Received: 29 March 2019 | Accepted: 26 September 2019

DOI: 10.1111/1365-2435.13462

AN INTEGRATIVE VIEW OF SENESCENCE IN NATURE

Functional Ecology | BRITISH ECOLOGICAL SOCIETY

Sex, senescence, sources and sinks

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Handling Editor: Jean-Michel Gaillard

Abstract

1. Why do most organisms age, and why do most of them reproduce sexually? Does sex rejuvenate? We review progress that has been made linking theories of senescence with those of sexual reproduction.
2. We show that there is a dearth of theory against the numerous questions waiting to be answered theoretically or empirically: observed senescence patterns are a result of past selection acting on individuals of different age categories and abundances, modes of reproduction (asexual, sexual, facultatively sexual, via buds or zygotes). Modular organisms present their own challenges. Assigning offspring an unambiguous age of zero at birth is often too simplistic.
3. We also comment on germline mutations as a form of ageing over generations (Lansing effect) and ask whether there is value in reinvigorating an old metaphor (1988, Sex and death in the protozoa. New York, NY, USA: Cambridge University Press) of chairs and their chairmakers who both are subject to deterioration over time, since endogenous repair is never foolproof.
4. Future theory could usefully revisit a known mathematical analogy between selection on senescence across age classes and source–sink theory. Some modes of reproduction (particularly asexuality) may yield offspring that are, in a sense, already aged, with the problem increasing over generations (ultimately leading to a demographic sink). Insofar as sexuality 'rejuvenates', it does so through a gamble that, with some frequency, produces 'exogenously repaired' individuals that act as a source of genes to future generations. This gamble has been argued to be managed best when life cycles include a unicellular stage (zygote), an argument that could be usefully complemented with an analysis of the relevant economic trade-offs between offspring size and number.

KEYWORDS
ageing, clonality, deleterious mutations, disposable soma, germline, repair

1 | INTRODUCTION

Life is about replication, but replication is not 100% faithful. Much of any faithfulness deficit is 'unintentional' in the sense that the vast majority of non-neutral mutations are deleterious (Lynch et al., 2016). Sex, in this framework, is somewhat of a mystery because it is a thoroughly non-faithful mode of reproduction. When defined as biparental sex (Lehtonen & Kokko, 2014), sex involves the fusion of gametic material from two different parents—a very strong deviation from even attempting 100% parent–offspring semblance.

The evolutionary maintenance of sex becomes easier to grasp when considering the fate of lineages reproducing purely asexually. Graham Bell's (1988) second book on sex is particularly insightful. He focuses on the consequences of accumulating deleterious

mutations in protozoan lineages, with much of the content devoted to understanding why it is difficult to keep protozoan lineages alive if kept singly, despite an a priori expectation of unlimited potential for asexual reproduction (Box 1). One proposed answer is that sex rejuvenates. Sex, by producing a large diversity of variants subject to selection, might provide effective means of so-called exogenous repair, potentially keeping germlines free of mutations over time (Box 1). While there is also a 'creative' side to sex, in alleviating clonal interference and thus speeding the process of bringing together beneficial mutations (McDonald, Rice, & Desai, 2016; Peabody, Li, & Kao, 2017), the problem of mutation accumulation control remains a central one in the maintenance and reproduction of organisms. This links the topics of senescence and sex—their interaction being the focus of our review.

BOX 1 Chairs and their chairmakers

Evolutionary ecology teems with metaphors (Olson, Arroyo-Santos, & Vergara-Silva, 2019), from green beards to red queens; many of them have their origins in the 1960s-80s (Bell, 1982; Hamilton, 1964; Van Valen, 1973). Graham Bell (1988), in *Sex and death in protozoa: the history of obsession* (the less well-known 'sequel' to his 1982 book on sex), used a metaphor of chairs and chairmakers that has had meagre success permeating the literature. Beneficial mutations may or may not spread, likewise the success of phrases is probably an imperfect measure of their actual usefulness, and we believe Bell's phrase might contain resurrection potential.

Imagine that a chairmaker's task is to produce numerous copies of good chairs, without being allowed to keep the original blueprint anywhere. One can take a pre-existing chair as a template and try to minimize the differences between old and new. If the new differs from the old, the assumption is that a mistake occurred during manufacture of the new one, and measures can be taken to minimize the difference. Here, Bell had DNA repair and mitotic cell division in mind, calling this *endogenous repair*. Alternatively, one could leave quality control to the customer: the chairmaker will now put together unique chairs from mixes of chair parts; some products will not withstand the force of someone actually sitting on it. Only chairs 'surviving' this selection process will then remain in the population and used as templates to produce further chairs. This is *exogenous repair*, analogous to selection on variation produced by recombination during meiosis and the mixing of genomes through sex. Both methods have their pros and cons, especially when one notes that the chairmaker's own procedures, too, might be unfaithfully copied to become a new chairmaker. In other words, if an organism's genome contains instructions how to repair DNA, these instructions themselves can become victim to deleterious mutations.

Senescence is defined here as a general decrease in organismal performance with age. It is thought to result from unequal 'visibility' of individuals differing in age to natural selection (Hamilton, 1966). Selection does not favour improvements of vital rates with equal strength across all ages because mortality itself, together with the capacity of a population to grow, causes populations to contain more young than old individuals (Caswell, 2010; Hamilton, 1966). Intriguingly, this is mathematically analogous to source–sink dynamics (Holt, 1996): individuals that reside in a habitat that contributes disproportionately to future generations 'weigh' more in determining the evolutionary trajectory of a population (and sink residents' traits do not count at all unless there is migration back to the source), a point we will return to.

Senescence models differ in whether they evaluate the strength of selection against deleterious mutations acting at different ages (mutation accumulation theory: Medawar, 1952), or whether they assume antagonistic pleiotropy, where mutations may bring about strong early performance at the expense of poor later performance (Hamilton, 1966; Williams, 1957). The disposable soma theory of senescence (Kirkwood 1977, Kirkwood, 2017; Kirkwood & Holliday, 1979; Kirkwood & Rose, 1991) encourages an explicit view on somatic repair. Here, the soma always deteriorates, but this can be slowed down with endogenous repair (Box 1); however, achieving slowing down (delaying senescence) requires reducing growth and/or reproduction. This trade-off, which involves *energy* allocations in a broad sense, has recently been complemented with a focus on *function*: here optimizing traits to good performance at a young age may lead to suboptimal performance when the same gene expression is used by the same organism at a later age (Maklakov & Chapman, 2019). Such integrative work appears welcome to us, as it makes sense to combine the antagonistic idea of mutations having different effects at different ages with an explicit temporal look where an organism always 'inherits' its current soma from its own earlier self.

Deleterious mutations are often modelled over generations (Bell, 1988; Ho & Agrawal, 2017). The disposable soma theory of senescence and empirical evidence both highlight that limiting DNA damage and somatic mutations is simultaneously key to creating a healthy soma of a multicellular organism. Despite organisms' efforts at DNA repair, cell lineages cannot be copied indefinitely without accumulating differences to the originator cell (e.g. humans: Martincorena et al., 2018, plants: Wang et al., 2019). Tissues, if left to divide indefinitely, are therefore predicted to succumb to loss of cooperation (Nelson & Masel, 2017) that may manifest itself as cancer (modelling of the age-dependent probabilities: Kokko & Hochberg, 2015) or homeostasis loss (Cohen, 2016; Kirkwood & Holliday, 1979; Li et al., 2015), both indications of a senescing phenotype.

Organisms are expected to respond strategically to somatic mutation accumulation. Even bacteria, where the concepts of germline and soma do not apply in the traditional sense (but see Aanen & Debets, 2019), exhibit asymmetries at cell division: each cell has an old and a new pole, and division leads to one daughter cell inheriting

the old pole, while the other is a 'new' daughter cell, initially freed from problems such as pre-existing misfolded proteins. Insofar as the rejuvenated daughter cell can be considered younger than the cell inheriting the old pole, the concept of a stable age distribution therefore applies to growing unicellular populations (Proenca, Rang, Buetz, Shi, & Chao, 2018). This example also shows the limits of solely focusing on DNA. In bacteria, the reason why one product of cell division is 'older' than the other is directly related to which end keeps the old cytoplasm with all its accumulated damage, and this asymmetry itself is a product of a regulatory cascade (Ackermann, Stearns, & Jenal, 2003; Lindner, Madden, Demarez, Stewart, & Taddei, 2008). Along similar lines, senescence in multicellular organisms cannot be viewed solely via the lens of DNA mutation: if no other factor played a role, irradiation experiments would increase ageing rates in a straightforward manner, but that this is not the case was already pointed out by Bell (1988); for an updated view, including tissue-specific issues, see Vijg, 2000.

Life's diversity, together with the diversity of potential repair mechanisms that can keep a soma from deteriorating (for a while at least), yields the interesting question of whether the deterioration problem is solved differently in unicellular organisms (e.g. bacteria), those that alternate unicellular and multicellular stages rather flexibly (e.g. slime moulds), modular organisms (e.g. corals, plants) in which vegetative reproduction is a viable option (and germline segregation patterns vary, Lanfear, 2018, Munné-Bosch, 2018, Radzvilavicius, Hadjivasiliou, Pomiankowski, & Lane, 2016, Wang et al., 2019), and, finally, unitary organisms with a clearly segregated germ line. Germ cells inside unitary organisms may live quite 'pampered' lives, and the maintenance of such conditions may in turn be costly for the soma (Kirkwood, 2017; Maklakov & Immler, 2016), but recent snippets of evidence tantalizingly suggest germline-like structures in vegetative forms of plant reproduction (runners of strawberries) (Wang et al., 2019).

We therefore here ask whether sex allows lineages and individuals who compose them to avoid accumulating mutations and damage with time, both within and across generation. Specifically, we first discuss the concept of age distributions in organisms with different modes of reproduction, before considering its consequences for damage accumulation across generations and selection on senescence.

2 | HOW OLD ARE YOU, REALLY?

Any analysis of senescence requires determining an individual's age. While this may sound straightforward, the bacterial example above, and the language of sex 'rejuvenating' (which implies that asexuality might fail to do so), highlights that even the most basic assumption should not be taken for granted: What age should we assign to newborns?

The age of a newly produced individual can be defined in (at least) two different ways. It can refer to the number of time units since this individual—defined as a physically independent unit—arose in the population; we call this the demographic age (which we judge to be a clearer term than chronological age, Buss, 1987). Sexually as well as asexually produced zygotes, as well as any form of vegetative growth that leads to physical separation of 'parent' and 'offspring', all lead to an individual being young (age 0) in this demographic sense. But individuals in the same cohort are not necessarily young in terms of all the processes that make their cells senesce. More flexible usage is apparent when the two daughter cells in asymmetric bacterial division are assigned the labels 'old' and 'new', in the fact that metazoans experience potentially long-term carryover life-history effects when 'old' germline cells produced the zygote (Bell, 1988; Priest, Mackowiak, & Promislow, 2002), as well as in the way a lineage that has persisted solely via asexuality over many generations can be said to be an 'ageing' one (Ho & Agrawal, 2017; Lynch, Bürger, Butcher, & Gabriel, 1993). All these are aspects of what Buss (1987) calls genetic age and Bell (1988) calls clonal age, but as the diversity of examples above and in Box 2 shows, not all effects are necessarily traceable to changes in the genome. For this reason, we prefer a more all-encompassing term. According to myth, the founder of the Liao Chinese dynasty was a man called Abaoji, who was remarkable for having been born as a 3-year-old child. We shall, in the remaining of our review, call Abaoji-age or A-age, for short, the dimension of an individual's age that refers to the state of its soma.

Importantly, despite A-age referring to the soma, an organism cannot always be expected to be at A-age zero when the zygote is freshly formed. For example, gametes in yeast are rejuvenated in a process where nuclear senescence factors and nucleoporins are sequestered away from chromosomes during meiosis II (King et al., 2019). Each set of simultaneously formed four gametes comes with one membrane-bound 'rubbish bin', which is subsequently destroyed. This finding is so recent that we do not yet know much about potential variation in the reliability of this process, but it is easy to envisage situations that not all damaged elements are sequestered correctly (e.g. if there is simply too much damage to deal with). Should this be the case, then even sexually produced young might start with zygotes that are less 'fresh' than others in the population.

Obviously, we do not claim that once the demographic age and the A-age are known, all the axes required to study senescence have been defined: if Wang et al.'s (2019) interpretation proves correct, a strawberry runner consists of parts that belong to a disposable soma and others that are germline-like. Each can harbour mutations, and aspects of ageing that are not captured by DNA might also be found (akin to the bacterial examples above, or epigenetic). Also, for certain problems, such as the analysis of natural populations where individuals are captured at an uncertain age, other measures of relative age may offer insight. Reed et al. (2008) provide such an analysis for sex-specific ageing in guillemots, by examining fecundity as a function of 'years before death', that is the time the individual lived *after* the focal breeding event. We use A-age simply to chart current theoretical efforts as well as outline a path towards theoretical developments when, for example, wanting to consider how a lineage

BOX 2 Endogenous repair: how to build a robust soma

Keeping a disposable soma going for longer (i.e. delaying senescence) is a multifaceted problem. While ageing does not only involve accumulating DNA damage (for a complex case, see Lind et al., 2019), somatic mutations make it impossible to maintain a soma in its pristine state (Martincorena et al., 2018). Delaying the accumulation of damage is possible as DNA can be repaired within a cell using pre-existing signalling pathways (Heijink, Krajewska, & Vugt, 2013).

At the organismal level, multicellularity offers more options; for example, one cell may be sacrificed for the greater good of a well-functioning soma. One such mechanism involves limiting the replicative potential of cells. There is an interesting inherent nonlinearity, as a limit restricts how long a soma can live (assuming mitotic divisions are necessary for continued life), but may be safer over reasonable life spans. Telomere attrition stops any one cell lineage from replicating endlessly (Risques & Promislow, 2018). Telomerase can counteract telomere shortening, and while at first sight useful as a way to keep homeostasis and prolong life (Haussmann et al., 2003), the potential for uncontrolled growth brings about an elevated cancer risk (Risques & Promislow, 2018). The abnormal telomerase that is associated with many cancers (Jafri, Ansari, Alqahtani, & Shay, 2016) is a clear case of a damaged chairmaker, if one expands Bell's analogy (Box 1) to any type of quality control applied at cell division. Other defences include numerous tumour suppressors found in genomes of large metazoans, the most famed (and ubiquitously studied) being p53, which may be particularly important for large-bodied and long-lived organisms (Abegglen et al., 2015). Damaged cells accumulate p53 and respond by committing suicide (apoptosis) for the benefit of the entire organism, when this mechanism is intact. The 'chairmaker' role of p53 is evidenced by humans with mutations in this gene, who are at very high risk of developing various cancers at an unusually young age (Nagy, Sweet, & Eng, 2004).

The process of tissue differentiation itself, organized as a hierarchy where some cell lineages are set aside to become a relatively quiescent germline, separate from metabolically active soma (where differentiation can also lead to specialized tissues that perform specific tasks), appears an adaptation that allows the next—possibly sexual—generation to arise from a less damaged lineage than if there was no such division of labour (Berger, Stångberg, Grieshop, Martinossi-Allibert, & Arnqvist, 2017; Goldsby, Knoester, Ofria, & Kerr, 2014; Maklakov & Immler, 2016). Germline cells segregated early in development do not go through many mitotic divisions before forming a gamete (though this statement has to be made with sex differences in mind: males typically contribute more mutations, Wilson Sayres & Makova, 2011). Germline cells also remain functionally quiescent, which limits cellular respiration and the accumulation of damages from reactive oxygen species (Goldsby et al., 2014).

of asexual organisms accumulates age as it spreads (Ho & Agrawal, 2017; Sköld, Asplund, Wood, & Bishop, 2011).

3 | REPRODUCTIVE MODES AND THEIR CONSEQUENCES FOR SENESCENCE

Do organisms with different modes of reproduction, germline segregation and growth (unitary or modular, determinate or indeterminate) differ in how selection shapes senescence? Classic senescence theories yield no clear answer, as they tend to assume that the zero age at birth is undisputable and that senescence is a problem of the soma only—a clear reflection of our general tendency to build theories with a life history in mind that resembles our own, whether or not it is commonly found in nature (Kokko, 2017).

Intuition suggests that the more clearly an organism's performance relies on different tissues complementing each other (e.g. the one irreplaceable heart and one liver of a vertebrate), the greater the potential for somatic alterations in one tissue to have substantial deleterious fitness effects for the whole soma (see also Dańko, Kozłowski, & Schaible, 2015). If we, however, take the above intuition to mean that modular organisms that even perhaps lack a clearly distinct germline will not senesce, then data have the annoying tendency to conflict such views (Martinez & Levinton, 1992). Consider, first, the complications produced by colonial organisms where vegetative reproduction is possible. In these, a genet refers to the collection of all the 'individual' ramets, that arise from a single origin, that still reside physically together, and can be considered part of the same clone (in a broad sense: it would be too much to require zero within-genet genetic diversity—if Martincorena et al., 2018 found much genetic diversity in skin samples from the same human individual, it is no surprise that modular genets are diverse too, Pineda-Krch & Lehtilä, 2004, Barfield, Aglyamova, & Matz, 2016, Bythell, Brown, & Kirkwood, 2018, Wang et al., 2019). Definitional questions may arise at the grey zone: although dispersing units are often sexually produced, vegetative units may also disperse (though often shorter distances, Gerber & Kokko, 2018) and lose interconnections (Schaible, Ringelhan, Kramer, & Scheuerlein, 2017).

How far can a ramet leave its original site of growth and still be considered part of the same genet? This is not mere semantics, but relevant for making predictions on senescence in genets and ramets. Predictions for senescence of entire genets being mild or completely absent are often based on an analogy with unitary organisms with life cycles where late demographic ages associate with high reproductive success—should the organism reach that age in the first place. Thus, if we consider physically well-delineated genets, and larger colonies less likely to disappear due to all ramets dying (Gardner & Mangel, 1997; Orive, 1995), then

the reason for clonal reproduction to select against senescence in genets (Caswell, 1985; Gardner & Mangel, 1997; Orive, 1995; Sköld & Obst, 2011) is similar to unitary organisms that experience strong selection against senescence when large size permits high fecundity and it takes time to reach large sizes (Baudisch & Vaupel, 2010; Marshall & White, 2019). In some unitary organisms, the late-life benefit might also relate to learning life skills (if this takes time, Angelier, Weimerskirch, Dano, & Chastel, 2007; DuVal, 2013) or other types of phenotypic plasticity (Ratikainen & Kokko, 2019), and further examples might be found, for example, in biparental birds whose coordination, and thus breeding success, improves with the number of times that both parents breed together (Black, 2002; Sánchez-Macouzet, Rodríguez, & Drummond, 2014). Selection against genet-level senescence, in this view, has little to do with sexuality versus clonality per se. The models tend to assume that fitness is ultimately measured as the growth of a population of genets in terms of production of dispersing propagules, which are sexually produced units of reproductive success, analogous to the growth rate of a population of a unitary indeterminate grower producing zygotes.

However, as explained above, modular organisms differ from unitary ones in that there is a grey zone: if a ramet becomes detached from its parent colony and disperses, it may proceed to found a new colony elsewhere, with consequent problems of defining the age of such a genet (or the new ramets within it). Dispersal may even be the norm, in which case ramets tend to exist in a solitary state: hydra do not form coral-like colonies (Schaible et al., 2017), and many trees likewise do not form colonies with as clear physical isolation from other colonies as coral genets are from each other (Buss, 1987). In such cases, it seems important to focus on the senescence of ramets, instead (or in addition to) genets (Larson, 2001). Specifically, we can ask how classical theories of senescence help apprehending ageing in physiologically and physically independent products of vegetative reproduction.

Let us, for a moment, simplify the situation by considering that an individual's state can be specified with just two ages: the demographic age, and a one-dimensional version of A-age— that is we assume that individuals vary in how damaged and/or mutated they are at birth, but this can be adequately captured with just one dimension. New individuals have a demographic age of zero regardless of whether they arise through sexual or asexual reproduction, but the mode of reproduction that created them may impact their A-age.

To provide an extreme but illustrative example, consider the fate of a deleterious mutation that is lethal, but only expressed late in life. Individuals with a small A-age take longer to express this mutation. For simplicity, we achieve this by assuming that the deleterious mutation is expressed deterministically once an individual reaches an A-age of 10, and some are born older using this measure (while A-age accumulates at the same pace for everyone after birth). To keep matters as simple as possible, let us first work within the mutation accumulation framework of Medawar (1952); thus, there are no early-life benefits. How efficiently will selection remove this mutation from the population? If A-age never differs from demographic age, and the organism is unitary, age distributions are easy to compute (though in practice, researchers disagree regarding the causal role of ecological factors such as density dependence, Dańko, Burger, Argasinski, & Kozlowski, 2018; Moorad, Promislow, & Silvertown, 2019; da Silva, 2018). If, however, some individuals begin their lives in a decayed state already, the two-dimensionality of the age distribution begins to show its effect: the phenotypic expression of the mutation will shift towards demographically younger ages in at least some individuals.

The exact details of the 2-dimensional age distributions require formal modelling, but intuition suggests that the scenario can give rise to different senescence predictions, depending on the details. All else being equal, the presence of demographically young but somatically 'aged' individuals means that the expression of the lethal allele begins to occur at demographically more numerous age classes, implying that selection against this deleterious mutation will intensify. However, all else is not necessarily equal, which brings us back to source–sink theory.

4 | YOUNG SOURCES AND OLD SINKS

The above verbal model oversimplified the complexities of the demography, since it did not track the production of different offspring types from different parents. In reality, the class of demographically numerous (young) but genetically or otherwise 'damaged' individuals is likely to perform poorly in terms of reproductive value, not only because of limited future life span, but also because their offspring might have accumulated even more damage. Such offspring might be expected to achieve even poorer reproductive success themselves: such a lineage will contribute little to future generations, even if the original parents were still relatively undamaged.

In this context, it is useful to discuss Holt's (1996) results in more detail. He pointed out a deep analogy between theories of senescence and adaptation to sink habitats in a source–sink system. Deleterious mutations are not efficiently removed from a sink habitat (Holt & Gaines, 1992). This effect occurs because sink populations by definition are unproductive (Holt, 1996; Kawecki & Holt, 2002), analogous to selection being blind to how well an individual survives after a parasite has castrated it (even if such individuals happened to be common). Sources and sinks may be spatially distinct habitats, or—more relevant to our current question—individual classes, such as a categorization involving demographic and A-age, with disproportionate chances to contribute to the long-term future of a population (i.e. with different reproductive values). For instance, if we consider 'sink' individuals equivalent to individuals of great A-ages, then the strength of selection on vital rates should decrease as A-age increases. The expected weakening in selection with increasing demographic age in classical theories of senescence becomes equivalent to selection being relatively blind to performance in the sink. Future generations are, by definition, mainly comprised of descendants of current 'source' residents, and 'sink' individuals will be continually produced by those in the 'source'. Exact predictions about the fate of favourable or deleterious mutations on vital rates will depend on gene flow from the source to the sink, and (potentially) *vice versa*. In source–sink models, sink-produced individuals may indeed sometimes migrate

back to the source, and this impacts their reproductive value. In our context, if sex rejuvenates, then A-old individuals' genes will go back to well-functioning bodies once sex has occurred.

What a particular class of individuals can achieve becomes particularly complex if, from deleterious mutations, one begins to consider trade-offs between trait expression at early versus later life. Predictions regarding optimal ontogenetic trajectories of reproduction and growth also depend on whether self-inspection is possible, permitting reaction norms of reproductive effort as a function of own A-age. Predictions may change again once modelling considers costs of germline maintenance (Maklakov & Immler, 2016), hierarchical tissue organization (Derényi & Szöllösi, 2017), maintenance of the soma in the presence of oxidative damage (Tan et al., 2012) and DNA repair needs (Dańko et al., 2015). It may be frustrating that so little can be said in terms of general, directional predictions, but this only highlights the need to work on theory in a field where empirical evidence shows mixed patterns so far.

For example, in *Daphnia*, asexual clones appeared to senesce slower than sexuals (Dudycha & Hassel, 2013) while an analysis of 181 species of plants showed the opposite pattern (Salguero-Gómez, 2018). Highly intriguingly, both sets of authors stated the patterns to be counterintuitive, implying that the intuition of the authors was opposing each other as well. It is difficult to know why intuition suggests one pattern to one person and another to someone else, but we suspect the following to play a role. If one can identify conditions where many individuals are in a state where they express deleterious traits, one person's intuition might suggest efficient selection against senescence and 'little senescence' as the ultimate outcome, while another's focuses on the immediately visible effect of many individuals succumbing to early death (selection in action). The pattern may be particularly complex in facultative sexuals that produce progeny via two reproductive modes from a (more or less) common gene pool. The efficiency of selection will also depend on effective population size: finite population sizes, with stronger drift in smaller populations, lead to the accumulation of deleterious mutations through Muller's ratchet. Senescence patterns in *Daphnia*, where small populations show faster senescence, have indeed been attributed to such differences in genetic load (Lohr, David, & Haag, 2014).

5 | DOES SEX TRULY REJUVENATE?

What the above tacitly assumed is that at some stage the genet will produce 'rejuvenated' dispersing propagules—*via* sex—that form new genets, or, in a source–sink worldview, that sex is the more reliable way to form new 'source' individuals. Rejuvenation is also an inherent part of the 'disposable soma' theory: it only makes sense to dispose of the old soma if the new one is in a more pristine state. But is it actually true that sex rejuvenates?

Because sex often associates with germline segregation early in development and the repair of DNA double-strand breaks via recombination, it seems a priori more efficient than asexual modes of reproduction at producing offspring from cells with little accumulated damage and deleterious mutations (Mirzaghaderi & Hörandl, 2016, King et al., 2019, Box 2, but see Tan et al., 2012, Wang et al., 2019). But even in organisms with a segregated germline, gametes do not come from a lineage that was cryogenically frozen in an immaculate state. The Lansing effect refers to a pattern where gametes of old parents yield offspring with lower lifetime fitness than offspring of younger parents (or singular 'parent' in case of asexuality). Lansing found this effect in a series of experiments on asexual bdelloid rotifers, where lineages experimentally forced to produce the next generation from old mothers senesced to such a degree that the entire 'geriaclone' eventually perished (Bell, 1988; Lansing, 1947, 1954, pp. 93–96). While the repeatability of the finding in this particular lineage has been questioned (see Bell, 1988, p. 94), the Lansing effect appears real in many sexual metazoans (Bouwhuis, Verhulst, Bauch, & Vedder, 2018; Eisenberg & Kuzawa, 2018; Priest et al., 2002; Schroeder, Nakagawa, Rees, Mannarelli, & Burke, 2015). The exact mechanisms at play are still mostly unknown, but possible candidates include higher mutation load in older parent germlines (Crow, 2006; Gao et al., 2019), the inheritance of short telomeres (Bouwhuis et al., 2018; Heidinger et al., 2016), epigenetic inheritance (Lamb, 1994) or other parental effects (Lind et al., 2019; Ronget et al., 2018).

One way to view these results is that the rejuvenating effect of sex, that is its ability to produce 'source' individuals, itself may come with a 'best before' date. This leads us to our last topic: if the production of 'source' individuals is not only unreliable but also challenging with respect to the parent's own ageing, what does life-history theory predict regarding creating new organisms from the parent's own cells: in short, why do zygotes exist?

6 | WHY DOES SEX (SO OFTEN) INVOLVE THE PRODUCTION OF ZYGOTES?

Grosberg and Strathmann (1998) asked why multicellular organisms still include a unicellular stage in their life cycle. Highly intriguingly, this is true for all forms of multicellular life only if we stretch the definition of a life cycle to the end of a long period of vegetative growth, budding, etc., that can all occur before a lineage forms gametes (as an extreme example, the ability to produce sperm has become a rather pointless trait for an invasive sea star that, via asexual fissiparity, forms 100% male populations in the Mediterranean, Karako, Achituv, Perl-Treves, & Katcoff, 2002). Also, note that sex is not necessarily temporally aligned with the single-cell stage: in mushroom-forming basidiomycete fungi, fertilization is followed by further vegetative growth, and the production of haploid is an entirely different part of the life cycle (Nieuwenhuis & Aanen, 2018).

Grosberg & Strathmann's short paper mentions two categories of hypotheses. The first one states that deleterious mutations are more efficiently purged when there is much variance between offspring rather than each being a large (and thus similar to each other) sample of all the genetic material of parental cell lineages. The second hypothesis is based on within-offspring variability being directly detrimental to offspring performance, due to within-organism

conflict when different cell lineages compete. Interestingly, new models contain aspects of both elements: Radzvilavicius et al., 2016 argue that the degree to which germlines are distinct is related to the need to 'farm' mitochondria in a way that guarantees that at least some gametes will perform well.

Variance arguments are necessary to understand why sex (via zygote production) might rejuvenate. Given the inherent unpredictability of meiosis, recombination and the uncertainties of who one might mate with, there is no guarantee of any one offspring being a potential 'source' (in the sense of being a fully rejuvenated 'A-age zero' individual). While this is unfortunate, what is the alternative? If these variances were minimized (e.g. mitosis instead of meiosis), and especially if young were produced from a large bulk (many cells) of the parent's body, one now has a near-deterministic guarantee that the offspring will inherit some of the damage that the parent had, to which the offspring adds its own. A single-cell stage, therefore, appears to bring about two interrelated benefits. Firstly, purely economically, it allows many more young to be produced (though this economic benefit is no longer reaped in organisms that allocate substantial energy in parental care and can, at the extreme, only raise one young at a time; however, here, too, ill-formed zygotes are often aborted early, e.g. in humans). Secondly, the large number of young improves the odds in a trial-and-error gamble where the hope is that at least some young might be classified as having belonged to a 'source' by more 'rejuvenated' than any of their competitors, have the highest reproductive value, and the least worry (at a young demographic age, at least) from deleterious mutations.

It is not surprising that most ideas discussed in this section remain without a formal model. One aspect is, however, modelled by Pichugin, Peña, Rainey, & Traulsen, 2017, who ignore genetics (and ageing) but consider varying ways to 'split' a parent (cell group) into cells that form the next generation, when fecundity and/or survival of cells depends on the size of the group of cells forming the parent. In their model, economic considerations alone are sufficient to produce a life cycle featuring a unicellular bottleneck. Under certain conditions, this evolves as the best way to guarantee that the 'parent' group remains as large as possible to reap maximum fecundity and/or survival advantages of group living. Since the parental group does not age in their model, it would be interesting to extend this work to multidimensional aspects of damage accumulation, especially since some (but not all) of the above benefits also apply, for example to asexual sporulation (Zhang et al., 2015).

7 | CONCLUSIONS

We would like to end by highlighting an old tongue-in-cheek paper asking why offspring are smaller than their parents (Ellstrand, 1983). The paper lists many sensible sounding hypotheses before proceeding to its final paragraph, where the author suggests a fruitful future research direction of why an offspring is always *younger* than its parent. This parody makes it clear that we should not look for fancy explanations for properties of life that just cannot be organized any other way. Even so, we would like to invite the reader to rethink a little: just like in the offspring size question there are more nuanced ways to ask the question (why is a kiwi's egg so large and those of salmon so tiny?), one can legitimately ask whether lineages and individuals who compose them, especially asexual ones, 'age' faster than those that are, in some sense, rejuvenated by sex (Ho & Agrawal, 2017).

Space issues forced us to leave many topics aside. Sex may involve the intriguing polymorphism of two sexes, creating the potential for sex-specific selection for fast or slow life histories (and the associated senescence patterns, Bonduriansky, Maklakov, Zajitschek, & Brooks, 2008; Brooks & Garratt, 2017; Maklakov & Lummaa, 2013; Tidière et al., 2015). Also, our understanding of the evolution of multicellularity itself has recently advanced via combinations of experimental evolution and modelling (Ratcliff et al., 2013; Staps, Gestel, & Tarnita, 2019; Zhang et al., 2015); time will tell if the various aspects of organismic age can be added to such approaches.

ACKNOWLEDGEMENTS

We wish to thank Jean-Michel Gaillard and Jean-François Lemaitre for inviting us to submit a manuscript to this special issue. We also thank Charles Fox, two anonymous reviewers and Thomas Aubier for insightful comments and discussions.

AUTHORS' CONTRIBUTIONS

M.G. and H.K. together decided upon the content of this review and both contributed to writing it.

DATA AVAILABILITY STATEMENT

This article does not include any data.

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SUPPORTING INFORMATION

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How to cite this article: Galipaud M, Kokko H. Sex, senescence, sources and sinks. *Funct Ecol*. 2020;34:55–64. https://doi.org/10.1111/1365-2435.13462