Review

Intergenerational Transfer of Ageing: Parental Age and Offspring Lifespan

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The extent to which the age of parents at reproduction can affect offspring lifespan and other fitness-related traits is important in our understanding of the selective forces shaping life history evolution. In this article, the widely reported negative effects of parental age on offspring lifespan (the 'Lansing effect') is examined. Outlined herein are the potential routes whereby a Lansing effect can occur, whether effects might accumulate across multiple generations, and how the Lansing effect should be viewed as part of a broader framework, considering how parental age affects offspring fitness. The robustness of the evidence for a Lansing effect produced so far, potential confounding variables, and how the underlying mechanisms might best be unravelled through carefully designed experimental studies are discussed.

Highlights

The quality and viability of offspring depends on the age at which the parents breed.

A reduced lifespan in offspring from older parents is known as the Lansing effect.

It is found across many taxa but does not always occur.

The many mechanisms that can produce this effect is highlighted.

The Lansing effect does not necessarily imply a reduction in offspring fitness.

The Lansing Effect and Why It Matters

Understanding how and why ageing occurs, what factors determine its rate, and why this varies among species and individuals, is a topic of intense research by scientists from diverse disciplines [1–3]. However, almost all of the focus has been on the time-dependent, somatic deterioration of individuals during their lifetimes. Studies of the relationship between age and reproductive performance have primarily been concerned with effects on parent rather than offspring fitness [4]. However, the age of parents can potentially have a multitude of effects on offspring phenotypes, both positive and negative, and not necessarily involving parental ageing [4]. Here we focus on the widely reported negative effect of the age of parents at a breeding event (hereafter termed 'parental age') on offspring lifespan, which has become known as the Lansing effect. Knowing whether, and under what conditions, parental age can have a negative effect on offspring lifespan has profound implications for our understanding of the evolution of animal life histories, and for more applied fields such as reproductive medicine and conservation biology.

In many iteroparous species, the relationship between the age of parents and their reproductive output is a bell-shaped curve (Box 1), but it has been known for a surprisingly long time that parental age can affect the lifespan of offspring as well as their number. In 1918 Alexander Graham Bell (inventor of the telephone) conducted pioneering demographic analyses based on the extensive family tree of an early American settler. He found that offspring of the youngest parents lived a decade longer than those of the oldest [5]. Subsequent experimental studies by Jennings and Lynch working with parthenogenic rotifers, found a similar negative relationship between parental age at the time of reproduction and offspring lifespan [6]. In 1947, Albert Lansing further showed that rotifer selection lines in which the parents were old breeders had shorter-lived offspring and died out much sooner than lines in which the parents were consistently young breeders [7]. Based on these results, Lansing envisioned a 'transmissible and cumulative ageing factor', more of which was transmitted to offspring of older parents and which could thereby accumulate across the generations.

Early studies that followed Lansing were primarily concerned with the potential cumulative effect over successive generations, with several failing to reproduce it [8,9]. More recently, lifespan has

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Box 1. The Changing Relationship between Parental Age and Reproductive Output

A bell-shaped relationship (Figure I) between parental age and breeding performance has been found in many iteroparous species, both in the laboratory and in studies of wild populations [4,63,64]. At a population level, this pattern can be generated by the selective disappearance of low-quality individuals at a young age, and a longer lifespan of individuals with lower investment per breeding attempt; a relationship between the age of first breeding and reproductive performance can also contribute, for example, if poor performing individuals tend to breed at a younger age. However, similar bell shaped patterns are also found within individual animals, as a result of gaining resources, experience and/or status in their early years, and then in later life having impaired breeding performance due to the effects of ageing [4]. While the same bell-shaped pattern can be evident in a range of reproductive traits, such as egg size or egg quality [65], most ecological-based studies have focussed on demographic parameters, most usually offspring number (taken as a measure of the reproductive success of the parents), with less attention being paid to offspring quality or fate [13]. Studies aiming to investigate whether a Lansing effect occurs need to be designed carefully to take account of the likely initial improvement in offspring quality with parental age; the comparison should not involve young breeders (Figure I, part A of curve), but instead should be a comparison between middle-aged and old breeders B versus C. Comparison at A and C, or A and B could erroneously suggest no effect, or a positive effect.
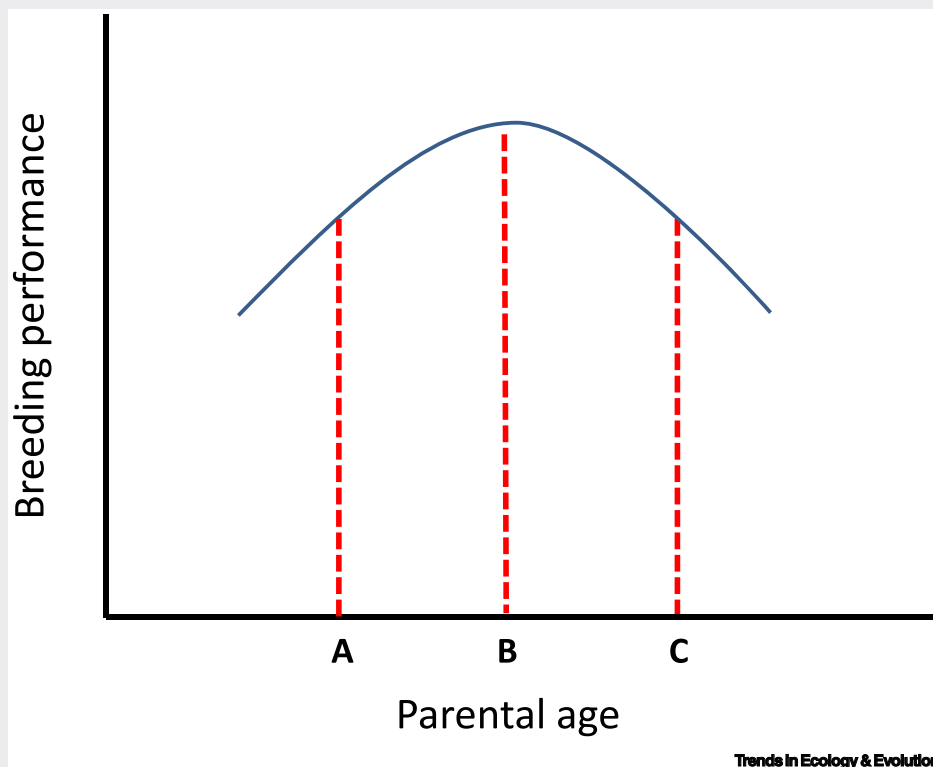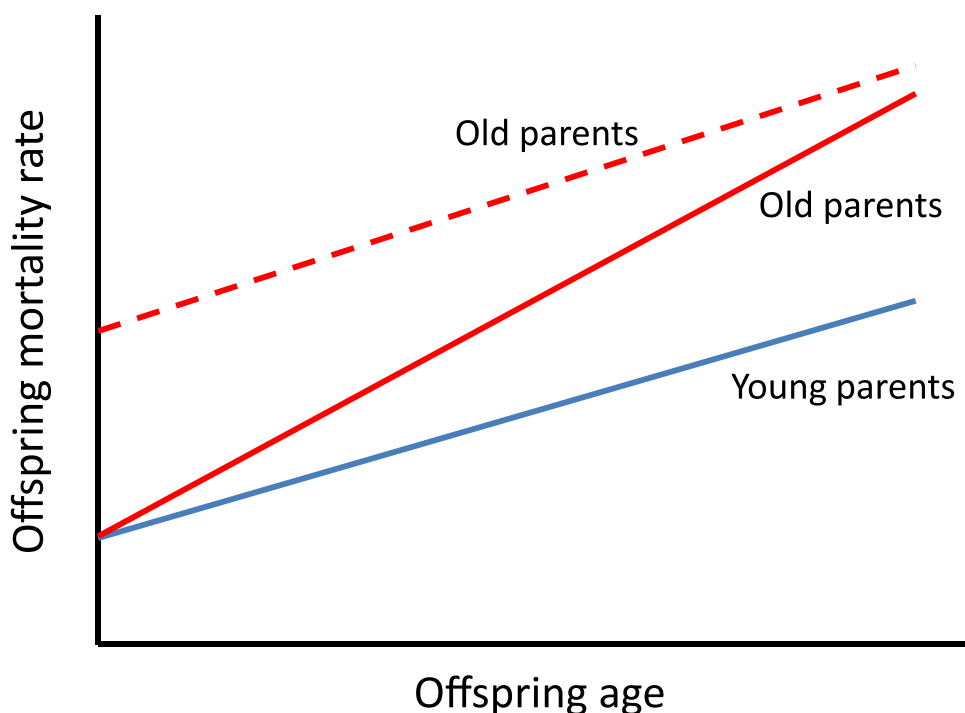

Figure I. The Relationship between a Parent's Age at the Time of Breeding and Its Reproductive Performance (e.g., the Number or Quality of Offspring It Produces).

mostly only been studied in the first (F1) generation of offspring (for exceptions see [10–12]), and the phenomenon of older parents giving rise to shorter-lived offspring has become known as the Lansing effect irrespective of any intergenerational accumulation. However, the existence of the Lansing effect has largely been ignored by theories on the evolution of ageing and life histories, which usually assume that the fitness of offspring is independent of parental age at conception [13]. There are exceptions (e.g., see [14–16,77]), but even a model that explicitly examines the link between parental age and offspring life expectancy [17] excludes the possibility that gametes can age, and so underplays the potential for the Lansing effect. The need for gametic ageing to be included has recently emphasised [18,19] since it is important that we understand the selection

pressures that shape optimal reproductive scheduling and thereby the pattern of parental invest-ment that organisms show across their lifetimes, which would be strongly influenced by intergen-erational transfer of ageing if this impacts offspring fitness [16]. It is also important to distinguish between the two quite distinct processes that can separately or together generate a shorter lifespan, namely an increased 'frailty', manifested as an increased mortality rate at any time of life, and an increased rate of ageing, which only becomes apparent later in life (Figure 1). We also need to understand the mechanisms underlying any Lansing effect, since this will help ex-plain whether the Lansing effect arises as a result of constraints (e.g., somatic or germline dete-rioration) or adaptations (e.g., changes in reproductive scheduling).

Documented Effects of Parental Age at Conception on Offspring Lifespan

The Lansing effect is defined here as an empirically demonstrated negative relationship between parental age and offspring lifespan, irrespective of whether this is due to an increase in offspring ageing rate or frailty. Discussed later, is the need to consider the Lansing effect as part of a broader framework encompassing parental age effects on offspring fitness. Table 1 gives exam-ples of studies that have found offspring early survival or average lifespan to be negatively related to the age of the parent(s); further examples are given in [10,12]. The table serves to illustrate the taxonomic breadth of the organisms in which Lansing effects have been found, that they occur in both field and laboratory studies, and that few have determined whether the shorter lifespan is

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Figure 1. The Relationship between the Mortality Rate of Offspring and the Age of Their Parents at Conception. The blue line shows offspring of young parents and the red lines offspring of old parents. Mortality rates are shown to increase linearly with offspring age, omitting the high immediate postnatal mortality for clarity. Offspring of old parents might have the same initial mortality rate as those of young parents, but the rate at which they die increases faster, indicating a faster rate of actuarial senescence (unbroken red line). Alternatively, offspring of older parents may show overall increased frailty compared with those of young parents, and survival is poorer at all ages (broken red line). In both cases, the average lifespan of the offspring of the older parents will be shorter. Note that the two processes are not mutually exclusive: it is possible that greater frailty can also increase the rate of ageing.

Table 1. Examples of the Lansing Effect (Where Offspring of Older Parents Have Shorter Lifespans), Chosen to Demonstrate the Breadth of Affected Taxa and Range of Approaches

Taxon and reference	Field or lab	Experimental or natural pairings[a]	Virgin parents?[b]	Paternal effect on lifespan[c]	Maternal effect on lifespan[c]	Nature of Lansing effect	Effects within individuals?[d]
Nematode *Heligmosomoides polygyrus* [69]	Lab	Natural	No	(Yes)	(Yes)	Reduced larval-adult survival in offspring of older worms (not known whether both parents contribute to effect)	No
Rotifer *Brachionus manjavacas* [70]	Lab	–	No		(Yes) - asexual	Higher initial mortality rate and lower median lifespan in offspring of older mothers, but slower rate of ageing	No
Water flea *Daphnia pulex* [10]	Lab	–	No		Yes	Shorter lifespan and faster rate of ageing in two of the three clones	No
Fruit fly *Drosophila melanogaster* [23]	Lab	Experimental	Yes	No	Yes	Shorter lifespan of daughters of older mothers in most strains; due to an increase in frailty rather than rate of ageing	No
Butterfly *Pieris brassicae* [71]	Lab	Experimental	No		Yes	Shorter lifespan in offspring of older mothers	No
Rice weevil *Sitophilus oryzae* [72]	Lab	Experimental	No		Yes	Shorter lifespan in offspring of older mothers	No
Common tern *Sterna hirundo* [57]	Field	Natural	No	Yes	No	Shorter lifespan in sons of older fathers; no effects in other combinations	No
Wandering albatross *Diomedea exulans* [58]	Field	Natural	No	Yes	No	Lower juvenile survival in offspring of older fathers; no effects of mother's age	Yes
Houbara bustard *Chlamydotis undulata* [55]	Lab then field	Experimental	No	Yes		Lower juvenile survival in daughters of older fathers (no effect in sons)	No
Mouse *Mus musculus* [54]	Lab	Experimental	Yes		Yes	Shorter lifespan in offspring of older females	No
Red squirrel *Tamiasciurus hudsonicus* [73]	Field	Natural	No		Yes	Lower juvenile post-weaning survival (to 1 year) of offspring of older females	No
Asian elephant *Elephas maximus* [74]	Field	Natural	No		Yes	Lower survival in offspring of older mothers and older grandmothers	No
Human *Homo sapiens* [75]	Field	Natural	No		Yes	Lower survival to adulthood in offspring of older mothers	Yes

[a]Whether parents were assigned mates by the researcher or formed their own natural pairings.
[b]Whether both young and old parents were virgins at time of producing the offspring whose survival was measured.
[c]Brackets indicate either that it is not known whether both sexes contribute to the effect, or that the species is asexual.
[d]Whether the analyses showed a Lansing effect within individual animals, (e.g., by using within-subject centring) [76]. Note that the majority of studies adopt a cross-sectional approach so cannot test for changes with age within individuals.

due to increased offspring frailty, more rapid ageing or both. It is important to emphasise that not all reported effects of parental age on offspring viability or lifespan are negative, and reported effects are sometimes inconsistent. For example, different studies of *Drosophila* have found the effect of increasing parental age on offspring survival or lifespan to be negative [20–23], positive [24], neutral [25], or strain-specific [26]. Some of this inconsistency could be due to aspects of study design: Table 1 emphasises that most studies to date have used a cross-sectional study design, which can lead to problems in data interpretation (Box 1). Older parents may produce

longer-lived offspring if they have increased access to resources and/or greater experience, or engage in increased reproductive effort if their life expectancy is short ('terminal investment') [27,28]. It is also the case that conspecifics will age at different rates, and that this will affect the pattern of intergenerational transfer.

Mechanisms That Could Underlie a Lansing Effect

Nonexclusive routes whereby the age of parents could affect the lifespan of their offspring are described herein. It is important to remember that the Lansing effect can arise via routes that have nothing to do with ageing of the parents or their offspring, as described in Box 2. It should be also noted that most of these potential mechanisms relate more to the biological rather than the chronological age of the parents, and may be influenced by, for instance, prior reproductive effort or environmental harshness, both of which influence rates of ageing [4,29]. However, in practice it tends to be the chronological age that is measured. A distinction is drawn here between direct effects of the age-related deterioration of the parents or their germ cells on offspring, and indirect effects of parental age such as changes in investment patterns by mates, or compensatory

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Box 2. Mechanisms That Can Generate Apparent Lansing Effects Even When There Is No Age-Related Change in Parents

Processes at both the tissue and population level can give the spurious impression of reproductive senescence within individuals reducing the viability of offspring. Firstly, consider the way in which a female's oocytes are used across her breeding lifespan. In species such as birds and mammals the full complement of a female's germ cells appears to be present at sexual maturity. If these vary in quality, then it would be adaptive to use the best oocytes first, and only use poorer oocytes later in life since the chances of living to breed in old age may be low [66]. This effect can arise if the last oocytes to be used have been through more cell divisions and have more genetic damage or reduced telomere length, as would appear to be the case, for example, in long-lived mammals [67]. Under this scenario, offspring quality would decline with maternal age even if there was no change over time in the state of each oocyte (or of the mother) (Figure IA). An alternative process operates in population level studies, where analyses can show a lower mean lifespan in the offspring produced by older parents, without any decline in the lifespan of offspring produced by individual parents. This can arise if there is a link between the level of investment in offspring per breeding event, and the probability of parents surviving into old age, as has been found for example, in wild red-billed choughs *Pyrrhocorax pyrrhocorax* [68]. Thus individual 1 in Figure IB invests heavily in each offspring but over a short reproductive lifespan, while individual 2 invests less per offspring but has a longer reproductive lifespan (and so produces more offspring) as a consequence. These two scenarios represent extremes of a continuum of alternative reproductive strategies that may have similar lifetime fitness. However, taking the population as a whole, there is then a decrease in the lifespan of offspring as mean parental age increases, because the only offspring being produced by older parents are those that receive less investment, and so are less viable [68].
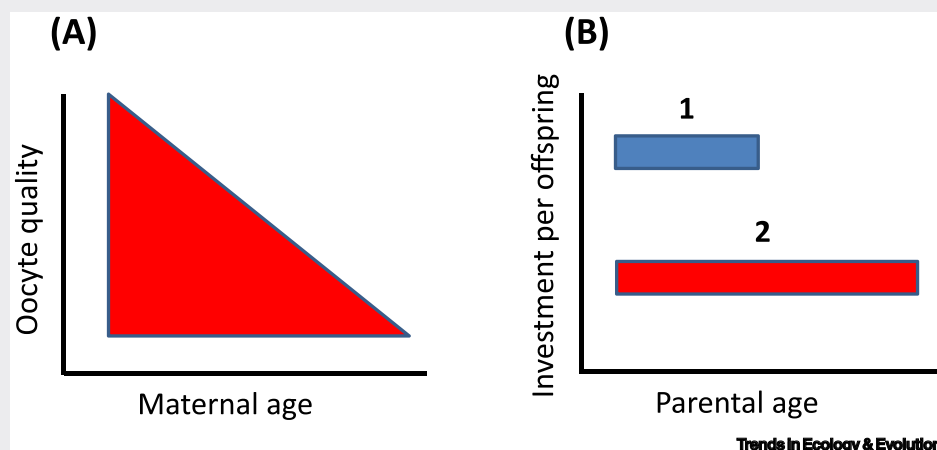

Figure I. Two Mechanisms That Can Generate an Apparent Lansing Effect. (A) The potential relationship between a mother's age and the quality of her remaining oocytes. (B) Alternative reproductive strategies that can generate a Lansing effect at a population level.

responses by the offspring themselves. Unless otherwise stated, the mechanisms discussed below could operate through either parent.

Direct Effects

(i) Decline in gamete quality: While it was once thought that gametes were 'ageless' it is now known that both eggs and sperm can show signs of deterioration with parental age. Gametes from old parents (i.e., those on the downward slope illustrated in Box 1) are more likely to have DNA mutations, impaired mitochondrial function and shorter telomeres, all of which could increase offspring frailty and/or hasten the rate of ageing of the offspring [30,31]. In theory, deleterious germline mutations could increase individual mutation rates, resulting in a positive feedback loop whereby poor quality genotypes will produce descendants of increasingly poor genetic and phenotypic quality [32,33]. There is also mounting evidence that epigenetic signatures such as DNA methylation patterns may be transmitted to the gametes, providing a mechanism of transfer of somatic ageing to offspring, and so the potential for intergenerational transfer and accumulation by this route [34–36].

(ii) Decline in the quality of parental care: As the parents begin to show age-related deterioration, their capacity to perform different components of reproduction may decline; older parents might have less ability to acquire resources, leading to reduced provisioning of eggs [37] and of offspring through either lactation or parental feeding [4], resulting in offspring that are of lower quality.

(iii) Greater cost of inbreeding depression in older breeders: The mutation accumulation theory of ageing predicts that the deleterious effects of inbreeding on fitness will increase with the age of the breeders [38,39]. This can have a negative effect on offspring viability, for example, because of lower levels of parental investment or care [40].

Indirect Effects

(i) Stored gamete deterioration: In some species, females mate once early in reproductive life and store sperm, which is then used over the remaining breeding lifespan, which in the case of some social insects can last for decades [41]. Any decline in offspring lifespan with maternal age, as has been documented in social insects [42], could be due to the deterioration of the stored sperm rather than any age-related change in the state of the mother or her gametes.

(ii) Differential allocation by the mate: If old animals are considered less attractive or are less competitive, then their mates may invest less in the offspring, following the prediction of the differential allocation hypothesis [43]. This reduced investment, for example, in provisioning or parental care, would lead to poorer quality offspring. Here the negative effect of parental age is indirect, being due to a strategic reduction in investment by the mate, who themselves could be a young individual. The supposed lower attractiveness of older mates could evolve due to their producing less viable offspring or contributing less to parental care. A counter argument of course is that older mates could be preferred, since by surviving into old age, they are demonstrating high genetic quality [44].

(iii) Greater likelihood of inbreeding in older breeders: Older adults may be more likely to mate with relatives, producing offspring suffering from inbreeding depression [45]. This can arise if one sex is philopatric; the dispersing sex (which is unlikely to move again after the initial natal dispersal) may then end up mating with its own (nondispersing) offspring. It can also occur if older adults are less attractive and so have a reduced choice of mates (see Indirect effect ii above).

(iv) Offspring response: A Lansing effect could be generated through the offspring compensating for age-related declines in parental provisioning [10]: if older parents either invest less in eggs or

provide less food to their offspring, then the offspring will be smaller at independence. This can trigger a compensatory accelerated growth response during juvenile life , which confers short-term benefits but comes at a cost of faster ageing [46,47]. Here both parents and offspring are contributing to a shortened offspring lifespan.

Cumulative Effects

There are few studies of the trans-generation cumulative effect of parental age envisioned by Lansing. It is necessary to measure traits in several successive generations to detect whether effects are becoming cumulatively magnified. However, it is clear that there can be both direct and indirect effects of the previous generation on maternal and paternal performance. For example, while multiple negative effects of maternal age on offspring traits were shown in *Drosophila melanogaster*, there was a complex interaction between maternal age and the age of the grandmother at the time of conception of the mother, with effects also being dependent on the strain of *Drosophila* used [11]. There is some evidence that detrimental maternal age effect on offspring juvenile viability can accumulate over two generations in *Drosophila serrata* [21]. Recently, Wylde *et al.* [12] showed in the neriid fly *Telostylinus angusticollis* that grandparental and parental age effects on lifespan interact across patrilines and matrilines. Offspring lifespan declined with parental age, and offspring of older parents lived shorter still if the grandparents were also old, although the relationship between grandparental and parental age effects was nonlinear and rather complex. The strength of selection against accumulation of these effects across multiple generations is likely to depend on the proportion of lifetime reproduction occurring at different parental ages, and might lead to selection on optimal breeding schedules.

Beyond Lifespan: The Lansing Effect and Offspring Fitness

It seems sensible to retain the term 'Lansing effect' as it is generally used at present - the empirical negative relationship between offspring lifespan and parental age at breeding. However, there are two main reasons why we should be interested in placing the Lansing effect in a more general framework of age-specific life-histories and assessing the effects of parental age on all components of offspring fitness rather than just lifespan. First, a reduction in offspring lifespan need not have negative fitness consequences if their reproductive scheduling is altered; this seems plausible given that the direct mechanisms listed above may affect offspring life-history traits in addition to lifespan, such as development, growth, and age-specific reproductive investment. Second, because the Lansing effect may result from adaptive phenotypic plasticity by mates or offspring (Indirect effects ii, iii, and iv), full knowledge of the fitness consequences of parental age may be a prerequisite for understanding why offspring of old parents are short-lived. The results of a recent study in water fleas (*Daphnia pulex*) illustrate the need for a broader approach. Older mothers produced larger offspring (i.e., increased their per capita investment into offspring with advancing age) and these offspring had faster growth rates and increased early-life fecundity [10]. However, these offspring also reached their reproductive peak earlier and senesced faster, so lived for a shorter time and produced fewer eggs over their lifetime. This study suggests that the short lifespan of 'old parent' offspring is a result of a phenotypically plastic switch to a 'live fast, die young' life-history strategy. The key issue in an evolutionary context is whether this offspring response is adaptive.

Further, given that the life history of many invertebrates is characterised by large fluctuations in population density, it is possible that increased investment in rapid development, and/or early reproduction at the cost of reduced lifespan and lifetime reproduction, is adaptive in an expanding population, or in one experiencing a high adult mortality rate [10]. This could be investigated directly by analysing rate-sensitive fitness, which considers the timing of reproduction and the

fact that offspring produced early in life are particularly important for individual fitness in a growing population. Another possibility is that, at the point when offspring are competing over resources, earlier-produced offspring will have reached an older age than later progeny and may thus have an age-related competitive advantage. In line with this argument, soil mites produce larger offspring late in life in order to increase their competitiveness in access to depleting resources [48]. The role of offspring life-history decisions in contributing to the Lansing effect becomes even more apparent if we consider that the re-analysis of Lansing's own data suggested that his 'Lansing effect' could result from rotifers from late-life lines reproducing earlier and at a higher rate [49].

Accelerated life histories in the offspring of older parents may be a general phenomenon that spans different taxonomic groups. For example, in great tits (*Parus major*), offspring of older mothers also reproduce at higher rate in early life but then pay the cost of accelerated reproductive ageing [50]. Similarly, daughters of older female yellow-bellied Marmots (*Marmota flaviventer*) had higher reproductive rates throughout their lives, but this increased rate of reproduction came at a cost of reduced lifespan when conditions were harsh [51].

Designing Experiments to Explore the Lansing Effect

Designing studies to examine intergenerational effects of ageing is not straightforward. Controlled experimental work, usually conducted in the laboratory, is needed in order to test hypotheses relating to the specific mechanism under study. Field studies are also important in order to examine the occurrence of the Lansing effect in natural populations, where lifespan is likely to be shorter and more variable than in benign laboratory conditions. Of paramount importance is careful selection of the parental age groups to be compared, as shown in Box 1. For this, information on the onset of ageing in the study animals is needed; it would be useful to know the biological state rather than only the chronological age of the parents. Experiments on ectothermic species need to consider the temperature at which they are kept since this can influence age of maturity and rate of ageing [52,53]. Cross-sectional studies potentially suffer from the problem of differential survival of parent phenotypes, such that only higher-quality individuals, or those adopting low investment reproductive strategies (Box 2), are still breeding at older ages. Within-individual longitudinal studies can solve this problem, but give rise to another: if the same individuals breed at young and old ages, we then need to take account of possible effects of reproductive history, since the young group may have bred only once, and the old group at least twice and the increased parental investment could have an effect on offspring irrespective of parental age. Experimentally assigning mates and comparing first-time breeders of different ages breeding with a mate of standardised age can be a good approach to get around this issue (e.g., [23,54]), while using *in vitro* fertilisation [55] can avoid complications arising from differential allocation. It is then also important to test in a separate cohort for effects of the number of breeding events on offspring viability/ageing rate by experimentally uncoupling this from age [56]. Further, examining whether Lansing effects are evident in semelparous species would help identify effects attributable to prior reproductive effort. Studies should examine whether older parents produce offspring that are of overall poor quality (offspring 'frailty') or whether the effect of parental age does not manifest itself until the offspring are themselves old, and is evident as an increase in their own rate of ageing (Figure 1).

If possible, the effect of maternal and paternal age should be examined separately, as should the effect on sons and daughters, since there is increasing evidence of sex-specific effects in both parents and offspring [23,57,58], (Table 1). If cumulative effects are to be investigated, it is important to check for the effect of the selection regime on other phenotypic traits such as size and age-specific reproduction. The extent to which offspring viability declines with parent age is itself likely

to be a trait subject to selection. Kern *et al.* [22] showed in a long-term selection experiment that selecting in *D. melanogaster* for old mothers led to the evolution of enhanced viability in their offspring, so leading to a mitigation (but not total elimination) of the Lansing effect. It is difficult to control for such effects, but important to measure them if possible.

The effect of parental age on offspring life-histories is likely to vary with the ecology of the species in question and on the environmental conditions experienced by the cohort. Studies of the Lansing effect ideally should include data on offspring fitness and grand-offspring quality rather than focussing only on offspring lifespan.

Concluding Remarks

There is clear evidence across a range of taxa that ageing parents may produce offspring that have reduced lifespan, but many issues remain to be explored (see Outstanding Questions). For instance, there are several potential mechanisms that can generate this Lansing effect, but very few studies to date have explored which of these are important. More work is also needed to identify the conditions under which the Lansing effect does and does not occur, and theoretical studies to explore whether it is likely to be an adaption or a constraint. No universal 'ageing factor' that could accumulate across generations has been identified, though increases in *de novo* germline mutations with parental age or shorter telomere length in offspring of older parents could act in this way [31]. More studies that test for cumulative effects across multiple generations are needed. Furthermore, sex-specific effects need to be explored: since males often have more germline cell divisions than females, it is generally expected that males will accumulate more germline mutations with age [59], leading to the prediction that the Lansing effect can be more pronounced in patrilines [12]. However, sex-specific germline cell division can have different patterns at different ages [60] so that knowledge of the study system is essential. Certain systems may be particularly valuable for such studies. Co-operative breeders and social insects for example, have many life history traits that make them especially interesting in the context of trans-generational ageing effects (e.g., contrasting ages of breeders and helpers rearing the same offspring, and long-lived queens with prolonged sperm storage, producing different types of offspring over their lifetime) but it is not apparent that any such studies have yet taken place. Ageing parents could potentially compensate for a reduced ability to invest in offspring by altering offspring sex ratios in favour of the less expensive sex. Reported changes in offspring sex ratio with parent age are more likely to relate to differences in embryo mortality or post-natal survival, so the adaptive significance is unclear [61,62]. Gametic deterioration with parental age is one route whereby this could occur. An important but neglected route for intergenerational accumulation could be due to changes in behaviour, but this has not been studied in this context. For example, if old parents show impaired parental care, this behavioural deficit may be repeated if their offspring learn parenting skills from their parents.

The Lansing effect is but one component of transgenerational effects of ageing, and much remains to be studied in this area. Understanding the extent to which age-related deterioration of the parents is a constraint that limits their reproductive lifespan, or can be overcome by strategic adjustments in investment patterns, is a field in which proximate and ultimate approaches, modelling, experiments, and field studies all have an important contribution to make.

Outstanding Questions

What are the most common mechanisms underlying the Lansing effect, whereby offspring of older parents tend to have shorter lives?

Is the shorter lifespan due to greater frailty or an earlier onset of senescence?

How does a shortened lifespan in offspring of older parents influence offspring fitness and to what extent is it an adaptation rather than a constraint?

Does the Lansing effect lead to altered offspring sex ratios?

Does the effect of parental age on offspring fitness last beyond the F1 generation, and does the effect accumulate or diminish?

Is prior reproductive effort more or less important than parental senescence?

Does age at breeding influence offspring lifespan in semelparous species?

References

1. Wolf, N.S., ed (2010) *The Comparative Biology of Aging*, Springer
2. López-Otín, C. *et al.* (2013) The hallmarks of aging. *Cell* 153, 1194–1217
3. Shefferson, R.P. *et al.,* eds (2017) *The Evolution of Senescence in the Tree of Life*, Cambridge University Press
4. Lemaître, J.F. and Gaillard, J.M. (2017) Reproductive senescence: new perspectives in the wild. *Biol. Rev.* 92, 2182–2199
5. Bell, A.G. (1918) *The duration of life and conditions associated with longevity - a study of the Hyde geneology*, Geneological Record Office
6. Jennings, H.S. and Lynch, R.S. (1928) Age, mortality, fertility, and individual diversities in the rotifer *Proales sordida* Gosse I Effect of age of the parent on characteristics of the offspring. *J. Exp. Zool.* 50, 345–407
7. Lansing, A.I. (1947) A transmissible, cumulative, and reversible factor in aging. *J. Gerontol.* 2, 228–239
8. Lints, F.A. and Hoste, C. (1974) Lansing effect revisited 1. Lifespan. *Exp. Gerontol.* 9, 51–69
9. Lints, F.A. and Hoste, C. (1977) Lansing effect revisited 2. Cumulative and spontaneously reversible parental age effects on fecundity in *Drosophila melanogaster*. *Evolution* 31, 387–404
10. Plaistow, S.J. *et al.* (2015) Offspring provisioning explains clone-specific maternal age effects on life history and life span in the water flea, *Daphnia pulex*. *Am. Nat.* 186, 376–389
11. Qazi, M.C.B. *et al.* (2017) Transgenerational effects of maternal and grandmaternal age on offspring viability and performance in *Drosophila melanogaster*. *J. Insect Physiol.* 100, 43–52
12. Wylde, Z. *et al.* (2019) Parental breeding age effects on descendants' longevity interact over 2 generations in matrilines and patrilines. *PLoS Biol.* 17, e3000556
13. Barks, P.M. and Laird, R.A. (2015) Senescence in duckweed: age-related declines in survival, reproduction and offspring quality. *Funct. Ecol.* 29, 540–548
14. McNamara, J.M. *et al.* (2009) Deterioration, death and the evolution of reproductive restraint in late life. *Proc. R. Soc. B* 276, 4061–4066
15. Moorad, J.A. and Nussey, D.H. (2016) Evolution of maternal effect senescence. *Proc. Natl. Acad. Sci. U. S. A.* 113, 362–367
16. Barks, P.M. and Laird, R.A. (2020) Parental age effects and the evolution of senescence. *Am. Nat.* 195, 886–898
17. van den Heuvel, J. *et al.* (2016) Disposable soma theory and the evolution of maternal effects on ageing. *PLoS ONE* 11, e0145544
18. Galipaud, M. and Kokko, H. (2020) Sex, senescence, sources and sinks. *Funct. Ecol.* 34, 55–64
19. Galipaud, M. and Kokko, H. (2020) Adaptation and plasticity in life-history theory: how to derive predictions. *Evol. Hum. Behav.* Published online June 19, 2020. https://doi.org/10.1016/j.evolhumbehav.2020.06.007
20. Price, D.K. and Hansen, T.F. (1998) How does offspring quality change with age in male *Drosophila melanogaster*? *Behav. Genet.* 28, 395–402
21. Hercus, M.J. and Hoffmann, A.A. (2000) Maternal and grandmaternal age influence offspring fitness in *Drosophila*. *Proc. R. Soc. B* 267, 2105–2110
22. Kern, S. *et al.* (2001) Decline in offspring viability as a manifestation of aging in *Drosophila melanogaster*. *Evolution* 55, 1822–1831
23. Priest, N.K. *et al.* (2002) The role of parental age effects on the evolution of aging. *Evolution* 56, 927–935
24. Krishna, M.S. *et al.* (2012) Offspring of older males are superior in *Drosophila bipectinata*. *Zool. Stud.* 51, 72–84
25. Yilmaz, M. *et al.* (2008) Maternal age effects on longevity in *Drosophila melanogaster* populations of different origin. *Biogerontology* 9, 163–168
26. Lee, J.H. *et al.* (2019) Strain-specific effects of parental age on offspring in Drosophila melanogaster. *Entomol. Res.* 49, 187–202
27. Poizat, G. *et al.* (1999) Empirical evidence of a trade-off between reproductive effort and expectation of future reproduction in female three-spined sticklebacks. *Proc. R. Soc. Lond. B* 266, 1543–1548
28. Boonekamp, J.J. *et al.* (2020) Experimentally increased brood size accelerates actuarial senescence and increases subsequent reproductive effort in a wild bird population. *J. Anim. Ecol.* 89, 1395–1407
29. Nussey, D.H. *et al.* (2013) Senescence in natural populations of animals: widespread evidence and its implications for bio-gerontology. *Ageing Res. Rev.* 12, 214–225
30. Gao, Z.Y. *et al.* (2019) Overlooked roles of DNA damage and maternal age in generating human germline mutations. *Proc. Natl. Acad. Sci. U. S. A.* 116, 9491–9500
31. Monaghan, P. and Metcalfe, N.B. (2019) The deteriorating soma and the indispensable germline: gamete senescence and offspring fitness. *Proc. R. Soc. B* 286, 20192187
32. Sharp, N.P. and Agrawal, A.F. (2012) Evidence for elevated mutation rates in low-quality genotypes. *Proc. Natl. Acad. Sci. U. S. A.* 109, 6142–6146
33. Sharp, N.P. and Agrawal, A.F. (2016) Low genetic quality alters key dimensions of the mutational spectrum. *PLoS Biol.* 14, e1002419
34. Seisenberger, S. *et al.* (2012) The dynamics of genome-wide DNA methylation reprogramming in mouse primordial germ cells. *Mol. Cell* 48, 849–862
35. Radford, E.J. *et al.* (2014) *In utero* undernourishment perturbs the adult sperm methylome and intergenerational metabolism. *Science* 345, 1255903
36. Xie, K. *et al.* (2018) Epigenetic alterations in longevity regulators, reduced life span, and exacerbated aging-related pathology in old father offspring mice. *Proc. Natl. Acad. Sci. U. S. A.* 115, E2348–E2357
37. Muller, D. *et al.* (2017) Maternal age affects offspring nutrient dynamics. *J. Insect Physiol.* 101, 123–131
38. Charlesworth, B. and Hughes, K.A. (1996) Age-specific inbreeding depression and components of genetic variance in relation to the evolution of senescence. *Proc. Natl. Acad. Sci. U. S. A.* 93, 6140–6145
39. Snoke, M.S. and Promislow, D.E.L. (2003) Quantitative genetic tests of recent senescence theory: age-specific mortality and male fertility in *Drosophila melanogaster*. *Heredity* 91, 546–556
40. Fox, C.W. and Reed, D.H. (2010) Inbreeding depression increases with maternal age in a seed-feeding beetle. *Evol. Ecol. Res.* 12, 961–972
41. Heinze, J. and Schrempf, A. (2008) Aging and reproduction in social insects - A mini-review. *Gerontology* 54, 160–167
42. Al-Lawati, H. and Bienefeld, K. (2009) Maternal age effects on embryo mortality and juvenile development of offspring in the honey bee (Hymenoptera: Apidae). *Ann. Entomol. Soc. Am.* 102, 881–888
43. Burley, N. (1986) Sexual selection for aesthetic traits in species with biparental care. *Am. Nat.* 127, 415–445
44. Hansen, T.F. and Price, D.K. (1995) Good genes and old age: do old mates provide superior genes? *J. Evol. Biol.* 8, 759–778
45. Soulsbury, C.D. *et al.* (2012) Restrictive mate choice criteria cause age-specific inbreeding in female black grouse, *Tetrao tetrix*. *Anim. Behav.* 83, 1497–1503
46. Metcalfe, N.B. and Monaghan, P. (2003) Growth versus lifespan: perspectives from evolutionary ecology. *Exp. Gerontol.* 38, 935–940
47. Lee, W.S. *et al.* (2013) Experimental demonstration of the growth rate - lifespan trade-off. *Proc. R. Soc. Lond. B* 280, 20122370
48. Plaistow, S.J. *et al.* (2007) How to put all your eggs in one basket: empirical patterns of offspring provisioning throughout a mother's lifetime. *Am. Nat.* 170, 520–529
49. King, C.E. (1983) A re-examination of the Lansing effect. *Hydrobiologia* 104, 135–139
50. Bouwhuis, S. *et al.* (2010) Trans-generational effects on ageing in a wild bird population. *J. Evol. Biol.* 23, 636–642
51. Kroeger, S.B. *et al.* (2020) Older mothers produce more successful daughters. *Proc. Natl. Acad. Sci. U. S. A.* 117, 4809–4814
52. Angilletta, M.J. (2009) *Thermal adaptation: a theoretical and empirical synthesis*, Oxford University Press
53. Munch, S.B. and Salinas, S. (2009) Latitudinal variation in lifespan within species is explained by the metabolic theory of ecology. *Proc. Natl. Acad. Sci. U. S. A.* 106, 13860–13864
54. Tarín, J.J. *et al.* (2005) Delayed motherhood decreases life expectancy of mouse offspring. *Biol. Reprod.* 72, 1336–1343

55. Vuarin, P. *et al.* (2019) Post-copulatory sexual selection allows females to alleviate the fitness costs incurred when mating with senescing males. *Proc. R. Soc. B* 286, 20191675

56. Vega-Trejo, R. *et al.* (2019) The effects of male age, sperm age and mating history on ejaculate senescence. *Funct. Ecol.* 33, 1267–1279

57. Bouwhuis, S. *et al.* (2015) Sex-specific pathways of parental age effects on offspring lifetime reproductive success in a long-lived seabird. *Evolution* 69, 1760–1771

58. Fay, R. *et al.* (2016) Paternal but not maternal age influences early-life performance of offspring in a long-lived seabird. *Proc. R. Soc. B* 283, 20152318

59. Bachtrog, D. (2008) Evidence for male-driven evolution in *Drosophila*. *Mol. Biol. Evol.* 25, 617–619

60. Bauer, V.L. and Aquadro, C.F. (1997) Rates of DNA sequence evolution are not sex-biased in *Drosophila melanogaster* and *D. simulans*. *Mol. Biol. Evol.* 14, 1252–1257

61. Blank, J.L. and Nolan, V. (1983) Offspring sex-ratio in red-winged blackbirds is dependent on maternal age. *Proc. Natl. Acad. Sci. U. S. A.* 80, 6141–6145

62. Vedder, O. *et al.* (2016) Male-biased sex allocation in ageing parents; a longitudinal study in a long-lived seabird. *Biol. Lett.* 12, 20160260

63. Forslund, P. and Pärt, T. (1995) Age and reproduction in birds - hypotheses and tests. *Trends Ecol. Evol.* 10, 374–378

64. Monaghan, P. *et al.* (2008) The evolutionary ecology of senescence. *Funct. Ecol.* 22, 371–378

65. Benton, T.G. *et al.* (2008) Maternal effects mediated by maternal age: from life histories to population dynamics. *J. Anim. Ecol.* 77, 1038–1046

66. Duran, H.E. *et al.* (2011) The association of reproductive senescence with mitochondrial quantity, function, and DNA integrity in human oocytes at different stages of maturation. *Fertil. Steril.* 96, 384–388

67. Polani, P.E. and Crolla, J.A. (1991) A test of the production line hypothesis of mammalian oogenesis. *Hum. Genet.* 88, 64–70

68. Reid, J.M. *et al.* (2010) Parent age, lifespan and offspring survival: structured variation in life history in a wild population. *J. Anim. Ecol.* 79, 851–862

69. Lippens, C. *et al.* (2017) Aging parasites produce offspring with poor fitness prospects. *Biol. Lett.* 13, 20160888

70. Bock, M.J. *et al.* (2019) Maternal age alters offspring lifespan, fitness, and lifespan extension under caloric restriction. *Sci. Rep.* 9, 3138

71. Ducatez, S. *et al.* (2012) Complex interactions between paternal and maternal effects: parental experience and age at reproduction affect fecundity and offspring performance in a butterfly. *Evolution* 66, 3558–3569

72. Opit, G.P. and Throne, J.E. (2007) Influence of maternal age on the fitness of progeny in the rice weevil, *Sitophilus oryzae* (Coleoptera: *Curculionidae*). *Environ. Entomol.* 36, 83–89

73. Descamps, S. *et al.* (2008) Age-specific variation in survival, reproductive success and offspring quality in red squirrels: evidence of senescence. *Oikos* 117, 1406–1416

74. Reichert, S. *et al.* (2020) Maternal age at birth shapes offspring life-history trajectory across generations in long-lived Asian elephants. *J. Anim. Ecol.* 89, 996–1007

75. Gillespie, D.O.S. *et al.* (2013) The effect of maternal age and reproductive history on offspring survival and lifetime reproduction in preindustrial humans. *Evolution* 67, 1964–1974

76. van de Pol, M.V. and Wright, J. (2009) A simple method for distinguishing within- versus between-subject effects using mixed models. *Anim. Behav.* 77, 753–758

77. Hernández, C.M. *et al.* (2020) A demographic and evolutionary analysis of maternal effect senescence. *Proc. Nat. Acad. Sci. U. S. A.* 117, 16431–16437