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

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**Abstract**

Broad and general what is Lansing effect and why is it relevant to study

Evidence for and against, we lack a theoretical framework

Here we present .. model for the Lansing effect.

We simulate different mechanisms, name them in a biological way.

What do the results show, some show Lansing effect, some don’t , for example an age-specific investment can counteract gamete decline. Intuituve for the reader.

We also demonstrate how the method of data collection might affect this.

Overall, we provide a mechanistic framework for understanding.. Potentially providing insight on the occurrence of a Lansing effect in empirical studies.

**Table of contents**

*Introduction*

*Methods*

*Results*

*Discussion*

*References*

*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. Moorad & Nussey (2016) have demonstrated in a quantitative genetics model and Hernández et al. (2020) in a demographic model that selection against deleterious maternal senescence effects declines with increasing age. 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). 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 and to predict the relative importance of them. In our models, we implemented multiple 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 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 : 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 |
| *ba* | 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 by assuming that, over time, each individual accumulates mutations in age-specific survival genes (Medawar, 1957). We assume that each individual carries diploid genes for each age from 0 to the maximum age *M* associated with gene values ranging between 0.0 and 1.0 (as in Kreider *et al* 2022). The average of the two homologous 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 the respective limit.

**Candidate mechanism 1: Decline of gamete quality**

We model damage accumulation in gametes by assuming that each individual carries twenty 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 will be one, 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 survival genes from above 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. We assume that the parental care quality value affects the survival of the offspring throughout its life. The parental care quality value is thus multiplied with the other survival effects from the other scenarios 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 to repair for their own survival or to 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

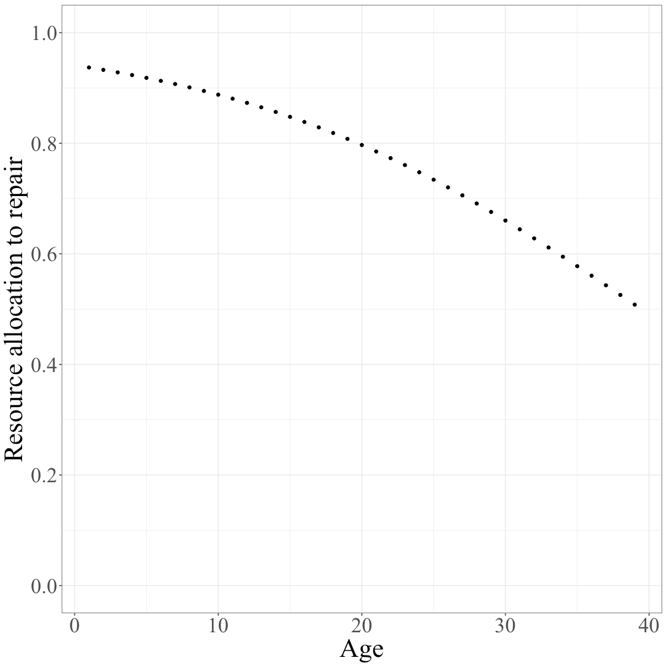
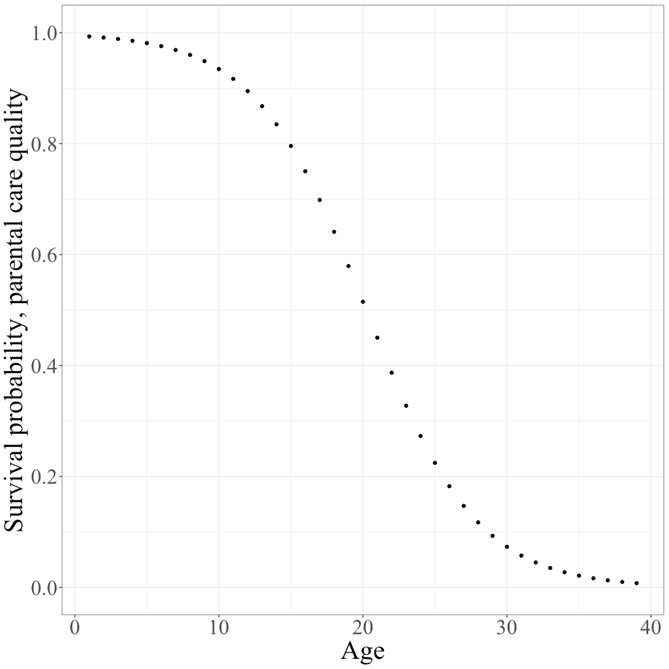
where *c* is a scaling parameter for the effect of resources invested in reproduction on survival. If *c =* 0, then the survival probability will be one, irrespective of the proportion of resources allocated to reproduction. The larger *c*, the more does the investment in reproduction affect the survival 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 sigmoidal 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 : Overview of mutations in model mechanisms. (A) A mutation occurring in an age-specific survival probability gene. (B) Mutation occurring in an age-specific resource allocation gene. (C) Mutation occurring a binary gene.

**Model analysis and statistics**

The model is 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 are *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 (quasi) equilibrium, where mean trait values no longer changed systematically. In order to estimate the evolved relation between offspring lifespan and parental age we gather data upon reaching the end of the time simulation. First, the cross-sectional data was gathered by having the final population make ten offspring per female. We simulate the generated offspring until all of them have died, the ages at death are then recorded. Next, we collect the longitudinal offspring lifespan over parental age, we let the final population reproduce at the end of the simulation. The offspring become the new population. We then simulate this population and record the age at which they themselves produce offspring and the lifespans that this offspring had.

For all scenarios and combinations, we run ten replicates and gather both the cross-sectional as the longitudinal data. For every run we fit a model. We use generalized additive models to model the relationship between offspring lifespan and parental age for both the cross-sectional data as for 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 as well as interactions among variables (Pedersen et al., 2019). For the longitudinal data sets, we first subset the data by choosing parents who have produced offspring at a minimum of six different ages, of this group, we randomly sample a hundred parents. Up next, we normalize the offspring’s ages at death to range between 0.0 and 1.0 and then logit transform them. These values become the response variable in the model equation. We model the response variable as a function of two smooth terms related to maternal age. The first is a smooth function for maternal age. The second smooth function we apply additional smoothing to maternal age by including the effect of the identifier ID, to model the longitudinal individual-specific effects. We do so by fitting a spline for every maternal ID by setting the smoothing basis to ‘fs’. We set the smoothing parameter estimation to Restricted Maximum Likelihood (REML), which takes into account both the fixed effects as the random effects in the model. For the cross-sectional analysis we first subset the data as well, in this case only by sampling a hundred random parents. The offspring’s ages at death are normalized and logit transformed and are used as the response variable in the model. In this case the response variable is modelled only as a function of the maternal age smooth 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) as well. Up next, we determine per scenario the 95th percentile of the maternal age over all replicates, so the maternal age class that contains 95% of the data. We make a new data set from maternal age of 0 to the percentile age class. Once the models are fitted to the data, we use it to predict the offspring lifespan over parental age using this new data set. We do so for every replicate, the expected values are averaged, the minimum and maximum is recorded as well. The mean is represented as the expected lifespan and the minimum and maximum serve as confidence intervals.

*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 papers. These slopes were determined for both all maternal age classes, as for only the older age classes. The models were fitted to correct for a time lag and publication bias (Figure 2). A Lansing effect is most apparent in insect species, however neither mammalian species show any Lansing effect. In mammalian species, older parents might have more experience, resulting in post-natal care of higher quality, potentially surpassing the negative effect of pre-natal parental effects. In general, as the wide confidence intervals show, there are a lot of inconsistencies within species. Also, between species the strength of the Lansing effect differs. This meta-analysis confirms the highly inconsistent results when examining the Lansing effect.

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Figure 2: Means of slope estimates with 95% confidence intervals for all age classes as well as only the two terminal age classes. 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 species.

**Baseline: Age-specific survival evolution**

We model evolving lifespans in the population by assuming that, over time, each individual accumulates mutations in 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 age classes. 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 offspring that depends on the parents’ age. With an increasing mutation load, the force of selection becomes weaker resulting in the lifespans to shorten, for both the parents as for the offspring (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 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 is detected (Figure 3F). However, when this mechanism is determined cross-sectional, this effect disappears. Due to selective disappearance of the weak individuals in the cross-sectional analysis. Combining this mechanism with the baseline scenario, no Lansing effect is detected; in this case, the are less longer-lived individuals, resulting in less time to accumulate damage in the gametes (Figure 3B). Resulting in a possible Lansing effect to be undetectable. By increasing the rate of gamete decline, the individuals’ lifespans shorten, for both the offspring as for the parents (Figure S2). However, the Lansing effect becomes stronger with more gamete damage accumulation.

**Candidate mechanism 2: Quality of parental care**

The quality of parental care might decrease over progressive age. Muller *et al* (2017)show this in parasitic wasps, where older parents are less successful in provisioning nutrients for their offspring compared to younger parents. We model a decline in quality of parental care by assuming that the age of the parent at conception and the corresponding survival gene value determines the parental care quality of the individual. Under this candidate mechanism, a Lansing effect is detected, for both the cross-sectional as the longitudinal analysis (Figure 3K). When this mechanism is combined with the baseline scenario, we detect a Lansing effect as well (Figure 3I). However, we still see less long-lived individuals, but in this case, this does not result in the disappearance of the Lansing effect (Figure 3C). When a decline in quality of parental care is combined with the first candidate mechanism, a decline in gamete quality we also detect the Lansing effect (Figure 3J). In this case, there are some long-lived individuals present (Figure 3G). If the mutation load increases, the Lansing effect is reinforced, and the offspring lifespans decrease even steeper over increasing parental age (Figure S3). However, again the parents’ lifespans shorten as well.

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

The 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). We model this by assuming the individuals carry another set of age-specific genes, the gene values represent the proportion of resources allocated to reproduction.

Under this candidate mechanism, we do not detect a Lansing effect (Figure 3P). Meaning, individuals do not invest more or less of their resources in reproduction near the end of their lives. For young age classes, individuals will invest more into somatic repair, this decreases with progressive parental age to the point where older individuals invest more or less the same proportion of resources in somatic repair as in reproduction (Figure S5). The genes of the older age classes are not expressed in the population, which explains why this decrease does not result in a Lansing effect. When we combine this mechanism with the baseline scenario, looking at it cross-sectionally, there is again no Lansing effect (Figure 3M). However, looking at it longitudinally, results in an increase of offspring quality over parental age, so the opposite of a Lansing effect. Meaning, individuals start investing more into reproduction at the end of their lifetime (Figure S6). When the age-specific resource allocation mechanism is enabled with a decline a gamete quality, no Lansing effect is can be detected (Figure 3N). Within this combination of candidate mechanisms, there are still longer-lived parents present (Figure 3H).

Meaning that in this case, the effect of the resource allocation mechanism balances out the effect of a decline in gamete quality in contributing to the Lansing effect. When the resource allocation mechanism is combined with the decline in quality of parental care, a Lansing effect is detected for both the cross-sectional as for the longitudinal analysis (Figure 3L). When the mutation load is increased, it does not influence either parental or offspring lifespan. Due to the unbiased nature of the mutations.

**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 the somatic deterioration. But germline deterioration could potentially also be of great importance in the study of senescence. This was studied by Albert Lansing in 1947. He discovered that offspring lifespan would decrease over an increasing parental age, which became known as the Lansing effect (Lansing, 1947). Using evolutionary individual-based simulation models, we demonstrate the evolution of a Lansing effect across various scenarios. We model multiple candidate mechanisms possibly contributing to a Lansing effect. Our model shows that a Lansing effect can evolve, however it can also be contradicted or undetectable. In addition to the candidate mechanisms resulting in different results, our study reveals that different ways of data collection methods for the model analysis can lead to a similar effect. These results show that there are multiple factors important in the evolution of the Lansing effect and could explain the inconsistencies within and between species found when examining the Lansing effect.

Several mechanisms have been suggested to play a part in contributing to 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 disappear and thus be contradicted when examined further. A decline in gamete quality can be caused by mutations accumulating (Ziyue Gao et al., 2018); telomeres shortening; and mitochondrial mutations in gametes (Monaghan & Metcalfe, 2019). The germline might also be affected by the soma, which is less protected for mutations since the barrier between the soma and the germ cells might be less impenetrable as 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 is combined with the baseline scenario. The individuals’ lifespans evolve to a point where there are less longer-lived individuals present, resulting in the disappearance of the Lansing effect.

A decline in quality of parental care is also suggested to contribute to a Lansing effect. We find that this is indeed the case; we find a strong Lansing effect when examining this candidate mechanism. Monaghan *et al* (2020) suggest a decline in quality of parental care might contribute to a Lansing effect. Muller *et al* (2017) show this in a study of parasitic wasps. Older mothers have less resources available for reproduction and therefore invest less nutrients in their eggs. 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 part in generating a Lansing effect, since the effect stays detectable and present, irrespective of other mechanisms we add or of which data collection methods use.

Finally, an 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; we do not find any decrease or increase of offspring lifespan over increasing parental age. Meaning, that the individuals do not invest more or less into reproduction at the end of their lives. However, when we include evolving lifespans of the individuals together with this candidate mechanism, we see a longitudinal increase of offspring lifespan. Meaning that the individuals actually invest more into reproduction at the end of their lives, which is in accordance with the life history theory ‘terminal investment’ (Clutton-Brock, 1984; Duffield et al., 2017). When combining the resource allocation mechanism with the decline in gamete quality mechanism, surprisingly we do not find a Lansing effect. Meaning that the resource allocation mechanism contradicts the previously found Lansing effect due to a decline in gamete quality. However, we do find a Lansing effect when we combine this mechanism with a decline in quality of parental care. Results concerning the terminal investment theory have been inconsistent. Fox & McCoy (2000) demonstrate that lizards confronted with a greater risk of death, produce heavier hatchlings. Meaning, the reproductive effort increases when confronted with death. Bonneaud *et al* (2004) demonstrate the same effect in house sparrows; immune system activation is expected to be the cue for a greater risk of death, resulting in the females laying bigger egg clutches and the offspring differed in size as well. However, Ilmonen *et al* (2000) demonstrate the opposite effect in female fly catchers, if a female’s immune system is activated, it results in a decrease of reproductive effort and an increase in repair for itself. Råberg *et al* (2000) demonstrate this as well; activated immune system results in a decrease in reproductive effort in blue tits. Our results confirm the inconsistencies, for future references it would be interesting to model the life-history theory for specific species to see whether we can theoretically proof the terminal investment theory.

Furthermore, our results show that the data collection method might also affect whether a Lansing effect can be detected. The presence of longitudinal parental age effects may be concealed by the selective disappearance of poor-quality individuals. These poor-quality individuals are taken into account in the longitudinal analysis. The higher-quality individuals are examined over their lifetime, meaning we can still detect a possible decrease in offspring lifespan over their lives. We therefore encourage future empirical studies to gather the data longitudinally.

Overall, our model demonstrates a Lansing effect can occur under different underlying mechanisms. However, due to contradicting effects of other scenarios the Lansing effect might become undetectable or disappear. The data collection method might also conceal an existing Lansing effect. These differences might explain the varying results between and within species of the Lansing effect.

[future reference]

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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. 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 S6: Average gene values over corresponding age classes 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 age classes are not expressed in the population, resulting in wide confidence intervals for these ages.

Figure S5: Average gene values over corresponding age classes 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 age classes are not expressed in the population, resulting in wide confidence intervals for these ages.