Sexual complexity as a screen for replication fidelity: the Everest effect

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Abstract

Sexual reproduction is widespread among complex organisms, yet its elaborate accompaniments—from courtship displays to molecular recognition systems—remain incompletely explained. The Everest hypothesis rests on four propositions: (1) environmental change can shift the drift-barrier equilibrium toward higher mutation rates, as beneficial mutations become more common and mutator alleles hitchhike during adaptation; (2) many lineages reproduce through systems whose elaboration exceeds what fertilization or viability-fecundity benefits can explain; (3) such elaboration enlarges the mutational target by involving additional coding or regulatory DNA; and (4) mutations affecting those loci often reduce mating or fertilization success. If these propositions are broadly correct, reproductive complexity should couple replication fidelity to reproductive success. This coupling is the Everest effect: complex reproductive systems expose even mild mutators—whose effects may be too weak for ordinary viability-fecundity selection to purge reliably—to appreciable reproductive penalties. The hypothesis casts sexual selection as both accelerator and brake: during environmental change, mate choice can accelerate adaptation by favoring high-performing individuals, often arising from mutator backgrounds. After conditions stabilize, demanding reproductive screens can expose accumulated damage from elevated mutation rates, allowing mate choice or fertilization success to purge mutator lineages. Recombination then allows adaptive alleles to be retained while high fidelity is restored. Everest complements Fisherian, handicap, and good-genes processes by proposing that traits elaborated by those processes can also be co-opted as fidelity-linked reproductive screens. An individual-based simulation and proposed experimental, comparative, and genomic tests show how the hypothesis can be evaluated.

Keywords: sexual selection, mutation-rate evolution, mate choice, reproductive complexity, adaptation, experimental evolution

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Introduction: mutation rates, drift-barrier theory, and unstable environments

DNA replication is remarkably accurate but not perfect. Each generation introduces new mutations, including mutations in the replication machinery itself. Alleles that increase the error rate—mutator alleles—form a spectrum from severe to mild. Strong mutators are relatively easy to eliminate because they remain linked to the deleterious mutations they generate. Mild mutators pose a harder problem: each may impose selective effects too small for ordinary viability-fecundity selection to purge reliably, even though their accumulation threatens the long-term maintenance of complex genomes. (Viability-fecundity selection refers here to selection based on survival or reproductive output, apart from mate choice.)

This spectrum is asymmetric. Mutator alleles can range from severe to mild, but antimutator alleles—those that improve replication fidelity—are expected to be mild because large improvements in fidelity are unlikely to arise in a single mutational step, except by reversion of a previous mutator mutation. Mechanisms that respond only to large fidelity differences will therefore eliminate strong mutators but fail to purge the mild mutators that erode fidelity or favor the mild antimutators that could restore it—yet long-term genome maintenance requires selection to act on both.

Replication fidelity is distinct from fitness. Fitness is a context-dependent measure of expected reproductive success; replication fidelity is a quantitative property of the replication machinery, measurable by sequencing as a per-base or per-genome mutation rate. An animal colonizing an island may instantly become fitter than its competitors if it already carries alleles suited to that environment, although its genetic composition and mutation rate remain unchanged. Viability-fecundity selection acts directly on realized fitness; it detects mutation rate only indirectly, through harmful mutations that remain linked to mutator alleles. When populations are well adapted, most new mutations are deleterious and lower mutation rates tend to be favored, but genetic drift may allow suboptimal fidelity to persist when the benefit of further fidelity gains is very small.

A key premise of the Everest hypothesis is that environmental change can shift the drift-barrier equilibrium toward higher mutation rates. Under drift-barrier theory [Sung et al., 2012; Lynch et al., 2023], selection reduces mutation rates only while improved fidelity confers an advantage large enough to overcome drift; equilibrium is reached when further gains are too small to be reliably favored. Environmental change can shift this equilibrium upward in two ways. First, novel conditions can change the distribution of fitness effects, increasing the proportion of beneficial mutations among new variants, as illustrated schematically in Figure 1, so mutator alleles are more likely to hitchhike during adaptation. Put another way, if selection against mutators normally depends on their linkage to the deleterious mutations they generate, then any shift in the distribution of mutation effects toward beneficial or weakly deleterious variants *must*, all else equal, weaken this purging mechanism and move the mutation-rate equilibrium upward. Second, environmental change often coincides with a reduction in effective population size, such as during colonization bottlenecks, thereby strengthening drift and reducing selection against mildly deleterious mutator alleles. As Duffy observed, a lineage “suddenly thrust into an environment that it is not well adapted to” may benefit from a slightly higher mutation rate [Duffy, 2018]. Environmental transitions can therefore generate episodes in which mutator lineages rise substantially.

Figure 2 illustrates this scenario with a stochastic individual-based simulation, detailed in Appendix S1. Environmental fluctuation repeatedly allows mutator lineages to rise during adaptation through weakened selection against mutators and hitchhiking with adaptive alleles; under plausible parameter values, these increases can be substantial.

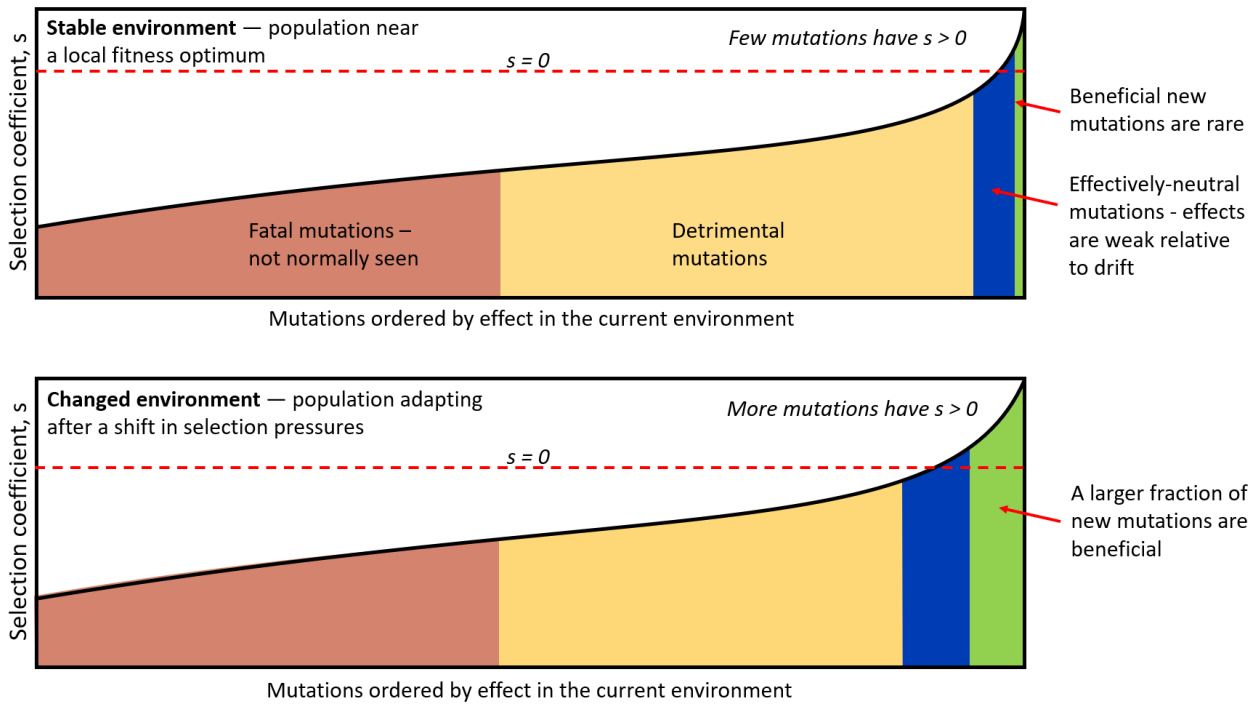


Figure 1. Mutation effects under different selection regimes. Mutations are grouped as fatal (brown, removed immediately), detrimental (beige, removed by purifying selection), effectively neutral (blue, fixed or lost by drift), and beneficial (green, fixed by positive selection). The dashed red line marks *s* = 0. It is shown shifted downward in the lower panel: environmental change alters the distribution of fitness effects, so mutations that were formerly neutral or deleterious may become beneficial. Top: In stable environments, populations are already well adapted, most readily available advantageous mutations have been fixed, and new beneficial mutations are rare; long-persisting lineages in relatively stable habitats, such as horseshoe crabs in shallow marine environments, illustrate this regime. Bottom: After environmental change, a greater proportion of new mutations may be beneficial; colonizing or radiating lineages, such as Darwin's finches in the Galápagos, illustrate this regime.

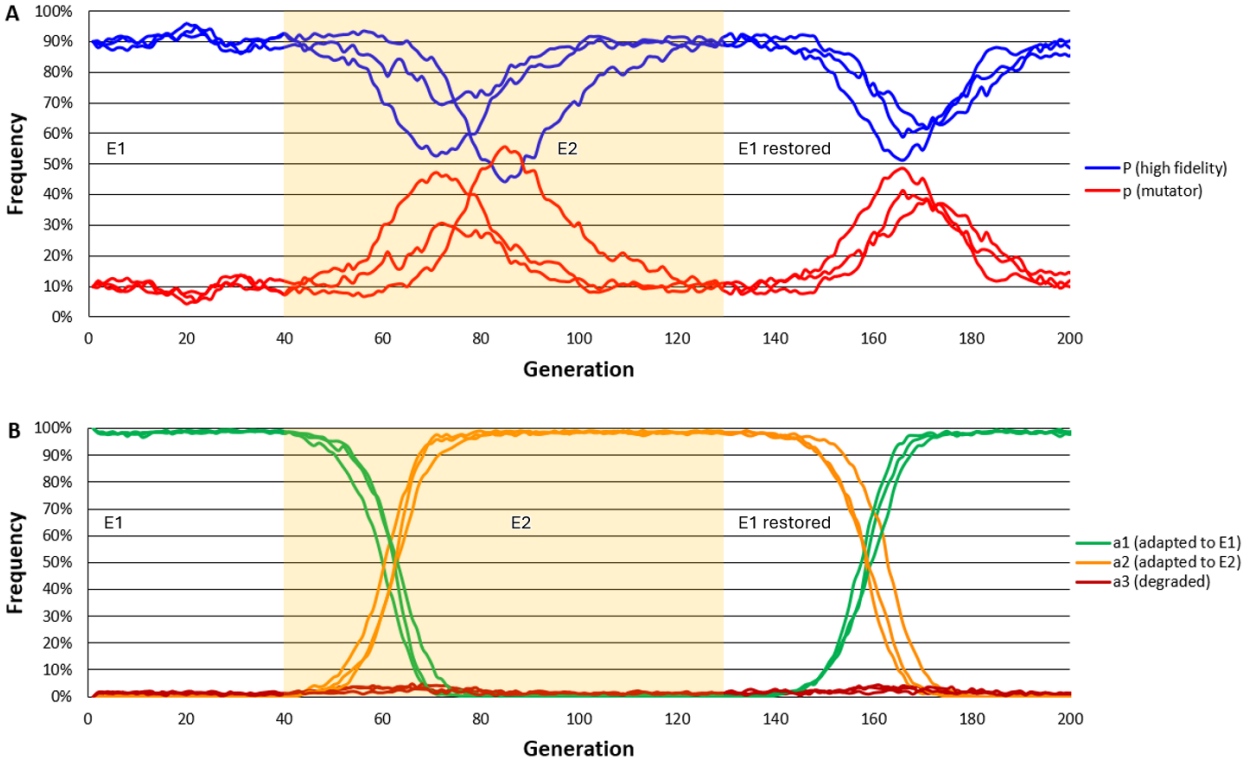


Figure 2. Individual-based simulation of mutator dynamics during environmental fluctuation. Three independent model runs are superimposed. (A) Replication fidelity allele frequencies: mutator (*p*) frequency rises after each environmental transition. (B) Adaptation allele frequencies: adaptation succeeds in all runs regardless of polymerase background. Shading indicates Environment 2; unshaded regions indicate Environment 1. Transitions occur at generations 40 and 130. In 36 of 50 runs, peak mutator frequency exceeded 30%; full results are given in Figure S2 and Table S4.

This dynamic is consistent with empirical evidence from several systems, although mainly indirectly for multicellular taxa. Spontaneously arising mutator lineages repeatedly rose to high frequency in *E. coli* populations adapting to new environments [Sniegowski et al., 1997]. Environmental stress has been associated with genome-wide accumulation of de novo mutations and epimutations in *Arabidopsis* [Jiang et al., 2014]. Avian lineages with higher diversification rates show faster molecular evolution, including higher synonymous substitution rates, consistent with a link between diversification and mutation rate [Lanfear et al., 2010]. While some microorganisms use stress-induced mutagenesis via error-prone polymerases [Galhardo et al., 2007], the animals and plants Everest focuses on are more likely to experience shifts in mutation rates due to drift-barrier and hitchhiking dynamics [Lynch et al., 2023].

These considerations imply a fundamental tension. Over geological timescales, lineages require accurate replication to maintain complex genomes; over shorter timescales, elevated mutation rates can accelerate adaptation. Mutator alleles may be transiently favored in unstable environments despite being harmful in the long run. Because mutations can arise in replisome genes, mutators may

generate additional mutator alleles, increasing the risk of mutational meltdown unless some mechanism limits their spread.

Sexual reproduction offers a possible resolution. Some lineages may be well adapted but low-fidelity, while others retain accurate replication machinery but are less well adapted. Recombination can generate offspring combining both advantages, but this process will be favored only if reproductive systems make the consequences of low fidelity visible to selection when immediate fitness differences are small. The Everest hypothesis proposes that the elaborate accompaniments of sexual reproduction are often co-opted for this function: reproductive complexity is, therefore, not merely ornamental, but part of a broader system of genomic quality control, as developed in the following section.

The Everest effect

The central question is therefore: how can sexual lineages make subtle differences in replication fidelity visible to selection, allowing higher-fidelity lineages to contribute disproportionately to future generations? I suggest that evolution is fundamentally a two-dimensional problem: one dimension is adaptation—how well an organism fits its environment—and the other is fidelity—how accurately its genome is copied. These dimensions can trade off: populations may gain adaptation at the cost of fidelity, and vice versa.

The Everest hypothesis (Figures 3 and 4) proposes that many features of sexual reproduction—including courtship, mating, fertilization, and their accompaniments—can function as fidelity-linked screens. The name refers to climbing Mount Everest as a test of integrated performance: reaching the summit provides a single readout of many systems working well together under demanding conditions. By analogy, complex multigenic reproductive traits can expose diffuse genome-wide mutational damage, making reproductive success a potential indicator of replication fidelity.

The logic follows from four premises.

> First, environmental change can produce substantial transient increases in mutator frequency through mutator hitchhiking and weakened selection against mutation (Figure 2).
>
> Second, many lineages reproduce through systems whose elaboration substantially exceeds what fertilization or direct viability-fecundity benefits alone can explain.
>
> Third, such elaboration generally enlarges the mutational target by increasing the amount of coding or regulatory DNA whose disruption can affect reproductive success.
>
> Fourth, mutations at those loci will often reduce mating or fertilization success.

Together, these premises imply that replication fidelity and reproductive success must be coupled to some degree. The empirical question is whether this association is strong and consistent enough to influence mutation-rate evolution. The core Everest prediction (Figure 3A) is that populations with greater reproductive complexity should maintain higher equilibrium replication fidelity than populations with simpler systems, because complex reproductive screens impose stronger penalties on mutator-linked damage.

The consequence is an amplification effect that ordinary viability-fecundity selection may often fail to generate. Viability selection typically exerts weak, diffuse pressure on mild defects. In a non-migratory bird, for example, a slightly compromised heart valve might reduce lifetime reproduction by 5–10%, with much of that effect masked by environmental variation. In a closely related migratory bird, the same defect could prevent completion of a demanding migration before breeding, reducing reproduction to zero. Reproductive complexity can thus amplify small differences in replication fidelity into large differences in reproductive success—this is the Everest effect. The empirical task is to determine its strength, taxonomic breadth, and importance relative to other forces shaping reproductive traits.

The same logic applies to antimutator alleles. Because fidelity improvements are usually incremental, mild antimutators may have effects that are too weak for ordinary viability-fecundity selection to reliably favor. By amplifying small differences in accumulated mutational damage, Everest tests can give antimutators a reproductive advantage: offspring accumulating fewer new mutations should be

more likely to pass demanding reproductive screens. Thus, reproductive complexity can both purge mutators and favor gradual restoration of high fidelity.

Everest's two-axis view helps reconcile two apparently opposite effects of sexual selection. Roberts and Petrie showed theoretically that female choice can favor elevated mutation rates during adaptation, because beneficial mutations may arise disproportionately on mutator backgrounds and sexual selection concentrates reproduction on high-performing males [Roberts & Petrie, 2022]. Everest accepts this accelerator logic but adds the corresponding brake. When environments stabilize, the value of increased mutation declines, but mutator alleles continue to generate harmful mutations. Complex reproductive traits can make the resulting damage disproportionately visible, converting weak selection against mutators into steep reproductive penalties. Sexual selection can therefore act sequentially: accelerating adaptation during environmental change, then restoring fidelity when mutation load becomes the greater threat.

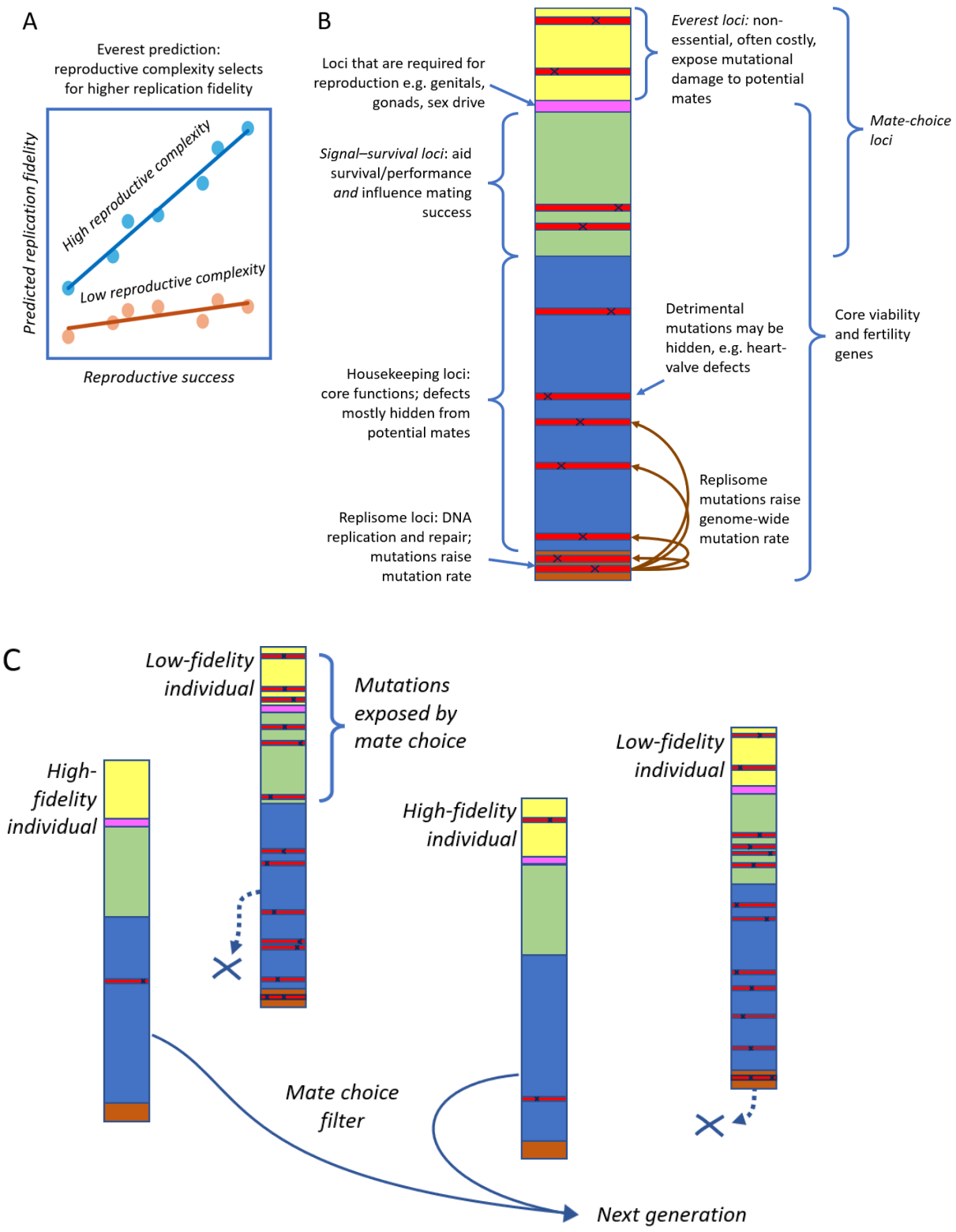


Figure 3. Everest hypothesis: predicted patterns, genomic architecture, and mechanism. Complex mate-choice systems can make mutator-linked damage visible to selection, coupling reproductive success to genome-wide genetic integrity. (A) Predicted equilibrium pattern. In stable environments, populations with high-complexity mate-choice systems (blue; many multigenic Everest tests) are predicted to maintain higher equilibrium replication fidelity than populations with low-complexity systems (brown). Complex reproductive screens impose stronger penalties on mutator-linked damage, so high reproductive success should be associated with higher replication fidelity, especially in high-complexity systems. Points and trend lines are schematic. (B) Loci by biological role. Yellow: Everest loci—non-essential, often costly, and elaborated to expose mutational damage. Green: signal–survival loci—influencing both mating and survival. Blue: housekeeping loci—core viability and fertility functions, largely hidden from mates. Brown: replisome loci—replication and repair machinery. Replisome mutations raise the genome-wide mutation rate, including at mate-choice loci where effects become visible. Red X's mark mutations. (C) Individual-level mechanism. Each vertical bar represents an individual. Low-fidelity individuals accumulate more mutations, including in mate-choice loci, where damage is exposed by the mate-choice filter and reduces mating success. High-fidelity individuals are more likely to mate and transmit accurate replication machinery.

To clarify the mechanism, I distinguish several classes of loci. A "locus" is any genomic region—including coding, intronic, or regulatory sequences—where variation alters phenotype. *Mate-choice loci* affect mating or fertilization success. Within this class:

- *Signal–survival loci* influence both mating success and survival/performance, for example through strength, endurance, or cognition.
- *Everest loci* have effects that are non-essential for survival or fertilization—often costly or risky—but elaborated by sexual selection partly to make mutational damage highly visible.

These categories are positions on a continuum rather than sharp classes; what matters for fidelity screening is how many loci are integrated into a single readable test. Traits derived from these loci amplify small differences in replication fidelity into large differences in reproductive success. I refer to the filters generated by these screening phenotypes as *Everest tests*.

Figure 3B orders locus types by function: Everest loci (yellow) and signal–survival loci (green) form a mate-choice sub-genome; housekeeping loci (blue) represent hidden core functions; and replisome loci (brown) encode replication machinery. Figure 3C shows the individual-level mechanism: error-prone replisomes generate mutations genome-wide, including at mate-choice loci, reducing mating success. Table 1 positions Everest relative to major frameworks for sexual traits, recombination, and mutation-rate dynamics. These include Fisherian and handicap models of sexual selection, classic accounts of recombination and mutation load—Hill–Robertson interference [Hill & Robertson, 1966], Muller's ratchet [Muller, 1964], and Kondrashov's "hatchet" [Kondrashov, 1988]—and Red Queen models of host–parasite coevolution [Hamilton et al., 1990]. The table summarizes which questions each framework addresses, not their overall explanatory power. The table is intended as an overview; fuller comparisons can be found in the section "Relations to previous work" and in Appendix S3.

Table 1. The position of Everest relative to major frameworks for the evolution of sexual traits, recombination, and mutation-rate dynamics.

Framework	*Fidelity explicit*	*†Complexity-as-screen*	*‡Two-axis: fidelity can decrease while adaptation increases*	*Diagnostic tests*	*Selection contributions*
Everest	☑	☑	☑	☑(E,C,G)	M
Fisherian runaway	—	—	—	◐(C)	S
Handicap/indicator	—	◐	—	◐(E,C)	S
Good genes / condition / genic capture	—	◐	—	◐(C,G)	S+V
Red Queen (parasites)	—	—	—	☑(E,C)	V
Mutation-rate evolution	☑	—	◐	☑(E,G)	V
Kondrashov's "hatchet" (purging mutation load)	◐	—	◐	☑(E,G)	V
Hill–Robertson (linkage)	—	—	—	☑(E)	V
Muller's ratchet	—	—	—	☑(E)	V+L

†*Complexity-as-screen:* reproductive complexity evolves because it increases sensitivity to mutational damage, acting as a fidelity screen; ◐ indicates that the framework is compatible with the idea (because certain traits depend on condition), but it does not explicitly predict that mating traits become complex *in order to* reveal mutational damage.

‡*Two-axis:* adaptation and fidelity are treated as distinct and potentially conflicting properties rather than components of a single fitness measure.

Symbol key:

☑ = Central/explicit in that framework

◐ = Compatible or implicit/secondary (can be accommodated, but not a main focus)

— = Not addressed / not a prediction of the framework

Diagnostic test codes ("Diagnostic tests" column)

E = Experimental evolution/selection experiments

C = Comparative (across taxa/populations)

G = Genomic signature (sequence-based; mutation spectrum/rate, DNA replication/repair genes)

Selection contribution codes ("Selection contributions" column)

S = Sexual selection (mate choice/competition)

V = Viability-fecundity selection (non-mating)

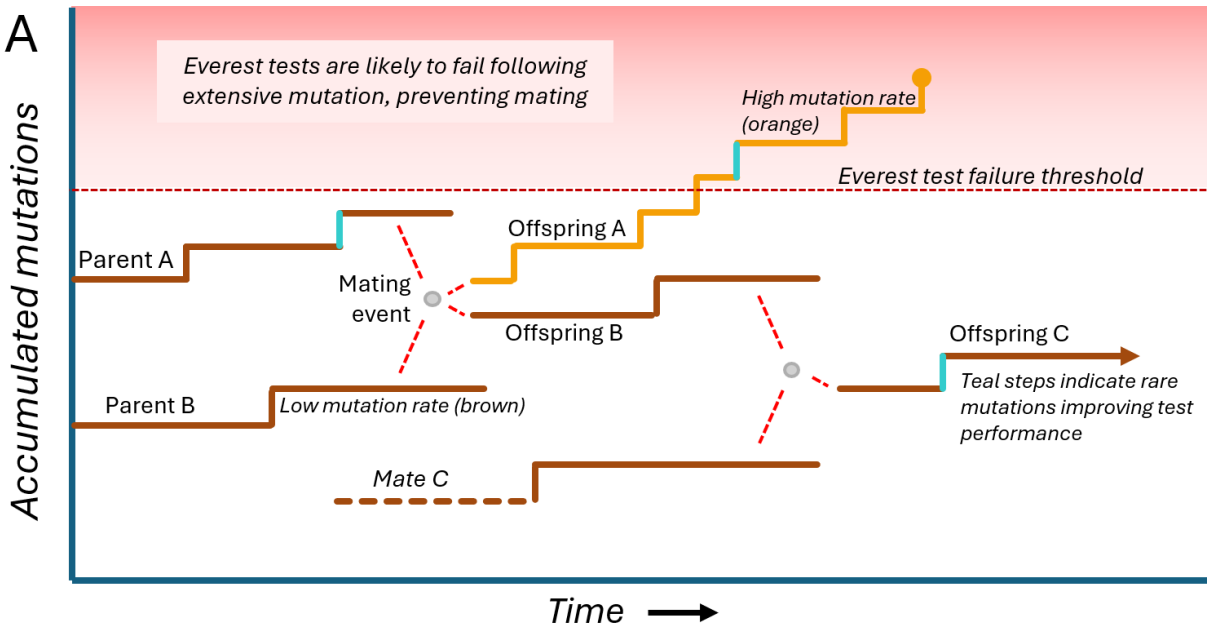
L = Lineage-level effects (persistence/extinction)

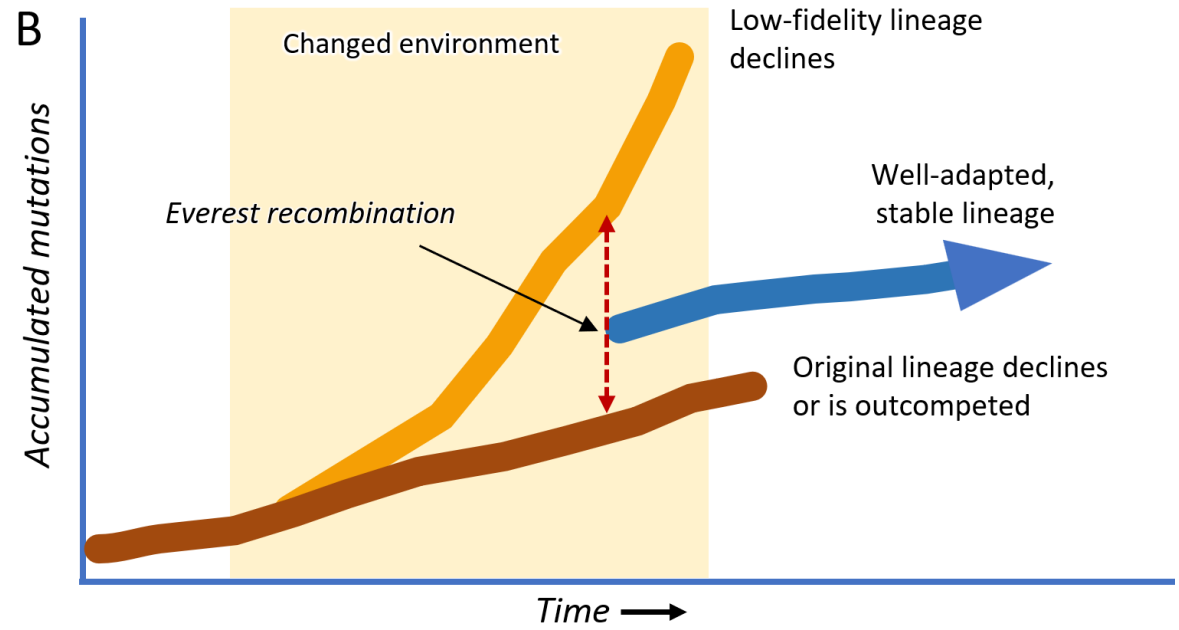
M = Mixed (more than one of S/V/L contributes)

Everest loci serve two functions. First, they expand the mutational target: adding non-essential components to reproductive traits increases the DNA regions where mutations affect mate choice. Second, they can stack their own mutation-sensitive effects together with signal–survival traits into a single reproductive test. A courtship display, song, dance, migration-linked breeding schedule, or molecular compatibility system may require several underlying capacities to be expressed together—sensory accuracy, motor coordination, metabolic regulation, learning, endurance, timing, or biochemical recognition. Some of these capacities may have ordinary viability or fecundity effects, but the reproductive screen integrates them into a shared outcome: successful display, arrival, mating, or fertilization. Thus, a mild mutator need not cause a large defect in any one component. If enough components are tested together, small increases in damage across many loci can produce appreciable differences in reproductive success.

While viability-fecundity selection should favor robustness and redundancy, sexual selection may favor demanding tests where fragility cannot be avoided, precisely because they discriminate genetic integrity. Critically, Everest tests must be shared widely enough within an interbreeding population to have statistical power. If preferences are fragmented—some individuals focusing on plumage, others on migration performance, others on biochemical compatibility—the Everest effect is weakened. However, Everest does not require a single universal test. It predicts convergence on one or a few dominant screens within each mating network, with the dominant screen varying across environments and regions. Where populations face different ecological demands, such as migratory versus resident strategies, different screens may predominate and contact zones may show mixtures of criteria.

Fidelity concerns mutation rate, not merely the total number of mutations an individual carries. A mutation rate is a slope—mutations accumulated per generation—so it cannot be read from a single phenotype in isolation. Selection on fidelity must operate across generations, through the repeated association between mutator alleles and the damage they generate. Figure 4 illustrates this logic across scales. At the individual level (panel A), parents who pass a demanding test establish a baseline; offspring that accumulate excessive damage since the parental generation may then fail the same test. At the population level (panels B and C), strong selection can initially favor mutator lineages that adapt quickly but accumulate mutations unsustainably; recombination with remaining or immigrant high-fidelity genotypes can later generate well-adapted lineages with restored fidelity, tracing a rise-and-fall trajectory in adaptation–fidelity space. I refer to this process—recombination between adapted, low-fidelity genotypes and high-fidelity partners, followed by mate choice favoring offspring that inherit both adaptive alleles and accurate replication machinery—as *Everest recombination*. The orange trajectory in Figure 4 represents the accelerator phase described by Roberts and Petrie [2022], in which sexual selection can favor mutator backgrounds during adaptation. The blue trajectory represents the Everest extension: reproductive screening after recombination can restore high fidelity without erasing adaptive gains.


A
Everest tests are likely to fail following extensive mutation, preventing mating
High mutation rate (orange)
Everest test failure threshold
Accumulated mutations
Parent A
Mating event
Offspring A
Offspring B
Offspring C
Parent B
Low mutation rate (brown)
Teal steps indicate rare mutations improving test performance
Mate C
Time


B
Changed environment
Low-fidelity lineage declines
Well-adapted, stable lineage
Everest recombination
Original lineage declines or is outcompeted
Accumulated mutations
Time

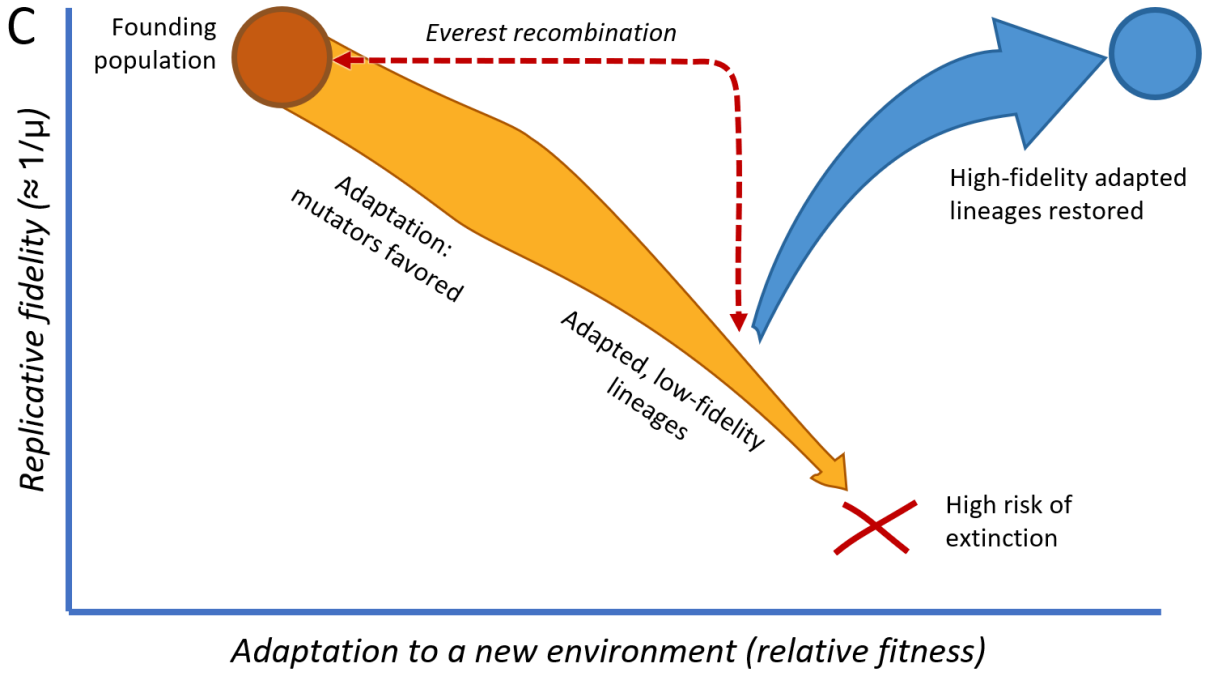

C
Founding population
Everest recombination
Adaptation: mutators favored
Adapted, low-fidelity lineages
High-fidelity adapted lineages restored
High risk of extinction
Replicative fidelity (≈ 1/μ)
Adaptation to a new environment (relative fitness)

Figure 4. Everest predicts that mate choice can expose mutator-linked damage and favor recombination that restores high fidelity without erasing adaptation. (A) Individual-level schematic of accumulated mutations through time. Low-mutation-rate parents produce offspring that differ in subsequent mutation accumulation: one acquires a mutator allele (orange), accumulates mutations rapidly, crosses the Everest test-failure threshold (red dashed line), and is unlikely to mate; another retains a low mutation rate (brown), remains below the threshold, and can produce low-mutation-rate descendants. Teal steps denote rare mutations improving test performance; dashed connectors show parentage. (B) Population-level mutation dynamics. During a changed environment (shading), a mutator lineage (orange) adapts quickly but accumulates mutations unsustainably and later declines; a low-mutation-rate lineage (brown) adapts more slowly and may decline or be outcompeted. Everest recombination—recombination between adapted low-fidelity genotypes and high-fidelity partners—followed by reproductive screening, yields a lineage (blue) that combines adaptation with restored fidelity. (C) The same scenario in adaptation–fidelity space, where fidelity ≈ 1/μ and μ is mutation rate. Mutator lineages move toward higher adaptation but lower fidelity (orange trajectory, red X); Everest recombination produces recovered lineages that retain adaptation while restoring high fidelity (blue). Curves are qualitative, not fitted data.

The population dynamics underlying the recovery in Figure 4B and C warrant emphasis. During a changed environment, low-fidelity mutator lineages may adapt quickly, rise to high frequency, and temporarily outcompete other lineages. However, their ongoing mutational load can erode fitness even in the new environment, eventually causing their numbers to decline. This reduces pressure on residual high-fidelity lineages, allowing their numbers to rebound and creating favorable conditions for Everest recombination. During colonization or metapopulation exchange, immigration can also replenish high-fidelity genotypes, providing further opportunities for Everest recombination without sacrificing local adaptation.

The hypothesis makes at least four testable predictions. First, under strong selection in a new environment, sexual populations should often evolve well-adapted but higher-mutation-rate lineages. Second, Everest recombination should be able to restore low mutation rates without sacrificing adaptation. Third, greater sexual and reproductive complexity should correlate with stronger Everest effects—lower mutation rates, reduced mutational load, or greater lineage persistence—relative to otherwise similar groups lacking such complexity. Fourth, where populations with different dominant screens interbreed, the Everest effect should be weakened; all else equal, such transition zones should exhibit higher mutation rates or mutational load than stable, well-screened populations on either side. Appendix S2 gives a quantitative argument showing how reproductive complexity can convert small per-locus mutation-risk differences into appreciable selection against mild mutators. When many mutation-sensitive loci contribute to a shared mating or fertilization screen, the implied selection coefficient against mild mutators can reach several percent.

The mate-selection strategies envisaged here can also reveal genetic defects beyond DNA replication. Complex behaviors and displays may be sensitive to weakly acting mutations in housekeeping genes, including those involved in ribosomal function, cell cycle control, mitochondrial metabolism, transcription, translation, and so on.

Relations to previous work

Before turning to natural phenomena that Everest might help explain, it is useful to compare the hypothesis with existing frameworks for sexual traits, recombination, and mutation-rate evolution.

Everest is closest to good-genes and condition-dependent signaling models [Hamilton & Zuk, 1982; Rowe & Houle, 1996], which treat elaborate traits as proxies for genome-wide mutation load. It differs by treating replication fidelity as a separable evolutionary variable rather than subsuming it into “condition,” by allowing adaptation and fidelity to move in opposite directions during environmental change, and by emphasizing that effective fidelity screens should be shared, complex, and multigenic within each mating network. This creates a Fisherian-like pressure toward shared standards, but with a different target: replication fidelity, a measurable property, rather than

attractiveness alone. This may help explain why sexual traits are often stereotyped locally yet geographically variable.

Everest complements rather than replaces Fisherian and handicap frameworks [Fisher, 1930; Zahavi, 1975]. Traits elaborated by runaway or handicap dynamics can be co-opted as fidelity-linked reproductive screens—a peacock's train may simultaneously reflect Fisherian selection, honest condition signaling, and sensitivity to genome-wide mutational damage. A key distinction from pure handicap models is that a handicap requires only that a signal be costly enough to preclude falsification, whereas Everest predicts that complexity itself is adaptive: multigenic traits amplify mutational damage into visible reproductive failure in a way that simple costly traits cannot.

On mutation-rate evolution, Everest proposes a phenotypic mechanism complementing drift-barrier theory: demanding reproductive screens translate small fidelity differences into large differences in mating or fertilization success. Roberts and Petrie [2022] describe the accelerator phase, in which mate choice can transiently favor mutator backgrounds during adaptation. Everest adds the brake: recombination with high-fidelity partners, followed by reproductive screening, can restore fidelity after adaptation. Fuller comparison with these and other frameworks appears in Appendix S3 and Table 1.

Natural phenomena that the Everest hypothesis may help to explain

Many traits and behaviors appear exceptionally costly, risky, or intricate, yet persist and often become highly stereotyped. Everest interprets such traits as potential fidelity-sensitive screens, without claiming to explain why any particular trait originated. Once a demanding, multigenic reproductive trait exists, whatever its origin, it can be co-opted as an Everest test, incidentally filtering lineages carrying elevated mutational damage or elevated mutation rates. The following examples illustrate this logic and generate testable predictions; they are not a comprehensive survey or replacements for existing explanations of trait origins.

(1) Long-distance migration. Arctic terns, Atlantic salmon, and Monarch butterflies undertake demanding journeys that require tight coordination of navigation, physiology, and timing (Figure 5). Individuals with even slightly compromised genomes may fail at one of many steps—orientation, endurance, homing, or timing—so persistent, repeated migration could disproportionately remove lineages carrying elevated mutational damage, with lower mutation rates emerging over time. Monarch butterflies pose a further challenge: their migration is a multi-generation relay, so northerly-breeding insects must faithfully transmit alleles that control overwintering behavior they themselves never experience.

(2) Elaborate displays in birds. Birds-of-paradise, peafowl, and many songbirds display striking combinations of plumage, courtship choreography, and complex song. These traits are developmentally and neurologically demanding, relying on many genes and extended learning. Their extravagance may make small losses in replication fidelity visible before mating. The peacock's train illustrates this: development depends on many genes acting in concert, so small defects can visibly degrade the result.

(3) Multifactorial mate selection. Observational data complicate simple predictions about ornament preferences. Takahashi et al. found that peahens in Japan did not prefer males with more symmetrical trains, more ocelli, or longer trains, and noted that male variation was, in any case, low [Takahashi et al., 2008]. One Everest interpretation is that past selection may already have removed many low-fidelity individuals so peahens now treat a well-formed train as an "entrance exam" and base final choice on additional traits—consistent with multifactorial mate selection that combines fidelity filtering with selection for current fitness.

(4) Lekking. In lekking systems, males aggregate at display sites where females choose mates freely, with no resource transfer. Lekking is an extreme form of sexual selection and one of its most persistent puzzles. The costs to males are severe, and the costs to females can also be substantial: female Galápagos marine iguanas incur significant physiological costs simply from searching and evaluating males on leks [Vitousek et al., 2007], female black grouse with higher body mass visit more males than lighter females [Rintamäki et al., 1995], and female pronghorn reduce mate-search effort markedly during dry summers when body condition is compromised [Byers et al., 2006].

Everest interprets lekking as a high-intensity version of the accelerator/brake logic. During environmental change, intense female choice can accelerate adaptation by concentrating reproduction on males carrying beneficial mutations, including those arising from mutator backgrounds. But the same system can later act as a powerful brake: once elevated mutation rates begin to degrade the many traits required for lek success—survival, stamina, display, coordination, competitive position, and attractiveness—female choice imposes steep reproductive penalties on mutator lineages. Lekking may therefore be especially effective at driving the rise-and-fall dynamic that Everest predicts in response to environmental fluctuation.

The framing offers a possible resolution of the lek paradox, which posits that if females choose only among ordinary fitness alleles, strong directional selection should erode genetic variation in male quality, eventually making choosiness self-defeating [Kirkpatrick & Ryan, 1991; Rowe & Houle, 1996]. Roberts and Petrie help explain why genetic variation is regenerated during episodes of rapid adaptation: mutator backgrounds produce beneficial variants, and sexual selection can amplify their success [Roberts & Petrie, 2022]. Everest completes the cycle by adding the corresponding purging phase. In this way, lekking can both regenerate variation during adaptation and later remove the low-fidelity backgrounds on which some of that variation arose.

When environments change, signals of current adaptation should become relatively more valuable than stringent fidelity filtering, and the cost of mate assessment may also rise. Everest therefore predicts that females should relax costly lek-based assessment under ecological stress—a pattern consistent with the reduced mate-search effort reported in pronghorn and black grouse.

(5) **Conformity of appearance.** Many birds and other animals within a species look remarkably similar; individual tits, jays, or gulls can be hard for humans to distinguish. Such conformity suggests strong stabilizing selection around a multigenic template of acceptable appearance: individuals whose development is perturbed by mutational damage are more likely to deviate from the template and suffer reduced mating success. Ridley reports that male black grouse observed on leks are difficult to distinguish unless injured or deformed, whereas females vary more visibly in barring and coloration [Ridley, 2025]. Where both sexes are choosy, Everest would predict that such templates are maintained in both sexes. Eurasian jays, for example, have small but conspicuous patches of black, white, and bright-blue barred feathers on the wing in both males and females, potentially providing a shared ornament in which developmental deviations become obvious. Although these observations are not direct tests of Everest, they are consistent with the prediction that shared mate-choice criteria can impose strong stabilizing selection on appearance, especially in the sex or sexes subject to the strongest reproductive screening.

(6) **Symmetry.** Bilateral and radial symmetry are widespread targets of mate choice across taxa, as illustrated in Figure 5. Producing a symmetrical body requires the same developmental program to be executed with precision on both sides—a demanding test sensitive to mutational noise. Partially defective proteins can lead to inconsistent developmental outcomes; compromised immunity can cause infections that affect one side more than the other; and aberrant behavior can cause asymmetric physical damage. Preferences for symmetry have been documented in humans evaluating potential partners [Rhodes et al., 2001], moths [Koshio, 2007], flower-visiting pollinators, female guppies, and female barn swallows (see the legend of Figure 5 for other references). The Everest hypothesis interprets these preferences as mechanisms that make mutator-linked developmental damage visible to mate choice: symmetry suggests that the many genes underlying development have functioned reliably during growth.

Figure 5. Examples of complex or stereotyped traits that could serve as integrity-sensitive screens under Everest. Top row: long-distance migrants—Arctic tern, Atlantic salmon, monarch butterfly—undertake journeys requiring coordinated navigation, physiology, and timing; successful migrants may therefore be biased toward lineages with lower accumulated damage or higher replication fidelity. Middle row: the peacock's train depends on coordinated expression of many developmental genes; bee orchid mimicry requires precise signal production; female barn swallows prefer males with long, symmetric tails [Bańbura, 2005]. Bottom row: female guppies favor symmetrical males with larger orange ornaments [Stephenson et al., 2020]; *Macrocilix maia* mimics flies near a bird dropping, yet retains near-perfect bilateral symmetry. Because avian predators can use symmetry as a detection cue [Merilaita & Lind, 2006], this maintained symmetry is consistent with countervailing selection for developmental precision or genomic integrity; pollinators prefer symmetrical flowers such as arugula (*Eruca vesicaria*) [Møller & Eriksson, 1995]. Everest interprets performance, precision, or symmetry as potential filters: successful execution or well-formed ornaments suggest that many underlying loci are intact, consistent with lower accumulated damage and, over generations, lower germline mutation rates. These examples illustrate Everest's logic and suggest testable predictions, not a systematic survey.

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(7) Ephemeral winged insects. Mayflies and periodical cicadas invest heavily in a brief winged adult phase, devoted almost entirely to mating, often with tightly synchronized emergence. Success depends on accurate development, precise timing, functional wings and sensory systems, and coordinated mating behavior—a sharp multigenic bottleneck in which lineages carrying subtle defects are more likely to fail.

(8) Complex signaling in invertebrates. Many invertebrates use elaborate, species-specific mating signals. Fireflies communicate via encoded light flashes; some flies and spiders perform complex dances; and fiddler crabs wave enlarged claws in stereotyped patterns while females assess multiple aspects of the claw signal and burrow quality. These behaviors depend on neural, muscular, and sensory systems encoded by many genes, functioning as real-time assays of genomic integrity.

(9) **Molecular compatibility in sessile organisms.** Corals and broadcast-spawning marine organisms rely on synchronized spawning and intricate gamete-recognition systems. Flowering plants use complex pollen–pistil recognition systems and multilayered signaling pathways to guide pollen tubes (Figure 6). Fungi and some protists have multigenic mating-type and compatibility systems. These mechanisms involve many interacting receptors, ligands, and regulatory genes—potential fidelity-linked screens expressed through fertilization success rather than behavioral choice.

(10) **Floral complexity in angiosperms.** Many angiosperm species have structurally complex blooms whose shapes, colors, scents, and pollination routines are learned by particular pollinators [Chittka & Raine, 2006]. Plants with slightly deformed flowers due to mutation are likely to receive fewer visits. Because most angiosperms lack an early-segregated germline, low mutational load in floral tissues likely correlates with low load in gametes, so floral complexity could act as a pollinator-mediated fidelity test.

(11) **Sperm competition.** In many animals, sperm from different males compete within the female tract, imposing strong selection on sperm number, form, and performance [Møller & Cuervo, 2003]. Lineages with elevated mutation rates are expected to produce defective sperm that fail to fertilize more often, so that high-fidelity lineages contribute disproportionately to fertilizations.

Two further extensions are discussed in the supplementary material. Appendix S4 considers how the Everest logic might apply to asexual organisms, where reproductive screening by mate choice is absent but selection may still act on complex performance traits or life-cycle bottlenecks. Appendix S5 considers pregnancy loss as a possible distributed fidelity-sensitive screen in viviparous animals, while emphasizing that this remains a speculative extension of the hypothesis.

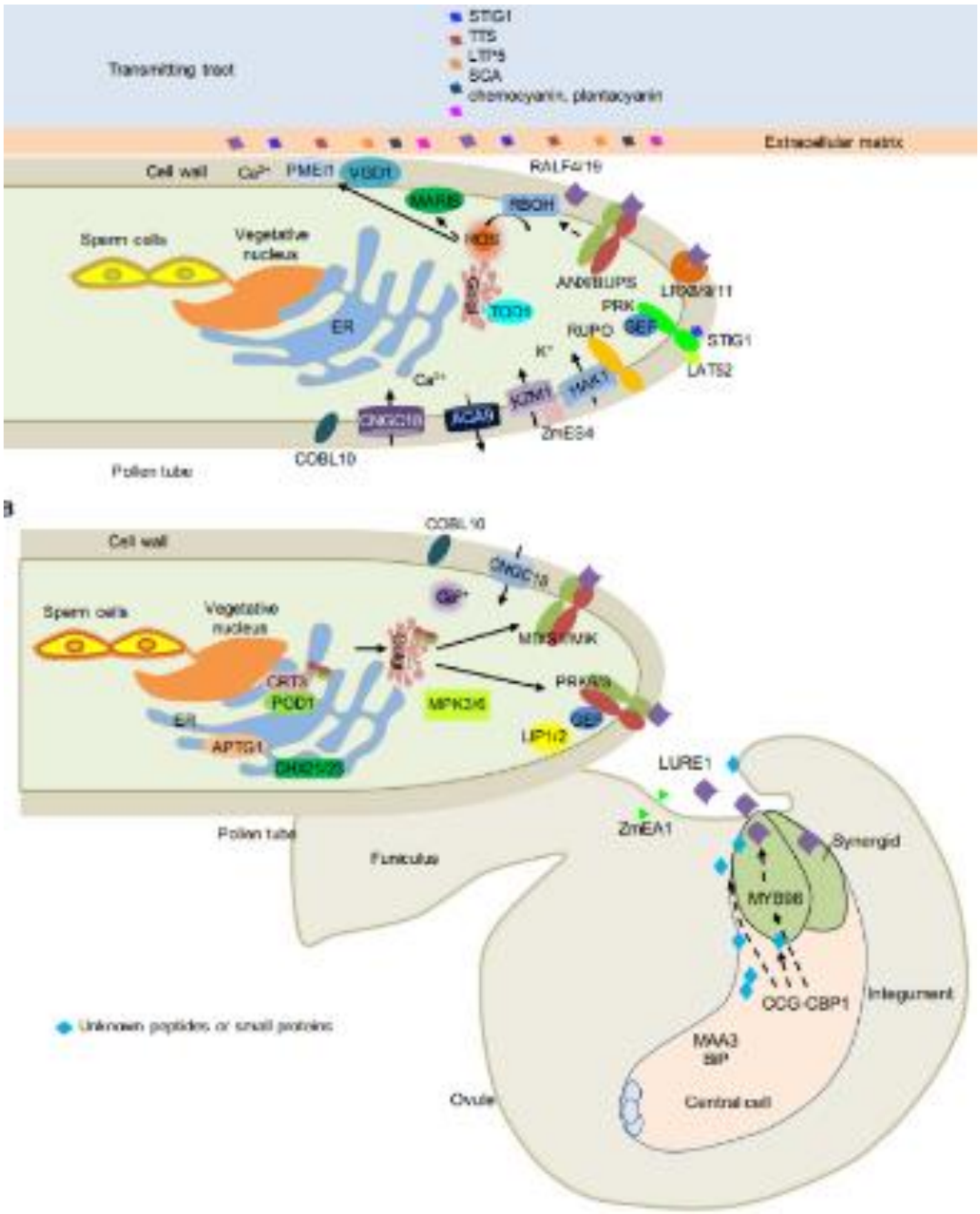


Figure 6. Pollen tube guidance as a molecular Everest test. Fertilization in flowering plants requires pollen tube growth through maternal tissue and precise navigation to the ovule, mediated by successive cell–cell interactions and multilayered biochemical signaling [Li et al., 2018]. Pollen grains function as mobile haploid organisms whose success depends on many gene products. This complexity could impose a molecular reproductive screen, making fertilization success sensitive to mutational damage and thereby illustrating how elaborate fertilization systems could act as fidelity-linked screens. Figure reproduced from Li HJ, Meng JG, Yang WC, "Multilayered signaling pathways for pollen tube growth and guidance," *Plant Reproduction* 31, 31–41 (2018), by permission of Springer Nature.

Levels of selection: individual selection and lineage sorting

The Everest hypothesis operates through individual-level selection, not group selection. Individuals with higher replication fidelity perform better on Everest tests and gain a direct reproductive advantage; individuals carrying mutator alleles perform worse and leave fewer descendants. This is the standard population-genetic argument that mutators are purged through linkage to the harmful mutations they generate [Kimura, 1967]. Everest does not change that logic but amplifies the

selective signal until selection that would otherwise be too weak to overcome drift can purge mild mutators and favor mild antimutators. No individual sacrifices fitness for group benefit, and no group-level selection coefficient is required.

This perspective has a lineage-level consequence: lineages lacking effective fidelity screens are expected to accumulate greater mutational load and face elevated extinction risk, not because rival groups outcompete them but because they collapse from within. This may explain why costly Everest tests persist despite their burden. Complex organisms with large genomes may be less able to persist in unstable environments without effective fidelity screens. On this view, Everest tests may be more than a refinement of sexual selection: they may contribute to the long-term survival of genomic complexity in a fluctuating world. This is better described as lineage sorting than group selection.

Existing experimental evidence: support and limitations

(1) **Experimental studies consistent with fidelity filtering.** A few experimental evolution studies provide indirect support for the Everest hypothesis, although it has not yet been tested directly. In flour beetles, sexual selection reduced extinction risk and purged deleterious mutations [Lumley et al., 2015]. In seed beetles, males from sexually selected lines transmitted smaller induced mutation loads following germline DNA damage, suggesting a link to germline maintenance [Baur & Berger, 2020]. In hermaphroditic freshwater snails, sexual selection similarly contributed to the purging of deleterious mutations across multiple generations [Noël et al., 2019]. Collectively, these studies show that sexual selection can reduce mutation load, but a direct test of the Everest hypothesis would require linking success in demanding reproductive traits to independently measured germline mutation rates—for example, through parent–offspring whole-genome sequencing.

(2) **Experimental studies that seem relevant to the Everest hypothesis without directly testing it.** Many experimental studies in sexual selection fall into this category. Carotenoid-supplementation experiments in zebra finches altered immune function and sexual attractiveness [Blount et al., 2003], but addressed current condition and ornament expression rather than heritable mutation rate or replication fidelity. Field experiments in common yellowthroats linking ornamentation to oxidative DNA damage likewise concern physiological maintenance rather than transmission of germline mutations [Freeman-Gallant et al., 2011]. Long-term experimental evolution studies under altered mating systems are also only indirectly relevant. In *Drosophila melanogaster*, enforced monogamy altered genome-wide gene expression after many generations [Hollis et al., 2014], and experimental hyper-promiscuity produced evolutionary responses to the altered sexual environment [Perry et al., 2016]. In *Drosophila pseudoobscura*, long-term manipulation of sexual selection generated substantial genomic divergence between mating regimes [Wiberg et al., 2021]. These studies demonstrate that sexual selection can reshape traits, gene expression, and genomes over time, but they do not test the narrower Everest claim that demanding reproductive traits function as fidelity-linked screens by making mutator-linked damage visible to selection.

Experimental and observational tests of the Everest hypothesis

(1) Experimental evolution in laboratory and captive populations. A variety of sexual organisms could be used to test the Everest hypothesis in laboratory evolution experiments, including protists, plants, insects (such as *Drosophila*, flour beetles, and seed beetles), fish, wild birds, and captive and farm mammals and birds. The design in Figure 7A and Box 1 asks whether strong selection affects replication fidelity (L2 versus L1) and whether recombination with wild-type strains can restore it (L4 versus L3), with whole-genome sequencing likely needed for unambiguous interpretation; similar approaches could be extended to zoo populations. Figure 7B maps the predicted trajectories of each line onto adaptation–fidelity space, with the L3–L4 comparison testing whether recombination can restore fidelity while preserving adaptation. In all such designs, replicated lines and controls for effective population size, inbreeding, and demographic bottlenecks would be essential, because these factors can also affect mutation load.

(2) **Sequencing existing laboratory-evolved lines.** A complementary approach would be to revisit laboratory lineages already evolved under altered mating systems—such as enforced monogamy, hyper-promiscuity, reduced sexual selection, or altered mate choice. Whole-genome sequencing of archived or repeatable lines could test whether relaxation or redirection of reproductive filtering changed mutation rate, mutational load, or allele frequencies in replication and repair genes. Experiments such as Perry et al.'s hyper-promiscuous *Drosophila* study are useful models because they show that genetic manipulation of the socio-sexual environment can rapidly reshape reproductive traits, but the authors did not directly measure replication fidelity [Perry et al., 2016].

(3) **Experimental manipulation of cricket song.** A workable investigation into the role of Everest loci could use crickets by disabling a major target of mate choice: the male calling song. In one line of a genetically mixed population captured before mating, males would be muted by applying non-toxic glue or filler to the file and scraper on their forewings, while control males would receive a sham treatment on non-stridulatory regions of the forewings, leaving their song intact. After several generations, whole-genome sequencing of parent–offspring trios (or pooled samples) from each line could estimate per-generation mutation rates and changes in replication and repair genes. The Everest hypothesis predicts that the song-muted line should more readily retain mutator alleles or accumulate a greater mutational load than the control, because removing the screen relaxes reproductive filtering.

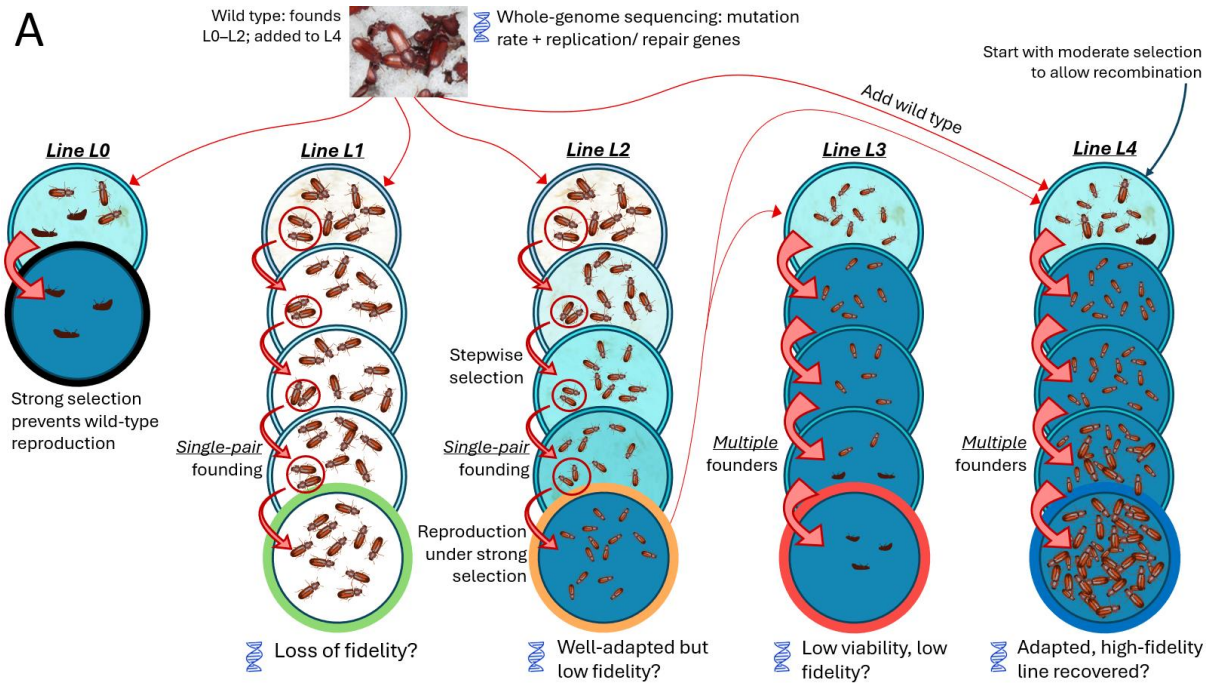
(4) **Extension to birds and mammals.** It would be valuable to extend experimentation to birds and mammals more broadly. The experimental approach of manipulating ornament elaboration has been established in widowbirds [Andersson, 1982] and peafowl [Petrie & Halliday, 1994], among others. Shortening or obscuring key ornamental traits to prevent females from using full elaboration in mate choice, then measuring germline mutation rates across males, could provide a direct test of whether ornamental elaboration functions as a fidelity-linked screen in these systems.

(5) **Experiments with lekking animals.** Lekking species might seem attractive for testing Everest because highly successful males are easy to identify in some systems, such as black grouse. However, the predicted relationship between lek success and replication fidelity is context-dependent. Highly successful males are expected to show lower mutation rates or lower mutational load only where the environment has been stable long enough for lekking to operate mainly as a fidelity filter. During environmental change, successful males may instead include individuals from mutator backgrounds carrying recent beneficial variants. This makes lekking a potentially informative but difficult test case. Moreover, if lekking already functions as a strong fidelity filter, surviving males may differ only subtly in fidelity, reducing statistical power. Any paternal signal will also be diluted because mothers contribute equally to offspring replication fidelity. Nevertheless, useful opportunities may exist. In black grouse and other intensively studied lekking species, long-term field datasets with archived genetic samples may allow tests for associations between male attractiveness, environmental context, and sequence variation in DNA replication and repair genes. Similar approaches may be feasible in other lekking or lek-like systems, including tephritid fruit flies, lek-forming *Drosophila*, and fiddler crabs.

(6) **Experimental manipulation of pollination in flowering plants.** A parallel test could be conducted in a plant that is easy to grow and manipulate experimentally. A genetically mixed population would be split into two lines: in a restricted pollination line, each maternal plant would be hand-pollinated with pollen from randomly chosen single donors, while in a control line, plants would be left to open pollination, allowing pollen from many donors to compete and interact with the pistil. After several generations in a common garden, lines would be compared by whole-genome sequencing with attention to replication and repair genes. The Everest hypothesis predicts that removing pollen competition and reducing pistil-level screening should allow greater accumulation of deleterious mutations (and possibly reduce vigor) in the restricted line (Figure 3A).

(7) **Comparative and population-genetic tests of migration.** The hypothesis predicts that long-distance migrants should have lower mutation rates than comparable non-migratory relatives—a prediction that can be approached in several complementary ways. First, existing ringing and mark–recapture survival data, combined with phylogenetic comparisons of

migratory and resident lineages, could test whether migratory species tend to be longer-lived than closely related non-migratory counterparts, since longevity is one possible consequence of more effective mutator purging. Second, and more directly, sequencing nestlings from migratory and non-migratory populations of partially migratory species—such as European robins—could estimate per-generation mutation rates and test whether migration is associated with lower germline mutation rates. Third, genome-wide data from transitional areas where migratory and resident populations interbreed could be used to ask whether genetic introgression is asymmetric—that is, whether alleles move more often from one population into the other than vice versa. After controlling for population size, demography, dispersal opportunity, and geographic structure, Everest predicts that alleles from migratory populations should more often enter resident populations than the reverse. Such a pattern would not by itself prove higher replication fidelity in migrants, but it would provide a testable population-genetic signature consistent with migration acting as a demanding fidelity-linked screen.



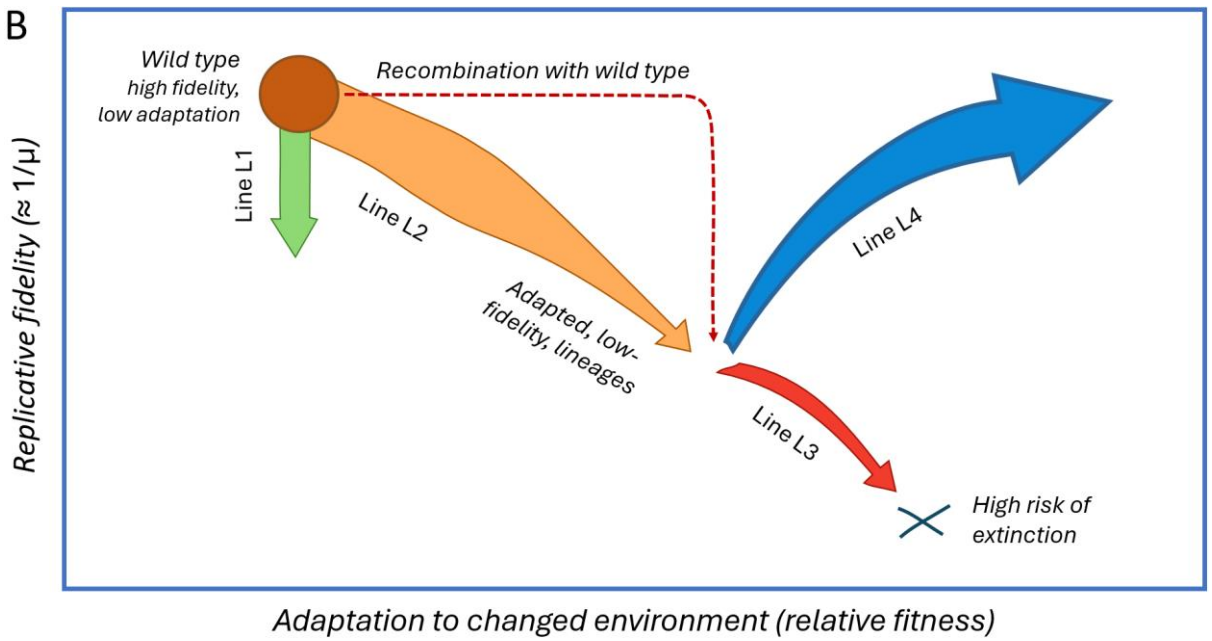


Figure 7. Experimental test of Everest in a sexual model system such as flour beetles. (A) Experimental design. Populations are exposed to increasingly severe selective environments, shown by darker blue circles. L0 identifies conditions that just prevent wild-type reproduction. In L1 and L2, each generation is founded by a single pair to restrict recombination: L1 is a bottleneck control without strong selection, whereas L2 experiences stepwise increasing selection and is predicted to favor mutator genotypes with reduced replication fidelity. From L2, two lines are derived. L3 continues strong selection without backcrossing but with multiple individuals transferred each generation, predicting further fidelity loss and increased extinction risk. L4 introduces wild type to allow recombination, then continues strong selection with multiple individuals transferred each generation, testing whether high fidelity can be restored without erasing adaptation. Whole-genome sequencing would estimate mutation rate and detect changes in replication and repair genes. (B) Predicted trajectories in adaptation–fidelity space, with axes as in Figure 4C. The wild-type population (brown) begins with high fidelity but low adaptation. L1 (green) represents bottlenecking without strong selection; L2 (orange), adaptation under stepwise selection with restricted recombination, yielding adapted but lower-fidelity lineages; L3 (red), continued strong selection without backcrossing, predicted to result in further fidelity loss and high extinction risk; and L4 (blue), recombination with wild type followed by strong selection, predicted to give restored fidelity with maintained adaptation. The key test is whether L4 restores fidelity relative to L3 while preserving adaptation. Other experiments discussed in the main text provide additional tests of these trajectories.

Box 1. Experimental readouts and predictions for the design shown in Figure 7

Measurements

1. Per-site mutation rate in individuals from each line (L0–L4), estimated from whole-genome sequencing data
2. Sequence changes and allele frequencies in replication and repair genes, including DNA polymerases and their associated factors
3. Distribution of fitness effects of new mutations, assessed through competition assays in the selective environment
4. Line viability and population dynamics under strong selection (extinction risk, population size, body size)

Null expectations: no detectable mutation-rate effect

If adaptation to the selective environment is independent of mutation-rate evolution:

- Wild type and L1, the bottleneck control, should show similar mutation rates and no consistent changes in replication or repair genes beyond drift
- L2, L3, and L4 may become better adapted to the selective environment but should not differ systematically in mutation rate or replication-gene profiles
- Recombination with wild type in L4 should not restore mutation rates below those in L2 and L3

Patterns supporting the Everest hypothesis

- L2 shows higher mutation rates and more changes in replication or repair genes than L0 and L1
- L3 shows continued elevation of mutation rate, increased mutational load, and reduced viability
- L4 shows lower mutation rates, fewer mutator alleles, and higher viability than L2 and L3, indicating recovery of high-fidelity lineages through recombination

Patterns that would challenge the hypothesis

- L2 and L3 show no consistent increase in mutation rate or changes in replication or repair genes relative to controls
- L4 fails to recover lower mutation rates or improved viability despite the opportunity for recombination with high-fidelity genotypes

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Conclusions

(1) The Everest hypothesis rests on four propositions: environmental change can shift mutation-rate evolution toward lower fidelity by increasing beneficial mutations, weakening selection against mutators, and allowing mutator alleles to hitchhike with adaptive sweeps; many lineages reproduce through systems whose elaboration exceeds what viability-fecundity benefits or fertilization requirements alone can explain; this elaboration enlarges the mutational target associated with reproduction; and mutations at those loci often reduce mating or fertilization success. If these propositions are broadly correct, replication fidelity and reproductive success must be coupled to some degree. Even mild mutators—whose effects may be too weak for ordinary viability-fecundity selection to purge reliably—should therefore suffer reproductive penalties. The question for future work is not merely whether such coupling can arise, but where it is strongest, how often it has evolutionary consequences, and how much it contributes to genomic integrity.

(2) The hypothesis suggests that evolutionary change may not be fully captured by a single fitness dimension. Short-term fitness and replication fidelity can move in opposite directions; mutation-rate evolution during environmental change therefore requires a two-dimensional framework in which fidelity is treated as an independent axis rather than absorbed into fitness (Figure 4C).

(3) Everest is motivated by the problem of conserving genomic integrity during environmental change—a problem that may be underappreciated because successful lineages have already evolved mechanisms that limit loss of fidelity. Elevated mutation rates can be advantageous when conditions shift, but threaten long-term genome maintenance if sustained [Lynch et al., 2023]. By making mutator-linked damage visible to mate choice or fertilization, reproductive complexity allows selection to act on variation that would otherwise be weakly expressed or hidden.

(4) A distinctive prediction is a rise-and-fall dynamic. Sexual selection can act first as an accelerator and later as a brake. During environmental change, mutator lineages may be transiently favored because they generate beneficial variation and high-performing individuals. After conditions stabilize, however, elevated mutation rates may degrade complex reproductive traits. Everest recombination—between adapted, low-fidelity genotypes and remaining or immigrant high-fidelity partners, followed by reproductive screening—then allows lineages to retain adaptive gains while restoring replication fidelity.

(5) Everest complements rather than replaces Fisherian, handicap, and good-genes models [Fisher, 1930; Zahavi, 1975; Hamilton & Zuk, 1982]. Its distinct claim is that traits elaborated by those processes can later be co-opted as fidelity-linked reproductive screens, making otherwise subtle mutator-linked damage visible to selection.

(6) The logic extends beyond animals with overt behavioral choice to taxa in which filtering occurs through fertilization success, gamete competition, or molecular recognition. Everest predicts that fidelity-linked reproductive screening based on excess reproductive complexity is widespread among sexually reproducing organisms, including forms where the relevant complexity is cryptic, molecular, or easily overlooked. The effect should be strongest where reproductive success depends on demanding, widely shared criteria, and weakest where those criteria are fragmented.

(7) The hypothesis is testable and falsifiable (Figure 7). Experimental evolution under strong selection, with and without recombination; manipulation of reproductive traits in animals and plants; and direct comparison of germline mutation rates between migratory and non-migratory lineages all provide ways to evaluate whether, and how strongly, reproductive complexity contributes to genomic integrity.

(8) The elaborate accompaniments of sexual reproduction—demanding migrations, intricate courtship displays, complex fertilization systems—should not be dismissed as arbitrary ornaments or isolated adaptations. Given the premises developed here, their role as potential instruments of genomic quality control is a logical prediction of the hypothesis. More broadly, Everest suggests that sexual reproduction may help complex organisms colonize and persist in unstable environments by allowing adaptation during environmental change while restoring replication fidelity afterward. The

question for future research is how often this filtering occurs, how strong it is, and how much it shapes the persistence and adaptability of sexual lineages.

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The Everest hypothesis: Supplementary Material

This Supplementary Material contains: Appendix S1, an individual-based stochastic simulation showing how environmental change can elevate mutator allele frequency through hitchhiking; Appendix S2, a quantitative multi-locus analysis of reproductive screening; Appendix S3, an expanded discussion of Everest in relation to previous work; Appendix S4, a discussion of adaptation–fidelity tradeoffs beyond sexual organisms; and Appendix S5, a speculative discussion of pregnancy loss as a possible distributed fidelity-sensitive screen.

Appendix S1. An Individual-Based Simulation of Mutator Dynamics During Environmental Change

Overview

This section presents an individual-based stochastic simulation that shows how environmental change can elevate mutator allele frequency via mutator hitchhiking. The model tracks 1,024 individuals across 200 generations, each carrying alleles at two loci: a polymerase locus that determines replication fidelity and an adaptation locus that determines fitness in the current environment. The simulation incorporates genetic drift, probabilistic selection, and stochastic mutation events, allowing outcomes to vary across runs. The model is intentionally asexual: permanent linkage between the polymerase and adaptation loci produces a strong hitchhiking signal when adaptive mutations arise on mutator backgrounds. In sexual populations, recombination would weaken individual hitchhiking events by decoupling p from the beneficial mutations that carry it to high frequency—but it would also weaken the subsequent purging of mutators after environmental change, increasing rather than reducing the need for Everest screening. An interactive Excel spreadsheet implementing this model is available as Supplementary File S1, allowing readers to regenerate runs by pressing F9.

Model Description

The model tracks a haploid asexual population of 1,024 individuals across 200 generations. Each individual carries alleles at two loci (genomic positions):

1. *Polymerase locus (P/p):* determines replication fidelity. P = high-fidelity polymerase (low mutation rate); p = low-fidelity mutator polymerase (high mutation rate).
2. *Adaptation locus (A):* determines fitness in the current environment. Three alleles are possible: a1 (adapted to environment E1), a2 (adapted to environment E2), and a3 (degraded, irreversible dead-end allele with low fitness in all environments).

The critical asymmetry is that a1↔a2 transition rates and a1/a2→a3 degradation rates both depend on which polymerase allele is carried: P individuals mutate at rate μ_P per locus per generation, while p individuals mutate at rate μ_p, which is 50-fold higher. Transitions to a3 are irreversible. The polymerase locus itself flips between P and p at a low background rate μ_pol. This is not intended to reflect a specific biological mechanism; it is a modeling convenience that maintains a continuous background frequency of both alleles (~10% p at equilibrium), preventing either allele from being permanently lost by drift.

Dynamics

Each generation proceeds in three steps:

Step 1 – Selection: Each generation, pairs of individuals compete in probabilistic tournaments. The probability that individual A defeats individual B is fitnessA / (fitnessA + fitnessB), where fitness is determined by the adaptation locus and current environment (Table S1). The winner reproduces; this is repeated until 1,024 offspring are produced.

Step 2 – Mutation: Each offspring's adaptation locus mutates with probability determined by its polymerase allele. A mutation at the adaptation locus is deleterious (→a3) with probability given by the degradation probability, and adaptive (a1→a2 or a2→a1) otherwise. Transitions to a3 are irreversible.

Step 3 – Environment: The environment switches from E1 to E2 at generation 40 and returns to E1 at generation 130, creating two successive episodes of environmental change.

Table S1. Parameters

Parameter	Symbol	Value	Description
Population size	N	1,024	Fixed throughout
Generations	—	200	Per simulation run
High-fidelity mutation rate	μ_P	0.002	Mutation rate at the A-locus for P individuals
Low-fidelity mutation rate	μ_p	0.1	Mutation rate at the A-locus for p individuals
Polymerase flip rate	μ_pol	0.01	Background P↔p rate
Deleterious:beneficial ratio	—	20:1	Ratio of degrading to adaptive mutations
Degradation probability	—	0.952	Calculated from that ratio
Environment 2 start	—	Generation 40	Onset of environmental change
Environment 2 end	—	Generation 130	Return to Environment 1

Table S2. Fitness values by genotype and environment

Adaptation allele	Environment E1	Environment E2
a1 (adapted to E1)	1	0.5
a2 (adapted to E2)	0.5	1
a3 (degraded)	0.1	0.1

Table S3. Derived transition rates by polymerase background

Transition	P background	p background
a1 → a2 (adaptive)	0.0001	0.0048
a1 → a3 (deleterious)	0.0019	0.0952
a2 → a1 (adaptive)	0.0001	0.0048
a2 → a3 (deleterious)	0.0019	0.0952
a3 → anything	0	0

Table S3 shows derived values that are not directly editable. The parameters above should be adjusted to update these values in the model.

Initial Conditions

All 1,024 individuals begin as a1 (adapted to E1). Approximately 90% carry P and 10% carry p, reflecting the equilibrium mutator frequency under the chosen polymerase flip rate in the absence of directional selection.

Results

The simulation was run 50 times with independent random seeds. Two single runs are shown in Figure S1, illustrating the two qualitatively distinct outcomes the model produces. For each run, the peak mutator (p) allele frequency reached during the first environmental switch (generations 40–130) was recorded; the resulting distribution across all 50 runs is shown in Figure S2, and individual values are listed in Table S4.

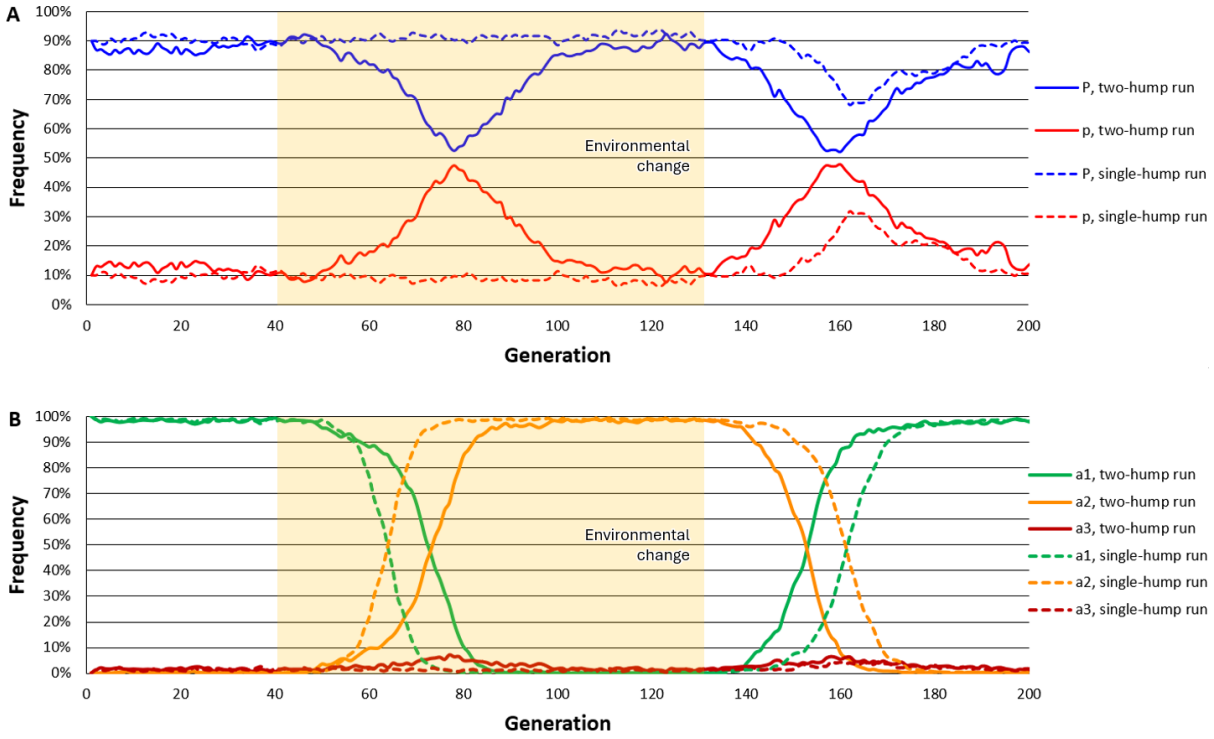

Figure S1. Example trajectories of the individual-based simulation, illustrating two qualitative outcomes summarized in Figure S2. Two runs are superimposed: a representative two-hump run (solid lines), in which the successful adaptive sweep arises on a mutator background at both environmental switches; and a representative single-hump run (dashed lines), in which the first adaptive sweep arises on a high-fidelity background, while the second arises on a mutator background. (A) Polymerase allele frequencies: P, high-fidelity polymerase, shown in blue; p, mutator polymerase, shown in red. When the successful adaptive allele arises on a p background, $p$hitchhikes upward with the spreading adaptive allele; when it arises on a P background, $p$remains near baseline. The single-hump outcome is therefore an expected stochastic result, not a failure of the model: the background that first supplies a successful adaptive variant can be either Por p, depending on mutation supply, starting frequency, degradation risk, and chance establishment. (B) Adaptation allele frequencies: a_1, adapted to E1, shown in green; a_2, adapted to E2, shown in orange; and a_3, degraded, shown in dark red. Adaptation succeeds in both runs; the runs differ in which polymerase background hosts the adaptive sweep, not in whether adaptation occurs. Recovery of high fidelity during stable phases reflects conventional selection against mutators through the adaptive locus; Everest predicts that this recovery should be amplified by multi-locus reproductive screens and steep mate-choice functions, as discussed in Appendix S2. Shading indicates the period of environmental change, generations 40–130. Parameter values are as in Tables S1–S3.

In this simulation, the recovery of high fidelity during stable phases is driven by conventional Kimura-style linkage logic: mutators are selected against because they remain linked to the deleterious mutations they generate at the adaptive locus. The Everest hypothesis predicts that this recovery should be stronger in sexual populations where many mutation-sensitive loci are stacked into shared reproductive screens, and where mate choice or fertilization success imposes a steeper reproductive penalty than ordinary viability–fecundity selection. Appendix S2 explicitly develops this quantitative

amplification, showing how increasing the number of mutation-sensitive loci and the steepness of the reproductive response can accelerate the recovery of high fidelity beyond that expected from conventional selection acting through ordinary adaptive or housekeeping loci.

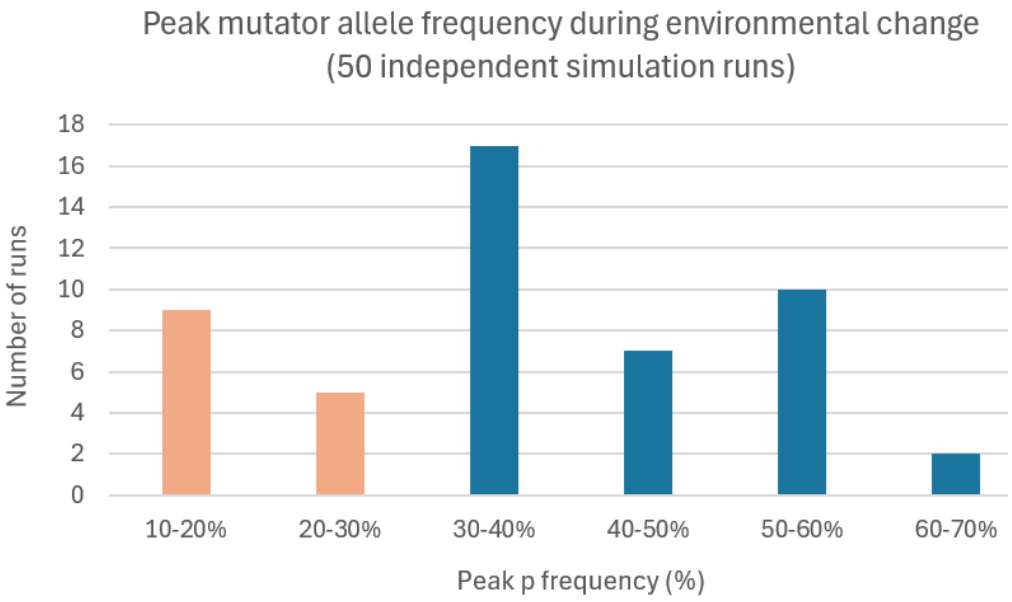

Figure S2. Distribution of peak mutator (p) allele frequency across 50 independent runs of the individual-based simulation. For each run, the maximum frequency reached by the mutator allele *p* during the first environmental switch (generations 40–130) was recorded; the 50 values are shown binned in 10% intervals. The distribution is bimodal, reflecting two qualitatively distinct outcomes. In 14 of 50 runs (orange bars, peak $p \leq 30\%$), the first adaptive $a_1 \rightarrow a_2$ mutation arose on a high-fidelity (P) background; the adaptive sweep carried no mutator with it, and peak *p* frequency remained at or near the pre-switch baseline of ~10%. In the remaining 36 of 50 runs (72%, blue bars, peak $p > 30\%$), the first adaptive mutation arose on a mutator (p) background; *p* hitchhiked with the spreading a_2 allele and rose substantially in frequency (mean peak across all 50 runs: 37.1%). The bimodality reflects the stochastic race between P- and p-background lineages to generate the first adaptive mutation: which background wins this race is decided early and largely determines the subsequent trajectory of mutator frequency.

Table S4. Peak mutator allele frequency recorded during 50 independent runs of the model during the first environmental switch (generations 40-130).

35.4%	43.1%	52.8%	33.0%	14.5%
33.0%	38.3%	34.0%	38.8%	39.3%
48.2%	44.3%	35.4%	27.9%	41.9%
35.3%	11.8%	38.1%	15.1%	21.7%
65.2%	64.3%	39.9%	51.0%	31.4%
44.1%	43.3%	52.7%	53.3%	24.8%
56.4%	50.0%	37.9%	31.7%	13.2%
37.4%	13.2%	56.4%	25.0%	52.4%
56.1%	57.9%	13.9%	14.6%	55.2%
14.9%	32.0%	27.0%	36.9%	19.5%

The distribution of peak p frequency was bimodal (Figure S2), reflecting the two outcomes shown in Figure S1. In 36 of 50 runs (72%), the adaptive sweep occurred predominantly on a mutator background (Figure S1, solid lines), driving peak p frequency above 30% during the first switch (mean peak across all runs: 37.1%). In the remaining 14 runs, adaptation proceeded on a high-fidelity (P) background during the first switch (Figure S1, dashed lines), and mutator frequency remained near the pre-switch baseline of ~10%. This bimodal pattern reflects the stochastic race between P-background and p-background individuals to generate the first adaptive a_2 mutation: when a p-

background individual wins, it hitchhikes with the spreading a_2 allele, substantially elevating mutator frequency; when a P-background individual wins, mutator frequency remains low despite the elevated mutation rate of p individuals.

The absence of mutator hitchhiking at the first switch in the single-hump run is an expected stochastic outcome, not a failure of the model. Although mutator-background individuals generate adaptive mutations at a higher per-individual rate, high-fidelity individuals are more numerous at baseline and are less likely to degrade to a_3. The successful adaptive mutation can therefore sometimes arise and establish on a P background, leaving p near its baseline frequency during that switch. The minority outcome should become progressively less common as the number of loci required for adaptation increases, because multi-locus adaptation gives mutator backgrounds more opportunities to generate the required variants. Since real adaptive episodes often involve many loci, the single-locus model likely underestimates mutator hitchhiking in nature. The 72% hitchhiking rate observed here is therefore best read as a conservative lower bound under these parameters.

Parameter Justification

Parameter values were chosen to be within the range observed in microbial experimental evolution systems, where mutator dynamics are best documented. The 50-fold difference between μ_P and μ_p is consistent with that observed in mutator strains of bacteria and RNA viruses [Sniegowski et al., 1997]. The background mutator frequency of ~10% is consistent with observations of mutator frequencies in natural and experimental bacterial populations [Sniegowski et al., 1997]. The deleterious:beneficial ratio of 20:1 is more favorable to beneficial mutations than most empirical estimates for stable environments, but is appropriate here as the model represents conditions of active environmental change, during which the proportion of beneficial mutations is expected to be substantially elevated [Lynch et al., 2023]. The population size of 1,024 is smaller than most natural populations; small Ne is conservative in the context of this model, as it represents conditions where selection against mutators is weakest, and Everest screening therefore matters most.

Model Limitations

This model deliberately simplifies several aspects of real biological systems: (1) only a single adaptation locus is modelled, whereas real adaptive episodes likely require changes at multiple loci, which would amplify the mutator hitchhiking effect; (2) the population is asexual, which maximises hitchhiking through permanent linkage — in sexual populations recombination partially decouples the polymerase and adaptation loci, weakening individual hitchhiking events but creating the problem that Everest screening is proposed to solve; (3) the model represents a single class of loci rather than a whole genome; (4) population size is fixed. Despite these simplifications, the model captures the essential dynamics: environmental change creates conditions under which mutator alleles rise substantially in frequency through hitchhiking with adaptive sweeps, demonstrating that elevated mutation rates during environmental change are a predictable population-genetic consequence rather than a theoretical possibility.

Methods and use of AI

This model was designed and implemented by the author. The model structure was developed and the model expanded to more individuals and generations with the help of Claude AI (Anthropic).

Appendix S2. Quantitative logic of multi-locus reproductive screening

The Everest hypothesis follows the conventional argument that mutator alleles can be purged because they remain linked to the deleterious mutations they generate. Its additional claim is that reproductive complexity can strengthen this purging by integrating many mutation-sensitive loci into a shared mating or fertilization screen. The amplification can be quantified with a simple probability argument.

Let n be the number of mutation-sensitive loci contributing to a reproductive screen, and let d be the per-locus probability of functionally relevant mutational damage over a defined interval. Assuming that damage events are independent, the probability that no component is damaged is $(1 - d)^n$. Therefore, the probability that at least one component is impaired is:

$$P \text{ (at least one impaired component)} = 1 - (1 - d)^n.$$

When d is small, this is approximately:

$$1 - (1 - d)^n \approx nd.$$

Thus, even small per-locus differences in mutation risk can become appreciable when many loci contribute to the same reproductive screen. For example, if high-fidelity genotypes have $d = 0.001$ and mild mutators have $d = 0.002$, the difference at one locus is only 0.001. Across 100 loci, however, the probability that at least one component is impaired is $1 - (0.999)^{100} = 0.095$ for high-fidelity genotypes and $1 - (0.998)^{100} = 0.181$ for mild mutators. The absolute difference in expected impairment is therefore nearly nine percentage points.

The evolutionary consequences of this nine-point differential also depend on the mating system. If expected reproductive success is written schematically as

$$W = V \times M,$$

where V is viability–fecundity performance and M is mating or fertilization success, then ordinary viability–fecundity selection acts mainly through V, whereas Everest tests act mainly through M. In weakly selective mating systems, impairment may cause only a modest reduction in M. In strongly selective systems, especially leks, the function can be much steeper: small declines in display, stamina, coordination, competitive position, or recognition can sharply reduce mating success, even to zero.

For example, if impairment of any one component reduces mating success by 50% in a steep mating system, the expected mating success of high-fidelity genotypes in the example above is $1 - 0.5\,(0.095) = 0.9525$, whereas that of mild mutators is $1 - 0.5\,(0.181) = 0.9095$. The implied selection coefficient against mild mutators is therefore $s = 1 - 0.9095/0.9525 \approx 0.045$, large enough, under simple constant-selection assumptions, to drive substantial mutator decline over tens of generations.

Thus, even a small per-locus difference in mutation risk can generate a selection coefficient of several percent when many loci are stacked into a steep reproductive screen. Everest therefore predicts that mutators should be removed more strongly when reproductive screens involve many mutation-sensitive components, and when small defects cause large drops in mating success.

This calculation is intentionally simple: real reproductive screens will include components with unequal effect sizes, partial redundancy, and correlated damage. These complications alter the details, but not the central expectation: reproductive screens that integrate many mutation-sensitive components should amplify small differences in replication fidelity into appreciable differences in mating or fertilization success, thereby strengthening selection against mild mutators and favoring the maintenance or recovery of high fidelity.

Appendix S3. Everest in relation to previous work

Everest builds on several existing lines of theory concerning sexual selection, recombination, mutation load, and mutation-rate evolution. This section places Everest alongside those frameworks in the same order as Table 1 in the main text, highlighting overlaps and key points of contrast.

Sexual selection frameworks

Fisherian runaway

Fisherian runaway explains the evolution of exaggerated ornaments through positive feedback between preference and display: attractive traits gain mates, and preferences for those traits spread because they produce attractive offspring [Fisher, 1930]. In its pure form, this process need not track any external measure of quality, so elaborated traits may be costly or arbitrary with respect to viability. Everest is compatible with runaway elaboration but suggests that such traits can later be co-opted as fidelity screens when their development depends on many mutation-sensitive loci.

Handicap and indicator models

Amotz Zahavi's handicap principle and related indicator models treat costly traits as honest signals of condition or underlying genetic state [Zahavi, 1975]. Everest is compatible with the general idea that mate choice can reveal otherwise hidden biological differences, but focuses on a specific property that is especially difficult to assess directly: replication fidelity. Everest also proposes a reason why some reproductive traits may become more complex than fertilization, signaling, or viability alone require: added complexity can increase sensitivity to mutational disruption, strengthening selection against error-prone lineages.

Good genes, condition dependence, and genic capture

Good-genes and genic-capture models treat elaborate traits as signals of genome-wide mutation load: recurrent deleterious mutations reduce condition, and condition-dependent ornaments are therefore expected to be less well formed when load is high [Hamilton & Zuk, 1982; Rowe & Houle, 1996]. Everest is close to these models in spirit, because both frameworks allow complex sexual traits to integrate genetic effects from many loci. The key difference is that genic capture usually routes this integration through the broad concept of condition, whereas Everest specifies a more explicit mechanism: stacking many mutation-sensitive components into a shared reproductive screen amplifies small differences in replication fidelity. Everest shifts the emphasis in three ways. First, it treats replication fidelity—the germline mutation rate—as a distinct target of selection, rather than subsuming all genetic quality into condition or short-term fitness. Second, it predicts that adaptation and fidelity will often move in opposite directions in response to environmental change. Third, it extends the same logic beyond overt mate choice to cases where filtering occurs through fertilization success, gamete competition, or biochemical compatibility.

Long-term consequences of costly elaboration

A related contrast concerns the long-term fate of highly costly traits. Under Fisherian runaway or handicap interpretations, extreme ornaments and demanding behaviors are generally expected to impose net viability costs, so lineages that evolve them may be disadvantaged relative to less elaborated lineages. Everest predicts an additional possibility: if costly traits also function as effective fidelity screens, they may improve long-term persistence by purging mild mutators more efficiently. Lineage sorting may therefore favor some elaborated reproductive systems despite their short-term costs, particularly when they help restore replication fidelity after episodes of environmental change.

Red Queen and fluctuating selection

Red Queen models emphasize ongoing coevolution, often with parasites, as a driver of sex and recombination [Hamilton et al., 1990]. Everest is compatible with the view that fluctuating selection is important but identifies a different quantity at risk: not simply adaptive variation against changing antagonists, but replication fidelity itself. In Everest, environmental change can favor mutator backgrounds during adaptation, while recombination and reproductive screening help restore fidelity after adaptation has been achieved.

Mutation rate evolution

A separate body of literature focuses on mutation-rate evolution and on the population-genetic mechanisms by which mutator alleles spread or are removed. In this literature, selection against mutators is usually indirect: mutator alleles are removed because they remain linked to the deleterious mutations they generate. Everest accepts this Kimura-style linkage logic but proposes a mechanism that can strengthen it. Complex reproductive screens can stack many mutation-sensitive components into a shared mating or fertilization test, so that small differences in replication fidelity generate larger differences in reproductive success.

Roberts and Petrie extend this mutation-rate logic to sexual selection, showing that mate choice can transiently support higher mutation rates when beneficial mutations are available [Roberts & Petrie, 2022]. Everest accepts this accelerator logic but adds the corresponding brake: once adaptation has been achieved, demanding reproductive screens can expose the damage generated by elevated mutation rates, allowing recombination and mate choice to restore high fidelity without erasing adaptive gains.

Mutation load and classic benefits of recombination

Kondrashov's "hatchet"

Kondrashov's "hatchet" emphasizes the purging of deleterious mutation load under strong selection, especially when deleterious mutations interact synergistically [Kondrashov, 1988; Kondrashov, 1993]. Everest shares the concern with mutation load but focuses on a different target: not only the load already present, but the mutation rate that generates it. Complex reproductive screens can make small differences in mutation rate visible by translating them into larger differences in mating or fertilization success.

Hill–Robertson interference

Hill–Robertson interference provides a classic rationale for recombination: linkage among selected loci can reduce the efficiency of selection, and recombination can relieve this interference [Hill & Robertson, 1966]. Everest is compatible with this logic but emphasizes a different step in the process. Recombination can separate adaptive alleles from mutator backgrounds, but selection must still favor the offspring that inherit both adaptation and high fidelity. Everest proposes that complex reproductive screens help do this by making small differences in replication fidelity visible through mating or fertilization success.

Muller's ratchet and lineage-level decay

Muller's ratchet emphasizes the irreversible accumulation of deleterious mutations in finite asexual populations and the lineage-level consequences of mutational decay [Muller, 1964; Felsenstein, 1974]. Everest shares the broader concern with long-term genomic integrity but applies it to sexual lineages facing environmental change. In Everest, mutator backgrounds may rise during adaptation, but later threaten persistence unless high fidelity is restored. Mate choice and fertilization filters provide one route for this restoration: they can expose mutational disruption in complex traits and reduce the reproductive contribution of low-fidelity lineages.

This logic also builds on classic work on recombination and mutator alleles. Muller emphasized that recombination can bring together advantageous mutations arising in different individuals [Muller, 1932], while Kimura showed that mutator alleles can be indirectly selected against through linkage to the deleterious mutations they generate [Kimura, 1967]. Everest accepts both points but adds that reproductive screening can amplify this indirect selection by converting small differences in replication fidelity into larger differences in mating or fertilization success.

Genomic integrity and sex

Another line of work treats sex as a maintainer of genomic integrity. Hörandl and others argue that regular sex, recombination, and segregation can restore higher-quality genomes and prevent mutational decay, particularly in complex eukaryotes [Hörandl, 2009; Hörandl & Hadacek, 2013]. Everest aligns with this broad view but adds a specific filtering mechanism: mate choice, fertilization success, and related reproductive screens can expose mutational disruption in complex traits, thereby favoring lineages that maintain higher replication fidelity.

Drift-barrier theory

Drift-barrier theory addresses a different question: not how mutators are selected against, but why mutation rates do not evolve indefinitely toward higher fidelity. Selection can reduce mutation rates

only while the incremental benefit of improved fidelity is large enough to overcome drift [Lynch et al., 2016]. Everest adopts this logic but proposes a mechanism that can shift the drift-barrier equilibrium toward higher fidelity. By stacking many mutation-sensitive components into shared reproductive screens and routing their effects through steep mating or fertilization success functions, reproductive complexity can amplify small fidelity differences into larger differences in reproductive success. In this sense, Everest does not replace drift-barrier theory; it proposes one way in which sexual lineages may strengthen selection for fidelity and push mutation rates closer to the lower bound set by molecular constraints.

The hypothesis integrates three ideas that are often treated separately: (i) replication fidelity as an independent dimension of variation, distinct from short-term fitness; (ii) the possibility that adaptation and fidelity can move in opposite directions during environmental change; and (iii) reproductive "excess" complexity as an integrity-sensitive screen that amplifies small fidelity differences by stacking many mutation-sensitive components into shared reproductive tests. For a concise summary of how conventional frameworks relate to Everest, and of the main diagnostic tests, see Table 1 in the main text.

Appendix S4. Adaptation–fidelity tradeoffs beyond sexual organisms

The adaptation–fidelity tradeoff emphasized by Everest should not be unique to sexually reproducing eukaryotes. Any replicating lineage must both adapt to changing conditions and maintain a functional genome, so mechanisms that transiently increase variation and later restore fidelity should be expected across the tree of life. Bacteria and viruses offer partial analogies: stress-induced mutagenesis, conjugation, recombination, and reassortment can all alter the balance between adaptation and fidelity. Conjugation systems such as the F factor in *E. coli* are especially suggestive, because they involve multiple plasmid-borne and host-dependent functions whose complexity may exceed the minimum required for DNA transfer. Crucially, these plasmids are replicated by host replication machinery, so an error-prone host background can generate disruptive mutations in the very genes required for plasmid transfer or maintenance. Like Everest traits, such complexity could increase the mutational target and make successful exchange more sensitive to replication errors, thereby biasing transfer toward higher-fidelity backgrounds. These are not Everest mechanisms in the strict sense, because they lack the regular, multigenic mate-choice screens emphasized here. They nevertheless support the broader premise that adaptation and fidelity can conflict, and that successful lineages require mechanisms for restoring genomic integrity after episodes of rapid change.

Appendix S5. Pregnancy loss as a possible distributed fidelity-sensitive screen

Mammalian reproduction provides a speculative example of how reproductive complexity could couple replication fidelity to reproductive success without any dedicated mechanism for detecting mutation rate. Successful mammalian pregnancy depends on a complex fetal–maternal interface involving implantation, trophoblast differentiation and invasion, decidual remodeling, immune tolerance, and early placentation; disruption of these processes is associated with implantation failure, miscarriage, and other pregnancy complications [Huang et al., 2023]. Everest would predict that, if elevated mutation rates increase the number of damaging variants in genes or regulatory elements involved in these processes, low-fidelity genotypes could experience increased reproductive loss due to developmental and placental failure. This argument is distinct from the well-established role of chromosomal abnormalities in early miscarriage, which are not themselves evidence of inherited mutator genotypes. The point is only that complex reproductive interfaces can act as distributed screens: they need not measure mutation rates directly to impose reproductive penalties on genotypes that generate a greater burden of damaging variants.

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