HAL open science

The hidden ageing costs of sperm competition

Jean-François Lemaître Lemaître, Jean-Michel Gaillard, Steven Ramm

To cite this version:

Jean-François Lemaître Lemaître, Jean-Michel Gaillard, Steven Ramm. The hidden ageing costs of sperm competition. Ecology Letters, 2020, 23 (11), pp.1573-1588. 10.1111/ele.13593 . hal-03060263

HAL Id: hal-03060263

https://hal.science/hal-03060263v1

Submitted on 14 Dec 2020

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

The hidden ageing costs of sperm competition

Jean-François LEMAÎTRE[1*], Jean-Michel GAILLARD[1] & Steven A. RAMM[2]

[1] *Université de Lyon, F-69000, Lyon; Université Lyon 1; CNRS, UMR5558, Laboratoire de Biométrie et Biologie Évolutive, F-69622, Villeurbanne, France.*
[2] *Evolutionary Biology, Bielefeld University, Konsequenz 45, 33615 Bielefeld, Germany*

* Correspondence: jean-francois.lemaitre@univ-lyon1.fr

Type of article: *Ideas and Perspectives*
Running title: *The hidden ageing costs of sperm competition*
Number of references: 213
Number of words in the abstract: 199
Number of words in the main text: 7790
Number of figures: 1
Number of tables: 2
Number of boxes: 1
Keywords: *Actuarial senescence - Ejaculate quality - Life history evolution - Longevity - Phenotypic plasticity - Reproductive senescence - Seminal fluid - Sexual selection - Spermatogenesis*

Data accessibility statement: This article contains no data

Authorship: JFL and SAR designed the study. JFL, JMG and SAR discussed all aspects of the research and contributed to writing the paper

Abstract

Ageing and sexual selection are intimately linked. There is by now compelling evidence from studies performed across diverse organisms that males allocating resources to mating competition incur substantial physiological costs, ultimately increasing ageing. However, although insightful, we argue here that to date these studies cover only part of the relationship linking sexual selection and ageing. Crucially, allocation to traits important in post-copulatory sexual selection, i.e. sperm competition, has been largely ignored. As we demonstrate, such allocation could potentially explain much diversity in male and female ageing patterns observed both within and among species. We first review how allocation to sperm competition traits such as sperm and seminal fluid production depends on the quality of resources available to males and can be associated with a wide range of deleterious effects affecting both somatic tissues and the germline, and thus modulate ageing in both survival and reproductive terms. We further hypothesize that common biological features such as plasticity, prudent sperm allocation and seasonality of ejaculate traits might have evolved as counter-adaptations to limit the ageing costs of sperm competition. Finally, we discuss the implications of these emerging ageing costs of sperm competition for current research on the evolutionary ecology of ageing.

INTRODUCTION

The ageing process is generally defined as a progressive decline in the age-specific contribution to fitness (Partridge & Barton 1993; Kowald & Kirkwood 2015; Gaillard & Lemaître 2020), which occurs due to a decrease in fertility and / or survival rates with advancing age (also called reproductive and actuarial senescence, respectively). Age-specific decreases in reproductive performance or survival are widespread in the animal kingdom (Nussey *et al.* 2013; Jones *et al.* 2014), governed by myriad physiological processes (López-Otín *et al.* 2013; Kennedy *et al.* 2014) and potentially largely uncoupled from each other (Gaillard & Lemaître 2017).

The trade-off between growth and reproductive effort early in life, and ageing in terms of reproduction and survivorship constitutes a major cornerstone of our current understanding of the evolution of ageing (Lemaître *et al.* 2015; Flatt & Partridge 2018). Based on the declining force of natural selection with increasing age (Medawar 1952), Williams (1957) proposed the *antagonistic pleiotropy* theory of ageing, positing that an allele with a positive effect on fitness during early adulthood will be selected even if this same allele is responsible for a decline in reproductive efficiency or survival prospects later in life (Williams 1957; Gaillard & Lemaître 2017; Austad & Hoffman 2018). One physiological mechanism by which the evolutionary trade-off between reproductive effort in early life and ageing can occur is a resource-based allocation trade-off, that constitutes the core of the *disposable soma* theory of ageing (Kirkwood 1977). This theory postulates that individuals have to allocate the limited resources they acquire to growth, reproduction and somatic maintenance (Kirkwood & Rose 1991). This means that substantial allocation to growth during early life leaves fewer resources to devote to reproduction or somatic maintenance mechanisms (e.g. protein clearance, Vilchez *et al.* 2014), which is then ultimately responsible for an earlier and/or faster rate of ageing (Kirkwood & Rose 1991; Kirkwood 2017). Another specific physiological pathway, the so-called *developmental* theory of ageing (Maklakov & Chapman 2019) offers an alternative mechanism underlying the genetic trade-off envisioned by Williams (1957). According to that theory, physiological processes that allow fast growth and intense reproduction early in life have long-lasting effects that generate pathologies at old ages through cellular hyperfunction (Gems & Partridge 2013). Decades of research performed under both laboratory and wild conditions support the existence of a trade-off between reproduction and ageing (Flatt & Promislow 2007; Lemaître *et al.* 2015; Austad & Hoffman 2018).

A striking feature of the many studies investigating the ageing consequences of a strong allocation to reproduction is their overwhelming focus on females (Lemaître *et al.* 2015). For instance, a first compilation of studies testing for such early- vs. late-life trade-offs in wild populations of vertebrates revealed that 85.3 % of the single-sex studies were focused on females (Lemaître *et al.* 2015). There are several causes of this bias, including the central role of females in population dynamics, the higher energetic costs of gamete production for females compared to males (Hayward & Gillooly 2011), the obvious physiological costs associated with maternal care (e.g. lactation in harbour seal, *Phoca vitulina*, Bowen *et al.* 1992), and the additional need for genetic analyses (and associated uncertainty) when it comes to assigning paternity rather than maternity. Even in laboratory settings where paternity can be assigned more easily, most studies still largely focused on females (but see below). Yet, the recent evidence that the intensity of ageing in both survival (e.g. Lemaître *et al.* 2020) and reproduction (e.g. Brengdahl *et al.* 2018) can differ widely between males and females across species strongly emphasizes that we need to investigate how both natural and sexual selection interact to shape male ageing patterns. Such studies could reveal new insights about the evolutionary roots of the sex-specific decline in physiological performance and health issues at late ages (Marais *et al.* 2018).

So far, all attempts to reconcile male life history strategies within the early- vs. late-life trade-off framework offered by the evolutionary theory of ageing have put forward the predominant role of sexual selection (Vinogradov 1998; Promislow 2003; Bonduriansky *et al.* 2008; Hooper *et al.* 2018). Basically, these studies rely on the traditional 'sex role' paradigm in evolutionary ecology (Bateman 1948; Trivers 1972), with males competing for gaining access to mating opportunities while females are the choosier sex (Dewsbury 2005; Janicke *et al.* 2016). Evidence that sexual selection favours conspicuous behaviours and extravagant sexual traits is widespread in nature (Andersson 1994). Moreover, male reproductive effort is known to be associated with some physiological correlates of ageing (e.g. a decrease in anti-oxidant defences, Alonso-Alvarez *et al.* 2004, an impaired immune response, Peters *et al.* 2004, or a stronger rate of telomere attrition, Giraudeau *et al.* 2016). Although the exact physiological pathways linking sexual selection to the degradation of the 'organismal somatic state' are likely to be complex and probably involve a complex role of androgens (Brooks & Garratt 2017; Metcalf *et al.* 2020), several empirical studies have evidenced an ageing cost of male allocation to sexual competition (see Table 1). Most of these studies have expressed this survival cost in terms of lifespan reduction, which may not directly translate into an ageing

cost in the strict sense (Péron *et al.* 2019). However, a few studies have also considered possible consequences in terms of ageing *per se*. For instance, in the wild, male red deer (*Cervus elaphus*) that allocate strongly to reproductive effort by displaying rutting behaviour during an extended period of the breeding season or by controlling large harems suffer from a stronger rate of reproductive ageing, measured through competitive abilities (Lemaître *et al.* 2014).

Is sperm competition a neglected cause of ageing? Earlier studies have provided important insights into how sexual selection influences lifespan and impacts age-specific reproduction and survival. However, in this perspective article, we argue that studies seeking to decipher the ageing costs of male allocation to sexual competition need to fully embrace its complexity. Specifically, male allocation to sexual competition can be divided into two consecutive episodes: competition over mating (i.e. pre-copulatory competition) and competition over fertilization (i.e. post-copulatory competition) (Parker 1998; Andersson & Simmons 2006; Simmons *et al.* 2017). So far, the physiological and ageing consequences of male allocation to post-copulatory competition have been extensively studied in females (see Box 1 for a synthesis), but remain almost totally overlooked in males, which prevents us from drawing any firm conclusion on how male reproductive allocation influences fitness-related traits in late life.

We thus aim to highlight the untapped potential of sperm competition studies for better understanding the ageing process. As we show, there are multiple ways in which allocation to sperm competition could alter physiological pathways ultimately linked to ageing in reproduction and survivorship (Figure 1). Although other components of the male reproductive phenotype linked to sperm competition success might be considered in the future (e.g. genital morphology; behavioural adaptations), we deliberately focus here on ejaculate-related adaptations to post-copulatory competition (both sperm and non-sperm components) as these seem likely to be most strongly associated with long-term ageing costs. After considering the various potential costs, conceptually as well as through a critical appraisal of the limited empirical evidence published so far, we further argue that plasticity in many of these traits can best be understood in the light of the evolutionary theory of ageing. Finally, we provide a roadmap for future studies aimed at deciphering the hidden ageing costs of sperm competition.

AGEING COSTS OF SPERM COMPETITION

153 **(a) Sperm quantity and quality**

154 Female multiple mating is widespread in nature (Birkhead & Møller 1998). As a
155 consequence, sperm competition (i.e. when sperm from two or more males compete to
156 fertilize a set of ova, Parker 1970) is found in the vast majority of species (Birkhead & Møller
157 1998). Across species, males can improve their fertilization success through several (non-
158 mutually exclusive) pathways. The most studied pathway is undoubtedly through producing
159 more sperm, as sperm competition game models generally predict that a higher number of
160 sperm within ejaculates provide a fertilization advantage over competitors (reviewed in
161 Pizzari & Parker 2009; Parker 2016). In line with these predictions, it has been repeatedly
162 observed that the production of larger ejaculates is indeed often associated with a higher
163 fertilization success (e.g. Gage & Morrow 2003; Boschetto *et al.* 2011). Unsurprisingly,
164 sexual selection has thus promoted the evolution of larger testes (Parker 2016) as well as a
165 higher proportion of sperm-producing tissue within it (e.g. Lüpold *et al.* 2009) and a faster
166 rate of spermatogenesis (Ramm & Stockley 2010), under intense levels of sperm competition
167 (e.g. in promiscuous species). Indeed, the selective pressure of sperm competition likely
168 shapes multiple aspects of spermatogenesis affecting sperm production rate, from the number
169 and activity of spermatogonial stem cells, the degree and architecture of their expansion and
170 their post-meiotic differentiation into highly specialised sperm cells (Ramm *et al.* 2014;
171 Ramm & Schärer 2014).

172 An increase in the number of sperm allocated per ejaculate is only one of the multiple
173 pathways boosting fertilization success. Several features of the sperm cell itself have evolved
174 in response to intense levels of sperm competition (Pizzari & Parker 2009; Fitzpatrick &
175 Lüpold 2014). Among these characteristics, the positive influence of sperm motility (i.e.
176 proportion of motile sperm within an ejaculate) and of average within-ejaculate sperm
177 velocity on male fertilizing ability has been repeatedly documented (Snook 2005; Pizzari &
178 Parker 2009). These effects are explained by the fact that faster sperm will often reach
179 female's gametes quicker than slower sperm (see for example Gage *et al.* 2004; Gasparini *et*
180 *al.* 2010 for evidence in external and internal fertilizers, respectively). Comparative analyses
181 performed across various taxa have revealed that sperm swimming velocity increases with
182 sperm size (e.g. Fitzpatrick *et al.* 2009 in fishes; Tourmente *et al.* 2011 in mammals),
183 highlighting that various structural aspects of sperm – especially flagellum or midpiece
184 length/volume – modulate sperm velocity (Cardullo & Baltz 1991; Pizzari & Parker 2009). In
185 primates, males from species where females mate with multiple partners display midpieces
186 with a larger volume (Anderson & Dixson 2002). As larger midpieces are associated with a

187 greater mitochondrial loading supporting sperm motion (Gu *et al.* 2019), this increased
188 midpiece volume appears to be adaptive when females mate with several males within a given
189 breeding attempt.
190 In the next sections, we consider the various pathways through which allocation to
191 these sperm traits imposes costs on males that likely result in accelerated ageing in
192 reproduction or survivorship.
193
194 *Energetic costs of sperm production.* One tenet of our current understanding of the evolution
195 of ageing relies on the energetically-demanding aspect of reproductive allocation (Kirkwood
196 2017), which is typically the case when it comes to producing an ejaculate and to growing and
197 maintaining an efficient reproductive machinery (Kenagy & Trombulak 1986). Although
198 earlier studies assumed that sperm were particularly cheap to produce (see Dewsbury 2005),
199 the influence of resource availability on sperm production has been demonstrated in a wide
200 spectrum of organisms. For instance, in fallow deer (*Dama dama*), a species in which females
201 mate with several males during a given reproductive attempt (Briefer *et al.* 2013), yearling
202 males with a diet supplemented with protein and amino acids have larger testes, exhibit
203 Sertoli cells – which play a key nutritional function during spermatogenesis – with higher
204 workload capacities, higher concentrations of sperm within the epididymis and sperm with a
205 longer midpiece than do non-supplemented yearling males (Ros-Santaella *et al.* 2019). This
206 positive effect of an enriched diet on sperm quantity and/or quality has also been repeatedly
207 found in agronomy research (e.g. Zhao *et al.* 2017) and in invertebrates. In cockroaches
208 (*Nauphoeta cinerea*), for example, the number of sperm found in the spermatophore delivered
209 to females (but not sperm viability) increases with the intake of both proteins and
210 carbohydrates (Bunning *et al.* 2015). The same relationships are observed in the Argentine ant
211 (*Linepithema humile*), where a protein restriction in the diet during the larval stage (i.e. when
212 the whole stock of sperm is produced) impairs sperm production but does not decrease sperm
213 viability (Dávila & Aron 2017). In this social insect, a stronger allocation of the decreased
214 quantity of resources to sperm quality (i.e. sperm viability) might be adaptive as sperm can be
215 stored for several years before fertilization in the female spermathecae. Overall, the
216 production of high-quality ejaculates largely depends on male condition and the quantity of
217 resources that individuals can allocate to the post-copulatory traits (Perry & Rowe 2010),
218 although the condition-dependent nature of ejaculate traits likely vary across species (see
219 Macartney *et al.* 2019 for a meta-analysis).

220 Limitations on resource availability that generally occur in the wild lead to an increase
221 in the physiological costs of spermatogenesis. This is well illustrated in adders (*Vipera berus*),
222 where the magnitude of testis growth prior to the reproductive season is positively correlated
223 with body mass loss (Olsson *et al.* 1997). In this species, the allocation to spermatogenesis
224 (i.e. post-copulatory traits) accounts for a higher proportion of mass loss than the allocation to
225 mate searching (i.e. pre-copulatory traits) (Olsson *et al.* 1997), which strongly suggests that
226 the long-term ageing costs of mass loss generated by repeated reproductive seasons
227 throughout life might not be trivial. It thus appears that males substantially allocating to
228 sperm competition might – following the life history of ageing framework (Lemaître *et al.*
229 2015; Kirkwood 2017) – suffer from strong physiological costs (e.g. immune costs, as
230 discussed in the next section) that will ultimately lead to a shorter lifespan and/or more
231 pronounced ageing in fitness components.
232
233 *Immune costs of sperm production.* Pathogen-infected males generally have less efficient
234 post-copulatory traits than uninfected males, whether pathogens are directly located in the
235 ejaculate component (e.g. Cunningham & Beagley 2008) or not (e.g. Polak 1998). The
236 negative association between the allocation to ejaculate-related traits and the performance of
237 the immune system (or vice-versa) likely accounts for this difference (see Table 2 for a
238 synthesis). For instance, in Arctic charr (*Salvelinus alpinus*) males, the density of
239 granulocytes (i.e. white blood cells belonging to the non-specific immune system) is
240 negatively correlated with the concentration of sperm within semen (Liljedal *et al.* 1999).
241 Overall, the trade-off between ejaculate quality and immune performance is pervasive, even if
242 a thorough investigation of the immune repertoire is required because the allocation to
243 ejaculate-related traits might be negatively related to some immune cells while being
244 positively associated with others (see Simmons & Roberts 2005 for a case study in crickets,
245 *Teleogryllus oceanicus*). This decreased activity of the immune system following an increased
246 sperm production is adaptive because it minimizes the risk of autoimmunity, since antigens at
247 the surface of sperm cells lead them to be recognized as foreign bodies by the immune cells
248 (see Skau & Folstad 2005). In line with this functional link, experimental evolution work in
249 yellow dung flies (*Scathophaga stercoraria*) revealed that males from lines selected under an
250 elevated sperm competition regime show lower phenoloxydase activity than males from
251 monandrous lines (Hosken 2001), which makes these males more likely to suffer from an
252 infection (McKean & Nunney 2001). In addition, the negative association between sperm
253 production and immunity might be directly reinforced by a resource-based allocation trade-

254 off. This is well-illustrated in crickets (*T. oceanicus*), where the trade-off between
255 antibacterial activity and sperm viability is only observed in individuals fed with a nutrient-
256 restricted diet (Simmons 2012).
257 Similar to what has already been reported for the allocation to growth and/or
258 reproduction in males, or the male allocation to the growth and maintenance of pre-copulatory
259 traits in males (Folstad & Karter 1992; Rödel *et al.* 2016), a strong allocation to post-
260 copulatory traits can be highly detrimental for male's immune performance (Table 2). As
261 immunity constitutes a key physiological function mediating trade-offs among life-history
262 traits (Sheldon & Verhulst 1996), an elevated allocation to post-copulatory competition
263 should have long-term consequences in terms of reduced lifespan and increased ageing rates
264 through the impairment of immune performance throughout life.
265
266 *Reproductive senescence costs of sperm production*. Similar to what is observed for the
267 allocation of resources to pre-copulatory traits in early life (Table 1), the long-term fitness
268 costs of a strong allocation to sperm production could also be expressed in terms of
269 reproductive ageing. Thus, in *Drosophila melanogaster*, an increased reproductive effort early
270 in life has a negative impact on subsequent fertility, which is mediated by a pronounced age-
271 specific decline in sperm numbers (Prowse & Partridge 1997). Similarly, in the domestic fowl
272 (*Gallus gallus domesticus*), males that produce the highest quality sperm (judged through
273 sperm velocity) during early life show the steepest decline in sperm quality in late life
274 (Cornwallis *et al.* 2014). This suggests that males allocating a lot to the production of high-
275 quality ejaculates during early life might suffer from a stronger decline in reproductive
276 success at late ages. Evidence that males naturally suffer from a decline in reproductive
277 success with increasing age is widespread across the tree of life (Johnson & Gemmell 2012;
278 Lemaître & Gaillard 2017), and the (epi)genetic and physiological mechanisms underlying
279 this decline are diverse (Evans *et al.* 2019; Fricke & Koppik 2019; Monaghan & Metcalfe
280 2019). Here we discuss four related aspects that seem especially relevant when sperm
281 competition occurs.
282 First, the accumulation of mutations on the male germline throughout life (e.g. Velando
283 *et al.* 2011; Kong *et al.* 2012) might play a key role in causing reproductive ageing
284 (Monaghan & Metcalfe 2019). In that context, is selection for a higher allocation to sperm
285 competition responsible for an increased mutation rate? And do elevated mating rates amplify
286 this mutation load, as males need to replenish their sperm storage quickly to avoid sperm
287 limitation (Dewsbury 1982; Nakatsuru & Kramer 1982)? There is good evidence that high

288 mating rates impair ejaculate quality in the short-run (e.g. Wedell & Ritchie 2004) but
289 whether the repeated and/or sustained activation of the spermatogenesis machinery influences
290 fitness-related traits in late life is much less clear (Vega-Trejo *et al.* 2019), as are the
291 underlying mechanisms (Fricke & Koppik 2019). The degree to which mutations impact stem,
292 transit or sperm cells themselves during spermatogenesis will be key to the long-term
293 consequences of sperm allocation, and this in turn will partly depend on the hierarchical
294 organisation of cell division within the testis, i.e. on its tissue architecture, which varies
295 widely among species (see Schärer *et al.* 2008; Ramm & Schärer 2014). But as testicular stem
296 cell activity increases to support greater sperm production under sperm competition, the
297 burden of mutation and its manifestation in terms of reproductive ageing is predicted to
298 increase (Blumenstiel 2007). To date, little work has been done to measure inter- and intra-
299 specific variation in relevant parameters in response to sperm competition levels, but we
300 know that in the flatworm *Macrostomum lignano*, an increased stem cell activity and
301 corresponding faster speed of spermatogenesis support the enhanced sperm production that is
302 selected when sperm competition occurs (Schärer *et al.* 2004; Giannakara *et al.* 2016).
303 Moreover, recent evidence in seed beetles, *Callosobruchus maculatus*, suggests that the
304 higher level of sperm production under sperm competition directly compromises the ability of
305 the testis to maintain germline integrity, resulting in increased mutation loads being passed to
306 offspring (Baur & Berger 2020).

307 Second, a plausible route linking sperm production to reproductive ageing is that
308 increased gonadal activity might have direct detrimental consequences on male reproductive
309 traits through an increased production of reactive oxygen species (ROS) (Reinhardt 2007;
310 Monaghan & Metcalfe 2019) - a factor known to influence ageing patterns (Finkel &
311 Holbrook 2000). Thus, in rats, *Rattus norvegicus*, an increased sexual activity directly leads to
312 an elevated level of oxidative stress within the testis (Salomon *et al.* 2013). While repeatedly
313 increased level of oxidative stress might cause impaired testicular function and sperm
314 fertilization efficiency over time (Aitken & Baker 2006), and thereby increase reproductive
315 ageing, empirical studies investigating this topic are currently lacking.

316 A third consideration regarding long-term consequences of increased sperm production
317 concerns the fact that sperm competition has likely selected for the overexpression of genes
318 promoting cell proliferation, notably in the spermatogonia (Kleene 2005; Lewis *et al.* 2008).
319 While the expression of such genes might convey an important competitive advantage in
320 terms of ejaculate size and number, they are likely also to increase the risk of developing
321 testicular cancer (see Kleene 2005; Lewis *et al.* 2008 for reviews), which could badly impair

322 reproductive success in late life (Huddart *et al.* 2005). So far, these relationships have been
323 almost solely investigated in *Drosophila spp.* (Hime *et al.* 2007), even though several studies
324 are now documenting cancers across very diverse species (see Madsen *et al.* 2017;
325 Albuquerque *et al.* 2018). However, whether selection for enhanced sperm production under
326 sperm competition is associated with an increased risk of getting cancer (and most
327 specifically testicular cancer) at both intra- or inter-specific level is yet to be determined
328 (Lewis *et al.* 2008).

329 Finally, the maintenance of accurate and efficient physiological and cellular processes
330 throughout life is fundamental to our current understanding of ageing (Kirkwood 2017;
331 Maklakov & Chapman 2019). In most animals, males have to allocate a substantial quantity
332 of resources to mechanisms involved in the maintenance of the genome and proteome of the
333 pool of germline stem cells (Maklakov & Immler 2016), and we expect that these costs should
334 scale with sperm production capacity. These costs are probably far from negligible, since in
335 laboratory mice the activity of enzymes repairing DNA is much greater in germ cells than in
336 somatic cells (Intano *et al.* 2001). Moreover, among somatic cells, the enzymatic activity is
337 higher in Sertoli cells than in those from other tissues (e.g. liver, brain), highlighting again the
338 substantial maintenance costs associated with the male reproductive machinery (Intano *et al.*
339 2001). With increasing age, males suffer from an age-specific decline in germline
340 maintenance (Maklakov & Immler 2016), which might be responsible for an increased
341 mutation rate on sperm in old males. However, whether the quantity of resources devoted to
342 sperm competition limits the allocation to germline maintenance throughout life and
343 ultimately triggers accelerated reproductive ageing remains to be investigated (but see Baur &
344 Berger (2020) for a notable case study and Chen et al. (2020) for experimental evidence that
345 preventing allocation to the germline results in an enhanced ability for somatic repair).

346 Overall, although there are several well understood aspects of male reproductive ageing,
347 the extent to which a strong resource allocation to traits shaping sperm competition in early
348 life amplifies ageing and thus exacerbates the widely observed decrease of the male gonadal
349 size and function with increasing age (Lemaître & Gaillard 2017; Fricke & Koppik 2019;
350 Santiago *et al.* 2019) remains an open question. Answering it is of crucial importance, since
351 such effects might not only reinforce male reproductive ageing (e.g. through a decrease in
352 sperm quantity) but could also lead to deleterious consequences more generally for male
353 condition and health (Bribiescas 2006) – for example, through a decline in testosterone
354 production over the life course (see Yeap 2009) – which could ultimately impact lifespan and
355 survival ageing patterns (Bribiescas 2006, see next section). These interactions between

356 ageing in reproduction and survivorship might be even more complex as organisms have to
357 partition a limited resource pool between germline and somatic maintenance (Figure 1;
358 Maklakov & Immler 2016) and selection to maintain the integrity of the germline might be
359 particularly strong (Kirkwood 2005; Maklakov & Immler 2016). Hence, maintaining germline
360 integrity under sperm competition likely gets harder (as so many more sperm need to be
361 produced), and this may lead somatic maintenance (see Chen *et al.* 2020 for recent evidence
362 in vertebrates) and ultimately lifespan to be traded against sperm competition to get
363 immediate benefits in terms of reproductive output.
364
365 *Lifespan and survival ageing costs of sperm production.* Despite the accumulation of
366 evidence that a strong allocation to sperm production can entail acute physiological costs, its
367 long-term consequences in terms of lifespan or survival ageing have been largely overlooked.
368 Yet, some studies performed on insects have revealed that high mating rates can shorten male
369 lifespan (Partridge & Farquhar 1981; Martin & Hosken 2004; Paukku & Kotiaho 2005; Dao
370 *et al.* 2010; Metzler *et al.* 2016; Jehan *et al.* 2020), which might be due to the down-
371 regulation of the expression of some specific genes (e.g. metabolic genes) straight after sexual
372 activity (Branco *et al.* 2017). However, although sperm competition and mating rate are often
373 related (Parker & Ball 2005), mating rate depends also on the number of receptive females in
374 the population rather than purely the number of competing males, and so a high mating rate
375 alone does not constitute the most accurate proxy of a strong resource allocation to sperm
376 competition (Vahed & Parker 2012). The relative influence of sperm production and mating
377 rate *per se* on lifespan was disentangled in a pioneering experiment carried out on
378 *Caenorhabditis elegans* (Van Voorhies 1992). Males (and hermaphrodites) carrying a mutant
379 *spe-26* gene showed a reduced rate of sperm production but a 65% increased lifespan
380 compared to wild-type worms, but this gain was not amplified when mutant males were
381 prevented from mating, suggesting that the long-term fitness costs strictly associated with
382 sperm production are far from negligible (Van Voorhies 1992). Despite these promising
383 results, the survival costs of sperm production have remained largely understudied. Yet, in
384 large milkweed bug (*Oncopeltus fasciatus*), males fed with an ancestral seed diet show a
385 higher sperm production at the expense of a reduced lifespan (Duxbury *et al.* 2018).
386 Moreover, in domestic fowl (*Gallus gallus domesticus*), males that produce the highest
387 quantity of sperm through life show a reduced lifespan (Cornwallis *et al.* 2014) while in the
388 guppy (*Poecilia reticulata*), the opposite pattern occurs (Gasparini *et al.* 2019).

389 We thus emphasize that studies at the interplay of sexual selection and ageing should
390 now seek to accurately quantify these long-term survival costs (Figure 1), which would
391 strongly contribute to understanding the diversity of life history strategies observed across the
392 tree of life. Importantly, such studies should investigate whether the survival costs of an
393 increased allocation to sperm competition is mediated by an acceleration of ageing rather than
394 an immediate rise of the risk of death (Partridge & Andrews 1985). The phylogenetically-
395 controlled comparison of male actuarial senescence patterns across species displaying
396 contrasted mating systems or - more accurately - a high variation in the rate of multiple
397 paternity (e.g. Soulsbury 2010 in mammals) would provide complementary insights on the
398 role played by sperm competition in shaping lifespan and actuarial senescence patterns in
399 males. In addition, analyses performed at the population level will enable tests of whether
400 these long-term survival costs are a consequence of an earlier age at the onset of ageing, an
401 increased rate of ageing, or both (Péron *et al.* 2019).
402
403 **(b) Seminal fluid**
404 Male fertilization success is also modulated by the non-sperm components of the ejaculate
405 (Perry *et al.* 2013), with comparative (e.g. Ramm *et al.* 2005) and experimental (e.g.
406 Simmons *et al.* 2020) data pointing towards the importance of increased allocation to seminal
407 fluid under heightened levels of sperm competition. Interestingly, the female ageing cost of
408 repeated exposure to male seminal fluid content has been studied extensively (see box 1). By
409 contrast, whether the selection acting on the production of complex seminal fluid is associated
410 with long-term ageing costs in males has largely been overlooked.
411 Multiple lines of evidence suggest that seminal fluid production is energetically costly
412 (see also Table 2). Metabolic labelling studies in mice indicate that seminal fluid-producing
413 tissues exhibit high rates of protein turnover (Claydon *et al.* 2012). In some taxa, seminal
414 fluid allocation can reach extreme proportions. This is well illustrated in several species of
415 bushcrickets where a single water- and protein-rich spermatophore transferred to a female can
416 represent as much as 40% of male body mass (Vahed 2006), but also in butterflies where the
417 production of the sphragis (i.e. an external plug secreted by the male accessory glands and
418 fixed to the female abdomen to impair further the access to the female genitalia) by males can
419 constitute up to 20% of the male body mass (Carvalho *et al.* 2017). In line with these
420 observations, a recent meta-analysis highlighted that the production of seminal fluid show a
421 higher degree of condition-dependence than sperm production *per se* (Macartney *et al.* 2019).
422 Moreover, studies of accessory gland size in *Drosophila,* bedbugs and snails all suggest that

423 seminal fluid production may limit male mating rate (De Boer *et al.* 1997; Bangham *et al.*
424 2002; Reinhardt *et al.* 2011), implying that replenishment of seminal fluid protein reserves
425 after mating is a non-trivial cost to males. Finally, while the male competitive advantage of
426 producing large amounts of seminal fluid as well as the deleterious consequences for females
427 (see Box 1) have been well described in the literature (e.g. Wigby *et al.* 2009), the potential
428 toxic effects and associated ageing consequences to their repeated storage in male's gonad are
429 yet to be investigated.

430 Because males usually produce both sperm and seminal fluid simultaneously,
431 however, it is difficult to tease apart the relative costs of these two ejaculate components. This
432 difficulty is circumvented in red-sided garter snakes, *Thamnophis sirtalis parietalis*, in which
433 spermatogenesis and seminal fluid (copulatory plug) production occurs at different times of
434 the year. Whilst sperm are produced in the late summer and then stored over-winter, seminal
435 fluid production does not occur until the onset of the mating (and fasting) season in spring.
436 Friesen and colleagues (2015) took advantage of these biological features to establish that the
437 elevated metabolic rate associated with plug production are considerable, comparable even to
438 those incurred by pregnant females. They further showed that these costs are especially
439 pronounced in small males and, importantly, were able to quantify the energetic costs of
440 producing a single copulatory plug (up to 18% of the daily energy expenditure). Their
441 estimate reinforces earlier evidence of substantial mating costs based on the heightened blood
442 lactate levels observed in mating vs. only courting males (Shine *et al.* 2004). These high
443 energetic costs mean males can only deposit 1-2 plugs per day (Friesen *et al.* 2013) and again
444 clearly challenge the traditional idea that seminal fluid production is a trivial expense to
445 males. Whether the substantial costs of seminal fluid production translate into increased
446 ageing is currently unclear, due simply to a paucity of relevant data. However, Koppik and
447 colleagues (2018) recently discovered that, at old ages, sexually active male *D. melanogaster*
448 display shorter mating, resulting in a lower number of eggs laid by females than following
449 mating with virgin males. However, this pattern might be more likely due to a decrease in
450 sperm number rather than in the quantity of seminal fluid proteins delivered to females, when
451 sexually active males are reaching old ages (see Sepil *et al.* 2020).
452

453 **MITIGATION OF THE AGEING COSTS OF SPERM COMPETITION IN THE**
454 **WILD**

455 The evidence compiled above suggests that a strong allocation to traits associated with an
456 advantage when sperm competition occurs may be associated with some underestimated

457 fitness costs, such as a shortened lifespan or increased ageing. One might thus expect that
458 counter-adaptations have evolved to buffer the cascade of physiological costs and their fitness
459 consequences. We discuss below some of these possible adaptations and evaluate how much
460 they can mitigate fitness costs.
461
462 **(a) Phenotypic plasticity in ejaculate production**
463 Within species, sperm competition levels can fluctuate considerably across reproductive
464 seasons and among populations. One way to minimize the various physiological costs
465 potentially associated with elevated ejaculate expenditure would be to adjust ejaculate quality
466 and quantity to the level of sperm competition. Such fine-scale adjustments require the
467 evolution of phenotypic plasticity in the male reproductive machinery. Experimental
468 manipulations of the social environment faced during the developmental period reveal that
469 such plasticity might be common. For instance, in house mice (*Mus musculus*) males that
470 were repeatedly in contact with the odour of multiple unrelated (i.e. rival) males had an
471 elevated sperm production rate compared to males that were in contact to the odour of only a
472 single competitor (Ramm & Stockley 2009). A similar experimental design revealed that in
473 bank voles (*Myodes glareolus*), males who developed under environmental conditions
474 mimicking high levels of sperm competition developed larger seminal vesicles (but similar
475 testis mass) than males who developed under environmental conditions mimicking low levels
476 of sperm competition (Lemaitre *et al.* 2011). Overall, this suggests that the allocation to post-
477 copulatory traits is modulated in response to (social) environmental conditions, a
478 characteristic that is widely shared across animal species for various sperm and non-sperm
479 ejaculate traits (e.g. Immler *et al.* 2010 in birds; Stockley & Seal 2001; Simmons &
480 Lovegrove 2017; Sloan *et al.* 2018 in insects; Nakadera *et al.* 2019 in snails; Janicke *et al.*
481 2013; Giannakara *et al.* 2016; Ramm *et al.* 2019 in flatworms; Tan *et al.* 2004 in leeches and
482 Crean & Marshall 2008 in a broadcast spawning ascidian). Such fine-tuning to match the
483 prevailing demand – or in other words, minimizing the expenditure where possible – may thus
484 be one of the means through which males can limit the substantial physiological costs
485 associated with the high metabolic demand of spermatogenesis and seminal fluid production.
486 In line with this prediction, an experiment performed in *D. melanogaster* has revealed that
487 males reared with rivals produced more offspring during early life (in non-competitive trials)
488 but paid a long-term survival cost with a median lifespan reduced by 19% (Bretman *et al.*
489 2013), which suggests that curtailing ejaculate production in absence of competitors might
490 limit long-term ageing costs.

491

492 **(b) Context-dependent sperm allocation**

493 In addition to fine-tuning how many resources to expend on ejaculate production, a further
494 level of male allocation likely occurs with respect to how many sperm and/or how much
495 seminal fluid is transferred in each individual ejaculate. Thus, whilst traditionally assumed to
496 be cheap, it has now been recognised for some time that males should prudently allocate
497 ejaculate components according to relevant cues of sperm competition at the time of mating
498 (see Wedell *et al.* 2002; Cameron *et al.* 2007; Parker & Pizzari 2010; Perry *et al.* 2013 for
499 reviews). For example, it typically pays to allocate more sperm per ejaculate under conditions
500 of high sperm competition risk (e.g. delBarco-Trillo & Ferkin 2004), to allocate fewer per
501 ejaculate at high sperm competition intensity (e.g. Pilastro *et al.* 2002), to adjust the proteins
502 compounds of the seminal fluid according to the degree of sperm competition (Hopkins *et al.*
503 2019) and to modulate ejaculate size depending on own social status (e.g. Bartlett *et al.* 2017)
504 or to the male mating order and female reproductive status (Thomas & Simmons 2007).
505 Whilst these responses can be understood in terms of maximising fitness returns given a
506 certain gamete number (or seminal fluid amount) available to allocate, they ultimately stem
507 from the fact that the ejaculate supply is limited – one reason for which being that producing
508 both sperm and non-sperm ejaculate components likely imposes substantial costs, many of
509 which may ultimately increase the intensity of ageing.

510

511 **(c) Sperm production seasonality**

512 Besides using cues from their social environment to fine-tune sperm production and
513 allocation, males of many taxa also show clear seasonal variation in traits such as testis size
514 and sperm in storage, with peak production or availability often timed to coincide with a
515 specific breeding season (reviewed in Ramm & Schärer 2014). In male fallow deer, for
516 example, there is a three-fold increase in testis volume from its minimum to its peak just
517 before the annual rut (Gosch & Fischer 1989; for a broader survey across mammals, see
518 Setchell & Breed 2006). Even more marked variation is reported in other taxa with highly
519 seasonal reproductive seasons such as the annual variation observed in male (and female)
520 gonado-somatic index in many broadcast spawners (see Parker *et al.* 2018 and references
521 therein) and some fishes (e.g. Turner 1919). As discussed by Ramm and Schärer (2014), such
522 seasonal variation can be accompanied by specific features of spermatogenesis, such as the
523 long branch lengths (many rounds of mitotic division) observed for example in cichlids,
524 meaning thousands of spermatids are produced per primary spermatogonium, supporting the

525 delivery of a large number of sperm at a specific time of the year (Fishelson 2003). These and
526 other features emphasize the expensive nature of spermatogenesis. Moreover, as we have
527 seen, the fact that sperm production sometimes occurs asynchronously with the breeding
528 season – when energetic demand from other traits such as mate searching, pre-copulatory
529 competition and courtship also peaks - is probably also indicative of its high costs (Olsson *et*
530 *al.* 1997). However, we cannot rule out that in some species, such uncoupling might be a
531 constraint associated with prolonged periods of torpor (Gustafson 1979).
532 Overall, we expect that the 'savings' to be made from seasonal fluctuation in ejaculate
533 production will be highly species-specific and depend on its particular reproductive ecology,
534 but that matching supply and demand in this manner is a widespread mechanism to minimise
535 the energetic and other costs outlined above, all of which would otherwise ultimately
536 feedback on accelerated survival and/or reproductive ageing.
537
538 **ROADMAP FOR FUTURE RESEARCH**
539 Overall, studies that seek to quantify the long-term ageing cost of a strong allocation to sperm
540 competition and to decipher the underlying physiological pathways are thus badly required.
541 Such studies would undeniably enhance our knowledge in two main areas of evolutionary
542 ecology, namely sexual selection and ageing. We propose below a few potential avenues for
543 future research.
544
545 **(a) Do production plasticity, resource allocation and seasonality limit male**
546 **ageing?**
547 Whilst at some level it is clear that any reduction in ejaculate demand must ultimately be less
548 costly than the alternative, few studies have to date sought to use either intra- or interspecific
549 variation in, for example, the incidence of sperm competition or length of the breeding season
550 to better understand the nature and magnitude of these costs, or to quantify their impact on
551 ageing of fitness components. Comparative work in this area is likely to be highly
552 informative, and one of the most promising of all would be experimental approaches that seek
553 to modify one or more of these axes and then measure the ageing consequences. Potentially
554 useful approaches here might include experimental evolution to test for the correlated
555 evolution of ageing effects in lines selected for higher or lower allocation toward sperm
556 competition traits or under different intensity of sexual conflict. Recent experiments
557 performed in *D. melanogaster* have revealed that males from monogamous lines (i.e. without
558 sperm competition) reduce their allocation to various post-copulatory traits. For instance, they

559 show a lower expression of genes coding for seminal fluid proteins compared to males from
560 polygamous lines (Hollis *et al.* 2019). Investigating whether these males also display a
561 delayed and/or reduced reproductive and/or survival senescence patterns compared to males
562 evolving under competitive conditions would be very informative to assess whether natural
563 and sexual selection have fine-tuned the allocation to post-copulatory traits to limit its
564 detrimental ageing consequences.

565 Where experimentally feasible, the direct manipulation of relevant physiological
566 pathways involved in ejaculate production using gene knockdown or gene-editing techniques
567 would also be insightful. By thereby manipulating the capacity for plasticity or allocation, one
568 could more precisely identify the mechanisms involved in both short- and long-term fitness
569 consequences. Precisely such an approach was recently adopted to test for a trade-off between
570 germline and somatic maintenance in the zebrafish *Danio rerio* (Chen *et al.* 2020). Un-
571 manipulated and germline-free knockdown individuals were exposed to sublethal low-dose
572 ionizing radiation, revealing that somatic recovery occurred much faster in the germline-free
573 individuals that were prevented from allocating to reproduction (Chen *et al.* 2020). These
574 results thus support the idea that germline maintenance is costly. As we emphasized above,
575 these costs likely scale with sperm competition level. Finally, experimental manipulations of
576 the quantity of resources available in the environment would also provide relevant insights.
577 As emphasized above, the allocation to ejaculate production increases with resource
578 availability (Zhao *et al.* 2017; Ros-Santaella *et al.* 2019). Therefore, for a given level of
579 sperm competition perceived in the environment, we can predict that males should their
580 increase their allocation to post-copulatory competition, at no or very limited costs in terms of
581 ageing.

582

583 **(b) Ageing consequences of allocation to both pre- and post-copulatory traits in a**
584 **trade-off world**

585 As pointed out in the Introduction, male allocation to sexual competition is a complex process
586 encompassing both a pre- and post-copulatory phase (Parker 1998). While we have
587 demonstrated in this perspective article that allocation to post-copulatory competition is likely
588 to be associated with long-term ageing costs, substantial evidence that the competition for
589 gaining mating opportunities also shortens lifespan and accelerates ageing in terms of both
590 reproduction and survival has accumulated (e.g. Bonduriansky *et al.* 2008; Table 1). The
591 physiological pathways involved in mediating the relationships between the allocation to pre-
592 or post-copulatory competition on the one hand and those linking them to ageing on the other

593 are also likely to strongly overlap. For instance, in three-spined sticklebacks (*Gasterosteus*
594 *aculeatus*), males with an increased carotenoid-based red coloration on their cheeks and
595 throats show the strongest levels of oxidative DNA damage in both sperm and somatic tissues
596 (Kim & Velando 2020), which highlights that a resource-based allocation trade-off between
597 sexual selection and somatic maintenance mediates the long-term ageing costs of both pre-
598 and post-copulatory sexual competition. Interestingly, as allocation to both primary (e.g.
599 sperm-producing tissue within the testes) and secondary (e.g. ornaments and armaments)
600 sexually-selected traits relies on the quantity of resources available in the environment, males
601 cannot maximize both aspects of allocation to sexual competition simultaneously (Parker
602 1998; Parker *et al.* 2013). Evidence for trade-offs between pre- and post-copulatory traits are
603 widespread in nature, at both inter- and intra-specific levels (see Simmons *et al.* 2017 for a
604 review). For instance, across pinnipeds, the relative testes mass and the degree of sexual size
605 dimorphism are negatively correlated (Fitzpatrick *et al.* 2012). Overall, the resource-
606 allocation strategy towards pre- and post-copulatory competition is likely fine-tuned by the
607 fitness payoff of favoring one set of sexual traits at the expense of the other, which is likely to
608 result from complex interactions between species- or population-specific traits (e.g. mating
609 system, mating tactic) (Simmons *et al.* 2017) and environmental characteristics determining
610 the nature and degree of sexual competition (Vahed & Parker 2012). In practice, it is
611 particularly difficult to predict what aspects of sexual competition will predominantly drive
612 the physiological costs of sexual selection (e.g. Olsson *et al.* 1997 in the case of adders),
613 especially since the relative allocation to each sexual trait might change over the male's life
614 course (Lemaître & Gaillard 2017). Nevertheless, any thorough investigation of the ageing
615 costs of sexual selection should consider the cumulative costs associated with both aspects of
616 a male's allocation to sexual competition, which is lacking to date.
617
618 **(c) Contribution to the sex differences in lifespan and ageing across species**
619 In most species, males and females differ in lifespan and ageing patterns (Austad 2011;
620 Austad & Fischer 2016; Marais *et al.* 2018). While some factors (e.g. sex chromosomes)
621 provide relevant explanations for the average direction in sex differences in lifespan across
622 species (Xirocostas *et al.* 2020), the evolutionary roots of the large magnitude of sex
623 differences in lifespan and ageing patterns observed across species remain poorly understood
624 (see Lemaître *et al.* 2020 in mammals). So far, most comparative analyses that seek to explain
625 this diversity have focused on the role of sexual selection (e.g. Clutton-Brock & Isvaran
626 2007), with the expectation that sex differences in lifespan and ageing rates should increase in

627 species where males devote a substantial amount of resources to pre-copulatory competition
628 (generally measured through the degree of sexual size dimorphism, see Tidière *et al.* 2015).
629 However, these results remain largely equivocal and current evidence that the allocation to
630 pre-copulatory competition influences the evolution of sex differences in lifespan and ageing
631 in wild populations is limited at best (Lemaître *et al.* 2020). Here, we argue that comparative
632 analyses should carefully consider the long-term costs associated with post-copulatory
633 competition, which might also contribute to shaping the magnitude of sex differences in
634 lifespan and ageing patterns. Species displaying high levels of sperm competition should
635 allocate a much higher quantity of resources towards germline maintenance, at the expense of
636 somatic maintenance (Maklakov & Immler 2016). So far, these long-term costs have only
637 been considered in an analysis focused on large herbivores and in this study, species-specific
638 allocation to post-copulatory competition - measured by the relative testes mass - did not
639 influence the degree of sexual size dimorphism in lifespan (Lemaître & Gaillard 2013a).
640 However, the mean allocation to testis size is weak and shows little variation among large
641 herbivores compared to other taxa (Gomendio *et al.* 2011), and similar analyses need to be
642 performed using a broader taxonomic range. Moreover, such studies should fully embrace the
643 multi-dimensional nature of allocation to post-copulatory competition by considering not just
644 relative testes mass but also several other costly sperm features (e.g. size, velocity) and the
645 level of allocation to the seminal fluid component of the ejaculate. In addition, such inter-
646 specific studies should control for the basal metabolic rate, as species displaying a high mass-
647 specific metabolic rate can more easily allocate to high quality ejaculates (e.g. through the
648 production of particularly long sperm, see Gomendio *et al.* 2011). Lastly, such comparative
649 analyses should include the socio-ecological context. As shown above, the environmental
650 conditions that affect the amount of available food resources, the habitat heterogeneity that
651 influences spatial distribution of both sexes and the level of sexual segregation, and the
652 population size and structure that define the operational sex ratio and female group size
653 distribution should all determine the male investment corresponding to a given allocation to
654 traits that shape the intensity of post-copulatory competition, and thereby fine-tune this
655 allocation (Figure 1).

656 Species-specific differences in the level of sperm competition could also shape diverse
657 patterns of reproductive ageing in males (and thus various patterns of sex differences in
658 reproductive ageing). Interestingly, a comparative analysis performed across 18 species of
659 rodents maintained in lab conditions revealed a positive relationship between levels of sperm
660 competition and DNA fragmentation among sperm (delBarco-Trillo *et al.* 2016), which

suggests that inter-specific differences in the level of sperm competition can explain between-species differences in sperm integrity. Whether these differences are exacerbated with increasing age and occur in the wild is unknown but this clearly deserves attention. Recently, delBarco-Trillo and colleagues (2018) compared different post-copulatory traits (e.g. relative testes mass, various sperm features) between a small sample of young and old males for three species of rodents, all belonging to the genus *Mus* and displaying contrasting levels of sperm competition. While this transversal study did not reveal any clear difference in the age-specific changes in post-copulatory traits among these three species, it clearly set out a promising framework for future longitudinal studies comparing the age-specific decline in post-copulatory traits over the life course of individuals and across a wider range of species. The study of male reproductive ageing is currently attracting significant attention in evolutionary ecology (Lemaître & Gaillard 2017; Fricke & Koppik 2019; Monaghan & Metcalfe 2019) and such approaches would be particularly relevant to understand the evolutionary roots of male reproductive ageing.

ACKOWLEDGMENTS

J. -F. L. and J. -M. G. were supported by a grant from the Agence Nationale de la Recherche (ANR-15-CE32-0002-01). This work was performed within the framework of the LABEX ECOFECT (ANR-11-LABX-0048) of Université de Lyon, within the program ‘‘Investissements d’Avenir’’ (ANR-11-IDEX-0007) operated by the French National Research Agency (ANR). We are extremely thankful to Mathieu Douhard, Alexei Maklakov and Geoff Parker for insightful comments on earlier drafts of this work. We also thank three anonymous referees for insightful and constructive comments that markedly improved our manuscript.

REFERENCES

Aitken, R.J. & Baker, M.A. (2006). Oxidative stress, sperm survival and fertility control. *Molecular and cellular endocrinology*, 250, 66–69.

Albuquerque, T.A., Drummond do Val, L., Doherty, A. & de Magalhães, J.P. (2018). From humans to hydra: patterns of cancer across the tree of life. *Biological Reviews*, 93, 1715–1734.

Alonso-Alvarez, C., Bertrand, S., Devevey, G., Prost, J., Faivre, B. & Sorci, G. (2004). Increased susceptibility to oxidative stress as a proximate cost of reproduction. *Ecology*

Letters, 7, 363–368.
Anderson, M.J. & Dixson, A.F. (2002). Sperm competition: motility and the midpiece in primates. *Nature*, 416, 496.
Andersson, M. & Simmons, L.W. (2006). Sexual selection and mate choice. *Trends in ecology & evolution*, 21, 296–302.
Andersson, M.B. (1994). *Sexual selection*. Princeton University Press.
Arnqvist, G. & Rowe, L. (2005). *Sexual conflict*. Princeton University Press.
Austad, S.N. (2011). Sex differences in longevity and aging. In: *The Handbook of the Biology of Aging*. Academic Press, San Diego, pp. 479–496.
Austad, S.N. & Fischer, K.E. (2016). Sex differences in lifespan. *Cell metabolism*, 23, 1022–1033.
Austad, S.N. & Hoffman, J.M. (2018). Is Antagonistic Pleiotropy Ubiquitous in Aging Biology? *Evolution, Medicine, and Public Health*, 1, 287-294.
Avila, F.W., Ram, K.R., Qazi, M.C.B. & Wolfner, M.F. (2010). Sex peptide is required for the efficient release of stored sperm in mated Drosophila females. *Genetics*, 186, 595–600.
Bangham, J., Chapman, T. & Partridge, L. (2002). Effects of body size, accessory gland and testis size on pre-and postcopulatory success in Drosophila melanogaster. *Animal Behaviour*, 64, 915–921.
Bartlett, M.J., Steeves, T.E., Gemmell, N.J. & Rosengrave, P.C. (2017). Sperm competition risk drives rapid ejaculate adjustments mediated by seminal fluid. *Elife*, 6, e28811.
Bateman, A.J. (1948). Intra-sexual selection in Drosophila. *Heredity*, 2, 349–368.
Baur, J. & Berger, D. (2020). Experimental evidence for effects of sexual selection on condition-dependent mutation rates. *Nature Ecology & Evolution*, 1–8.
Birkhead, T.R. & Møller, A.P. (1998). *Sperm competition and sexual selection*. Elsevier.
Blumenstiel, J.P. (2007). Sperm competition can drive a male-biased mutation rate. *Journal of theoretical biology*, 249, 624–632.
Bonduriansky, R., Maklakov, A., Zajitschek, F. & Brooks, R. (2008). Sexual selection, sexual conflict and the evolution of ageing and life span. *Functional ecology*, 22, 443–453.
Boschetto, C., Gasparini, C. & Pilastro, A. (2011). Sperm number and velocity affect sperm competition success in the guppy (Poecilia reticulata). *Behavioral Ecology and Sociobiology*, 65, 813–821.
Bowen, W.D., Oftedal, O.T. & Boness, D.J. (1992). Mass and energy transfer during lactation in a small phocid, the harbor seal (*Phoca vitulina*). *Physiological Zoology*, 65, 844–866.
Branco, A.T., Schilling, L., Silkaitis, K., Dowling, D.K. & Lemos, B. (2017). Reproductive activity triggers accelerated male mortality and decreases lifespan: genetic and gene expression determinants in Drosophila. *Heredity*, 118, 221–228.
Brengdahl, M., Kimber, C.M., Maguire-Baxter, J., Malacrinò, A. & Friberg, U. (2018). Genetic quality affects the rate of male and female reproductive aging differently in Drosophila melanogaster. *The American Naturalist*, 192, 761–772.
Bretman, A. & Fricke, C. (2019). Exposure to males, but not receipt of sex peptide, accelerates functional ageing in female fruit flies. *Functional Ecology*, 33, 1459–1468.
Bretman, A., Westmancoat, J.D., Gage, M.J. & Chapman, T. (2013). Costs and benefits of lifetime exposure to mating rivals in male Drosophila melanogaster. *Evolution*, 67, 2413–2422.
Bribiescas, R.G. (2006). On the evolution, life history, and proximate mechanisms of human male reproductive senescence. *Evolutionary Anthropology: Issues, News, and Reviews: Issues, News, and Reviews*, 15, 132–141.
Briefer, E.F., Farrell, M.E., Hayden, T.J. & McElligott, A.G. (2013). Fallow deer polyandry is related to fertilization insurance. *Behavioral ecology and sociobiology*, 67, 657–665.

Brooks, R.C. & Garratt, M.G. (2017). Life history evolution, reproduction, and the origins of sex-dependent aging and longevity. *Annals of the New York Academy of Sciences*, 1389, 92–107.

Bunning, H., Rapkin, J., Belcher, L., Archer, C.R., Jensen, K. & Hunt, J. (2015). Protein and carbohydrate intake influence sperm number and fertility in male cockroaches, but not sperm viability. *Proceedings of the Royal Society of London B: Biological Sciences*, 282, 20142144.

Cameron, E., Day, T. & Rowe, L. (2007). Sperm competition and the evolution of ejaculate composition. *The American Naturalist*, 169, E158–E172.

Cardullo, R.A. & Baltz, J.M. (1991). Metabolic regulation in mammalian sperm: mitochondrial volume determines sperm length and flagellar beat frequency. *Cell motility and the cytoskeleton*, 19, 180–188.

Carvalho, A.P.S., Orr, A.G. & Kawahara, A.Y. (2017). A review of the occurrence and diversity of the sphragis in butterflies (Lepidoptera, Papilionoidea). *ZooKeys*, 41.

Chapman, T., Bangham, J., Vinti, G., Seifried, B., Lung, O., Wolfner, M.F., *et al.* (2003). The sex peptide of Drosophila melanogaster: female post-mating responses analyzed by using RNA interference. *Proceedings of the National Academy of Sciences*, 100, 9923–9928.

Chapman, T., Liddle, L.F., Kalb, J.M., Wolfner, M.F. & Partridge, L. (1995). Cost of mating in Drosophila melanogaster females is mediated by male accessory gland products. *Nature*, 373, 241–244.

Chen, H., Jolly, C., Bublys, K., Marcu, D. & Immler, S. (2020). Trade-off between somatic and germline repair in a vertebrate supports the expensive germ line hypothesis. *Proceedings of the National Academy of Sciences*.

Claydon, A.J., Ramm, S.A., Pennington, A., Hurst, J.L., Stockley, P. & Beynon, R. (2012). Heterogenous turnover of sperm and seminal vesicle proteins in the mouse revealed by dynamic metabolic labeling. *Molecular & Cellular Proteomics*, 11, 1-13.

Clutton-Brock, T. & Langley, P. (1997). Persistent courtship reduces male and female longevity in captive tsetse flies Glossina morsitans morsitans Westwood (Diptera: Glossinidae). *Behavioral Ecology*, 8, 392–395.

Clutton-Brock, T.H. & Isvaran, K. (2007). Sex differences in ageing in natural populations of vertebrates. *Proceedings of the Royal Society of London B: Biological Sciences*, 274, 3097–3104.

Cordts, R. & Partridge, L. (1996). Courtship reduces longevity of maleDrosophila melanogaster. *Animal Behaviour*, 52, 269–278.

Cornwallis, C.K., Dean, R. & Pizzari, T. (2014). Sex-specific patterns of aging in sexual ornaments and gametes. *The American Naturalist*, 184, E66–E78.

Crean, A.J. & Marshall, D.J. (2008). Gamete plasticity in a broadcast spawning marine invertebrate. *Proceedings of the National Academy of Sciences*, 105, 13508–13513.

Crudgington, H.S. & Siva-Jothy, M.T. (2000). Genital damage, kicking and early death. *Nature*, 407, 855–856.

Cunningham, K.A. & Beagley, K.W. (2008). Male genital tract chlamydial infection: implications for pathology and infertility. *Biology of reproduction*, 79, 180–189.

Dao, A., Kassogue, Y., Adamou, A., Diallo, M., Yaro, A.S., Traore, S.F., *et al.* (2010). Reproduction-longevity trade-off in Anopheles gambiae (Diptera: Culicidae). *Journal of medical entomology*, 47, 769–777.

Dávila, F. & Aron, S. (2017). Protein restriction affects sperm number but not sperm viability in male ants. *Journal of Insect Physiology*, 100, 71–76.

De Boer, P., Jansen, R.F., Koene, J.M. & Ter Maat, A. (1997). Nervous control of male sexual drive in the hermaphroditic snail Lymnaea stagnalis. *Journal of Experimental*

Biology, 200, 941–951.
Dewsbury, D.A. (1982). Ejaculate cost and male choice. *The American Naturalist*, 119, 601–610.
Dewsbury, D.A. (2005). The Darwin-Bateman paradigm in historical context. *Integrative and Comparative Biology*, 45, 831–837.
Duxbury, A.E., Weathersby, B., Sanchez, Z. & Moore, P.J. (2018). A study of the transit amplification divisions during spermatogenesis in Oncopetus fasciatus to assess plasticity in sperm numbers or sperm viability under different diets. *Ecology and evolution*, 8, 10460–10469.
Eberhard, W.G. (1985). *Sexual selection and animal genitalia*. Harvard University Press Cambridge, MA.
Edward, D.A. & Chapman, T. (2011). Mechanisms underlying costs of reproduction.
Evans, J., Wilson, A., Pilastro, A. & Garcia-Gonzalez, F. (2019). Ejaculate-mediated paternal effects: evidence, mechanisms and evolutionary implications. *Reproduction*, 157,R109-R126.
Finkel, T. & Holbrook, N.J. (2000). Oxidants, oxidative stress and the biology of ageing. *Nature*, 408, 239.
Fishelson, L. (2003). Comparison of testes structure, spermatogenesis, and spermatocytogenesis in young, aging, and hybrid cichlid fish (Cichlidae, Teleostei). *Journal of Morphology*, 256, 285–300.
Fitzpatrick, J.L., Almbro, M., Gonzalez-Voyer, A., Kolm, N. & Simmons, L.W. (2012). Male contest competition and the coevolution of weaponry and testes in pinnipeds. *Evolution: International Journal of Organic Evolution*, 66, 3595–3604.
Fitzpatrick, J.L. & Lüpold, S. (2014). Sexual selection and the evolution of sperm quality. *Molecular Human Reproduction*, 20, 1180–1189.
Fitzpatrick, J.L., Montgomerie, R., Desjardins, J.K., Stiver, K.A., Kolm, N. & Balshine, S. (2009). Female promiscuity promotes the evolution of faster sperm in cichlid fishes. *Proceedings of the National Academy of Sciences*, 106, 1128–1132.
Flatt, T. & Partridge, L. (2018). Horizons in the evolution of aging. *BMC Biol.*, 16, 93.
Flatt, T. & Promislow, D.E. (2007). Still pondering an age-old question. *Science*, 318, 1255–1256.
Folstad, I. & Karter, A.J. (1992). Parasites, bright males, and the immunocompetence handicap. *The American Naturalist*, 139, 603–622.
Franceschi, C., Bonafè, M., Valensin, S., Olivieri, F., De Luca, M., Ottaviani, E., *et al.* (2000). Inflamm-aging. An evolutionary perspective on immunosenescence. *Annals of the New York Academy of Sciences*, 908, 244.
Fricke, C. & Koppik, M. (2019). Male reproductive ageing-a tale of the whole ejaculate. *Reproduction*, 158, R219-R229.
Friesen, C.R., Powers, D.R., Copenhaver, P.E. & Mason, R.T. (2015). Size dependence in non-sperm ejaculate production is reflected in daily energy expenditure and resting metabolic rate. *Journal of Experimental Biology*, 218, 1410–1418.
Friesen, C.R., Shine, R., Krohmer, R.W. & Mason, R.T. (2013). Not just a chastity belt: the functional significance of mating plugs in garter snakes, revisited. *Biological Journal of the Linnean Society*, 109, 893–907.
Gage, M.J., Macfarlane, C.P., Yeates, S., Ward, R.G., Searle, J.B. & Parker, G.A. (2004). Spermatozoal traits and sperm competition in Atlantic salmon: relative sperm velocity is the primary determinant of fertilization success. *Current Biology*, 14, 44–47.
Gage, M.J. & Morrow, E.H. (2003). Experimental evidence for the evolution of numerous, tiny sperm via sperm competition. *Current Biology*, 13, 754–757.
Gaillard, J.-M. & Lemaître, J.-F. (2017). The Williams' legacy: A critical reappraisal of his

nine predictions about the evolution of senescence. *Evolution*, 71, 2768–2785.
Gaillard, J.-M. & Lemaître, J.-F. (2020). An integrative view of senescence in nature. *Functional Ecology*, 34, 4–16.
Gasparini, C., Devigili, A. & Pilastro, A. (2019). Sexual selection and ageing: interplay between pre-and post-copulatory traits senescence in the guppy. *Proceedings of the Royal Society B*, 286, 20182873.
Gasparini, C., Simmons, L.W., Beveridge, M. & Evans, J.P. (2010). Sperm swimming velocity predicts competitive fertilization success in the green swordtail *Xiphophorus helleri*. *PloS one*, 5.
Gems, D. & Partridge, L. (2013). Genetics of longevity in model organisms: debates and paradigm shifts. *Annual Review of Physiology*, 75, 621–644.
Giannakara, A., Schärer, L. & Ramm, S.A. (2016). Sperm competition-induced plasticity in the speed of spermatogenesis. *BMC Evolutionary Biology*, 16, 60.
Giraudeau, M., Friesen, C.R., Sudyka, J., Rollings, N., Whittington, C.M., Wilson, M.R., *et al.* (2016). Ageing and the cost of maintaining coloration in the Australian painted dragon. *Biology Letters*, 12, 20160077.
Gomendio, M., Tourmente, M. & Roldan, E.R. (2011). Why mammalian lineages respond differently to sexual selection: metabolic rate constrains the evolution of sperm size. *Proceedings of the Royal Society of London B: Biological Sciences*, 278, 3135-3141.
Gosch, B. & Fischer, K. (1989). Seasonal changes of testis volume and sperm quality in adult fallow deer (*Dama dama*) and their relationship to the antler cycle. *Reproduction*, 85, 7–17.
Gu, N.-H., Zhao, W.-L., Wang, G.-S. & Sun, F. (2019). Comparative analysis of mammalian sperm ultrastructure reveals relationships between sperm morphology, mitochondrial functions and motility. *Reproductive Biology and Endocrinology*, 17, 66.
Gustafson, A.W. (1979). Male reproductive patterns in hibernating bats. *Reproduction*, 56, 317–331.
Hayward, A. & Gillooly, J.F. (2011). The cost of sex: quantifying energetic investment in gamete production by males and females. *PLoS One*, 6, e16557.
Hime, G.R., Loveland, K.L. & Abud, H.E. (2007). Drosophila spermatogenesis: insights into testicular cancer. *International journal of andrology*, 30, 265–274.
Hollis, B., Koppik, M., Wensing, K.U., Ruhmann, H., Genzoni, E., Erkosar, B., *et al.* (2019). Sexual conflict drives male manipulation of female postmating responses in Drosophila melanogaster. *Proceedings of the National Academy of Sciences*, 116, 8437–8444.
Hooper, A.K., Lehtonen, J., Schwanz, L.E. & Bonduriansky, R. (2018). Sexual competition and the evolution of condition-dependent ageing. *Evolution Letters*, 2, 37–48.
Hopkins, B.R., Sepil, I., Thézénas, M.-L., Craig, J.F., Miller, T., Charles, P.D., *et al.* (2019). Divergent allocation of sperm and the seminal proteome along a competition gradient in Drosophila melanogaster. *Proceedings of the National Academy of Sciences*, 116, 17925–17933.
Hosken, D.J. (2001). Sex and death: microevolutionary trade-offs between reproductive and immune investment in dung flies. *Current Biology*, 11, R379–R380.
Huddart, R.A., Norman, A., Moynihan, C., Horwich, A., Parker, C., Nicholls, E., *et al.* (2005). Fertility, gonadal and sexual function in survivors of testicular cancer. *British Journal of Cancer*, 93, 200–207.
Hunt, J., Brooks, R., Jennions, M.D., Smith, M.J., Bentsen, C.L. & Bussiere, L.F. (2004). High-quality male field crickets invest heavily in sexual display but die young. *Nature*, 432, 1024.
Immler, S., Pryke, S.R., Birkhead, T.R. & Griffith, S.C. (2010). Pronounced within-individual plasticity in sperm morphometry across social environments. *Evolution: International*

Journal of Organic Evolution, 64, 1634–1643.
Intano, G.W., McMahan, C.A., Walter, R.B., McCarrey, J.R. & Walter, C.A. (2001). Mixed spermatogenic germ cell nuclear extracts exhibit high base excision repair activity. *Nucleic acids research*, 29, 1366–1372.
Janicke, T., Häderer, I.K., Lajeunesse, M.J. & Anthes, N. (2016). Darwinian sex roles confirmed across the animal kingdom. *Science advances*, 2, e1500983.
Janicke, T., Marie-Orleach, L., De Mulder, K., Berezikov, E., Ladurner, P., Vizoso, D.B., *et al.* (2013). Sex allocation adjustment to mating group size in a simultaneous hermaphrodite. *Evolution*, 67, 3233–3242.
Jehan, C., Chogne, M., Rigaud, T. & Moret, Y. (2020). Sex-specific patterns of senescence in artificial insect populations varying in sex-ratio to manipulate reproductive effort. *BMC evolutionary biology*, 20, 18.
Jennions, M.D. & Petrie, M. (2000). Why do females mate multiply? A review of the genetic benefits. *Biological Reviews*, 75, 21–64.
Johnson, S.L. & Gemmell, N.J. (2012). Are old males still good males and can females tell the difference? Do hidden advantages of mating with old males off-set costs related to fertility, or are we missing something else? *Bioessays*, 34, 609–619.
Jones, O.R., Scheuerlein, A., Salguero-Gómez, R., Camarda, C.G., Schaible, R., Casper, B.B., *et al.* (2014). Diversity of ageing across the tree of life. *Nature*, 505, 169.
Kenagy, G.J. & Trombulak, S.C. (1986). Size and function of mammalian testes in relation to body size. *Journal of Mammalogy*, 67, 1–22.
Kennedy, B.K., Berger, S.L., Brunet, A., Campisi, J., Cuervo, A.M., Epel, E.S., *et al.* (2014). Geroscience: linking aging to chronic disease. *Cell*, 159, 709–713.
Kerr, A.M., Gershman, S.N. & Sakaluk, S.K. (2010). Experimentally induced spermatophore production and immune responses reveal a trade-off in crickets. *Behavioral Ecology*, 21, 647–654.
Kim, S.-Y. & Velando, A. (2020). Attractive male sticklebacks carry more oxidative DNA damage in the soma and germline. *Journal of evolutionary biology*, 33, 121–126.
Kirkwood, T.B. (1977). Evolution of ageing. *Nature*, 270, 301.
Kirkwood, T.B. (2005). Understanding the odd science of aging. *Cell*, 120, 437–447.
Kirkwood, T.B. (2017). The Disposable Soma Theory. In: *The evolution of senescence in the tree of life*. p. 23.
Kirkwood, T.B. & Rose, M.R. (1991). Evolution of senescence: late survival sacrificed for reproduction. *Phil. Trans. R. Soc. Lond. B*, 332, 15–24.
Kleene, K.C. (2005). Sexual selection, genetic conflict, selfish genes, and the atypical patterns of gene expression in spermatogenic cells. *Developmental biology*, 277, 16–26.
Kong, A., Frigge, M.L., Masson, G., Besenbacher, S., Sulem, P., Magnusson, G., *et al.* (2012). Rate of de novo mutations and the importance of father's age to disease risk. *Nature*, 488, 471.
Kowald, A. & Kirkwood, T.B. (2015). Evolutionary significance of ageing in the wild. *Experimental gerontology*, 71, 89–94.
Lemaître, J.-F., Berger, V., Bonenfant, C., Douhard, M., Gamelon, M., Plard, F., *et al.* (2015). Early-late life trade-offs and the evolution of ageing in the wild. *Proc. R. Soc. B*. 282, 20150209.
Lemaître, J.-F., Cheynel, L., Douhard, F., Bourgoin, G., Débias, F., Ferté, H., *et al.* (2018). The influence of early-life allocation to antlers on male performance during adulthood: Evidence from contrasted populations of a large herbivore. *Journal of Animal Ecology*, 87, 921–932.
Lemaître, J.-F. & Gaillard, J.-M. (2013a). Male survival patterns do not depend on male allocation to sexual competition in large herbivores. *Behavioral Ecology*, 24, 421–428.

Lemaître, J.-F. & Gaillard, J.-M. (2013b). Polyandry Has No Detectable Mortality Cost in Female Mammals. *PLoS ONE*, 8, e66670.

Lemaître, J.-F. & Gaillard, J.-M. (2017). Reproductive senescence: new perspectives in the wild: Reproductive senescence in the wild. *Biological Reviews*, 92, 2182–2199.

Lemaître, J.-F., Gaillard, J.-M., Pemberton, J.M., Clutton-Brock, T.H. & Nussey, D.H. (2014). Early life expenditure in sexual competition is associated with increased reproductive senescence in male red deer. *Proceedings of the Royal Society of London B: Biological Sciences*, 281, 20140792.

Lemaitre, J.-F., Ramm, S.A., Hurst, J.L. & Stockley, P. (2011). Social cues of sperm competition influence accessory reproductive gland size in a promiscuous mammal. *Proceedings of the Royal Society B: Biological Sciences*, 278, 1171–1176.

Lemaître, J.-F., Ronget, V., Tidière, M., Allainé, D., Berger, V., Cohas, A., *et al.* (2020). Sex differences in adult lifespan and aging rates of mortality across wild mammals. *Proceedings of the National Academy of Sciences*, 117, 8546–8553.

Lewis, Z., Price, T.A.R. & Wedell, N. (2008). Sperm competition, immunity, selfish genes and cancer. *Cellular and molecular life sciences*, 65, 3241.

Liljedal, S., Folstad, I. & Skarstein, F. (1999). Secondary sex traits, parasites, immunity and ejaculate quality in the Arctic charr. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 266, 1893–1898.

López-Otín, C., Blasco, M.A., Partridge, L., Serrano, M. & Kroemer, G. (2013). The Hallmarks of Aging. *Cell*, 153, 1194–1217.

Lüpold, S., Linz, G.M., Rivers, J.W., Westneat, D.F. & Birkhead, T.R. (2009). Sperm competition selects beyond relative testes size in birds. *Evolution*, 63, 391–402.

Macartney, E.L., Crean, A.J., Nakagawa, S. & Bonduriansky, R. (2019). Effects of nutrient limitation on sperm and seminal fluid: a systematic review and meta-analysis. *Biological Reviews*, 94, 1722–1739.

Madsen, T., Arnal, A., Vittecoq, M., Bernex, F., Abadie, J., Labrut, S., *et al.* (2017). Cancer prevalence and etiology in wild and captive animals. In: *Ecology and evolution of cancer*. Elsevier, pp. 11–46.

Maklakov, A.A. & Chapman, T. (2019). Evolution of ageing as a tangle of trade-offs: energy versus function. *Proceedings of the Royal Society B*, 286, 20191604.

Maklakov, A.A. & Immler, S. (2016). The expensive germline and the evolution of ageing. *Current Biology*, 26, R577–R586.

Marais, G., Gaillard, J.-M., Vieira, C., Plotton, I., Sanlaville, D., Gueyffier, F., *et al.* (2018). Sex-specific differences in aging and longevity: can sex chromosomes play a role? *Biology of Sex Differences*, 9, 33.

Martin, O.Y. & Hosken, D.J. (2004). Copulation reduces male but not female longevity in Saltella sphondylli (Diptera: Sepsidae). *Journal of Evolutionary Biology*, 17, 357–362.

McKean, K.A. & Nunney, L. (2001). Increased sexual activity reduces male immune function in Drosophila melanogaster. *Proceedings of the National Academy of Sciences*, 98, 7904–7909.

McNamara, K.B., Van Lieshout, E., Jones, T.M. & Simmons, L.W. (2013). Age-dependent trade-offs between immunity and male, but not female, reproduction. *Journal of Animal Ecology*, 82, 235–244.

Medawar, P.B. (1952). *An unsolved problem of biology*. College.

Metcalf, C.J.E., Roth, O. & Graham, A.L. (2020). Why leveraging sex differences in immune tradeoffs may illuminate the evolution of senescence. *Functional Ecology*, 34, 129-140.

Metzler, S., Heinze, J. & Schrempf, A. (2016). Mating and longevity in ant males. *Ecology and Evolution*, 6, 8903–8906.

Monaghan, P. & Metcalfe, N.B. (2019). The deteriorating soma and the indispensable

germline: gamete senescence and offspring fitness. *Proc. R. Soc. Lond. B*, 286, 20192187.

Nakadera, Y., Giannakara, A. & Ramm, S.A. (2019). Plastic expression of seminal fluid protein genes in a simultaneously hermaphroditic snail. *Behavioral Ecology*, 30, 904–913.

Nakatsuru, K. & Kramer, D.L. (1982). Is sperm cheap? Limited male fertility and female choice in the lemon tetra (Pisces, Characidae). *Science*, 216, 753–755.

Nunn, C.L., Gittleman, J.L. & Antonovics, J. (2000). Promiscuity and the primate immune system. *Science*, 290, 1168–1170.

Nussey, D.H., Froy, H., Lemaitre, J.-F., Gaillard, J.-M. & Austad, S.N. (2013). Senescence in natural populations of animals: widespread evidence and its implications for bio-gerontology. *Ageing research reviews*, 12, 214–225.

Olsson, M., Madsen, T. & Shine, R. (1997). Is sperm really so cheap? Costs of reproduction in male adders, Vipera berus. *Proceedings of the Royal Society of London B: Biological Sciences*, 264, 455–459.

Orr, T.J. & Brennan, P.L. (2016). All features great and small—the potential roles of the baculum and penile spines in mammals. *Integrative and comparative biology*, 56, 635–643.

Papadopoulos, N.T., Liedo, P., Müller, H.-G., Wang, J.-L., Molleman, F. & Carey, J.R. (2010). Cost of reproduction in male medflies: the primacy of sexual courting in extreme longevity reduction. *Journal of Insect Physiology*, 56, 283–287.

Parker, G.A. (1970). Sperm competition and its evolutionary consequences in the insects. *Biological Reviews*, 45, 525–567.

Parker, G.A. (1998). Sperm competition and the evolution of ejaculates: towards a theory base. *Sperm competition and sexual selection*, 3–54.

Parker, G.A. (2016). The evolution of expenditure on testes. *Journal of Zoology*, 298, 3–19.

Parker, G.A. & Ball, M.A. (2005). Sperm competition, mating rate and the evolution of testis and ejaculate sizes: a population model. *Biology Letters*, 1, 235–238.

Parker, G.A., Lessells, C.M. & Simmons, L.W. (2013). Sperm competition games: a general model for precopulatory male–male competition. *Evolution*, 67, 95–109.

Parker, G.A. & Pizzari, T. (2010). Sperm competition and ejaculate economics. *Biological Reviews*, 85, 897–934.

Parker, G.A., Ramm, S.A., Lehtonen, J. & Henshaw, J.M. (2018). The evolution of gonad expenditure and gonadosomatic index (GSI) in male and female broadcast-spawning invertebrates. *Biological Reviews*, 93, 693–753.

Partridge, L. & Andrews, R. (1985). The effect of reproductive activity on the longevity of male Drosophila melanogaster is not caused by an acceleration of ageing. *Journal of Insect Physiology*, 31, 393–395.

Partridge, L. & Barton, N.H. (1993). Optimally, mutation and the evolution of ageing. *Nature*, 362, 305.

Partridge, L. & Farquhar, M. (1981). Sexual activity reduces lifespan of male fruitflies. *Nature*, 294, 580–582.

Paukku, S. & Kotiaho, J.S. (2005). Cost of reproduction in Callosobruchus maculatus: effects of mating on male longevity and the effect of male mating status on female longevity. *Journal of Insect Physiology*, 51, 1220–1226.

Péron, G., Lemaître, J.-F., Ronget, V., Tidière, M. & Gaillard, J.-M. (2019). Variation in actuarial senescence does not reflect life span variation across mammals. *PLoS biology*, 17, e3000432.

Perry, J.C. & Rowe, L. (2010). Condition-dependent ejaculate size and composition in a ladybird beetle. *Proceedings of the Royal Society B: Biological Sciences*, 277, 3639–

3647.
Perry, J.C., Sirot, L. & Wigby, S. (2013). The seminal symphony: how to compose an ejaculate. *Trends in Ecology & Evolution*, 28, 414–422.
Peters, A., Delhey, K., Denk, A.G. & Kempenaers, B. (2004). Trade-offs between immune investment and sexual signaling in male mallards. *The American Naturalist*, 164, 51–59.
Pilastro, A., Scaggiante, M. & Rasotto, M.B. (2002). Individual adjustment of sperm expenditure accords with sperm competition theory. *Proceedings of the National Academy of Sciences*, 99, 9913–9915.
Pizzari, T. & Parker, G.A. (2009). Sperm competition and sperm phenotype. In: *Sperm biology*. Elsevier, pp. 207–245.
Poiani, A. (2006). Complexity of seminal fluid: a review. *Behavioral Ecology and Sociobiology*, 60, 289–310.
Polak, M. (1998). Effects of ectoparasitism on host condition in the *Drosophila-Macrocheles* system. *Ecology*, 79, 1807.
Preston, B.T., Jalme, M.S., Hingrat, Y., Lacroix, F. & Sorci, G. (2011). Sexually extravagant males age more rapidly. *Ecology Letters*, 14, 1017–1024.
Promislow, D. (2003). Mate choice, sexual conflict, and evolution of senescence. *Behavior genetics*, 33, 191–201.
Prowse, N. & Partridge, L. (1997). The effects of reproduction on longevity and fertility in male Drosophila melanogaster. *Journal of Insect Physiology*, 43, 501–512.
Radhakrishnan, P. & Fedorka, K.M. (2012). Immune activation decreases sperm viability in both sexes and influences female sperm storage. *Proceedings of the Royal Society B: Biological Sciences*, 279, 3577–3583.
Ramm, S.A., Lengerer, B., Arbore, R., Pjeta, R., Wunderer, J., Giannakara, A., *et al.* (2019). Sex allocation plasticity on a transcriptome scale: Socially sensitive gene expression in a simultaneous hermaphrodite. *Molecular ecology*, 28, 2321–2341.
Ramm, S.A., Parker, G.A. & Stockley, P. (2005). Sperm competition and the evolution of male reproductive anatomy in rodents. *Proceedings of the Royal Society of London B: Biological Sciences*, 272, 949–955.
Ramm, S.A. & Schärer, L. (2014). The evolutionary ecology of testicular function: size isn't everything. *Biological Reviews*, 89, 874–888.
Ramm, S.A., Schärer, L., Ehmcke, J. & Wistuba, J. (2014). Sperm competition and the evolution of spermatogenesis. *Molecular Human Reproduction*, 20, 1169–1179.
Ramm, S.A. & Stockley, P. (2009). Adaptive plasticity of mammalian sperm production in response to social experience. *Proceedings of the Royal Society B: Biological Sciences*, 276, 745–751.
Ramm, S.A. & Stockley, P. (2010). Sperm competition and sperm length influence the rate of mammalian spermatogenesis. *Biology Letters*, 6, 219–221.
Reinhardt, K. (2007). Evolutionary consequences of sperm cell aging. *The Quarterly review of biology*, 82, 375–393.
Reinhardt, K., Naylor, R. & Siva-Jothy, M.T. (2011). Male mating rate is constrained by seminal fluid availability in bedbugs, Cimex lectularius. *PloS one*, 6.
Richard, M., Massot, M., Clobert, J. & Meylan, S. (2012). Litter quality and inflammatory response are dependent on mating strategy in a reptile. *Oecologia*, 170, 39–46.
Rödel, H.G., Zapka, M., Stefanski, V. & Holst, D. von. (2016). Reproductive effort alters immune parameters measured post-partum in European rabbits under semi-natural conditions. *Functional Ecology*, 30, 1800–1809.
Rodríguez-Muñoz, R., Boonekamp, J.J., Liu, X.P., Skicko, I., Fisher, D.N., Hopwood, P., *et al.* (2019). Testing the effect of early-life reproductive effort on age-related decline in a wild insect. *Evolution*, 73, 317–328.

Rolff, J. & Siva-Jothy, M.T. (2002). Copulation corrupts immunity: a mechanism for a cost of mating in insects. *Proceedings of the National Academy of Sciences*, 99, 9916–9918.

Ros-Santaella, J.L., Kotrba, R. & Pintus, E. (2019). High-energy diet enhances spermatogenic function and increases sperm midpiece length in fallow deer (Dama dama) yearlings. *Royal Society open science*, 6, 181972.

Salomon, T.B. & Benfato, M.S. (2018). Sexual activity affects the redox profile along the aging process in male rats. *Biogerontology*, 19, 13–21.

Salomon, T.B., Hackenhaar, F.S., Almeida, A.C., Schüller, A.K., Alabarse, P.V.G., Ehrenbrink, G., *et al.* (2013). Oxidative stress in testis of animals during aging with and without reproductive activity. *Experimental gerontology*, 48, 940–946.

Santiago, J., Silva, J.V., Alves, M.G., Oliveira, P.F. & Fardilha, M. (2019). Testicular aging: an overview of ultrastructural, cellular, and molecular alterations. *The Journals of Gerontology: Series A*, 74, 860–871.

Schärer, L., Da Lage, J.-L. & Joly, D. (2008). Evolution of testicular architecture in the Drosophilidae: a role for sperm length. *BMC evolutionary biology*, 8, 143.

Schärer, L., Ladurner, P. & Rieger, R.M. (2004). Bigger testes do work more: experimental evidence that testis size reflects testicular cell proliferation activity in the marine invertebrate, the free-living flatworm Macrostomum sp. *Behavioral Ecology and Sociobiology*, 56, 420–425.

Sella, G. & Lorenzi, M.C. (2003). Increased sperm allocation delays body growth in a protandrous simultaneous hermaphrodite. *Biological Journal of the Linnean Society*, 78, 149–154.

Sepil, I., Hopkins, B.R., Dean, R., Bath, E., Friedman, S., Swanson, B., *et al.* (2020). Male reproductive aging arises via multifaceted mating-dependent sperm and seminal proteome declines, but is postponable in Drosophila. *PNAS.*

Setchell, B. & Breed, W. (2006). Anatomy, vasculature, and innervation of the male reproductive tract. Elsevier.

Sheldon, B.C. (1993). Sexually transmitted disease in birds: occurrence and evolutionary significance. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 339, 491–497.

Sheldon, B.C. & Verhulst, S. (1996). Ecological immunology: costly parasite defences and trade-offs in evolutionary ecology. *Trends in ecology & evolution*, 11, 317–321.

Shine, R., Phillips, B., Langkilde, T., Lutterschmidt, D.I., Waye, H. & Mason, R.T. (2004). Mechanisms and consequences of sexual conflict in garter snakes (Thamnophis sirtalis, Colubridae). *Behavioral Ecology*, 15, 654–660.

Simmons, L.W. (2012). Resource allocation trade-off between sperm quality and immunity in the field cricket, Teleogryllus oceanicus. *Behavioral Ecology*, 23, 168–173.

Simmons, L.W. & Lovegrove, M. (2017). Socially cued seminal fluid gene expression mediates responses in ejaculate quality to sperm competition risk. *Proceedings of the Royal Society B: Biological Sciences*, 284, 20171486.

Simmons, L.W., Lüpold, S. & Fitzpatrick, J.L. (2017). Evolutionary trade-off between secondary sexual traits and ejaculates. *Trends in ecology & evolution.*

Simmons, L.W. & Roberts, B. (2005). Bacterial immunity traded for sperm viability in male crickets. *Science*, 309, 2031–2031.

Simmons, L.W., Sloan, N.S. & Firman, R.C. (2020). Sexual Selection Shapes Seminal Vesicle Secretion Gene Expression in House Mice. *Molecular Biology and Evolution*, 37, 1114–1117.

Skau, P.A. & Folstad, I. (2005). Does immunity regulate ejaculate quality and fertility in humans? *Behavioral Ecology*, 16, 410–416.

Sloan, N.S., Lovegrove, M. & Simmons, L.W. (2018). Social manipulation of sperm

1147 competition intensity reduces seminal fluid gene expression. *Biology letters*, 14,
1148 20170659.
1149 Snook, R.R. (2005). Sperm in competition: not playing by the numbers. *Trends in ecology &*
1150 *evolution*, 20, 46–53.
1151 Soulsbury, C.D. (2010). Genetic patterns of paternity and testes size in mammals. *PLoS One*,
1152 5, e9581.
1153 South, S.H., Steiner, D. & Arnqvist, G. (2009). Male mating costs in a polygynous mosquito
1154 with ornaments expressed in both sexes. *Proceedings of the Royal Society B: Biological*
1155 *Sciences*, 276, 3671–3678.
1156 Stockley, P. (1997). Sexual conflict resulting from adaptations to sperm competition. *Trends*
1157 *in Ecology & Evolution*, 12, 154–159.
1158 Stockley, P. & Seal, N.J. (2001). Plasticity in reproductive effort of male dung flies
1159 (Scatophaga stercoraria) as a response to larval density. *Functional Ecology*, 96–102.
1160 Stürup, M., Baer, B. & Boomsma, J.J. (2014). Short independent lives and selection for
1161 maximal sperm survival make investment in immune defences unprofitable for leaf-
1162 cutting ant males. *Behavioral ecology and sociobiology*, 68, 947–955.
1163 Tan, G.N., Govedich, F.R. & Burd, M. (2004). Social group size, potential sperm competition
1164 and reproductive investment in a hermaphroditic leech, Helobdella papillornata
1165 (Euhirudinea: Glossiphoniidae). *Journal of evolutionary biology*, 17, 574–580.
1166 Thomas, M.L. & Simmons, L.W. (2007). Male crickets adjust the viability of their sperm in
1167 response to female mating status. *The American Naturalist*, 170, 190–195.
1168 Thomsen, R., Soltis, J., Matsubara, M., Matsubayashi, K., Onuma, M. & Takenaka, O.
1169 (2006). How costly are ejaculates for Japanese macaques? *Primates*, 47, 272–274.
1170 Thrall, P.H., Antonovics, J. & Bever, J.D. (1997). Sexual transmission of disease and host
1171 mating systems: within-season reproductive success. *The American Naturalist*, 149,
1172 485–506.
1173 Tidière, M., Gaillard, J.-M., Müller, D.W., Lackey, L.B., Gimenez, O., Clauss, M., *et al.*
1174 (2015). Does sexual selection shape sex differences in longevity and senescence
1175 patterns across vertebrates? A review and new insights from captive ruminants.
1176 *Evolution*, 69, 3123–3140.
1177 Tourmente, M., Gomendio, M. & Roldan, E.R. (2011). Sperm competition and the evolution
1178 of sperm design in mammals. *BMC Evolutionary Biology*, 11, 12.
1179 delBarco-Trillo, J. & Ferkin, M.H. (2004). Male mammals respond to a risk of sperm
1180 competition conveyed by odours of conspecific males. *Nature*, 431, 446.
1181 delBarco-Trillo, J., García-Álvarez, O., Soler, A.J., Tourmente, M., Garde, J.J. & Roldan,
1182 E.R.S. (2016). A cost for high levels of sperm competition in rodents: increased sperm
1183 DNA fragmentation. *Proc Biol Sci*, 283.
1184 Trivers, R.L. (1972). *Parental investment and sexual selection.[W:] B. Campbell (red.)*
1185 *Sexual selection and the descent of man, 1871-1971*. Chicago: Aldine.
1186 Turner, C.L. (1919). *The seasonal cycle in the spermary of the perch*.
1187 Vahed, K. (2006). Larger ejaculate volumes are associated with a lower degree of polyandry
1188 across bushcricket taxa. *Proceedings of the Royal Society B: Biological Sciences*, 273,
1189 2387–2394.
1190 Vahed, K. & Parker, D.J. (2012). The evolution of large testes: sperm competition or male
1191 mating rate? *Ethology*, 118, 107–117.
1192 Van Voorhies, W.A. (1992). Production of sperm reduces nematode lifespan. *Nature*, 360,
1193 456–458.
1194 Vega-Trejo, R., Fox, R.J., Iglesias-Carrasco, M., Head, M.L. & Jennions, M.D. (2019). The
1195 effects of male age, sperm age and mating history on ejaculate senescence. *Functional*
1196 *Ecology*.

1197 Velando, A., Noguera, J.C., Drummond, H. & Torres, R. (2011). Senescent males carry
1198 premutagenic lesions in sperm. *Journal of evolutionary biology*, 24, 693–697.
1199 Vilchez, D., Saez, I. & Dillin, A. (2014). The role of protein clearance mechanisms in
1200 organismal ageing and age-related diseases. *Nature communications*, 5, 5659.
1201 Vinogradov, A.E. (1998). Male reproductive strategy and decreased longevity. *Acta*
1202 *biotheoretica*, 46, 157–160.
1203 Voigt, C.C., Michener, R. & Kunz, T.H. (2005). The energetics of trading nuptial gifts for
1204 copulations in katydids. *Physiological and Biochemical Zoology*, 78, 417–423.
1205 Wedell, N. (2010). Variation in male courtship costs in butterflies. *Behavioral ecology and*
1206 *sociobiology*, 64, 1385–1391.
1207 Wedell, N., Gage, M.J.G. & Parker, G.A. (2002). Sperm competition, male prudence and
1208 sperm-limited females. *Trends in Ecology & Evolution*, 17, 313–320.
1209 Wedell, N. & Ritchie, M.G. (2004). Male age, mating status and nuptial gift quality in a
1210 bushcricket. *Animal Behaviour*, 67, 1059–1065.
1211 Wigby, S. & Chapman, T. (2005). Sex peptide causes mating costs in female Drosophila
1212 melanogaster. *Current Biology*, 15, 316–321.
1213 Wigby, S., Sirot, L.K., Linklater, J.R., Buehner, N., Calboli, F.C., Bretman, A., *et al.* (2009).
1214 Seminal fluid protein allocation and male reproductive success. *Current Biology*, 19,
1215 751–757.
1216 Williams, G.C. (1957). Pleiotropy, natural selection, and the evolution of senescence.
1217 *Evolution*, 11, 398–411.
1218 Xirocostas, Z.A., Everingham, S.E. & Moles, A.T. (2020). The sex with the reduced sex
1219 chromosome dies earlier: a comparison across the tree of life. *Biology Letters*, 16,
1220 20190867.
1221 Yasukawa, K. (1987). Breeding and nonbreeding season mortality of territorial male red-
1222 winged blackbirds (*Agelaius phoeniceus*). *The Auk*, 104, 56–62.
1223 Yeap, B.B. (2009). Testosterone and ill-health in aging men. *Nature clinical practice*
1224 *Endocrinology & metabolism*, 5, 113–121.
1225 Zhao, J., Jin, Y., Du, M., Liu, W., Ren, Y., Zhang, C., *et al.* (2017). The effect of dietary
1226 grape pomace supplementation on epididymal sperm quality and testicular antioxidant
1227 ability in ram lambs. *Theriogenology*, 97, 50–56.
1228

1229

1230 **BOX 1: Sperm competition influences ageing in females**

1231

1232 In contrast to males, the consequences of sperm competition for female lifespan and ageing
1233 have been well studied. By aiming to answer the question "*why do females mate with multiple*
1234 *males in most species?*", behavioural ecologists have provided compelling evidence that
1235 polyandry (i.e. the female propensity to mate with several males and thus to incite sperm
1236 competition among males) is associated with both benefits and costs (Jennions & Petrie
1237 2000). In males, sperm competition has led to the evolution of many behavioural, anatomical
1238 and physiological adaptations that increase the probability to fertilize a female's eggs
1239 (Birkhead & Møller 1998). However, several of these adaptations can have detrimental effects
1240 on female reproductive success and survival (reviewed in Stockley 1997; Arnqvist & Rowe
1241 2005), notably in late life. Among these adaptations, the role played by male seminal fluid
1242 proteins constitutes an iconic example.
1243 Seminal fluid proteins can have various detrimental effects for females (Poiani 2006)
1244 and those are particularly well studied in the fruit fly, *D. melanogaster*. In this species, the
1245 repeated exposure to seminal fluid proteins transferred during copulation increases female
1246 death rate (Chapman *et al.* 1995). More specifically, the decrease in female survival prospects
1247 appears to be predominantly caused by the sex-peptide (Wigby & Chapman 2005) - a protein
1248 produced by the male accessory glands - that enhances male reproductive success in a
1249 competitive context by reducing the degree of female receptivity and increasing sperm storage
1250 by females (Chapman *et al.* 2003; Avila *et al.* 2010). The female-specific deleterious effects
1251 of male sex peptide on lifespan and late-life reproductive success are mediated by several
1252 physiological pathways, notably through a rise in the release of juvenile hormones (Edward &
1253 Chapman 2011). Strikingly, it has been recently shown that - independently of the transfer of
1254 sex peptide - both mating and the amount of exposure to males not only decrease female
1255 survival but also increase the degree of physiological ageing (measured by the age-specific
1256 decline in climbing activity and starvation resistance) (Bretman & Fricke 2019). These results
1257 strongly emphasize the complexity of the interactions between mating rates, physiological
1258 processes and both reproductive and survival ageing in females.
1259 While the production of seminal fluid proteins in response to sperm competition and
1260 its consequence for female's ageing are well studied - at least in flies - studying other
1261 instances of sexual conflict over mating and fertilization might shed interesting light on the
1262 evolution of demographic ageing in females. This is well illustrated by the very peculiar
1263 genitalia that have evolved to improve sperm transfer abilities, and more generally,
1264 fertilization efficiency in many species (Eberhard 1985) and that are already known to
1265 influence female survival in the short run (e.g. Crudgington & Siva-Jothy 2000). Across
1266 mammals, the level of polyandry (measured by the species-specific relative testes mass) does
1267 not seem to increase the female rate of ageing in survivorship (Lemaître & Gaillard 2013b).
1268 However, keratinized spines found at the surface of the penis of several mammalian species
1269 (e.g. some primates, rodents and felids) can be responsible for lesions of the female
1270 reproductive tract (Orr & Brennan 2016), which could ultimately compromise the late-life
1271 reproductive success of the most polyandrous females.
1272 Finally, high levels of polyandry are associated with a higher risk of contracting
1273 sexually transmitted diseases (Thrall *et al.* 1997) that can impair reproductive success and
1274 survival in the long-run (Sheldon 1993). To counteract these adverse effects, polyandrous
1275 females have evolved immune systems that might mitigate these fitness costs through an
1276 increased resistance to infections (e.g. Nunn *et al.* 2000 in primates). This is well illustrated in
1277 lizards (*Zootoca vivipara*) where polyandrous females show a greater ability to mount an
1278 inflammatory immune response than monandrous females (Richard *et al.* 2012). While a

1279 greater allocation of resources to immune defences in polyandrous females is likely to convey
1280 fitness benefits at a short-time scale, they might also lead to an overall increased rate of
1281 ageing through an over-activation of the immune system (e.g. inflammaging) (Franceschi *et*
1282 *al.* 2000). Yet, how much this and many other consequences of male allocation to sperm
1283 competition contribute to the observed differences in female ageing at both inter- and intra-
1284 specific levels is yet to be determined.
1285

1286 **Table 1: Evidence of long-term lifespan and ageing costs associated to an increased allocation to pre-copulatory competition. Such**
1287 **relationships have been assessed across different types of populations (L: laboratory, W: wild populations, SC: semi-captive**
1288 **populations).**

1289

Taxon	Common name		Population	Measure of allocation to pre-copulatory competition[1]	Late-life trait impacted	Reference
Insect	Mediterranean fruit fly	*Ceratitis capitata*	L	Courtship effort	Lifespan	Papadopoulos *et al.* 2010
Insect	Fruit fly	*Drosophila melanogaster*	L	Courtship effort	Lifespan	Cordts & Partridge 1996
Insect	Tsetse fly	*Glossina morsitans morsitans*	L	Courtship effort	Lifespan	Clutton-Brock & Langley 1997
Insect	Field cricket	*Gryllus campestris*	W	Searching activity	Rate of reproductive senescence[2]	Rodríguez-Muñoz *et al.* 2019
Insect	Green-veined white	*Pieris napi*	L	Courtship effort	Lifespan	Wedell 2010
Insect	Mosquito	*Sabethes cyaneus*	L	Courtship effort	Lifespan	South *et al.* 2009
Insect	Black field cricket	*Teleogryllus commodus*	L	Calling effort	Lifespan	Hunt *et al.* 2004
Bird	Red-winged Blackbird	*Agelaius phoeniceus*	W	Wing length	Lifespan	Yasukawa 1987
Bird	Houbara bustard	*Chlamydotis undulata*	SC	Sexual display effort	Rate of reproductive senescence[3]	Preston *et al.* 2011
Mammal	Red deer	*Cervus elaphus*	W	Harem size and rutting duration	Rate of reproductive senescence[4]	Lemaître *et al.* 2014

1290

1291 [1] Articles linking horn length (or horn growth) to lifespan in bovids were deliberately omitted because these studies have been subjected to some controversies, due to the
1292 possible confounding effects of trophy hunting (see Lemaître *et al.* 2018 for a compilation of these studies).
1293 [2] Reproductive senescence measured by the age-specific decline in calling activity.
1294 [3] Reproductive senescence measured by the age-specific decline in ejaculate quality.
1295 [4] Reproductive senescence measured by the age-specific decline in harem size.
1296
1297

1298

1299 **Table 2: Compiled evidence of physiological costs associated to an increased allocation to sperm competition. This table reports the**
1300 **relationships that have been established between the allocation to an ejaculate-related trait (e.g. sperm viability) and the impaired**
1301 **physiological function (e.g. body growth, immune activity). Such relationships have been assessed across different types of populations**
1302 **(L: laboratory, WC/L: wild-caught population brought to the laboratory, W: wild populations).**
1303

Taxon	Species		Population	Ejaculate-related trait	Physiological trait	Reference
Polychaeta		*Ophryotrocha diadema*	L	Sperm allocation and replenishment	Body growth	Sella & Lorenzi 2003
Insect	Fruitfly	*Drosophila melanogaster*	L	Sperm viability	Immune activity	Radhakrishnan & Fedorka 2012
Insect	Cotton bollworm	*Helicoverpa armigera*	L	Sperm allocation	Immune activity	McNamara *et al.* 2013[1]
Insect	Field cricket	*Teleogryllus oceanicus*	L	Sperm viability	Lysozyme activity	Simmons & Roberts 2005
Insect	Tropical house cricket	*Gryllodes sigillatus*	WC/L	Spermatophore size and production rate	Immune activity	Kerr *et al.* 2010
Insect	Gemeine Plumpschrecke	*Isophya kraussi*	WC/L	Spermatophore production	Energy expenditure	Voigt *et al.* 2005
Insect	Leaf-cutting ant	*Atta colombica*	WC/L	Sperm viability	Immune activity	Stürup *et al.* 2014
Insect	Leaf-cutting ant	*Acromyrmex echinatior*	WC/L	Sperm viability	Immune activity	Stürup *et al.* 2014
Insect	Mealworm	*Tenebrio molitor*	L	Sexual activity	Immune activity	Rolff & Siva-Jothy 2002[2]
Mammal	Japanese macaque	*Macaca fuscata*	W & L	Ejaculate production	Basal metabolic rate	Thomsen *et al.* 2006
Mammal	Wistar rats	*Rattus norvegicus*	L	Sexual activity	Age-specific redox profile	Salomon & Benfato 2018
Mammal	Wistar rats	*Rattus norvegicus*	L	Sexual activity	Oxidative damage in the testes	Salomon *et al.* 2013[3]
Fish	Arctic charr	*Salvelinus alpinus*	WC/L	Spermatocrit	Granulocyte density	Liljedal *et al.* 1999
Fish	Arctic charr	*Salvelinus alpinus*	WC/L	Spermatocrit	Spleen mass	Liljedal *et al.* 1999
Fish	Arctic charr	*Salvelinus alpinus*	WC/L	Sperm motility	Granulocyte density	Liljedal *et al.* 1999
Reptile	Red-sided garter snake	*Thamnophis sirtalis parietalis*	WC/L	Production of copulatory plugs	Energy expenditure / Resting metabolic rate	Friesen *et al.* 2015
Reptile	Red-sided garter snake	*Thamnophis sirtalis parietalis*	WC/L	Number of copulations	Body mass loss	Friesen *et al.* 2015
Reptile	Adder	*Vipera berus*	W	Testes growth	Body mass loss	Olsson *et al.* 1997

1304 [1] The sperm allocation is used as a proxy of sperm production.

1305 [2] The activity of the phenoloxidase is reduced but there was no effect on the hemocyte load.
1306 [3] Damages found on proteins (carbonylation levels) but sexual activity of males shows an increase in anti-oxidant defences that could probably buffer oxidative costs.

CAPTION FOR FIGURES
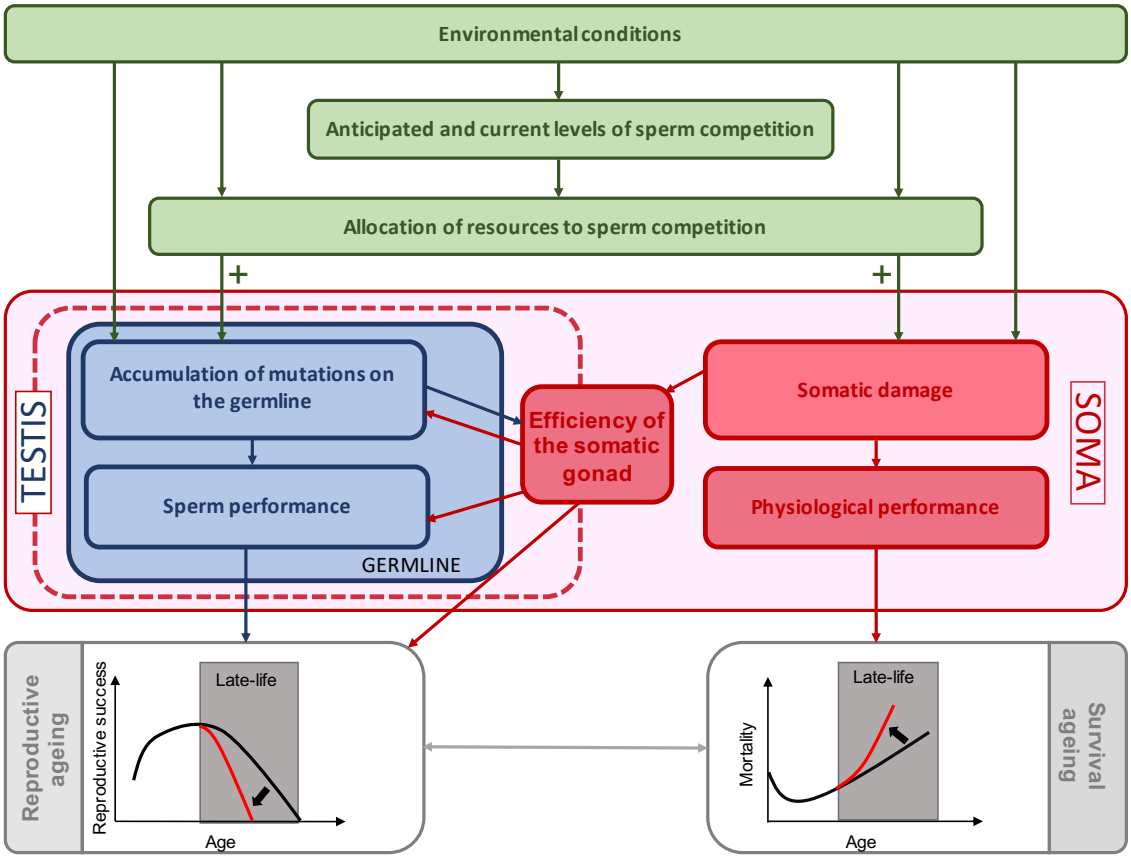
Figure 1: Diagram of the functional relationships linking the allocation of resources to sperm competition traits with increased ageing in both reproduction and survivorship (i.e. denoted by the shift from the black to the red trajectories on the ageing curves). While some environmental conditions (e.g. pollution) can directly increase the mutation load on both germline and somatic tissues, they also largely modulate the socio-environmental context (e.g. adult sex ratio) that determines the level of sperm competition within a population. These ageing consequences are likely to be mediated by multiple physiological pathways leading to a progressive deterioration of the germline (e.g. through an accumulation of mutations on sperm DNA) and the soma (e.g. a decrease in the efficiency of the somatic gonad and an impairment of some key physiological functions such as immunity).