

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

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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

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

96 The social behaviour and group living of termites reduce the risks of disease and
97 starvation in reproductives. Termites limit the spread of infection within the nest; the
98 antennae recognize pathogenic conidia [35], followed by initiation and maintenance of
99 allogrooming (social grooming) of contaminated body parts [36–38]. The additive effects of
100 individual immune responses and their social amplification are also important in terms of
101 anti-pathogen defence. Grouped termites cope better with disease than do isolated individuals
102 [36,39], suggesting that resistance is socially enhanced [40,41]. Group foraging may reduce
103 the risk of starvation in reproductives; especially in some foraging termite species, non-
104 reproductives forage actively, discover new food sources and store foods [42–46]. If termite
105 non-reproductives have residual nutrients to be shared via trophallaxis, the reproductives are
106 prioritised; the reproductives thus survive for longer even within a starved colony [47].

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

226 levels of the mTOR, eIF4, and RPS6 genes involved in TOR signalling and IIS than did
227 workers, suggesting that long-lived reproductives may downregulate these growth signalling
228 [87]. In general, these nutrient-sensing growth pathways effect juvenile hormone (JH)
229 production followed by upregulation of vitellogenin (Vg), a yolk protein required for egg
230 production [88,89]. Recent studies outlined the view that re-wiring of the IIS-JH-Vg circuit
231 has occurred in social insects which may explain the re-shaping of the fecundity/longevity
232 trade-off [90]. However, most data has come from the honeybee where JH has no further
233 function in the maintenance of the reproductive status in queens [91,92]. The positive
234 correlation between JH levels and vitellogenesis has been well studied in termites [93–97],
235 but almost nothing is known about the details of the IIS-JH-Vg circuit. Further research on
236 the relationship between the IIS-JH-Vg circuit in termites is needed.

237 There is also possibility that termite reproductives, especially queens, may perhaps
238 reduce these signalling without sacrificing high-level reproductive performance, by
239 employing other signals to control reproduction. The transcription factor termed
240 carbohydrate-responsive element-binding protein (ChREBP), a glucose sensor regulating the
241 expression of genes that drive fatty acid biosynthesis, was highly expressed in mature queens
242 of eight different termite species, compared to the levels in sterile workers and soldiers [98].
243 ChREBP critically mediates the glucose-dependent induction of glycolytic and lipogenic
244 genes in metabolic tissues [99], and may thus provide essential precursors of oogenesis via
245 redirection of a significant proportion of glucose carbons to de novo lipogenesis and
246 nucleotide biosynthesis. Thus, in addition to growth hormone signalling, IIS and TOR
247 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 O₂; approximately 15%) and hypercapnic (high in CO₂; approximately 4%) [34]. Oxidative stress is influenced by the level of O₂ present, shown by the fact that increases in the level of O₂ in the atmosphere that the insects are breathing lead to enhanced rates of oxidative damage and reduced longevity [100]. Atmospheric O₂ 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

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

299 Since termite reproductives have extraordinary longevity, it is difficult to track their
300 entire life, and there is not so much information on the lifespan of termites [5,11–14].
301 Therefore, reliable and valid biomarkers of chronological and biological age are essential for
302 future advanced studies of termites. This would allow us to take into account the effects of
303 age, which is one of the difficulties in termite researches. Moreover, molecular studies of
304 termites have identified several genes that may be involved in longevity assurance, but their
305 functions remain unclear. Since it is necessary to carefully assess how termite reproductives
306 achieve extended lifespan, further studies are required to evaluate the biological function of
307 these genes in the longevity of termites using genetic tools, such as RNA interference
308 [110,111] and transgenic systems [112].

309 The quest for understanding the proximate mechanisms of extraordinary longevity in
310 termite reproductives is only the beginning. We are now at the dawn of a big paradigm shift
311 in the ageing study of termites having the great opportunity to uncover the unexplored
312 longevity-mechanisms that cannot be reached by studies using intrinsically short-lived model
313 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.

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figure 1

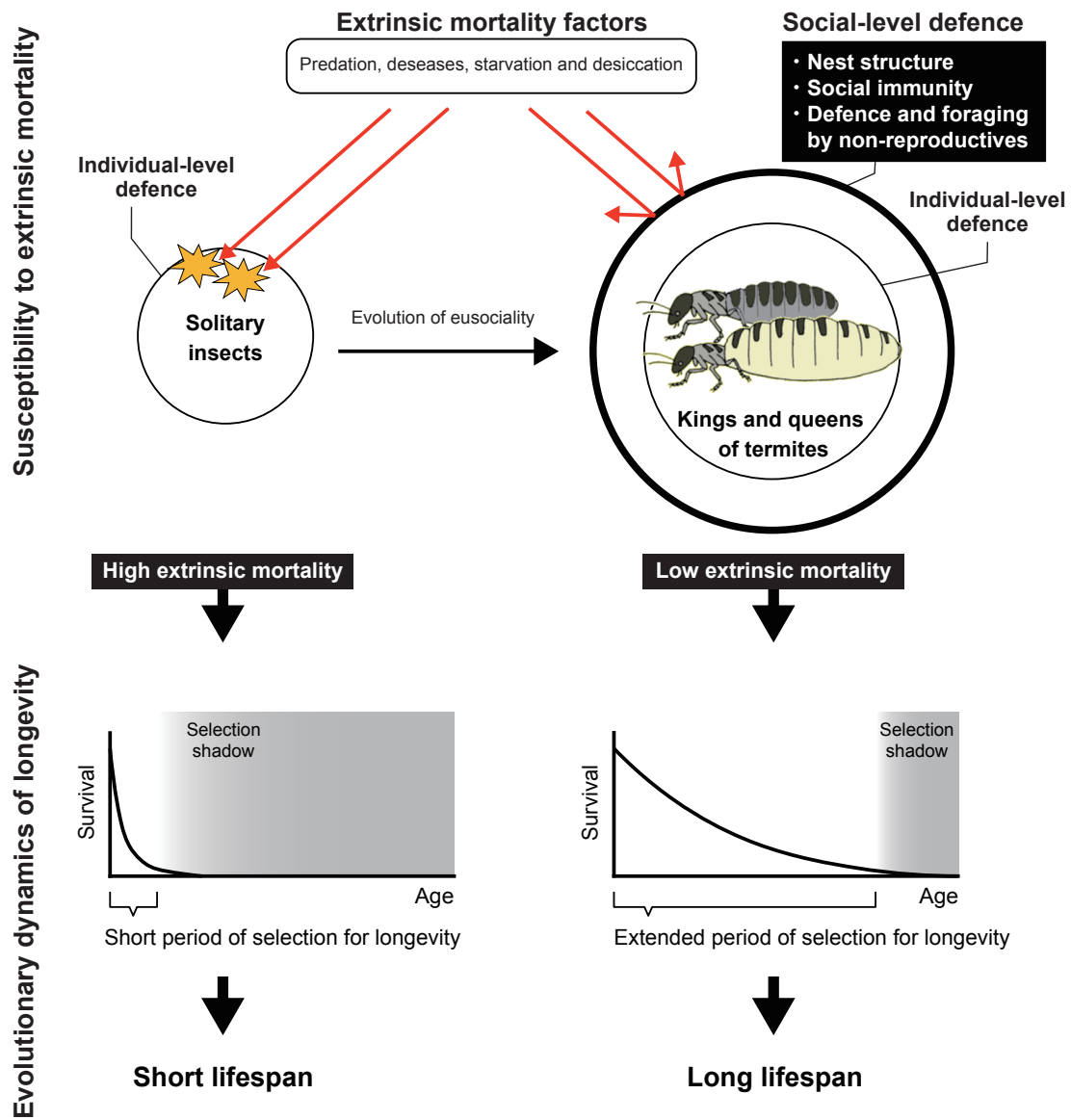
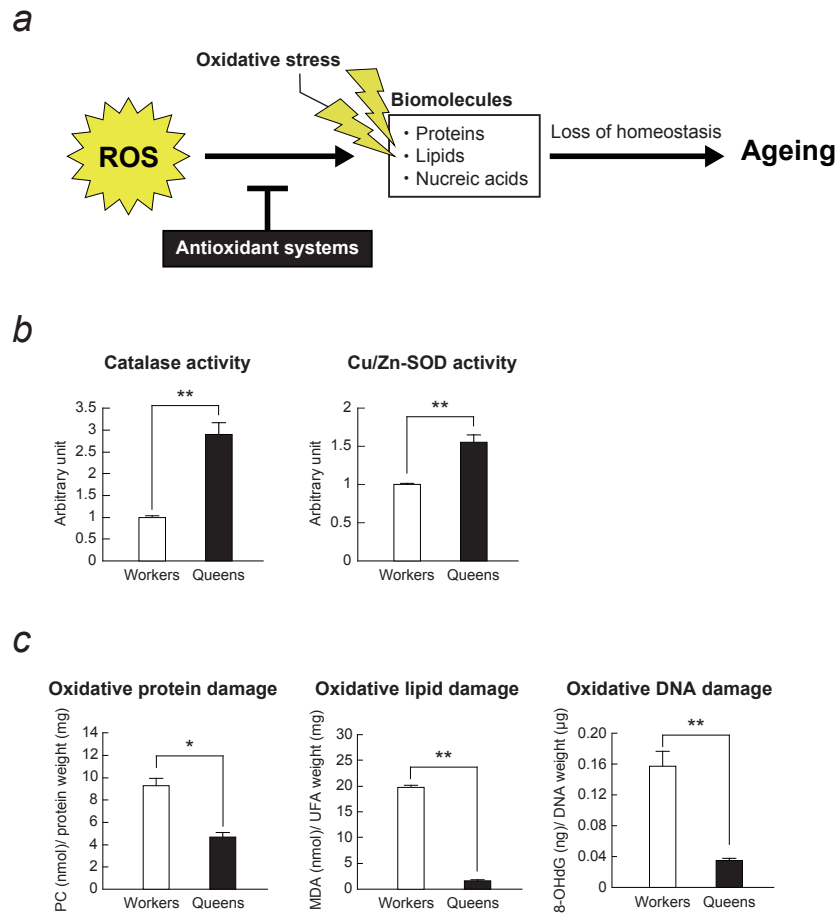


Figure 1. Schematic of the evolution of extended longevity in termite kings and queens. The upper panel shows the susceptibilities to extrinsic mortality of solitary insects (left) and termite reproductives (right). Black solid circles indicate defence systems; termite reproductives implement both individual- 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
<i>Ecological characteristics</i>		
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]
<i>Physiological characteristics</i>		
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]

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