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Parental age and offspring lifespan: the Lansing Effect and its underlying mechanisms

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*Abstract*

Biological ageing, or senescence, is the physiological deterioration of an organism. In addition to the negative effects on the senescing individual’s own survival and fecundity, there is evidence that the offspring of older parents may also suffer from reduced survival. This negative correlation of parental age and offspring lifespan is known as the Lansing effect. There is not a lot of data available on the Lansing effect and it seems to vary between taxa, however we lack a theoretical framework. Here, we present an evolutionary individual-based simulation model to examine which mechanisms can contribute to a Lansing effect. We model several candidate mechanisms: a decline of gamete quality with age, a decline in quality of parental care with age, and an age-specific resource allocation to repair vs. reproduction. We show that a decline in quality of parental care can readily cause a Lansing effect. A decline in gamete quality can cause a Lansing effect as well, however, this effect can also be counteracted when this candidate mechanism co-occurs with age-specific resource allocation to repair vs. reproduction. We also demonstrate how the method of data collection and analysis can alter whether a Lansing effect can be detected, i.e., cross-sectional, or longitudinal analysis. Overall, we provide a mechanistic framework for understanding the Lansing effect and its underlying mechanisms. Potentially providing insight on the disparate findings on the occurrence of a Lansing effect in empirical studies.

Taxonomic differences in parental care could account for the variations in the occurrence of a Lansing effect.

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# Introduction

Biological ageing, or senescence, is characterized by the physiological deterioration of an organism, which negatively affects the reproductive capacity and fitness of an individual (Maklakov et al., 2015). The evolution of senescence is a central topic of evolutionary biology. The biological study of senescence typically focusses on the deterioration of the soma throughout the lifespan of an individual. However, also germline deterioration can have important consequences on senescence, and potentially lead to transgenerational senescence effects (Monaghan et al., 2020). A negative correlation of parental age on offspring lifespan is known as the Lansing effect. In 1947, Albert Lansing investigated the relation between parental age and decline in offspring quality in rotifers, demonstrating that offspring die younger when their parents were older (Lansing, 1947; Monaghan et al., 2020). Following this seminal work, the Lansing effect has been studied in a range of organisms; though with highly inconsistent results. In European rabbits increasing maternal age results in reduced lifetime reproductive success, but increased survival of offspring (Rödel et al., 2009). In Asian elephants the opposite occurs; an increase in maternal age results in reduced survival of their offspring but an increase in the offspring’s lifetime reproductive success (Reichert et al., 2020). Such an increase has also been found in yellow-bellied Marmots (Kroeger et al., 2020). In both the common tern as well as in great tits a decrease in lifetime reproductive success with increasing maternal age has been demonstrated (Bouwhuis et al., 2010, 2015). In house sparrows increasing parental age results in a negative effect on lifetime fitness of the offspring (Schroeder et al., 2015). In the Seychelles warbler, maternal age negatively affects offspring lifespan, although such a correlation does not seem to occur between paternal age and offspring lifespan (Sparks et al., 2022). In *Drosophila* fruit flies, both positive (Krishna et al., 2012), negative (Hercus & Hoffmann, 2000; Kern et al., 2001; Price & Hansen, 1998; Priest et al., 2002), population-specific (Yılmaz et al., 2008) and strain-specific (Lee et al., 2019) relations between parental age and offspring lifespan have been found.

Theoretical models have demonstrated that a Lansing effect can readily evolve because selection against deleterious maternal senescence effects on offspring declines with increasing maternal age (Moorad & Nussey 2016; Hernández et al. 2020). However, a Lansing effect might also be counteracted – at least to a certain extent – by an age-specific parental investment into reproduction vs. somatic repair. The theory of ‘terminal investment’ predicts that individuals should invest more in reproduction at the end of their lifetime to prevent resources going to waste (Clutton-Brock, 1984; Duffield et al., 2017). However, as some theoretical models also predict the opposite to be true – individuals show reproductive restraint later in life instead of an increase in reproductive effort – a Lansing effect might also be reinforced by age-specific parental reproductive investment (McNamara et al., 2009; van den Heuvel et al. 2009).

Mechanistically, the Lansing effect could be explained by a variety of proximate factors (Monaghan et al. 2020). For instance, the Lansing effect could be caused by a decline in gamete quality. It has long been believed that gametes are ageless; however, this is not the case (Monaghan et al., 2020). In most of the metazoan animals, females produce gametes early in development and store them for later use. During this storage, damage accumulation can occur, leading to a decline in gamete quality (Monaghan & Metcalfe, 2019; Ziyue Gao et al., 2018). Males, however, produce their gametes from stem cells as needed over their lifetime. A proliferation phase is entered upon requirement. This can result in spermatogonia stem cell exhaustion over time and thus a decrease in gamete quality (Monaghan & Metcalfe, 2019; Pohl et al., 2021). In some species, e.g. ants, females also store sperm, then damage could accumulate in the stored sperm (Den Boer et al., 2009). Another proposed mechanism for the Lansing effect is a decline in the quality of parental care; for instance, older parents might have lower foraging success and therefore provide less food to their offspring (Monaghan et al., 2020; Muller et al., 2017; Sparks et al., 2022). Another possibility is that age-specific parental investment into repair vs. reproduction causes a Lansing effect (McNamara et al. 2009; van den Heuvel et al., 2016). In order to better understand why and how parental age can affect offspring quality and lifespan, it is necessary to evaluate whether these candidate mechanisms can indeed cause a Lansing effect.

Here, we present evolutionary individual-based simulation models to examine which mechanisms can contribute to the Lansing effect. In our models, we implemented multiple candidate mechanisms, such as a decline in gamete quality, a decline in parental care with increasing parental age, or a parental age-specific allocation of resources to repair vs. reproduction, which all could generate or modulate a Lansing effect. We examine the effect of parental age on offspring lifespan both cross-sectional as well as longitudinal to gain a better understanding of how the method of data acquisition can influence whether a Lansing effect is detected or not.

# Methods

## Individual-based simulation model

We developed an individual-based simulation model representing a population of *N* females and *N* males (overview of model parameters in Table 1). Females produce gametes at the beginning of their lives. Males possess a constant number of stem cells *nsc*, from which they produce gametes as needed. Recombination happens upon generation of gametes. Every time step, female gametes and male stem cells undergo mutations, and females mate with a random male using their gametes to generate *o* offspring. The offspring replace adult females or males that die.

We model different candidate mechanisms that could potentially cause a Lansing effect. Each of these mechanisms can be enabled and disabled in the model, and thus the mechanisms can be examined individually or in combination. The focal trait of the simulations is the lifespan of the individuals, which ranges from 0 to the maximum age *M*, at which point the individuals die in any case. The lifespan is determined by the survival effects of the different candidate mechanisms for a Lansing effect (*m*1 to *m*4). The probability that an individual at age *i* survives is given by

where the different mechanisms have a multiplicative effect on survival.

An overview of the occurrence of mutation per mechanism can be found in Figure 1.

Table 1: Model parameters and the default values.

|  |  |  |
| --- | --- | --- |
| Parameter | Value | Meaning |
| *N* | 10.000 | Number of breeding females,  Number of males |
| *tend* | 100.000 | Number of time steps |
| *M* | 40 | Maximum age |
|  | 1 | Number offspring per female per time step |
| *nsc* | 30 | Number of male stem cells |
|  | 0.0024 | Mutation rate for binary genes |
| *s* | 0.05 | Scaling parameter for effect of binary genes on survival |
|  | 0.002 | Mutation rate for age-specific genes |
| *bs* | -0.02 | Mutation bias for survival / parental care genes |
| *br* | 0 | Mutation bias for allocation genes |
|  | 0.02 | Mutational effect size for age-specific genes |
| *c* | 0.3 | Scaling parameter for effect of allocation on parental survival |
| *a* | 3 | Scaling parameter for effect of allocation on offspring survival |
| *d* | 1 | Scaling parameter for effect of allocation on offspring survival |

## Baseline: Age-specific survival evolution

We model evolving lifespans in the population due to the accumulation of mutations, over time, in each individual’s age-specific survival genes (Medawar, 1957). We assume that each individual carries diploid genes for each age from 0 to the maximum age *M*, each associated with gene values ranging between 0.0 and 1.0 (as in Kreider *et al* (2022). The average of the diploid gene values represents the age-specific survival probability of an individual. The genes were initialised with a survival probability of 0.9. The age-specific survival genes in the gametes and stem cells can mutate with a mutation probability of . If a mutation occurs, the effect was drawn from a normal distribution with a mean of *bs* < 0 (“mutation bias”) and a standard deviation of (“mutational effect size”). If the gene value decreases 0.0 or exceeds 1.0 due to a mutation, the gene value is set back to the respective limit.

## Candidate mechanism 1: Decline of gamete quality

We model damage accumulation in gametes by assuming that each individual carries 20 genes that can take the value 0 (no damage) or 1 (damage). The binary genes in the gametes and stem cells can mutate from 0 to 1 with a mutation probability *.* The survival probability multiplier for an individual, based on the binary genes, was calculated as

where *D* is the number of damaged genes and *s* is a scaling parameter that represents the strength of selection. The strength of selection determines how much the damaged genes affect the survival probability. If *s* = 0, the survival probability is 1.0, irrespective of the number of damaged genes. At initialisation of the simulation, 10% of the genes were damaged.

## Candidate mechanism 2: Quality of parental care

In this scenario, the age-specific genes from the “Baseline”-scenario determine the quality of parental care; for instance, senescence effects that reduce survival (as in the scenario above) could also have an effect on foraging success and parental care quality. We assume that the age of the parent at conception of the offspring determines the parental care quality value. The maternal and paternal care quality equally affect the offspring. The parental care quality value is multiplied with the other survival effects from the other mechanisms to determine an individual’s survival probability at a given age.

## Candidate mechanism 3: Age-specific resource allocation to repair vs. reproduction

In this scenario, we assume that individuals can distribute resources between repair for their own survival and reproduction. We assume that individuals carry a set of genes for resource allocation for each age from 0 to the maximum age *M*, each associated with a gene value ranging between 0.0 and 1.0. The gene values determine the proportion of resources allocated to reproduction. The remainder of resources are allocated to somatic repair. Upon initialisation, we assume an equal division of resources. The age-specific resource allocation genes in the gametes and stem cells can mutate with a mutation probability . If a mutation occurs, the effect of the mutation on the gene value is drawn from a normal distribution with a mean of zero and a mutational effect size of . If the gene value decreases below 0.0 or exceeds 1.0 due to a mutation, it is cut off at the respective limit. The survival probability multiplier for an individual, based on the proportion of resources allocated to reproduction *x* vs. repair 1-x, is

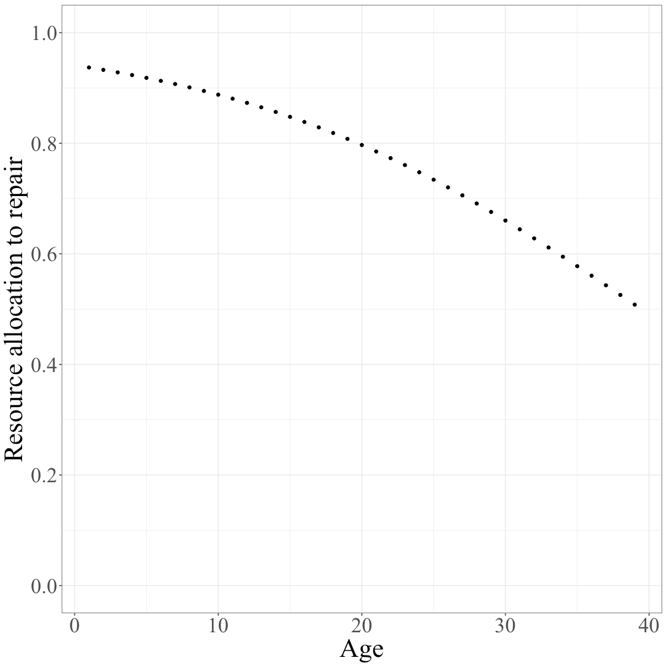
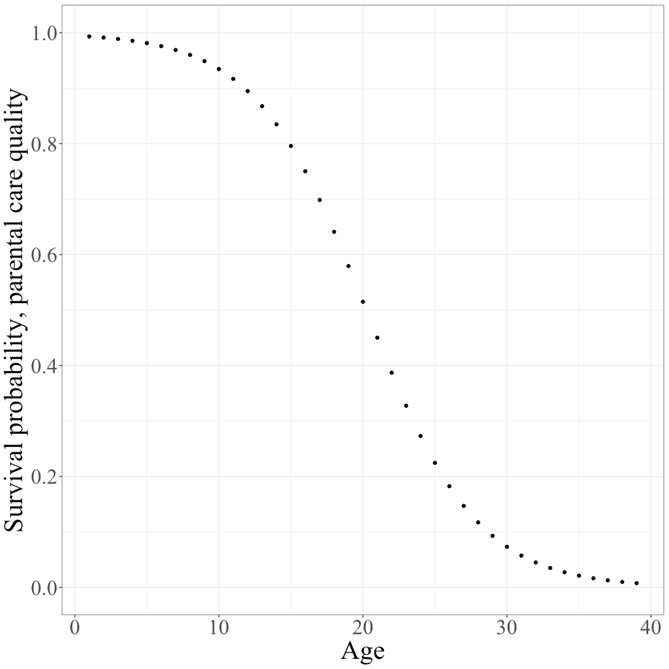
where *c* is a scaling parameter for the effect of resources invested in reproduction on survival. If *c =* 0, then the survival probability is 1.0, irrespective of the proportion of resources allocated to reproduction. The larger *c*, the more does the investment in reproduction affect the survival probability.

The proportion of resources allocated to reproduction *x* affects the survival probability of the offspring by the logistic function

where *a* and *d* are scaling parameters. Parameter *a* affects the steepness of the curve. If *a* = 0 then allocation to reproduction will not affect the survival probability of the offspring. Parameter *d* determines the location of the inflection point of the logistic function.

(A)

(B)



0 1 0 1 0 1 0 0 0 1 0 0 0 1 0 1 0 1 0 0



(C)

Figure 1: Overview of mutations in model mechanisms. (A) A mutation occurring in an age-specific survival probability of parental care quality gene. (B) Mutation occurring in an age-specific resource allocation gene. (C) Mutation occurring a binary gene.

## Model analysis and statistics

The model was implemented using C++ and compiled with g++ 8.5.0. The model analysis and statistics were performed using R 4.2.2 (R Core Team, 2022). The packages used for the analysis were *tidyverse* (Wickham et al., 2019), *mgcv* (Wood, 2011), *cowplot* (Wilke, 2020), *MetBrewer* (Blake, 2022), *ggpubr* (Kassambara, 2023) and *gridExtra* (Auguie, 2017)*.*

All simulations were run until time step *tend*. At this point, the simulations had reached an evolutionary equilibrium, where mean trait values no longer changed systematically. In order to estimate the evolved relation between offspring lifespan and parental age we obtained data at the end of the simulations cross-sectionally and longitudinally. In the cross-sectional analysis, data is collected from different individuals at a single point in time, whereas the longitudinal analysis collects data for the same individuals over an extended period of time. For the cross-sectional data, we obtained the lifespans and parental ages at reproduction for 10 offspring produced by each female parent. For the longitudinal data, we followed the parents throughout their lifespan and recorded their ages at which they produced offspring as well as the lifespans of those offspring.

For all scenario combinations, we ran ten replicate simulations. For each replicate simulation, we fitted a generalized additive model (GAM) to examine the relationship between offspring lifespan and parental age for both the cross-sectional and the longitudinal data. Generalized additive models are used to model smooth relationships between the explanatory and the response variable while taking non-linear relationships into account (Pedersen et al., 2019).

For the longitudinal data sets, we randomly sampled 100 parents who have produced offspring at a minimum of six different ages. We normalized the offspring lifespans to range between 0.0 and 1.0 and logit transform them. These values act at response variable in the GAM model, which is modelled as a function of two smoothing terms related to maternal age. The first is a smoothing function for maternal age. In the second smoothing function, we include the effect of the individual identity of the mother to model the longitudinal individual-specific effects. We do so by fitting a spline for every maternal identity by setting the smoothing basis to ‘fs’.

For the cross-sectional data sets, we randomly sampled 100 parents. Again, the offspring lifespans are normalized and logit transformed. We modelled offspring lifespan in this case only as a function of the maternal age smoothing function since we do not have individual-specific effects in the cross-sectional analysis. The smoothing parameter estimation is set to Restricted Maximum Likelihood (REML).

From both the longitudinal and cross-sectional GAM models, we use the 95th percentile of the maternal ages over all replicates of the same parameter settings, and set the age at the 95th percentile to 1.0. The offspring lifespan at parental age 0 is set to 1.0. From the models we predicted the offspring lifespan over parental age. In the figures, we depict the mean across replicates and the range of the offspring lifespan as a function of maternal age.

# Results

## Meta-analysis

Ivimey-Cook *et al* (2022) performed a meta-analysis on the occurrence of the Lansing effect across 15 different species. The estimated slopes of a linear maternal effect on offspring lifespan were determined based on data from 22 published studies or directly taken from the studies (Figure 2). Monaghan *et al* (2020) suggest a bell-shaped relationship between parental age and offspring lifespan; as parental age increases, offspring lifespan initially increases before subsequently declining. Because of this bell-shaped relationship, a Lansing effect might be difficult to identify if all maternal ages are considered; therefore, are the models applied to both all maternal ages (‘All Ages’) as well as the older maternal ages (‘Terminal Ages’). Overall, when considering all species together, there is an observed negative correlation between maternal age and offspring lifespan. This correlation is most apparent in insect species; in mammal species on the other hand, there is little to no Lansing effect apparent. In most of the species, if the model is applied to terminal ages, the effect size seems to become less negative; suggesting the relationship between parental age and offspring lifespan might not follow a bell-shaped pattern but rather exhibits an exponential decline. Overall, the meta-analysis shows taxonomic variation; in some taxa there is stronger evidence of a Lansing effect than in others. These differences could be due to biological effects. The meta-analysis underlines the disparate findings on the occurrence of the Lansing effect in empirical studies.

Figure 2: Means of slope estimates with 95% confidence intervals for all ages as well as the terminal ages. The model was also fitted to correct for a time lag and publication bias. Reprinted and modified from Ivimey-Cook et al. (2022) with the addition of the pooled group.

## Baseline: Age-specific survival evolution

****We model evolving lifespans in the population due to the accumulation of mutations, over time, in each individual’s age-specific survival genes (Medawar, 1957). When the baseline mechanism is solely enabled in the model, the survival of the individuals is only affected by the age-specific survival genes. The force of natural selection declines with age, resulting in mutation accumulation for the older ages. Under the baseline scenario no Lansing effect occurs (Figure 3A). Which is as expected, since the age-specific survival genes only affect the survival of the individual and have no parental effect on the offspring that depends on the parents’ age. An increasing mutation load leads to more mutation accumulation, and thus shorter lifespans (Figure S1).

Figure 3: (lower triangular) Offspring lifespan over parental age. Parental age normalized to the 95th percentile of parental age. The offspring lifespan normalized to the expected offspring lifespan at a parental age of 0. For both the cross-sectional (blue) and the longitudinal (orange) study design. Confidence bands show the range and lines the mean across ten replicate simulations. (Upper triangular, without diagonal) The parental age distribution. Confidence bands show the range and lines the mean across ten replicate simulations.

## Candidate mechanism 1: Decline of gamete quality

With an increasing parental age, gametes are expected to have accumulated more damage, resulting in a decrease in offspring lifespan (Monaghan & Metcalfe, 2019; Ziyue Gao et al., 2018).We model damage accumulation in the individual’s gametes by assuming individuals carry a set of binary genes affecting survival. Under this candidate mechanism, a Lansing effect occurs (Figure 3F). However, when the Lansing effect is evaluated cross-sectional under this mechanism, this effect disappears. This is probably due to the selective disappearance of individuals with low quality genes in higher ages in the cross-sectional analysis. When a decline in gamete quality co-occurs with the baseline scenario, no Lansing effect occurs (Figure 3E). In this case, the are less longer-lived individuals, resulting in less time to accumulate damage in the gametes (Figure 3B), resulting in the Lansing effect to be undetectable. By increasing the rate of gamete decline, the individuals’ lifespans shorten, for both the offspring and for the parents (Figure S2). However, the Lansing effect becomes stronger with more gamete damage accumulation.

## Candidate mechanism 2: Quality of parental care

It has been hypothesized that the quality of parental care could decrease with increasing age (Muller et al. 2017, Monaghan et al. 2020). We model a decline in quality of parental care by assuming that the age of the parent at conception determines the parental care quality of the individual. Under this candidate mechanism, a Lansing effect occurs, for both the cross-sectional and the longitudinal analysis (Figure 3K). When a decline in quality of parental care co-occurs with the baseline scenario, a Lansing effect occurs as well (Figure 3I). Finally, when a decline in quality of parental care co-occurs with a decline in gamete quality, again a Lansing effect occurs (Figure 3J). If the mutation load increases, the Lansing effect is reinforced, and the offspring lifespans decrease even steeper over increasing parental age (Figure S3).

## Candidate mechanism 3: Age-specific resource allocation to repair vs. reproduction

Age-specific resource allocation to repair vs. reproduction could contribute to a Lansing effect if individuals show reproductive restraint later in life (McNamara et al., 2009; van den Heuvel et al., 2016). Alternatively, a Lansing effect could also be counteracted if individuals evolve terminal investment. We model this by assuming that individuals carry age-specific genes for the proportion of resources allocated to reproduction. Under this candidate mechanism, we do not detect a Lansing effect (Figure 3P); i.e., individuals do not invest more or less of their resources in reproduction near the end of their lives (Figure S5). If age-specific resource allocation co-occurs with mutation accumulation, the Lansing effect cannot be detected cross-sectionally (Figure 3M). However, longitudinally, there is an increase in offspring lifespan with an increase in parental age; thus, the opposite of a Lansing effect. This is reflected in the age-specific investment of individuals, who start investing more into reproduction at the end of their lifetime (Figure S6). When the age-specific resource allocation mechanism co-occurs with a decline in gamete quality, no Lansing effect occurs (Figure 3N). In this case, the effect of the resource allocation mechanism balances out the effect of a decline in gamete quality, making the Lansing effect disappear. When the resource allocation mechanism co-occurs with the decline in quality of parental care, a Lansing effect is detected for both the cross-sectional and the longitudinal analysis (Figure 3O). The mutation rate does not affect these results qualitatively (Figure S4).

# Discussion

Biological ageing, or senescence can be characterized by the physiological deterioration of an organism (Maklakov et al., 2015). This is a central topic in evolutionary biology; however, these studies mostly focus on somatic deterioration. Germline deterioration could potentially also be of great importance for the study of senescence. In 1947, Albert Lansing discovered that offspring lifespan decreases as parental age increases, which became known as the Lansing effect (Lansing, 1947). Using evolutionary individual-based simulations, we model multiple candidate mechanisms that have been hypothesized to contribute to a Lansing effect. Our model shows that a variety of mechanisms can lead to a Lansing effect; however, the Lansing effect can also be counteracted if particular mechanisms co-occur. Our study also reveals that different data collection methods can render a Lansing effect undetectable. Our model therefore suggests that different mechanisms causing a Lansing effect in different groups of organisms and different data collection approaches could explain the highly disparate findings on the occurrence of a Lansing effect across animals (Ivimey-Cook et al. 2023).

Several mechanisms have been suggested to cause a Lansing effect. In our model, we show that damage accumulation in the individual’s gametes can contribute to a Lansing effect. However, the effect of this mechanism can also disappear and thus be counteracted. A decline in gamete quality can be caused by mutations accumulating (Ziyue Gao et al., 2018), telomere shortening, and mitochondrial mutations in gametes (Monaghan & Metcalfe, 2019). The germline might also be affected by the soma, which is less protected against mutations since the barrier between the soma and the germ cells might be less impenetrable than once thought (Monaghan & Metcalfe, 2019). Age-related germline damage accumulation is likely to depend on the lifespan of the individuals since there needs to be enough time to accumulate damage in the gametes (Hood et al., 2019). In our model we see this when this candidate mechanism co-occurs with the baseline scenario. The individuals evolve relatively short lifespans, resulting in the disappearance of the Lansing effect.

A decline in quality of parental care is also suggested to contribute to a Lansing effect (Monaghan et al. 2020). Our model demonstrates that a decline in parental care with increasing parental age can readily cause a Lansing effect. In parasitic wasps, older mothers have less resources available for reproduction and therefore invest less nutrients in their eggs (Muller et al. 2017). As a consequence, the offspring of older mothers contain lower levels of nutrients, although they can feed on host larva to overcome some of these shortages. Our results show that this mechanism might play an important role in generating a Lansing effect, since the effect stays detectable and present, irrespective of co-occurring mechanisms or of which data collection method we use.

Finally, age-specific resource allocation to repair vs. reproduction is suggested to contribute to a Lansing effect. We find that this mechanism does not result in a Lansing effect on its own; thus, the individuals do not evolve age-specific differences in their investment into reproduction vs. repair. However, when we include evolving lifespans of the individuals co-occurring with this candidate mechanism, we see an increase of offspring lifespan with increasing parental age. This means that the individuals actually invest more into reproduction at the end of their lives, which is in accordance with the ‘terminal investment’ idea from life history theory (Clutton-Brock, 1984; Duffield et al., 2017). When the resource allocation mechanism co-occurs with the decline in gamete quality, we do not find a Lansing effect. This is because the evolved resource allocation strategy counteracts the effect of the accumulated damage in the gametes. However, we do find a Lansing effect when this mechanism co-occurs with a decline in quality of parental care. The terminal investment theory has only found partial support by empirical studies. Lizards confronted with a greater risk of death, produce heavier hatchlings; thus, increasing reproductive effort when confronted with death (Fox & McCoy 2000). House sparrow immune system activation is assumed to be a cue for a greater risk of death, resulting in the females laying larger clutches of eggs (Bonneaud et al. 2004). However, if a female’s immune system is activated in fly catchers and blue tits, then this results in a decrease of reproductive effort and an increase in the investment in repair (Ilmonen et al. 2000; Råberg *et al* 2000). These “inconsistencies” are also found in our model, where the co-occurrence with other mechanisms can lead to terminal investment, no age-specific investment or reproductive restraint in late life. Our model can therefore potentially explain why terminal investment might be found in some cases but not in others.

Furthermore, our results show that the data collection method also affects whether a Lansing effect can be detected. The presence of longitudinal parental age effects may be concealed by the selective disappearance of individuals with “bad genes”. This problem does not exist in a longitudinal analysis, where the individuals are examined over their lifetime, and where a Lansing effect can be detected over their lives. We therefore encourage future studies to gather their data longitudinally (van de Pol & Verhulst, 2006).

Overall, our model demonstrates that a Lansing effect can occur under different underlying mechanisms. However, a Lansing effect can also be counteracted or reinforced if particular mechanisms co-occur. The data collection method might also conceal an existing Lansing effect. Our model thus provides a potential explanation for the disparate results from empirical studies of the Lansing effect.

In mammals, older parents might benefit from having more experience in raising their young, resulting in post-natal care of higher quality, potentially surpassing the negative effect of pre-natal parental effects.

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# Data availability

Data generated and analyzed during this study, including the R code for the figures and the C++ simulation code of the model are available in: [repository](https://github.com/WFOudijk/TheLansingEffect).

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# Supplementary material

Figure S2: Offspring lifespan over maternal age with differing rates of gamete decline; the mutation probability for the binary genes for candidate mechanism 1: Decline of gamete quality. Confidence bands show the range and lines the mean across ten replicate simulations.

Figure S1: Offspring lifespan over maternal age with differing mutation probabilities for the baseline scenario: age-specific survival evolution. Confidence bands show the range and lines the mean across ten replicate simulations.

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Figure S4: Offspring lifespan over maternal age with differing mutation probabilities of the age-specific resource allocation genes for candidate mechanism 3: Age-specific resource allocation to repair vs. reproduction. Confidence bands show the range and lines the mean across ten replicate simulations.

Figure S3: Offspring lifespan over maternal age with differing mutation probabilities of the age-specific survival genes for candidate mechanism 2: Quality of parental care. Confidence bands show the range and lines the mean across ten replicate simulations.

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Figure S5: Average gene values over corresponding ages for candidate mechanism 3: Age-specific resource allocation to repair vs. reproduction. Confidence bands show the range and lines the mean across ten replicate simulations. The genes corresponding to older ages are not expressed in the population, resulting in wide confidence intervals for these ages.

Figure S6: Average gene values over corresponding ages for candidate mechanism 3 in combination with the baseline scenario. Confidence bands show the range and lines the mean across ten replicate simulations. The genes corresponding to older ages are not expressed in the population, resulting in wide confidence intervals for these ages.

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Figure S7: Offspring lifespan over parental age. Parental age normalized to the 95th percentile of parental age. The offspring lifespan normalized to the expected offspring lifespan at a parental age of 0. For both the cross-sectional (blue) and the longitudinal (orange) study design. Confidence bands show the range and lines the range across ten replicate simulations. (A) Baseline scenario + candidate mechanism 1 + candidate mechanism 2. (B) Baseline scenario + candidate mechanism 1 + candidate mechanism 3. (C) Candidate mechanism 1 + candidate mechanism 2 + candidate mechanism 3. (D) Baseline scenario + candidate mechanism 2 + candidate mechanism 3. Baseline scenario: age-specific survival evolution; candidate mechanism 1: decline of gamete quality; candidate mechanism 2: quality of parental care; candidate mechanism 3: age-specific resource allocation to repair vs. reproduction.

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Figure S8: Offspring lifespan over parental age for all four mechanisms combined: baseline age-specific survival evolution + decline of gamete quality + quality of parental care + age-specific resource allocation to repair vs. reproduction. Parental age normalized to the 95th percentile of parental age. The offspring lifespan normalized to the expected offspring lifespan at a parental age of 0. For both the cross-sectional (blue) and the longitudinal (orange) study design. Confidence bands show the range and lines the range across ten replicate simulations.