives, where workers are as protected as reproductives. On the other hand, the foraging termites are characterised by multiple pieces nesting and form large and complex societies where irreversible workers perform risky tasks such as foraging outside of the nest. In foraging termites, the high mortality of workers is expected to result in earlier

ageing and shorter lifespan compared to the workers of wood-dwelling termites. Currently, there is little evidence that their lifestyles have an effect on longevity of non-reproductives (and also reproductives) as a general rule, but the reproductives of *Macrotermes* termites, foraging termites, are thought to reach 20 years of age while workers can live only a few months [45], whereas the lifespan of reproductives and workers is about the same 4–5 years in *Zootermopsis* termites, wood-dwelling termites [49] (an overview of longevities for termite reproductives and workers was reviewed in [5]).

3. The physiological and molecular mechanisms of extended longevity

An understanding of the physiological and molecular mechanisms in play would illuminate how termite reproductives achieve both high reproductive outputs and extraordinarily long lives. The evolutionary theory of ageing proposes two proximate theories; these are the "energy" and "function" trade-offs between growth, reproduction and longevity (the disposable soma theory and the developmental theory of ageing, respectively) [50]. The former theory considers that ageing is a trade-off between reproduction and somatic maintenance [24,51,52]. This suggests that delayed ageing and extended longevity can be achieved via greater allocation of resources to longevity assurance mechanisms, such as antioxidant and DNA repair systems, that slow the age-associated accumulation of physiological damages. In the alternative to damage accumulation, the latter theory suggests that ageing reflects suboptimal gene function in later life, mechanistically linked to the idea

that superfluous nutrient-sensing growth pathways during adulthood can cause excessive biosynthesis that triggers functional decline (the hyperfunction hypothesis) [53–56]. Together, these trade-off theories are thought to be not mutually exclusive [57] and, broadly speaking, both sides suggest that loss of homeostasis causes ageing.

(a) Oxidative stress resistance

Loss of homeostasis is caused, in part, by oxidative stress. Reactive oxygen species (ROS) that are by-products of mitochondrial aerobic energy metabolism [58] enhance immunity and cellular signalling when they are present at certain levels. However, when enzymatic and non-enzymatic antioxidants cannot fully neutralise overproduced ROS, the resulting imbalance seriously damages important biomolecules such as proteins, lipids and nucleic acids [59–64]. The basic homeostatic balance is compromised by either increased ROS production per se or reduction in the effectiveness of defences.

An efficient antioxidant system as a longevity assurance mechanism may explain why termite queens enjoy long lifespans. If mutations causing oxidative stress-induced DNA damage are not eliminated from the germline, they may pass to the offspring, reducing the fitness. Therefore, the germline must be highly protected against oxidative stress in long-lived organisms. The long-lived queens of *R. speratus* show significantly higher activities of antioxidant enzymes (catalase and superoxide dismutase) than short-lived non-reproductives, and suffer significantly less oxidative protein, lipid and DNA damage (Figure 2) [65,66]. In

particular, the catalase activities of termite queens were higher than those of certain solitary insects and social Hymenoptera, reflected by an increased expression of the catalase gene *RsCAT1* [65]. In addition, a study on another subterranean termite *R. chinensis* revealed that differentiation of workers into neotenic reproductives was associated with increased catalase gene expression [67]. These studies partially support the hypothesis that transition to a reproductive state is associated with a gradual decrease in the extent of oxidative damage to body tissues (the oxidative shielding hypothesis) [68].

Lifestyles may influence the level of investment in their antioxidant system. Contrary to the results of the study using *R. speratus* [65], comparative studies of young and old individuals of the less-socially complex (wood-dwelling) termite *Cryptotermes secundus* revealed that protein oxidative damage levels were lower in workers than in reproductives [69] and that workers increase their protection with age but not reproductives [70]. In addition, another study using *C. secundus* found a clear oxidative stress defence signal under stress conditions, inducing ageing, that was stronger in workers than in queens [71]. These suggest that workers which are totipotent to become reproductives, like in *C. secundus*, should invest more in longevity assurance mechanisms than sterile workers because the former can still reproduce and have not reached maturity yet [71].

(b) DNA integrity and transposon defence

Genomic instability and DNA damage can trigger ageing in multicellular organisms [72].

DNA damage is repaired via various pathways [73]. In *R. speratus*, reproductives expressed higher levels of DNA repair genes than did other castes [74]. Notably, the BRCA1 gene *RsBRCA1* expression in somatic tissues was higher in long-lived kings than short-lived workers. Although *BRCA1* is one of the best-studied DNA repair genes particularly in the context of cancer research, any role of the gene in terms of organismal aging or longevity remains unclear. The cited termite study proposed that the BRCA1-associated DNA repair pathway contributed to longevity.

DNA damage occurs during germline meiotic DNA replication. The DNA repair proteins BRCA1, MCPH1, XRCC3, and MLH1 are associated with meiotic progression and maintenance of genomic stability [75–78]. These genes *RsBRCA1*, *RsMCPH1*, *RsXRCC3*, and *RsMLH1* were significantly upregulated in reproductive tissues compared to their expression in somatic tissues of *R. speratus* reproductives [74]. The expression pattern of these DNA repair genes suggests that it may protect the germline from accumulation of progressive DNA damage. The strong evolutionary pressures placed on long-lived organisms with long reproductive periods may have enhanced the expression of DNA repair genes in reproductive tissues.

Transposon defence may also be involved in the exceptional longevity of termite reproductives. Transposable elements (TEs), also termed “jumping genes”, are DNA sequences that move from one location on the genome to another and thus enhance genomic instability [79]. Increased TE activation has been linked to ageing in the mouse [80], a fly [81] and a yeast [82]. A recent study on the termite *Macrotermes bellicosus* found that although TE expression increased with advancing age in short-lived workers, this was not

the case in long-lived reproductives [12]. The study also performed age-estimates and used reproductives (around 9 years for old and 3-4 years for young) and non-reproductives (weeks to months) with large differences in age. Notably, the reproductives upregulated PIWI-interacting RNA (piRNA) biosynthesis; these RNAs silence TEs [83]. Thus, the extended lifespan of termite reproductives may be explained in part by TE suppression; this ensures longevity.

(c) Downregulation of growth signalling

The nutrient-sensing growth pathways (e.g. growth hormone signalling, insulin/IGF-1 signalling (IIS) and target of rapamycin (TOR) signalling) at the heart of the trade-off theories would be an important determinant of ageing in termite reproductives. Down regulation of these growth signaling pathways as well as dietary restriction is thought to reduce resource allocation to reproduction, growth and biosynthesis, thus prolong lifespan in organisms [84–86]. Recent study using *C. secundus* investigating transcriptome between young and old individuals revealed that aged primary reproductives (more than seven-year-old) have lower expression levels of IMP-L2 and PRMT1 genes involved in IIS suppression and ATPsynD genes related to protein homeostasis under activated TOR signalling than young ones (one-year-old) [70]. This suggests that lifespan determination and aging processes might be modulated by the typical aging pathways IIS and TOR in termites as in other organisms. In addition, four-year-old reproductives of the termite *R. chinensis* expressed significantly lower

levels of the mTOR, eIF4, and RPS6 genes involved in TOR signalling and IIS than did workers, suggesting that long-lived reproductives may downregulate these growth signalling [87]. In general, these nutrient-sensing growth pathways effect juvenile hormone (JH) production followed by upregulation of vitellogenin (Vg), a yolk protein required for egg production [88,89]. Recent studies outlined the view that re-wiring of the IIS-JH-Vg circuit has occurred in social insects which may explain the re-shaping of the fecundity/longevity trade-off [90]. However, most data has come from the honeybee where JH has no further function in the maintenance of the reproductive status in queens [91,92]. The positive correlation between JH levels and vitellogenesis has been well studied in termites [93–97], but almost nothing is known about the details of the IIS-JH-Vg circuit. Further research on the relationship between the IIS-JH-Vg circuit in termites is needed.

There is also possibility that termite reproductives, especially queens, may perhaps reduce these signalling without sacrificing high-level reproductive performance, by employing other signals to control reproduction. The transcription factor termed carbohydrate-responsive element-binding protein (ChREBP), a glucose sensor regulating the expression of genes that drive fatty acid biosynthesis, was highly expressed in mature queens of eight different termite species, compared to the levels in sterile workers and soldiers [98]. ChREBP critically mediates the glucose-dependent induction of glycolytic and lipogenic genes in metabolic tissues [99], and may thus provide essential precursors of oogenesis via redirection of a significant proportion of glucose carbons to de novo lipogenesis and nucleotide biosynthesis. Thus, in addition to growth hormone signalling, IIS and TOR signalling, the other signalling pathways such as ChREBP-mediated glucose signalling need

to be further investigated.

4. Hypoxic adaptation and longevity evolution in termite reproductives

The physiological and ecological mechanisms of extended termite longevity may be evolutionarily linked via hypoxic adaptation. Termite reproductives are protected from the external environment because they live in closed nests, where the hypoxic condition can be viewed as a by-product of the nest structure. A recent study using *R. speratus* revealed that the royal chambers in termite nests were hypoxic (low in O_2; approximately 15%) and hypercapnic (high in CO_2; approximately 4%) [34]. Oxidative stress is influenced by the level of O_2 present, shown by the fact that increases in the level of O_2 in the atmosphere that the insects are breathing lead to enhanced rates of oxidative damage and reduced longevity [100]. Atmospheric O_2 can be toxic and the levels must be carefully regulated to avoid oxidative stress [101]. Indeed, the discontinuous gas-exchange cycle is known as a respiratory adaptation to avoid oxygen toxicity in insects [102] (also in a termite *Z. nevadensis* [103]). Hypoxic nests thus may protect reproductives from oxidative stress through interacting with their antioxidant system.

Termite reproductives exhibit higher-level reproductive activities, survival, and expression of antioxidants and vitellogenin under hypoxia compared to normoxia, suggestive of hypoxic adaptation [34,104]. Intriguingly, during physiological adaptation to hypoxia, the development of anaerobic energy-producing systems operative in the conditions of low

oxidative stress may extend the lifespans of termite reproductives. Glycolysis that is a form of anaerobic metabolism is considered to produce fewer ROS than mitochondrial aerobic metabolism; large amounts of glucose are required to create energy [105,106]. Termite queens exhibit glucose signalling [98]; workers may deliver glucose to reproductives via trophallaxis. The long lifespan of queens may be attributable in part to optimisation and adaptation of their physiological responses to the hypoxia of their closed chambers, but it still needs to be further tested.

An evolutionary linkage between adaptation to hypoxia and longevity may also be in play in other long-lived animals, thus the naked mole rat [107], parasitic nematodes [108] and the ocean quahog [109], adapted to protected habitats that are underground, inside the body of the host, and muddy bottom sediments, respectively. Although adaptation to hypoxia may be physiologically useful, this does not necessarily delay ageing. When exploring the relationship between longevity and metabolic adaptation to hypoxia in termite reproductives, it is essential to define the pathways of energy metabolism, and the metabolic burdens imposed by (for example) oxidative stress during metabolism.

5. Perspectives

The ecological and physiological studies infer that the striking reproductive activity and lifespan of termite reproductives evolved under selection pressures that accompanied the development of eusociality. However, it remains unclear whether factors associated with such

evolution increased termite longevity. The life-history traits (the reproductive outputs and longevities) of various termite species featuring different levels of social defence should be further compared. For instance, there are wood-dwelling termites in which all castes are protected against predators as workers do not forage and in which also all castes stay in the nest and probably experience hypoxia. These direct comparisons would yield important insights. Probably, it is not a single factor but several traits that explain potential differences between wood-dwelling and foraging termite species.

Since termite reproductives have extraordinary longevity, it is difficult to track their entire life, and there is not so much information on the lifespan of termites [5,11–14]. Therefore, reliable and valid biomarkers of chronological and biological age are essential for future advanced studies of termites. This would allow us to take into account the effects of age, which is one of the difficulties in termite researches. Moreover, molecular studies of termites have identified several genes that may be involved in longevity assurance, but their functions remain unclear. Since it is necessary to carefully assess how termite reproductives achieve extended lifespan, further studies are required to evaluate the biological function of these genes in the longevity of termites using genetic tools, such as RNA interference [110,111] and transgenic systems [112].

The quest for understanding the proximate mechanisms of extraordinary longevity in termite reproductives is only the beginning. We are now at the dawn of a big paradigm shift in the ageing study of termites having the great opportunity to uncover the unexplored longevity-mechanisms that cannot be reached by studies using intrinsically short-lived model organisms. As the possibility that termite queens activate ChREBP-mediated glucose

signaling to attenuate TOR signaling and achieve longevity without sacrificing reproduction, these unexpected physiological and molecular pathways explaining the remarkable lifespan extension of termite reproductives may be identified in the future.

Data accessibility

This review has no additional data.

Author's contributions

E.T., M.T. and K.M. wrote the paper together.

Competing interests

We declare that we have no competing interests.

Funding

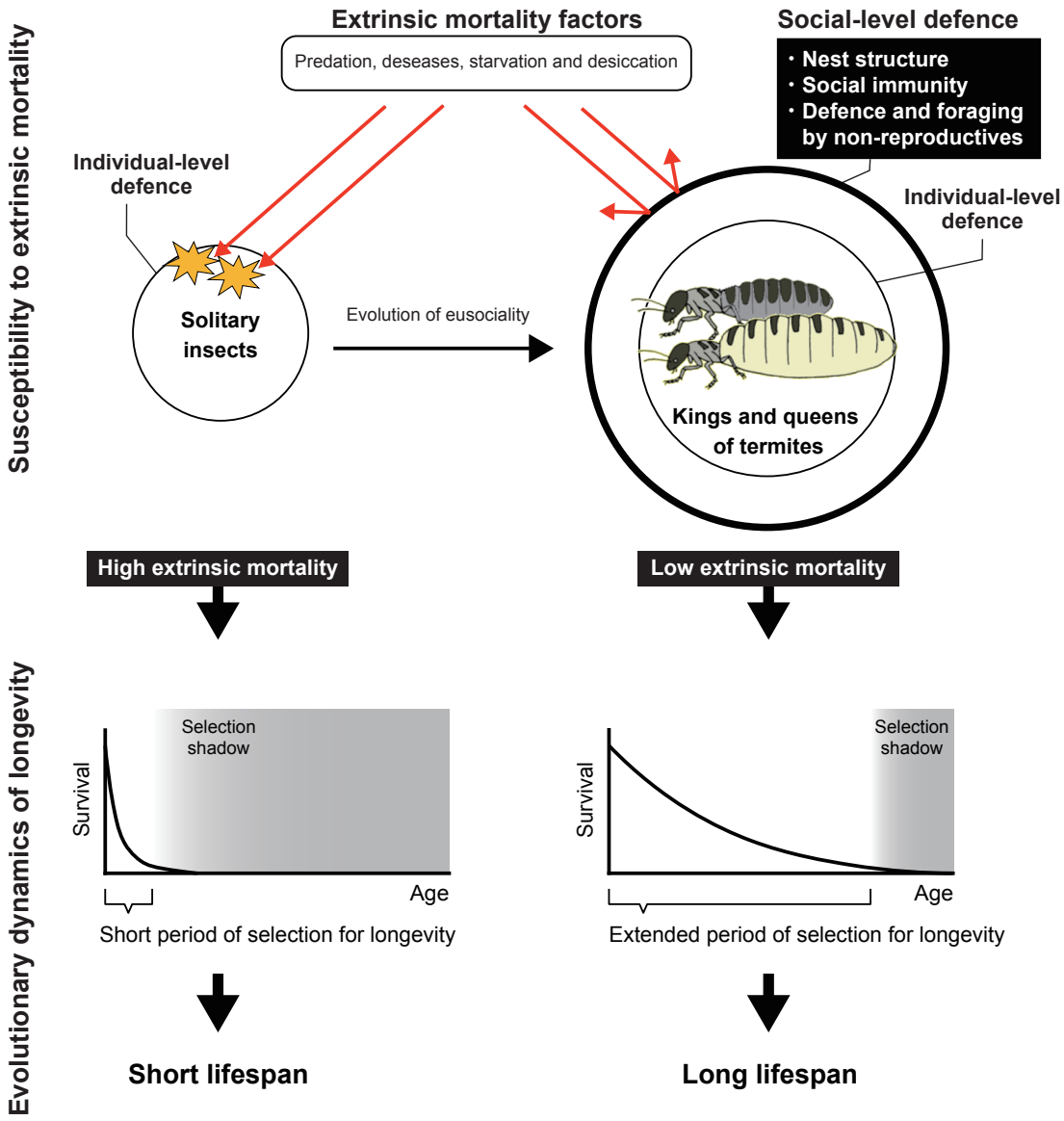
This work was supported by the Japan Society for the Promotion of Science (Kiban Kenkyu S: 18H05268) to K.M.

Acknowledgments

We thank all members of the Laboratory of Insect Ecology, Kyoto University for valuable discussions and comments.

336 **Figures**

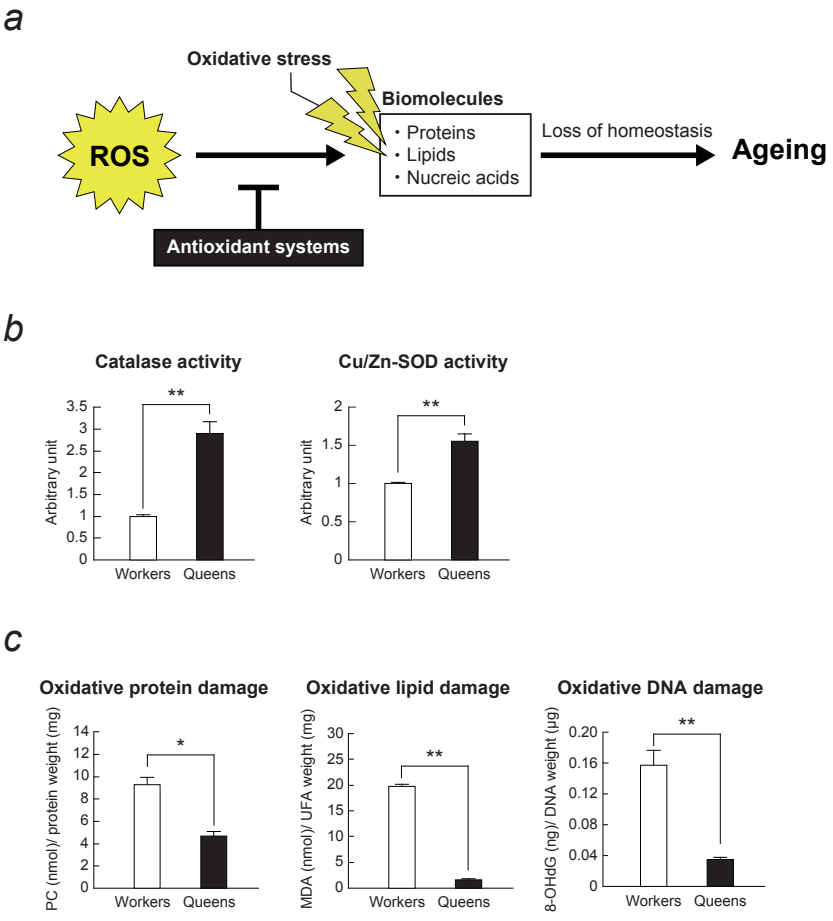
figure 1



337

Figure 1. **Schematic of the evolution of extended longevity in termite kings and queens.** The upper panel shows the susceptibilities to extrinsic mortality of solitary insects (left) and termite reproductives (right). Black solid circles indicate defence systems; termite reproductives implement both individual- and social-level defences (the latter by the evolution of eusociality). The red arrows indicate the effects of extrinsic mortality factors on individuals. Termite reproductives may be better protected than solitary insects against such attacks. The lower panel shows the evolutionary dynamics of longevity in short-lived solitary insects (left) and long-lived termite reproductives (right). The black solid curves indicate survival levels in the wild, and the grey areas the "selection shadows". The reduced extrinsic mortality of termite reproductives extends the period available for longevity selection.

figure 2



350

351

Figure 2. Oxidative stress resistance in termite queens. (*a*) The ageing process associated with oxidative stress. (*b*) Comparisons of the catalase and Cu/Zn-superoxide dismutase (Cu/Zn-SOD) enzyme activities (antioxidant systems) of short-lived *R. speratus* workers and long-lived queens. (*c*) Comparisons of the oxidative damage levels of various biomolecules in *R. speratus* short-lived workers and long-lived queens after free radical formation by ultraviolet irradiation (left and right boxes respectively). The error bars denote standard errors of the means. The statistical significances are: * $p<0.05$ and ** $p<0.01$. White and black bars indicate workers and queens, respectively. PC, protein carbonyl; MDA, malondialdehyde; 8-OHdG, 8-hydroxy-2′-deoxyguanosine. Modified from Tasaki *et al*. (2017) [65] and Tasaki *et al*. (2018) [66].

Table 1. Ecological and physiological characteristics in termites, and the predicted effect on extended longevity of the reproductives.

Characteristics	Expected effect on extended longevity of termite reproductives	References
Ecological characteristics		
Nest defence	Nest defence, which involves nest structures in combination with the defensive behaviour of the non-reproductives, contributes to the decrease in the risks of predation, thermal stress and desiccation in the reproductives.	[30,33,113]
Social immunity	Social immunity, which is one adaptive mechanism that helps to alleviate the risks of pathogen infection in group-living termites, contributes to a reduced risk of mortality due to diseases in the reproductives.	[36–39,41]
Group foraging	Group foraging by non-reproductives involves high foraging activity and new food discovery and storage, thus avoiding the risk of starvation in the reproductives.	[42–47]
Hypoxic nest	Hypoxic nest could protect the reproductives from oxygen toxicity.	[34,104]
Physiological characteristics		
Oxidative stress resistance	Antioxidant system neutralises the overproduction of reactive oxygen species associated with oxidative stress, which contributes to delayed ageing and long lifespan of the reproductives.	[65–67]
DNA repair	Various repair pathways repairing DNA damages associates to be long lifespan of the reproductives.	[74]
Transposon defence	Transposon defence such as PIWI-interacting RNA inhibits transposon activity, which contributes to improve genomic instability and slow ageing in the reproductives.	[12]
Downregulation of growth signalling	Downregulation of insulin/IGF-1 signalling (IIS) and target of rapamycin (TOR) signalling prevents excessive biosynthesis resulting in loss of homeostasis and contributes to longevity in the reproductives.	[87]

366 **References**

367 1. Korb J. 2008 Termites, hemimetabolous diploid white ants? *Front. Zool.* **5**, 15.
368 (doi:10.1186/1742-9994-5-15)
369 2. Bar-On YM, Phillips R, Milo R. 2018 The biomass distribution on Earth. *Proc. Natl.*
370 *Acad. Sci.* **115**, 6506–6511. (doi:10.1073/pnas.1711842115)
371 3. Korb J, Hartfelder K. 2008 Life history and development - a framework for
372 understanding developmental plasticity in lower termites. *Biol. Rev.* **83**, 295–313.
373 (doi:10.1111/j.1469-185X.2008.00044.x)
374 4. Roisin Y, Korb J. 2010 Social organisation and the status of workers in termites. In
375 *Biology of Termites: a Modern Synthesis*, pp. 133–164. Dordrecht: Springer
376 Netherlands. (doi:10.1007/978-90-481-3977-4_6)
377 5. Korb J, Thorne B. 2017 Sociality in termites. In *Comparative Social Evolution* (eds
378 DR Rubenstein, P Abbot), pp. 124–153. Cambridge: Cambridge University Press.
379 (doi:10.1017/9781107338319.006)
380 6. Korb J. 2016 Genes underlying reproductive division of labor in termites, with
381 comparisons to social Hymenoptera. *Front. Ecol. Evol.* **4**, 1–10.
382 (doi:10.3389/fevo.2016.00045)
383 7. Thorne BL. 1997 Evolution of eusociality in termites. *Annu. Rev. Ecol. Syst.* **28**, 27–
384 54. (doi:10.1146/annurev.ecolsys.28.1.27)
385 8. Keller L, Genoud M. 1997 Extraordinary lifespans in ants: a test of evolutionary
386 theories of ageing. *Nature* **389**, 958–960. (doi:10.1038/40130)
387 9. Inward D, Beccaloni G, Eggleton P. 2007 Death of an order: a comprehensive

388 molecular phylogenetic study confirms that termites are eusocial cockroaches. *Biol.*
389 *Lett.* **3**, 331–335. (doi:10.1098/rsbl.2007.0102)
390 10. Engel MS, Grimaldi DA, Krishna K. 2009 Termites (Isoptera): their phylogeny,
391 classification, and rise to ecological dominance. *Am. Museum Novit.* **3650**, 1–27.
392 (doi:10.1206/651.1)
393 11. Keller L. 1998 Queen lifespan and colony characteristics in ants and termites.
394 *Insectes Soc.* **45**, 235–246. (doi:10.1007/s000400050084)
395 12. Elsner D, Meusemann K, Korb J. 2018 Longevity and transposon defense, the case
396 of termite reproductives. *Proc. Natl. Acad. Sci.* **115**, 5504–5509.
397 (doi:10.1073/pnas.1804046115)
398 13. Thorne BL, Breisch NL, Haverty MI. 2002 Longevity of kings and queens and first
399 time of production of fertile progeny in dampwood termite (Isoptera; Termopsidae;
400 *Zootermopsis*) colonies with different reproductive structures. *J. Anim. Ecol.* **71**,
401 1030–1041. (doi:10.1046/j.1365-2656.2002.00666.x)
402 14. Long CE, Thorne BL, Breisch NL. 2007 Termite colony ontogeny: supplemental
403 data in the long-term assessment of reproductive lifespan, female neotenic
404 production and colony size in *Reticulitermes flavipes* (Isoptera: Rhinotermitidae).
405 *Bull. Entomol. Res.* **97**, 321–325. (doi:10.1017/S0007485307004919)
406 15. Hartke TR, Baer B. 2011 The mating biology of termites: a comparative review.
407 *Anim. Behav.* **82**, 927–936. (doi:10.1016/j.anbehav.2011.07.022)
408 16. Monroy Kuhn JM, Korb J. 2016 Social insects: aging and the re-shaping of the
409 fecundity/longevity trade-off with sociality. *Curr. Opin. Insect Sci.* **16**, vii–x.

(doi:10.1016/j.cois.2016.06.002)

17. Kirkwood TBL, Austad SN. 2000 Why do we age? *Nature* **408**, 233–238. (doi:10.1038/35041682)

18. Lemaître J-F, Berger V, Bonenfant C, Douhard M, Gamelon M, Plard F, Gaillard J-M. 2015 Early-late life trade-offs and the evolution of ageing in the wild. *Proc. R. Soc. B Biol. Sci.* **282**, 20150209. (doi:10.1098/rspb.2015.0209)

19. Salguero-Gómez R, Jones OR. 2017 Life history trade-offs modulate the speed of senescence. In *The Evolution of Senescence in the Tree of Life* (eds RP Shefferson, OR Jones, R Salguero-Gomez), pp. 403–421. Cambridge: Cambridge University Press. (doi:10.1017/9781139939867.020)

20. Korb J. 2016 Why do social insect queens live so long? Approaches to unravel the sociality-aging puzzle. *Curr. Opin. Insect Sci.* **16**, 104–107. (doi:10.1016/j.cois.2016.06.004)

21. Williams GC. 1957 Pleiotropy, natural selection, and the evolution of senescence. *Evolution (N. Y).* **11**, 398–411.

22. Hamilton WD. 1966 The moulding of senescence by natural selection. *J. Theor. Biol.* **12**, 12–45.

23. Moorad J, Promislow D, Silvertown J. 2019 Evolutionary ecology of senescence and a reassessment of Williams' 'extrinsic mortality' hypothesis. *Trends Ecol. Evol.* **34**, 519–530. (doi:10.1016/j.tree.2019.02.006)

24. Kirkwood TBL, Holliday R. 1979 The evolution of ageing and longevity. *Proc. R. Soc. London - Biol. Sci.* **205**, 531–546. (doi:10.1098/rspb.1979.0083)

25. Kirkwood TBL, Melov S. 2011 On the programmed/non-programmed nature of ageing within the life history. *Curr. Biol.* **21**, R701–R707. (doi:10.1016/j.cub.2011.07.020)
26. Chen H, Maklakov AA. 2012 Longer life span evolves under high rates of condition-dependent mortality. *Curr. Biol.* **22**, 2140–2143. (doi:10.1016/j.cub.2012.09.021)
27. Medawar P. 1952 *An Unsolved Problem of Biology*. London: H.K. Lewis and Company.
28. Partridge L, Barton NH. 1993 Optimality, mutation and the evolution of ageing. *Nature* **362**, 305–311. (doi:10.1038/362305a0)
29. Bourke AFG. 2007 Kin selection and the evolutionary theory of aging. *Annu. Rev. Ecol. Evol. Syst.* **38**, 103–128. (doi:10.1146/annurev.ecolsys.38.091206.095528)
30. Noirot C, Darlington JPEC. 2000 Termite nests: architecture, regulation and defence. In *Termites: Evolution, Sociality, Symbioses, Ecology*, pp. 121–139. Dordrecht: Springer Netherlands. (doi:10.1007/978-94-017-3223-9_6)
31. Darlington JPEC. 1984 Two types of mound built by the termite *Macrotermes subhyalinus* in Kenya. *Int. J. Trop. Insect Sci.* **5**, 481–492. (doi:10.1017/S1742758400004914)
32. Darlington JPEC. 1985 The structure of mature mounds of the termite *Macrotermes michaelseni* in Kenya. *Insect Sci. Its Appl.* **6**, 149–156. (doi:10.1017/S1742758400006536)
33. Yanagihara S, Suehiro W, Mitaka Y, Matsuura K. 2018 Age-based soldier polyethism: old termite soldiers take more risks than young soldiers. *Biol. Lett.* **14**,

20180025. (doi:10.1098/rsbl.2018.0025)

34. Tasaki E, Komagata Y, Inagaki T, Matsuura K. 2020 Reproduction deep inside wood: a low O_2 and high CO_2 environment promotes egg production by termite queens. *Biol. Lett.* **16**, 20200049. (doi:10.1098/rsbl.2020.0049)

35. Yanagawa A, Yokohari F, Shimizu S. 2009 The role of antennae in removing entomopathogenic fungi from cuticle of the termite, *Coptotermes formosanus*. *J. Insect Sci.* **9**, 1–9. (doi:10.1673/031.009.0601)

36. Rosengaus RB, Maxmen AB, Coates LE, Traniello JFA. 1998 Disease resistance: a benefit of sociality in the dampwood termite *Zootermopsis angusticollis* (Isoptera: Termopsidae). *Behav. Ecol. Sociobiol.* **44**, 125–134. (doi:10.1007/s002650050523)

37. Yanagawa A, Shimizu S. 2007 Resistance of the termite, *Coptotermes formosanus* Shiraki to *Metarhizium anisopliae* due to grooming. *BioControl* **52**, 75–85. (doi:10.1007/s10526-006-9020-x)

38. Wilson-Rich N, Stuart RJ, Rosengaus RB. 2007 Susceptibility and behavioral responses of the dampwood termite *Zootermopsis angusticollis* to the entomopathogenic nematode *Steinernema carpocapsae*. *J. Invertebr. Pathol.* **95**, 17–25. (doi:10.1016/j.jip.2006.11.004)

39. Calleri D V., Rosengaus RB, Traniello JFA. 2010 Disease resistance in the drywood termite, *Incisitermes schwarzi*: does nesting ecology affect immunocompetence? *J. Insect Sci.* **10**, 1–12. (doi:10.1673/031.010.4401)

40. Traniello JFA, Rosengaus RB, Savoie K. 2002 The development of immunity in a social insect: evidence for the group facilitation of disease resistance. *Proc. Natl.*

476 *Acad. Sci.* **99**, 6838–6842. (doi:10.1073/pnas.102176599)
477 41. Rosengaus RB, Traniello JFA, Bulmer MS. 2010 Ecology, behavior and evolution of
478 disease resistance in termites. In *Biology of Termites: a Modern Synthesis*, pp. 165–
479 191. Dordrecht: Springer Netherlands. (doi:10.1007/978-90-481-3977-4_7)
480 42. Waller DA, Fage JP La. 1987 Food quality and foraging response by the
481 subterranean termite Coptotermes formosanus shiraki (isoptera: Rhinotermitidae).
482 *Bull. Entomol. Res.* **77**, 417–424. (doi:10.1017/S0007485300011883)
483 43. Reinhard J, Hertel H, Kaib M. 1997 Systematic search for food in the subterranean
484 termite *Reticulitermes santonensis* De Feytaud (Isoptera, Rhinotermitidae). *Insectes*
485 *Soc.* **44**, 147–158. (doi:10.1007/s000400050037)
486 44. Hedlund JC, Henderson G. 1999 Effect of available food size on search tunnel
487 formation by the formosan subterranean termite (Isoptera: Rhinotermitidae). *J. Econ.*
488 *Entomol.* **92**, 610–616. (doi:10.1093/jee/92.3.610)
489 45. Traniello JFA, Leuthold RH. 2000 Behavior and ecology of foraging in termites. In
490 *Termites: Evolution, Sociality, Symbioses, Ecology*, pp. 141–168. Dordrecht:
491 Springer Netherlands. (doi:10.1007/978-94-017-3223-9_7)
492 46. Howard KJ, Thorne BL. 2010 Eusocial evolution in termites and Hymenoptera. In
493 *Biology of Termites: a Modern Synthesis*, pp. 97–132. Dordrecht: Springer
494 Netherlands. (doi:10.1007/978-90-481-3977-4_5)
495 47. Chouvenc T. 2020 Limited survival strategy in starving subterranean termite
496 colonies. *Insectes Soc.* **67**, 71–82. (doi:10.1007/s00040-019-00729-5)
497 48. Korb J, Poulsen M, Hu H, Li C, Boomsma JJ, Zhang G, Liebig J. 2015 A genomic

comparison of two termites with different social complexity. *Front. Genet.* **6**. (doi:10.3389/fgene.2015.00009)

49. Heath H. 1907 The longevity of members of the different castes of *Termopsis angusticollis*. *Biol. Bull.* **13**, 161–164. (doi:10.2307/1535601)

50. Maklakov AA, Chapman T. 2019 Evolution of ageing as a tangle of trade-offs: energy versus function. *Proc. R. Soc. B Biol. Sci.* **286**, 20191604. (doi:10.1098/rspb.2019.1604)

51. Kirkwood TBL. 1977 Evolution of ageing. *Nature* **270**, 301–304. (doi:10.1038/270301a0)

52. Kirkwood TBL. 2017 The disposable soma theory: origins and evolution. In *The Evolution of Senescence in the Tree of Life* (eds RP Shefferson, OR Jones, R Salguero-Gomez), pp. 23–39. Cambridge: Cambridge University Press. (doi:10.1017/9781139939867.002)

53. Gems D, Partridge L. 2013 Genetics of longevity in model organisms: debates and paradigm shifts. *Annu. Rev. Physiol.* **75**, 621–644. (doi:10.1146/annurev-physiol-030212-183712)

54. Blagosklonny M V. 2006 Aging and immortality: quasi-programmed senescence and its pharmacologic inhibition. *Cell Cycle* **5**, 2087–2102. (doi:10.4161/cc.5.18.3288)

55. Gems D, de la Guardia Y. 2013 Alternative perspectives on aging in *Caenorhabditis elegans* : reactive oxygen species or hyperfunction? *Antioxid. Redox Signal.* **19**, 321–329. (doi:10.1089/ars.2012.4840)

56. Blagosklonny M V. 2012 Answering the ultimate question “what is the proximal

cause of aging?" *Aging (Albany. NY).* **4**, 861–877. (doi:10.18632/aging.100525)

57. de Verges J, Nehring V. 2016 A critical look at proximate causes of social insect senescence: damage accumulation or hyperfunction? *Curr. Opin. Insect Sci.* **16**, 69–75. (doi:10.1016/j.cois.2016.05.003)

58. Murphy MP. 2009 How mitochondria produce reactive oxygen species. *Biochem. J.* **417**, 1–13. (doi:10.1042/BJ20081386)

59. Schieber M, Chandel NS. 2014 ROS function in redox signaling and oxidative stress. *Curr. Biol.* **24**, R453–R462. (doi:10.1016/j.cub.2014.03.034)

60. Hensley K, Robinson KA, Gabbita SP, Salsman S, Floyd RA. 2000 Reactive oxygen species, cell signaling, and cell injury. In *Free Radical Biology and Medicine*, pp. 1456–1462. Pergamon. (doi:10.1016/S0891-5849(00)00252-5)

61. Dröge W. 2002 Free radicals in the physiological control of cell function. *Physiol. Rev.* **82**, 47–95. (doi:10.1152/physrev.00018.2001)

62. Ray PD, Huang BW, Tsuji Y. 2012 Reactive oxygen species (ROS) homeostasis and redox regulation in cellular signaling. *Cell. Signal.* **24**, 981–990. (doi:10.1016/j.cellsig.2012.01.008)

63. D'Autréaux B, Toledano MB. 2007 ROS as signalling molecules: mechanisms that generate specificity in ROS homeostasis. *Nat. Rev. Mol. Cell Biol.* **8**, 813–824. (doi:10.1038/nrm2256)

64. Finkel T, Holbrook NJ. 2000 Oxidants, oxidative stress and the biology of ageing. *Nature* **408**, 239–247. (doi:10.1038/35041687)

65. Tasaki E, Kobayashi K, Matsuura K, Iuchi Y. 2017 An efficient antioxidant system

542 in a long-lived termite queen. *PLoS One* **12**, e0167412.
543 (doi:10.1371/journal.pone.0167412)
544 66. Tasaki E, Kobayashi K, Matsuura K, Iuchi Y. 2018 Long-lived termite queens
545 exhibit high Cu/Zn-superoxide dismutase activity. *Oxid. Med. Cell. Longev.* **2018**,
546 5127251. (doi:10.1155/2018/5127251)
547 67. Ye C, Rasheed H, Ran Y, Yang X, Xing L, Su X. 2019 Transcriptome changes
548 reveal the genetic mechanisms of the reproductive plasticity of workers in lower
549 termites. *BMC Genomics* **20**, 702. (doi:10.1186/s12864-019-6037-y)
550 68. Blount JD, Vitikainen EIK, Stott I, Cant MA. 2016 Oxidative shielding and the cost
551 of reproduction. *Biol. Rev.* **91**, 483–497. (doi:10.1111/brv.12179)
552 69. Kramer B *et al.* 2021 Oxidative stress and senescence in social insects – a significant
553 but inconsistent link. *Philos. Trans. R. Soc. B Biol. Sci.* **XXX**, XXX.
554 70. Monroy Kuhn JM, Meusemann K, Korb J. 2019 Long live the queen, the king and
555 the commoner? Transcript expression differences between old and young in the
556 termite *Cryptotermes secundus*. *PLoS One* **14**, e0210371.
557 (doi:10.1371/journal.pone.0210371)
558 71. Rau V, Korb J. 2021 The effect of environmental stress on ageing in a termite
559 species with low social complexity. *Philos. Trans. R. Soc. B Biol. Sci.* **XXX**, XXX.
560 72. Hoeijmakers JHJ. 2009 DNA damage, aging, and cancer. *N. Engl. J. Med.* **361**,
561 1475–1485. (doi:10.1056/NEJMra0804615)
562 73. Ciccia A, Elledge SJ. 2010 The DNA damage response: making it safe to play with
563 knives. *Mol. Cell* **40**, 179–204. (doi:10.1016/j.molcel.2010.09.019)

564 74. Tasaki E, Mitaka Y, Nozaki T, Kobayashi K, Matsuura K, Iuchi Y. 2018 High
565 expression of the breast cancer susceptibility gene BRCA1 in long-lived termite
566 kings. *Aging (Albany. NY).* **10**, 2668–2683. (doi:10.18632/aging.101578)
567 75. Broering TJ *et al.* 2014 BRCA1 establishes DNA damage signaling and pericentric
568 heterochromatin of the X chromosome in male meiosis. *J. Cell Biol.* **205**, 663–675.
569 (doi:10.1083/jcb.201311050)
570 76. Liang Y *et al.* 2010 BRIT1/MCPH1 is essential for mitotic and meiotic
571 recombination DNA repair and maintaining genomic stability in mice. *PLoS Genet.*
572 **6**, e1000826. (doi:10.1371/journal.pgen.1000826)
573 77. Liu Y, Tarsounas M, O'Regan P, West SC. 2007 Role of RAD51C and XRCC3 in
574 genetic recombination and DNA repair. *J. Biol. Chem.* **282**, 1973–1979.
575 (doi:10.1074/jbc.M609066200)
576 78. Kneitz B *et al.* 2000 MutS homolog 4 localization to meiotic chromosomes is
577 required for chromosome pairing during meiosis in male and female mice. *Genes*
578 *Dev.* **14**, 1085–1097. (doi:10.1101/gad.14.9.1085)
579 79. Kazazian HH. 2004 Mobile elements: drivers of genome evolution. *Science* **303**,
580 1626–1632. (doi:10.1126/science.1089670)
581 80. De Cecco M, Criscione SW, Peterson AL, Neretti N, Sedivy JM, Kreiling JA. 2013
582 Transposable elements become active and mobile in the genomes of aging
583 mammalian somatic tissues. *Aging (Albany. NY).* **5**, 867–883.
584 (doi:10.18632/aging.100621)
585 81. Chen H, Zheng X, Xiao D, Zheng Y. 2016 Age-associated de-repression of

586 retrotransposons in the *Drosophila* fat body, its potential cause and consequence.
587 *Aging Cell* **15**, 542–552. (doi:10.1111/acel.12465)
588 82. Maxwell PH, Burhans WC, Curcio MJ. 2011 Retrotransposition is associated with
589 genome instability during chronological aging. *Proc. Natl. Acad. Sci.* **108**, 20376–
590 20381. (doi:10.1073/pnas.1100271108)
591 83. Brennecke J, Aravin A a., Stark A, Dus M, Kellis M, Sachidanandam R, Hannon GJ.
592 2007 Discrete small RNA-generating loci as master regulators of transposon activity
593 in *Drosophila*. *Cell* **128**, 1089–1103. (doi:10.1016/j.cell.2007.01.043)
594 84. Gesing A *et al.* 2016 A long-lived mouse lacking both growth hormone and growth
595 hormone receptor: a new animal model for aging studies. *Journals Gerontol. Ser. A*
596 *Biol. Sci. Med. Sci.* **72**, glw193. (doi:10.1093/gerona/glw193)
597 85. Fontana L, Partridge L, Longo VD. 2010 Extending healthy life span-from yeast to
598 humans. *Science* **328**, 321–326. (doi:10.1126/science.1172539)
599 86. Partridge L, Alic N, Bjedov I, Piper MDW. 2011 Ageing in *Drosophila*: the role of
600 the insulin/Igf and TOR signalling network. *Exp. Gerontol.* **46**, 376–381.
601 (doi:10.1016/j.exger.2010.09.003)
602 87. Haroon, Ma X-M, Li Y-X, Zhang H-X, Liu Q, Su X-H, Xing L-X. 2020
603 Transcriptomic evidence that insulin signalling pathway regulates the ageing of
604 subterranean termite castes. *Sci. Rep.* **10**, 8187. (doi:10.1038/s41598-020-64890-9)
605 88. Flatt T, Tu M-P, Tatar M. 2005 Hormonal pleiotropy and the juvenile hormone
606 regulation of Drosophila development and life history. *BioEssays* **27**, 999–1010.
607 (doi:10.1002/bies.20290)

89. Toivonen JM, Partridge L. 2009 Endocrine regulation of aging and reproduction in *Drosophila*. *Mol. Cell. Endocrinol.* **299**, 39–50. (doi:10.1016/j.mce.2008.07.005)
90. Rodrigues MA, Flatt T. 2016 Endocrine uncoupling of the trade-off between reproduction and somatic maintenance in eusocial insects. *Curr. Opin. Insect Sci.* **16**, 1–8. (doi:10.1016/j.cois.2016.04.013)
91. Rueppell O, Aumer D, Moritz RF. 2016 Ties between ageing plasticity and reproductive physiology in honey bees (*Apis mellifera*) reveal a positive relation between fecundity and longevity as consequence of advanced social evolution. *Curr. Opin. Insect Sci.* **16**, 64–68. (doi:10.1016/j.cois.2016.05.009)
92. Hartfelder K, Tiberio GJ, Lago DC, Dallacqua RP, Bitondi MMG. 2018 The ovary and its genes—developmental processes underlying the establishment and function of a highly divergent reproductive system in the female castes of the honey bee, *Apis mellifera*. *Apidologie* **49**, 49–70. (doi:10.1007/s13592-017-0548-9)
93. Scharf ME, Ratliff CR, Wu-Scharf D, Zhou X, Pittendrigh BR, Bennett GW. 2005 Effects of juvenile hormone III on *Reticulitermes flavipes*: changes in hemolymph protein composition and gene expression. *Insect Biochem. Mol. Biol.* **35**, 207–215. (doi:10.1016/j.ibmb.2004.12.001)
94. Brent CS, Schal C, Vargo EL. 2005 Endocrine changes in maturing primary queens of *Zootermopsis angusticollis*. *J. Insect Physiol.* **51**, 1200–1209. (doi:10.1016/j.jinsphys.2005.06.009)
95. Elliott KL, Stay B. 2007 Juvenile hormone synthesis as related to egg development in neotenic reproductives of the termite Reticulitermes flavipes, with observations on

630 urates in the fat body. *Gen. Comp. Endocrinol.* **152**, 102–110.
631 (doi:10.1016/j.ygcen.2007.03.003)
632 96. Cornette R, Gotoh H, Koshikawa S, Miura T. 2008 Juvenile hormone titers and caste
633 differentiation in the damp-wood termite *Hodotermopsis sjostedti* (Isoptera,
634 Termopsidae). *J. Insect Physiol.* **54**, 922–930. (doi:10.1016/j.jinsphys.2008.04.017)
635 97. Korb J. 2015 Juvenile hormone: a central regulator of termite caste polyphenism. In
636 *Advances in Insect Physiology*, pp. 131–161. Academic Press Inc.
637 (doi:10.1016/bs.aiip.2014.12.004)
638 98. Sillam-Dussès D, Hanus R, Poulsen M, Roy V, Favier M, Vasseur-Cognet M. 2016
639 The role of the glucose-sensing transcription factor carbohydrate-responsive
640 element-binding protein pathway in termite queen fertility. *Open Biol.* **6**, 160080.
641 (doi:10.1098/rsob.160080)
642 99. Filhoulaud G, Guilmeau S, Dentin R, Girard J, Postic C. 2013 Novel insights into
643 ChREBP regulation and function. *Trends Endocrinol. Metab.* **24**, 257–268.
644 (doi:10.1016/j.tem.2013.01.003)
645 100. Yan L-J, Sohal RS. 1998 Mitochondrial adenine nucleotide translocase is modified
646 oxidatively during aging. *Proc. Natl. Acad. Sci.* **95**, 12896–12901.
647 (doi:10.1073/pnas.95.22.12896)
648 101. Fridovich I. 1977 Oxygen is toxic! *Bioscience* **27**, 462–466. (doi:10.2307/1297527)
649 102. Hetz SK, Bradley TJ. 2005 Insects breathe discontinuously to avoid oxygen toxicity.
650 *Nature* **433**, 516–519. (doi:10.1038/nature03106)
651 103. Lighton JRB, Ottesen EA. 2005 To DGC or not to DGC: oxygen guarding in the

652 termite *Zootermopsis nevadensis* (Isoptera: Termopsidae). *J. Exp. Biol.* **208**, 4671–
653 4678. (doi:10.1242/jeb.01934)
654 104. Tasaki E, Matsuura K, Iuchi Y. 2018 Hypoxia adaptation in termites: hypoxic
655 conditions enhance survival and reproductive activity in royals. *Insect Mol. Biol.* **27**,
656 808–814. (doi:10.1111/imb.12519)
657 105. Vander Heiden MG, Cantley LC, Thompson CB. 2009 Understanding the Warburg
658 effect: the metabolic requirements of cell proliferation. *Science* **324**, 1029–1033.
659 (doi:10.1126/science.1160809)
660 106. Harris AL. 2002 Hypoxia - A key regulatory factor in tumour growth. *Nat. Rev.*
661 *Cancer* **2**, 38–47. (doi:10.1038/nrc704)
662 107. Buffenstein R. 2008 Negligible senescence in the longest living rodent, the naked
663 mole-rat: Insights from a successfully aging species. *J. Comp. Physiol. B Biochem.*
664 *Syst. Environ. Physiol.* **178**, 439–445. (doi:10.1007/s00360-007-0237-5)
665 108. Gems D. 2000 Longevity and ageing in parasitic and free-living nematodes.
666 *Biogerontology* **1**, 289–307. (doi:10.1023/A:1026546719091)
667 109. Strahl J, Brey T, Philipp EER, Thorarinsdottir G, Fischer N, Wessels W, Abele D.
668 2011 Physiological responses to self-induced burrowing and metabolic rate
669 depression in the ocean quahog *Arctica islandica*. *J. Exp. Biol.* **214**, 4223–4233.
670 (doi:10.1242/jeb.055178)
671 110. Zhou X, Wheeler MM, Oi FM, Scharf ME. 2008 RNA interference in the termite
672 *Reticulitermes flavipes* through ingestion of double-stranded RNA. *Insect Biochem.*
673 *Mol. Biol.* **38**, 805–815. (doi:10.1016/j.ibmb.2008.05.005)

111. Zhou X, Oi FM, Scharf ME. 2006 Social exploitation of hexamerin: RNAi reveals a major caste-regulatory factor in termites. *Proc. Natl. Acad. Sci. U. S. A.* **103**, 4499–4504. (doi:10.1073/pnas.0508866103)

112. Modisett KL, Robinson CD, Raina AK, Lax AR, Michael SF, Lsern S. 2008 Foreign gene transfer in termite cells using a recombinant vesicular stomatitis virus. *J. Insect Sci.* **8**, 1–10. (doi:10.1673/031.008.5201)

113. Bonabeau E, Theraulaz G, Deneubourg J, Franks NR, Rafelsberger O, Joly J, Blanco S. 1998 A model for the emergence of pillars, walls and royal chambers in termite nests. *Philos. Trans. R. Soc. London. Ser. B Biol. Sci.* **353**, 1561–1576. (doi:10.1098/rstb.1998.0310)