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A critical look at proximate causes of social insect senescence: damage accumulation or hyperfunction?

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Social insects have received attention for their extreme lifespan variation and reversal of the fecundity/longevity trade-off. However, proximate causes of senescence in general are disputed, and social insects often fail to meet the predictions of prevailing models. We present evidence for and against the long-held free radical theory of aging in social insects, and consider the application of the competing hyperfunction theory. Current results present problems for both theories, and a more complex picture of the biological processes involved emerges. The eusocial life style might allow colonies to allocate damage in ways that create seemingly senescence-free life histories. Only experimental approaches characterizing multiple senescence factors simultaneously will shed light on how social insects defy the conventions of senescence.

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Introduction

Virtually all organisms show signs of senescence, a functional decline over time. A limited reproductive lifespan is among the most fundamental life history traits of an organism. In most animals, reproduction and longevity are negatively correlated, allowing for evolutionary theories like the disposable soma theory to explain senescence as a strategic trade-off [1] (Kuhn and Korb, this issue). However, social insects appear to defy this generalization, often with extreme differences in lifespans of related individuals (reproductives outlive workers by large margins [2]). This observation presents social insects as ideal model systems to investigate the physiological, genetic, and behavioral correlates of lifespan variation. A generally accepted framework delineates senescence as caused by the interaction of random and inevitable destabilizing forces, counteracted by adaptive and genetically controlled maintenance measures. However, surprisingly little agreement can be found among the mechanistic theories of senescence, some of which are championed with strict exclusivity [3]. For social insects, a wealth of empirical evidence exists (particularly for honeybees), which can be interpreted as for or against the prevailing theories, but taken together paint a much more complex picture of many interacting processes, some of which may be remodeled from their relationships in solitary organisms. In this paper we offer a brief review of two theories of senescence, the prevailing free radical theory and the more recent hyperfunction theory. We suggest how these theories might be invoked or rejected from the symptoms of senescence found in social insects.

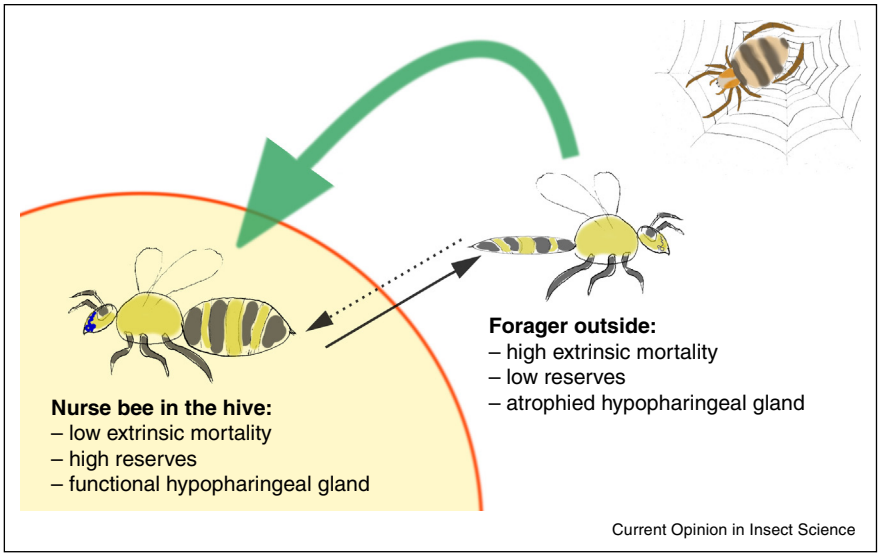
Senescence in social insects

We know little about functional degeneration in social insects with age, in particular in queens. Behavioral experiments have provided surprisingly little evidence for functional decline with age in most social insects, with the exception of honey bee workers [4,5,6•,7,8••,9,10]. Honeybee foragers show an age-related decline in learning ability and bee nurses can lose the ability to produce jelly [5,11–14].

In the hypopharyngeal glands, pars intercerebralis, and calyx of honeybee foragers, lipofuscins — aggregations of essentially non-degradable residues of lysosomal waste — accumulate with age [5]. Mitochondrial energy utilization declines with age, which is counteracted by increasing the density of mitochondria [22]. Older individuals are typically more frail than younger ones in that they suffer more from starvation and oxidative or heat stress [13,23,24••]. In contrast, a recent study with ants showed no signs for increased brain apoptosis and no decrease in microglomerular density [8••].

The immune system changes with age. While hemocyte counts decrease in honeybees, phenoloxidase levels in bees and leaf-cutting ant workers and antimicrobial peptide gene expression in honeybees increase [25–27]. While these increases could in theory be caused by individuals making up for decreasing efficiency of the immune system due to senescence, they could also simply reflect an increase in pathogen load due to changing behavior. Honeybee workers begin their lives as nurses and later switch to foraging (Figure 1). The transition is associated with increased resistance against entomopathogenic fungi and hematocyte loss [28]. Interestingly,

Figure 1



The typical temporal polyethism of honeybee workers includes a switch from nursing the brood inside the hive to foraging outside (solid arrow). During the transition individuals lose weight (see skinny abdomen), which could optimize them for flying, but may also constitute a divestment of resources in these foragers. As extrinsic mortality outside the hive is high, resources invested in foragers are constantly at risk. Resources would be safe if workers could transfer them back into the hive before they become foragers (green arrow), for example in the form of vitellogenin fed to larvae. The hypopharingeal gland (blue, in the heads) is heavily used during nursing but accumulates damage and loses its function when the nursing phase lasts too long. In foragers, the gland is typically atrophied, suggesting that its maintenance is only provided as long as the organ is needed. As functional loss to the gland typically only occurs once it is not used any more, the loss will go undetected in most studies. The dotted arrow indicates a reversal of foragers to nurse work, which can be experimentally induced. The reversal rescues some symptoms of senescence in honeybee workers, which provides an excellent opportunity to challenge the theory that senescence is exclusively due to passively accumulated damage.

this loss is reversible through the proliferation of new cells when foragers are manipulated to revert to nurses [29]. In subsocial burying beetles, personal immune function declines with age but is still upregulated when beetles breed [30]. Such flexibility indicates that changes in immunity are likely to be context-dependent instead of being subject to inevitable senescence.

Worn out mandibles can reduce behavioral performance in ants, bees, and termites [6•,15]. Wing wear increases honeybee worker mortality in the field, but not the metabolic flight cost. Instead, the functional decline in flying ability is likely to cause extrinsic mortality. Foragers with worn-down wings are more susceptible to predation due to slower flight and thus longer foraging bouts, and due to decreased ability to escape predators [15–17]. Whether intrinsic mortality rates of social insects typically increase with age is unclear [4,18–21]. While many studies make lifespan estimates, they are often only reporting averages but provide little information on the shape of the underlying survivorship curves.

ROS theory of aging

With the identification of genes that modulate lifespan, research on senescence and its prevention entered a new era [31]. The genes are thought to code for protective mechanisms preventing the eventually lethal accumulation of damage through non-adaptive, non-targeted, and inevitable destabilizing forces. Most of the resulting damage accumulation hypotheses have since been discarded or joined and are now typically treated as a single free radical theory of aging [32–34].

Living requires active mitochondrial respiration, and errors in the underlying reactions produce reactive oxygen species (ROS, Figure 2 [35]). At low levels, ROS fulfill important functions (signaling) and may have beneficial effects (hormesis [36]), but in high doses ROS exert oxidative stress on the cell and, if left unchecked, can damage macromolecules (Figure 2 [32,33]). The free radical theory of aging evokes a model of senescence as the effect of chemical degradation of unstable machinery over time (often crudely depicted as the rusting of a car). By this theory, interventions that reduce levels of ROS and subsequent oxidative damage should retard senescence, those that increase damage accelerate it. Interestingly, ROS production is often highest when energy expenditure is lowest, and across taxa long lives are correlated with high metabolic rates [33,34]. These findings contradict the live fast — die young emphasis of the rate of living theory [32,37] and instead point to a less direct connection of metabolism and lifespan.