Evolution of female choice and age-dependent male traits with paternal germ-line mutation

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Several studies question the adaptive value of female preferences for older males. Theory and evidence show that older males carry more deleterious mutations in their sperm than younger males carry. These mutations are not visible to females choosing mates. Germ-line mutations could oppose preferences for “good genes.” Choosy females run the risk that offspring of older males will be no more attractive or healthy than offspring of younger males. Germ-line mutations could pose a particular problem when females can only judge male trait size, rather than assessing age directly. I ask whether or not females will prefer extreme traits, despite reduced offspring survival due to age-dependent mutation. I use a quantitative genetic model to examine the evolution of female preferences, an age-dependent male trait, and overall health (“condition”). My dynamical equation includes mutation bias that depends on the generation time of the population. I focus on the case where females form preferences for older males because male trait size depends on male age. My findings agree with good genes theory. Females at equilibrium always select above-average males. The trait size preferred by females directly correlates with the direct costs of the preference. Direct costs can accentuate the equilibrium preference at a higher rate than mutational parameters. Females can always offset direct costs by mating with older, more ornamented males. Age-dependent mutation in condition maintains genetic variation in condition and thereby maintains the selective value of female preferences. Rather than eliminating female preferences, germ-line mutations provide an essential ingredient in sexual selection.

I. INTRODUCTION

Sexual selection theory yields predictions for evolution of extravagant signals in males and preferences in females. Researchers categorize signals into two categories: (1) traits that signal direct benefits, i.e. choosy females themselves gain higher fitness; or (2) traits that signal indirect (genetic) benefits to females. Choosy females incur viability or fecundity costs during mate choice, but matings yield higher offspring fitness (Andersson, 1994; Jones and Ratterman, 2009a). Costs to females may occur in the form of exposure to predators, pathogens and parasites, or reduced chances of mating. Research on indirect benefits often focuses on costly preferences for traits that signal heritable condition of trait-bearing males (“condition-dependent” traits; Andersson [1986]. As the name suggests, the size of condition-dependent traits depends directly on the health of the male carrying the trait. Thus females can produce healthy offspring by exercising preferences for extreme values of condition-dependent traits. If selection were to act unchecked by mutation, genetic variation in condition of males would disappear, since the healthiest males survive best and gain most matings. A female in this situation optimally mates randomly, since all males carry the same genes for overall health. Selection would act against females expressing costly choices, since they gain no net benefits (the “lek paradox”; Kirkpatrick and Ryan [1991], Rowe and Houle [1996]. Selection maintaining female preferences therefore requires biased mutation in condition, i.e. repeated appearance of deleterious mutations in male offspring (Iwasa et al., 1991).

Another line of research focusing on male traits suggests that males benefit from age-dependent investment in their signals (Kokko [1997, 1998]. Proulx et al. (2002) showed that a male’s optimal life-history strategy depends on his condition: high-condition males will conserve resources and signal less at young ages, then increase their signaling into old age. Lower-condition males optimally signal at a high rate when they are young, since they have less chance of surviving to old age. These studies predict that females evolve preferences for older-aged males. Theory and empirical work throughout the history of research on sexual selection support the hypothesis that selection favors preferences for older- or middle-aged males (Brooks and Kemp [2001]). I previously showed that reduced adult mortality and age-dependent signals promote sexual selection by reducing selection against costly traits (Adamson [2013]). When male traits start out small, selection cannot eliminate traits that grow large later in life. Long-term data sets in mammals support this hypothesis by showing increased heritability and greater marginal benefits of sexually selected traits later in life (Courtiol et al. [2012], Pemberton et al. [2004], Poissant et al. [2008]).

Reduced adult mortality and age-dependent expression thus favor preferences for older males. This prediction seems to provide a convenient explanation for older-male preferences.
However, [Hansen and Price (1995)] raised four objections to the feasibility of selection favoring older-male preferences. Among these objections was the substantial number of deleterious mutations in the paternal germ-line (Bartosch-Härild et al., 2003; Ellegren, 2007; Goefting-Minesky and Makova, 2006; Hansen and Price, 1999; Kong et al., 2012; Möller and Cuervo, 2003; Radwan, 2003; Sayres et al., 2011). Spermato
gonia, the stem cells that mature into spermatocytes, undergo frequent mitotic divisions during the reproductive life of a male animal. Mutations in sperm cells increase with male age, even when mutation rate remains constant over the lifespan. These germ-line mutations negatively affect the heritability of male condition. Males who survive into attractive old age could yield offspring who do not survive well (see Hansen and Price, 1995, who showed this could be true even without germ-line mutations). Germ-line mutations could therefore negatively impact the evolution of costly male preferences. Maintenance of preferences depends on the genetic correlation between preferences and condition (Iwasa et al., 1991; Mead and Arnold, 2004). If females with strong preferences give rise to mutation-ridden offspring with lowered condition, preferences could correlate weakly or even negatively with condition.

Here I ask whether or not selection will maintain costly female preferences for older males when (1) females can only assess trait size; and (2) a male’s mutational contribution depends on his age. I focus on cases where trait size explicitly depends on age. Will a population under sexual selection achieve equilibria with females expressing directional sexual preferences, rather than favoring average or optimal males? When age-dependent traits and age-dependent mutation occur together, females preferring above-average traits could select males yielding less fit, mutant offspring. Natural selection could therefore eliminate costly female preferences. I will show, using a quantitative genetic model of preferences, traits and condition, that the process supplying necessary mutation bias (germ-line mutation) coincides with the process facilitating sexual selection (age-dependent traits). Selection maintains female preference for male traits in this model, including cases with preferences for older males. Age-dependent paternal mutation reinforces the evolution of costly female preferences, rather than hindering it.

II. MODEL

A. Phenotypic and population model

I will explore the above questions using the evolutionary dynamics of mean phenotypes (Lande, 1982). Individuals in the large population express a phenotype composed of a set of characters with non-overlapping genetic components (i.e. no pleiotropy). I assume that environmental variance has mean 0 and therefore ignore it when considering the evolution of mean phenotypes. The above considerations imply the equality of genetic and phenotypic variances, covariances and correlations (Iwasa et al., 1991). Quantitative genetic models typically ignore the effects of epistasis and dominance for simplicity, as I do here. I wish to isolate the effects of selection and mutation and therefore ignore the potential effects of drift and migration.

The phenotype consists of three traits: (1) a growth parameter \( \kappa \), (2) a female preference \( p \) and (3) intrinsic viability or “condition” \( C \) (Iwasa et al., 1991) used a similar model of condition-dependent traits. Males of condition \( C \) and growth parameter \( \kappa \) express the signaling trait \( s \) as a function of age \( x \):

\[
s(x) = \theta + C \kappa x
\]

where \( x \) represents age and \( \theta \) represents the optimal trait value for viability selection. Note that all juvenile males produce the optimal trait size.

Only females express the preference \( p \) and females display the trait at the optimum \( \theta \). Females express a preference independent of age and condition. A unimodal function expresses the relative frequency of matings of males with phenotype \( s \) to females of phenotype \( p \):

\[
\phi(s|p) = \exp\left(-\frac{(s-p)^2}{2\sigma^2}\right)
\]

where \( \sigma \) represents the width (“standard deviation”) of the preference function (Lande, 1981). Smaller values of \( \sigma \) indicate preferences more tightly concentrated around \( p \). Females have the highest relative frequency of mating with males whose size \( s \) matches their preferred value \( p \).

Phenotypic condition \( C \) remains constant throughout an individual’s life, but an individual male’s germ-line genotypic value (breeding value) of \( C \) decays according to the linear function

\[
\mu(x) = -\mu x
\]

where \( \mu \) represents a constant, phenotypically standardized mutational effect (e.g. one could measure \( \mu \) in units of \( \theta \)). A male of age \( x \) on average delivers genetic value \( C - \mu x \) to his offspring.

I assume that the population grows exponentially according to

\[
\frac{dN}{dt} = rN \quad \text{(4)}
\]

where \( r \) represents the largest real root in \( r \) of

\[
\int_0^\infty e^{-rx}I(x)m(x)dx = 1. \quad \text{(5)}
\]

The hazard function \( I(x) \) represents survival to age \( x \), \( m(x) \) represents fecundity at age \( x \) and \( \omega \) symbolizes the oldest age in the population. I assume these vital rates remain roughly constant over short periods of little phenotypic change (as in Lande, 1982). I use \textit{generation time}

\[
T = \int_0^\infty xe^{-rx}I(x)m(x)dx \quad \text{(6)}
\]
as a parameter expressing the basic structure of the life-history. Readers can also conceptualize $T$ as the average age of breeding adults. Small values of $T$ represent populations of individuals with relatively short lives, whereas larger values of $T$ represent populations of individuals with longer lifespans. Equation (3) holds at stable age distribution, hence I assume weak selection. By weak selection, I mean that any disturbance of stable age distribution caused by change in the mean phenotype will quickly converge before more phenotypic change occurs. For example, if mean condition increases, survival to old age could also increase, taking the population temporarily out of stable age distribution. Then I assume that the age distribution will restabilize within a few generations, before mean condition changes again and brings about the next demographic disturbance. Therefore I can assume the population remains in stable age distribution over the course of evolutionary change (see Charlesworth 1993, Iwasa et al. 1991 and others have derived similar equations).

The mating system follows a polygynous model. Males gain total male fitness by multiplying viability by mating success. Viability selection on females depends on condition, preference value, and the average male signaling trait:

$$W_f = \exp \left( -\frac{b(p - \bar{s})^2}{2\sigma^2} \right).$$

I assume that female fecundity varies independently of age and condition, and that all females (across the distribution of $C$) gain the same average mating success during any time interval $\Delta t$. Female fecundity as a function of condition could seem more realistic, but my goal is to isolate the effects of costly female preferences. I therefore assume that female fitness varies only due to viability costs generated by mate choice.

I calculate selection gradients by differentiating the natural logarithm of total fitness of males and females with respect to specific phenotypic traits and evaluating these partial derivatives at the population mean for the trait, with age set to the generation time:

$$\frac{\partial \log W_m}{\partial s} = -\kappa x^2 - \frac{C x}{\sigma^2} \int_{-\infty}^{\infty} (s - p) f(p) \phi(s|p)dp$$

I calculate the covariance of $\theta$ with itself at age $x$, and with condition, and that all females (across the distribution of $C$) gain the same average mating success during any time interval $\Delta t$. Female fecundity as a function of condition could seem more realistic, but my goal is to isolate the effects of costly female preferences. I therefore assume that female fitness varies only due to viability costs generated by mate choice.

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$$U(s) = \int_{-\infty}^{\infty} f(p) \phi(s|p)dp$$

## B. Fitness

Viability selection on the trait $s$ at age $x$ follows the Gaussian function

$$w(s, C) = \exp \left( -\frac{(s(x) - \theta)^2}{2\sigma^2} \right)$$

such that males carrying $s$ smaller and larger than $\theta$ suffer viability costs, dependent on the condition of the bearer. The fitness of males with higher $C$ will slope off more gradually than that of males with lower $C$ (Kotiaho 2001). I calculate total male fitness by multiplying viability by mating success. The mating system follows a polygynous model. Males gain mating success

$$W_m = \exp \left( -\frac{(x \theta)^2}{2} \right) \int_{-\infty}^{\infty} f(p) \phi(s|p)dp. \tag{11a}$$

during any short time interval $\Delta t$.

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I calculate selection gradients by differentiating the natural logarithm of total fitness of males and females with respect to specific phenotypic traits and evaluating these partial derivatives at the population mean for the trait, with age set to the generation time:

$$\frac{\partial \log W_m}{\partial \kappa} = -\kappa x^2 - \frac{C x}{\sigma^2} \int_{-\infty}^{\infty} (s - p) f(p) \phi(s|p)dp$$

$$\frac{\partial \log W_f}{\partial p} = -\frac{b(p - \bar{s})}{C^2}$$

$$\frac{\partial \log W_m}{\partial C} = \frac{1}{C^3} \int_{-\infty}^{\infty} (s - p)^2 f(p) \phi(s|p)dp$$

$$\frac{\partial \log W_f}{\partial C} = \frac{b(p - \bar{s})}{C^3}.$$
Accordingly I obtain the covariances between the trait \((s)\) and condition and growth:

\[
G_{sk} = \tau T G_{\kappa}, \quad (14a) \\
G_{sc} = \kappa T G_{C}. \quad (14b)
\]

Growth and trait always positively covary. The males with the largest traits in the population will tend to have the largest growth coefficients. Equation (14b) can become negative only if \(\kappa < 0\). Increasing generation time will increase the absolute value of these covariances in both cases. This accords with the higher variance in trait sizes afforded by longer generation time. Longer generation time means more old males in the population, hence more large traits and larger variances.

III. RESULTS

A. Selection gradient

The selection gradient illustrates the basic direction and magnitude of evolutionary change in the three phenotypic characters. The largest component of character change comes from the multiplication of the selection gradient for each character \((\beta_i)\) with its variance \((G_i)\). I refer to all other effects in any equation as “indirect.” Therefore looking directly at the selection gradient shows the basic structure of the model:

\[
\beta_{\kappa} = \frac{\partial \log W_m}{\partial \kappa} |_{x,T} = T \left( \frac{C(p - \bar{p})}{\sigma^2} - \kappa T \right) \\
\beta_p = \frac{\partial \log W_f}{\partial p} |_{x,T} = -\frac{b(p - \bar{p})}{C^2} \\
\beta_C = \frac{\partial \log W_{m}}{\partial C} |_{x,T} + \frac{\partial \log W_{f}}{\partial C} |_{x,T} = (1 + b) \left( \frac{(p - \bar{p})^2}{C^2} \right)
\]

Equation (15a) shows that (1) selection on males intensifies as generation time \((T)\) increases; (2) the life-history accentuates splitting selection on males into sexual (the first term in parentheses) and viability components (the second term in parentheses); (3) males carrying different somatic values of condition \((C)\) or growth \((\kappa)\) can display similar trait values with varying linearized fitness in the \(\kappa\)-dimension. Direct selection on female preference (Equation (15b)) leads to negative character change unless the preferred size \((p)\) falls below the average trait \((\bar{p})\). Intensity of selection against choice attenuates with increasing condition. Selection on condition (Equation (15c)) remains strictly positive at all trait values and intensifies with the direct costs of choice. Only selection on trait growth \((\kappa)\) depends on generation time \((T)\). More intense (sexual and viability) selection occurs on male traits in populations with longer-lived life-histories. The selection gradient here predicts lower values of \(\kappa\) at longer generation times. This follows from the model of trait growth, as populations with longer generation times will have more old males with larger traits.

B. Equilibria

I find equilibria in \(\bar{z}\) by setting the left-hand side of Equation (7) equal to 0 and using Cramer’s Rule, substituting the values in Equation (15) for \(\beta\):

\[
\frac{\bar{C}(\bar{p} - \bar{q})}{\sigma^2} - \kappa T = \frac{\mu|G^x|}{|G|} \\
\frac{b(p - \bar{p})}{C^2} = \frac{T \mu |G^p|}{|G|} \\
(1 + b) \left( \frac{(p - \bar{p})^2}{C^2} \right) = \frac{T \mu |G^C|}{|G|}
\]

where

\[
|G^x| = G_{kp}G_{Cp} - G_{kC}G_p \quad (17a) \\
|G^p| = G_{kC}G_p - G_{kC}G_{kp} \quad (17b) \\
|G^C| = G_{kp}G_p - G_{kC}G_{kp} \quad (17c)
\]

form the principal minors of the upper two rows of \(G\). Readers can find \(|G^i|\) by replacing column \(i\) of \(G\) with \(Tu\) and taking the determinant of the resulting matrix. Equation (17c) measures the basic intensity of direct correlation between male growth \((\kappa)\) and female preference \((p)\). This expression simplifies to \(|G^C| = G_{kC}G_p(1 - \rho_{kp}^2)\) and remains non-negative over all values of \(\rho_{kp}\).

\[
\delta = \theta + (G_{kC} + \kappa \kappa) T
\]

describes the equilibrium value of \(\bar{z}\), found by substituting from Equations (16) and averaging over the male population, using the approximation \(E[CKx] \approx C\kappa T\). I have assumed here that \(Cov(Ckx, x) \approx 0\) due to the slow rate of phenotypic change. Assuming that \(b > 0\) then solve Equations (16) for equilibria in all three variables, yielding

\[
k = \frac{b^3|G|^3|G^C|^2}{\sigma^2(1 + b)^2T^2 \mu |G^p|^3} \frac{\mu |G^x|}{|G|} \quad (19a) \\
\hat{\beta} = \frac{b^3|G|^3|G^C|^2}{(1 + b)^2T^2 \mu |G^p|^3} + \hat{\delta} \quad (19b) \\
\hat{C} = \frac{b^2|G||G^C|^2}{(1 + b)T^2 \mu |G^p|^2} \quad (19c)
\]

The equilibrium in male trait growth \((\kappa)\) includes a positive component (as long as \(|G|\) remains positive) and a negative component, mirroring the mating and viability components of the selection coefficient (Equation (15a)). The negative component increases with mutation size \((\mu)\) and decreases with generation time. Here the two forces oppose each other, whereas in the other equilibria they multiply together.

The equilibrium in female preference \((p)\) lies above the equilibrium value of the male trait \((\bar{p})\) as long as \(|G^p| > 0\), easily seen in this alternate representation:

\[
\hat{\beta} - \hat{\delta} = \frac{\hat{C}^2 T \mu |G^p|}{b}. \quad (20)
\]
The difference between the equilibrium preference and the equilibrium trait will shrink as mutation size ($\mu$) and generation time ($T$) increase, or as the scaling parameter of costs ($b$) gets smaller.

The equilibrium given by Equation (19c) shows that mutation-selection balance in $C$ remains positive and diminishes as mutation rate increases. The mutation-selection balance value for $C$ will increase as the scaling parameter of female viability costs ($b$) increases, or as the correlation ($\rho_{\kappa p}$) between preference ($p$) and growth ($\kappa$) decreases.

C. Interpretation

I will interpret the model in terms of what I call basic good genes theory (“good genes”). Selection will favor female preferences that improve offspring condition relative to the offspring condition of females with other preferences [Jones and Ratterman 2009b; Kokko 2001]. I will phrase the interpretations in terms of the position of mean female preference relative to the mean male trait, and the effects of life-history parameters (e.g. increasing generation time).

The three genetic correlations from $G$ (Figure 1) affect the hypothetical position of female preference relative to the male trait. The terms of Equation (17) translate into correlations using the relation $\rho_{ij} = \frac{q_{ij}}{\sqrt{q_{ee_i}q_{ee_j}}}$, which forms an upper bound for genetic covariances. I assume throughout this analysis that $\rho_{Cp} > 0$ since this condition forms a prerequisite for the evolution of preferences for indicator traits. I especially focus on cases where selection favors females that choose males with small growth parameters ($\kappa$), since trait size ($s$) does not reveal either condition ($C$) or growth rate ($\kappa$) directly. This situation occurs when males with high growth parameters tend to have low-condition offspring, i.e. $\rho_{Cp} < 0$. Negative correlations might evolve under trade-offs between trait size and viability. Readers should keep in mind that Figure 1 and Figure 2 were created using a fairly large mutational effects. For example, for a mutational effect of $\mu = \theta$ (Figure 1 and Figure 2a) the difference in trait size between a father of age $x$ and his son at that same age equals $-\theta \kappa x$. Differences attributable to mutation are even larger in Figure 2b where with short to intermediate generation times we still see females with fairly strong preferences.

Good genes predicts a non-negative difference between equilibrium female preference and equilibrium male trait. I base this interpretation on Iwasa et al. [1991], who found equilibrium female preference proportional to mutational input and inversely proportional to the scaling parameter of costs ($b$ here and in Iwasa et al.). Positive mutational bias enables an honest signal by depressing average male condition (i.e. increasing $G_C$ on the right-hand side of Equation (14b)). Females with positive preferences ($p > \bar{s}$) will produce higher quality offspring than females with average preferences ($p = \bar{s}$, i.e. random mating). My model differs from theirs in that female viability costs are proportional to the difference $\bar{p} - \bar{s}$, and so I evaluate the predictions of the theory slightly differently.

When growth and condition positively correlate ($\rho_{\kappa p} > 0$) the equilibrium $\kappa$ will lead to average traits above $\theta$, due to positively reinforcing viability and mating advantages. Selection will favor females whose preferences lead to larger $\kappa$ values (since $\kappa$ and $C$ positively correlate). The predicted equilibrium $p$ increases with $\rho_{\kappa p}$. Good genes predicts balancing selection at negative values of $\rho_{\kappa p}$, since females with extraordinarily low preferences will tend to choose small-$\kappa$ males as mates (see Equation (14a)), producing low-condition offspring.

When growth and condition negatively correlate ($\rho_{\kappa p} < 0$) the equilibrium $\kappa$ will lead to small traits, with average trait smaller than $\theta$ under some values of $\rho_{\kappa p}$. Selection will favor females whose preferences lead to smaller $\kappa$ values, since low-$\kappa$ offspring will have higher $C$. The predicted equilibrium $p$ decreases with $\rho_{\kappa p}$. Again good genes predicts balancing selection as $\rho_{\kappa p}$ becomes more strongly negative, since females with especially large preferences will tend to choose large-traited males as mates, leading to offspring with low condition (see Equation (14b)). Longer generation time means more old males will be available for mating, and females could gain good genes by mating with even older males. Therefore good genes universally predicts an increasing divergence between preference and trait values. Figure 1 on the other hand, shows that increasing generation time reduces the equilibrium deviation of preference from average trait, rather than increasing it (see Equation (19b)).

IV. DISCUSSION

The evolution of age-dependent signals appears to follow the basic guidelines set down by good genes theory. Age-dependent sexual signals could facilitate sexual selection by reserving the production of costly traits until older ages. Life-history strategy theory shows that honest signaling favors delaying development of costly signals, and favors preferences for older males. Older-male preferences, however, may come with costs arising from the higher mutation load of older males’ sperm. I have shown that viability selection does not eliminate sexual selection. Female preferences can remain positive despite considerable mutational effects. Furthermore, my results show that (1) sexual selection intensifies as generation time increases and (2) selection on condition intensifies as direct costs of choice increase.

The results of the model coincide with existing interpretations of good genes theory, but also contains information about the action of life-histories. Basic interpretations of good genes do not include any effects of age-dependent mutation. My interpretation of good genes places limits on the equilibrium deviation of preference from trait, but does not produce accurate predictions regarding the action of generation time. Increasing generation time reduces the departure of the preference from the trait, rather than increasing it (see Equa-
The failure of the basic theory leads me to consider two additional hypotheses. What I call mutational effects balancing selection (“mutational effects”) predicts that along with the predictions of basic good genes, the effects of mutation place limits on the adaptive value of extreme preferences. This theory roughly corresponds to the objections to good genes raised by Hansen and Price (1995). Direct costs balancing selection (“direct costs”) includes some of the predictions of both prior theories, but predicts that direct costs to preferences set the limit on the deviation of preference from average trait.

The mutational effects theory of balancing selection emphasizes that although selection favors female preferences, mutation places limits on the adaptive value of preferring older males. At some point the marginal benefit in offspring condition will maximize owing to the higher mutation rate of older males. The basic predictions of good genes theory still hold. Female preferences still lie above male trait sizes. The difference between the hypotheses lies in more accurate predictions based on increasing mutation rate (higher $T\mu$). Mutational rate augmentation will decrease the deviation between trait and preference. This prediction holds true in the model (see Equations ([19b]) and Figure 1). Mutational theory also predicts reduction in the absolute size of the equilibria (Figure 1). More old males in the population (larger $T$) leads to larger mutational input, meaning a smaller average benefit. Correspondingly, at short generation time, few males live long enough to accumulate many mutations. Selection will favor females selecting the oldest (i.e. largest) males they can find.

Direct costs balancing selection forms a third possibility. Considering direct costs of female choice makes the prediction of upper limits on preference more precise. Direct costs can account for the observation that as the scaling coefficient of mate choice costs ($b$) increases, the equilibrium preference increases (see Equation ([19b])). As choice becomes more directly costly, selection favors females that secure better genes for their offspring. Despite the risk of greater mutational input from larger-trait males, augmenting costs ($b$) creates stronger selection to find good genes. The fitness differential in offspring...
FIG. 2: Equilibrium trait (solid black) and preference (dashed green) as a function of generation time, with varying mutational effect sizes ($\mu$). The blue dashed line indicates the optimal male trait size $\theta$. Comparison of the two panels shows that increasing mutational effect size decreases the difference between equilibrium trait and equilibrium preference.

spring condition will offset any mutational losses in condition and losses in female viability. Readers can verify this in Equation (19c) by noting that equilibrium condition ($C$) also increases with $b$. Although some results of my model are consistent with the effects predicted by a theory of mutational effects, direct costs to females form the best explanation for my results.

I can also consider the effectiveness of the age-as-indicator
model [Brooks and Kemp 2001]. Although age does not constitute an indicator in this model, age does influence the reliability of the indicator trait (Equation 14b). When growth and condition positively correlate, older males will tend to have larger traits. Selection will also favor females that prefer larger traits. Good genes predicts that females will choose older males in all cases. Direct costs theory adds to the precision of this prediction. Despite mutation pressure, older males signal condition in a more reliable manner. On the other hand, in some cases when growth and condition negatively correlate, selection still favors females that choose the largest traits (Figures 1a and 1c). We can interpret this as selection against older males, since older males will show smaller traits, and the equilibrium still lies above $\bar{\mathcal{P}}$

Age-dependence and paternal mutation facilitate and maintain selection for female preferences. First, increasing lifespan facilitates sexual selection by reducing the power of selection against the trait [Adamson 2013]. Secondly, increasing lifespan increases the mutational input into condition, maintaining genetic variance and selection for female preferences. As selection weakens over the lifespan, traits can become more accentuated without impacting fitness as strongly. Mate choice for older males also contributes to the life-stage separation enabled by the weakening of selection. As females produce a broader range of condition in offspring, viability selection has more variance to work with in the early stages of life. Increased lifespan and age-dependence with mate choice therefore introduce a negative feedback in terms of genetic variation, viability selection and mate choice. The lek paradox disappears as the process enabling trait exaggeration simultaneously introduces genetic variation into the next generation.

Generalizing my results requires some caution. If viability selection weakens with age, enabling more extravagant traits, I expect that selection will also allow greater mutation accumulation in the trait itself and loss of variation through genetic drift. My model makes several assumptions that do not cover this possibility. I completely neglect drift, as well as the genetic mechanisms of dominance, epistasis and pleiotropy. Traits and preferences can correlate much more tightly than I’ve supposed here, including pleiotropy [Grace and Shaw 2011]. I chose particular functions for mathematical convenience that limit the scope of the application. Furthermore, since I seek the elucidation of theory, I have neglected details of male and female mating behavior.

I began with the question of whether selection can maintain costly preferences in the face of mutation pressure. Age-dependent mutation appears to supply the necessary genetic variation for sexual selection to continue. Contrary to some expectations, direct costs to females produce the greatest selective incentive for females to express preferences for extreme male traits. Direct costs and the continued input of variation in indirect benefits interact to reinforce sexual selection on an indicator trait. Selection maintains costly preferences supported by, rather than despite, age-dependent mutation. The question of whether or not age-dependent preferences display the same patterns remains open.

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Appendix A: Stability

I intend to show that the equilibria given in Equations (16) are asymptotically stable. Particularly I wish to show that all equilibria with $\phi > 0$ are asymptotically stable from any initial condition if $|G'\phi| > 0$. Consider Figure 3 where $\hat{\phi}$ represents a quadratic function of $\phi'$ for some fixed $\kappa$. Choose a point $(\phi', \phi')$ above the curve where $\phi' > \hat{\phi}$. By representing the time derivative of $\phi$ as a scalar product

$$\frac{d\phi}{dt} = \left( \frac{1}{2} \right) (\beta \cdot g_\phi) = \left( \frac{1}{2} \right) (|\beta| |g_\phi| \cos \gamma) \quad (A1)$$

where

$$g_\phi = \begin{pmatrix} G_{\phi p} \\ G_p \\ G_{C_p} \end{pmatrix} \quad (A2)$$

we can more easily calculate the direction of the path in Figure 3. Equation (A1) will be less than zero if and only if $\gamma > \frac{\pi}{2}$ (see Figure 4). At equilibrium the two vectors are perpendicular and $\frac{d\phi}{dt} = 0$. However, if we raise $\phi$ to $\phi'$ then $\gamma > \frac{\pi}{2}$ by making $\beta_p$ more strongly negative (see Equation (15b)). A similar argument shows that $\frac{dC}{dt} > 0$ such that the solution will approach the equilibrium depicted by the curve. The only difference in the argument lies in the non-zero equilibrium value of the scalar product with $g_{C_\phi}$, defined analogously to $g_\phi$.

Now consider the other side of the curve. We can apply a similar argument to show that $\frac{d\phi}{dt} > 0$ on this side of the curve. Again at equilibrium, $\gamma = \frac{\pi}{2}$ and if we decrease $\phi$ to $\phi''$ (see Figure 4) then we are increasing the value of $\beta_p$ to greater than $\frac{G_{\phi p}}{|G_{C_\phi}|}$, such that $\beta$ is closer in $\mathbb{R}^3$ to $g_\phi$. Thus $\gamma < \frac{\pi}{2}$ and $\frac{d\phi}{dt} > 0$. A similar argument in $C_\phi$ shows that $\frac{dC}{dt} < 0$ to the right of the curve. The solution approaches the curve in Figure 3 regardless of the magnitude of $T$ and $\mu$. 

FIG. 3: The equilibrium preference as a function of $C_\phi$ for constant $R$. Points on either side of this nullcline show component vectors of the solution of Equation (7).
FIG. 4: The position of three vectors describing Equation (A1) at the points in Figure 3 with a corresponding number of primes. When $\frac{dp}{dt} = 0$ the two vectors are perpendicular, corresponding to the vector $\hat{\beta}$. When $\beta$ moves to $\beta'$, $\gamma$ increases to greater than $\frac{\pi}{2}$, meaning $\frac{dp}{dt} < 0$. A similar argument shows that $\frac{dp}{dt} > 0$ when $\beta$ moves to $\beta''$. 