The Everest hypothesis: sexual complexity as a test of replication fidelity

Running title: Everest hypothesis

Keywords:

- sexual selection
- mutation rate evolution
- mate choice
- reproductive complexity
- adaptation

Number of words in the main text: 7261

Elements of the manuscript:

- Main text
- 6 figures
- 1 table
- 1 information box
- References
- Text describing the toy model in the Supplementary Information (deposited at Dryad)
- Excel spreadsheet showing the toy model (deposited at Dryad)

Article type: major article

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Abstract

Sexual reproduction is nearly universal among complex organisms, yet the reasons for its elaborate accompaniments—from grueling migrations to intricate courtship displays—remain puzzling. The Everest hypothesis proposes that this reproductive "excess" complexity evolved to expose subtle defects in replication fidelity to selection. It rests on three key ideas: first, because natural selection acts on immediate fitness, it is often blind to mild mutator alleles that slightly degrade germline DNA replication accuracy. Second, environmental instability exacerbates this problem by transiently favoring elevated mutation rates for short-term adaptation, even as they threaten long-term genomic integrity. Third, complex, multigenic reproductive traits function as demanding tests that magnify the phenotypic impact of mutational noise, allowing mate choice or selective fertilization to purge high-mutation lineages. The hypothesis therefore predicts a rise-and-fall dynamic in which mutator lineages increase during environmental change but are later eliminated, and that recombination with residual high-fidelity genotypes can restore fidelity without erasing adaptation. The paper also makes concrete predictions and proposes specific experiments to test these mechanisms. On this view, the elaborate accompaniments of sex across the tree of life are not arbitrary ornaments, but evolved instruments of quality control that help lineages maintain genomic integrity in fluctuating environments.

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

DNA replication is remarkably accurate but not perfect. Each generation introduces new mutations, some in ordinary genes and others in the replication machinery itself. Alleles that increase the error rate (mutator alleles) must form a spectrum from severe to mild. Strong mutators are relatively easy to eliminate because they remain linked to the deleterious mutations they generate, but mild mutators may persist because their effects are too subtle to detect through ordinary viability selection—even though the long-term maintenance of complex genomes requires their removal.

Crucially, this spectrum is asymmetric. While mutator alleles can range from severe to mild, antimutator alleles—those that improve replication fidelity—can only be mild. It is implausible that any single mutation, or even a small set of simultaneous mutations, could radically increase fidelity; improvements must come incrementally. (The exception would be reversion of a previous mutator mutation, which restores rather than creates fidelity.) This asymmetry has an important consequence: mechanisms that can detect only large differences in fidelity will eliminate strong mutators but miss both the mild mutators that erode fidelity and the mild antimutators that could restore it. For long-term genome maintenance, mechanisms must exist that can detect even very small differences in replication fidelity in both directions.

Replication fidelity is distinct from fitness. Fitness is expected reproductive success in a given environment; replication fidelity is the accuracy of the replication machinery, measured directly by sequencing as a per-base or per-genome mutation rate. For example, a plant colonizing a new island may instantly become much fitter than its competitors on arrival if it already carries alleles suited to that environment, even if its mutation rate remains unchanged. Natural

(viability) selection typically acts on fitness rather than on mutation rate. When populations are well adapted, most new mutations are deleterious, and lower mutation rates are favored—but if the fitness benefit of a small reduction in mutation rate is extremely weak, genetic drift may allow suboptimal fidelity to persist.

Drift-barrier theory formalizes this idea: mutation rates decline until further reductions are too weakly favored to overcome drift. In large populations, selection is efficient, and mutation rates can fall to relatively low values; in small populations, higher mutation rates persist because selection cannot "see" the tiny advantages of further improvement [Lynch et al., 2016]. The theory thus predicts a lower bound on mutation rates but says little about the phenotypic mechanisms that might expose very small fidelity differences to selection.

Environmental stability is also important. In stable niches where populations are already well adapted, beneficial mutations are rare, and most new mutations are harmful, thereby favoring lower mutation rates. But in novel or rapidly changing environments, the fraction of beneficial mutations increases, and a lineage "suddenly thrust into an environment that it is not well adapted to" may benefit from a slightly elevated mutation rate [Bank, 2022; Duffy, 2018]. Phylogenetic evidence supports this view: avian lineages with higher diversification rates tend to show faster molecular evolution [Lanfear et al., 2010]. Figure 1 illustrates this contrast schematically.

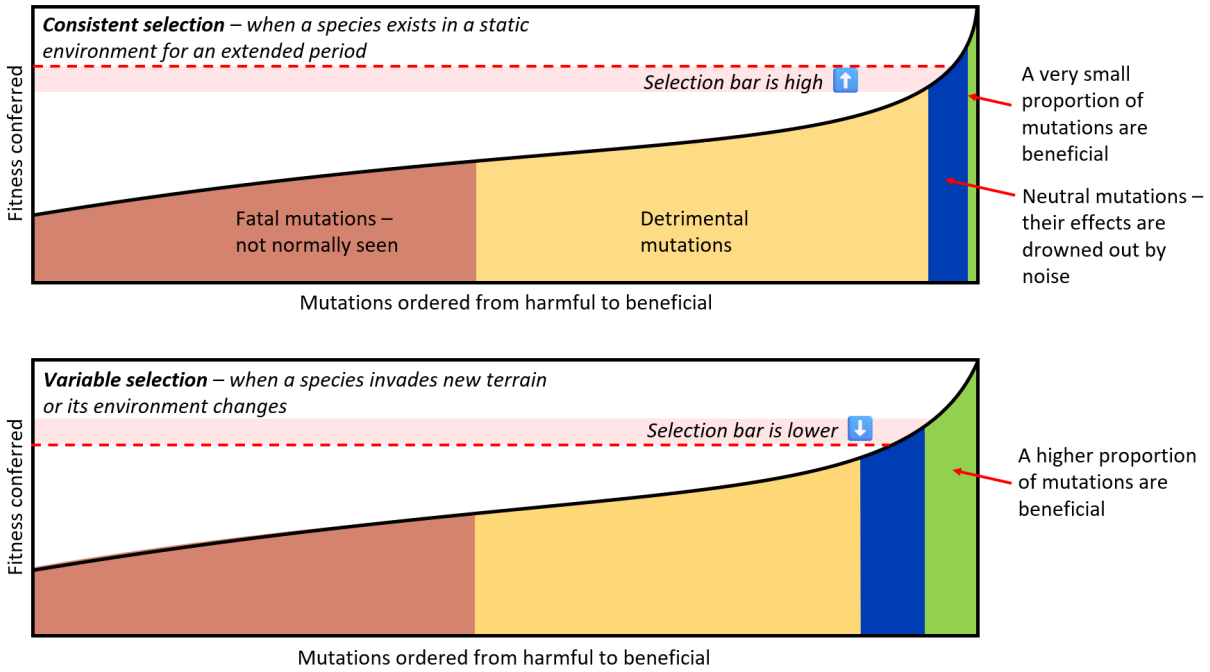


Figure 1. Mutation effects under different selection regimes. Mutations are grouped into four classes: fatal (brown, removed immediately), detrimental (beige, removed by purifying selection), effectively neutral (blue, conserved or lost by drift), and beneficial (green, conserved by positive selection). Top: In stable environments where populations are well adapted, most advantageous mutations have already been fixed, so new beneficial mutations are rare (e.g., clams in stable marine mud banks). Bottom: In novel or rapidly changing environments, selective pressures shift, increasing the proportion of beneficial mutations among new variants (e.g., birds or insects colonizing islands).

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 to new conditions. Mutator alleles may therefore be transiently favored in unstable environments even though they are harmful in the long run. Moreover, because mutations can occur in genes encoding the replisome itself, there is a risk of positive feedback—mutators promoting further mutations in replication genes—potentially leading to mutational meltdown unless some mechanism limits their spread.

In many microorganisms, stress-induced mutagenesis offers a partial solution: under nutrient limitation, DNA damage, or other stressors, bacteria and some unicellular eukaryotes upregulate error-prone polymerases, producing bursts of mutation that can accelerate adaptation, followed by a return to baseline fidelity when stress abates [Galhardo et al., 2007]. By contrast, in complex multicellular organisms, there are no well-established examples of whole-organism stress causing a comparably reversible increase in germline mutation rate. Although some somatic cells deliberately increase mutation for specific functions, there is little evidence that whole-organism stress triggers reversible germline mutagenesis. Complex animals and plants, therefore, cannot rely on stress-induced mutagenesis alone to balance short-term adaptation against long-term fidelity.

Sexual reproduction offers a possible resolution. In a sexual population, some lineages may be well adapted but have low replication fidelity, while others retain high-fidelity replisomes but are less well adapted. Recombination between such lineages can generate offspring that combine locally adapted alleles with high-fidelity replication machinery, allowing populations to exploit the short-term benefits of mutator alleles while later recovering low mutation rates. For this to work, however, there must be reliable means to distinguish individuals or gametes that carry high-fidelity machinery from those that carry mild mutator alleles, even when their immediate fitness is similar.

The Everest hypothesis

The central question of this paper is therefore: by what mechanisms can sexual species detect—and preferentially reproduce from—individuals with high replication fidelity, especially when mutator effects are subtle? 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 across generations). These dimensions can trade off—populations may gain adaptation at the cost of fidelity, or vice versa.

The Everest hypothesis (Figure 2) proposes that much of sexual reproduction—courtship, mating, fertilization, and their elaborate accompaniments—functions as a demanding screen. These complex, multigenic traits act as performance tests that expose genome-wide mutational damage. Individuals with slightly error-prone replication machinery accumulate disruptions across many loci; the resulting degradation becomes visible in traits that mates (or gametes) evaluate. Over time, this links mating success to replication fidelity.

Crucially, 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 detecting it requires comparing mutational damage at two points in time. The logic is illustrated in figure 3A at the individual level: parents who pass a demanding test establish the first point; when offspring face the same test, failures reflect mutational damage accrued since the parental generation. At the population level (figures 3B–C), this creates dynamics in which adaptation can rise while fidelity falls, and in which recombination can restore high-fidelity genomes without erasing adaptation.

Critically, Everest screens must be shared widely enough within an interbreeding population to have statistical power. If preferences are completely fragmented—some individuals weighting plumage, others migration performance, others biochemical compatibility—the link between genome-wide integrity and mating success is weakened. However, Everest does not require a single universal test. Instead, it predicts convergence on one or a few dominant screens within each mating network, with the dominant screen potentially varying across environments and regions. Where populations face different ecological demands (for example, migratory versus resident strategies), different screens may predominate, and transitional or contact zones may show mixtures of criteria.

This creates Fisherian-like pressure toward shared standards, with two key differences: (1) the target is replication fidelity—a measurable property—rather than arbitrary attractiveness; and (2) effective screens must be complex and multigenic, requiring many intact loci for proper development or performance. This interplay may partly explain why sexual traits are often stereotyped locally yet geographically variable across species. A simple prediction is that mutation-rate filtering should be weakest in transition zones where neighboring populations mix, so mutator lineages may persist more easily and, all else equal, average mutation rates may be higher.

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
Müller'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 experiment
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)

To clarify the mechanism, I distinguish several locus classes (Figure 2A). A "locus" is any genomic region—including open reading frames, introns, and regulatory sequences—where variation alters the phenotype. Mate-choice loci are those that affect mating or fertilization success. Within this class:

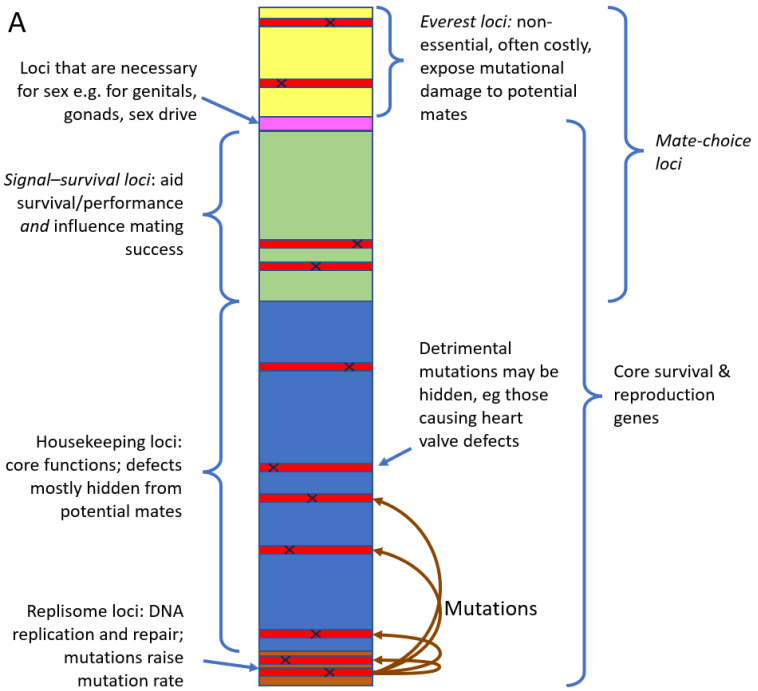
- *Signal–survival loci* influence both mating success and survival/performance (e.g., via strength, endurance, cognition)
- *Everest loci* are non-essential for survival or fertilization—often costly or risky—but elaborated by sexual selection to make mutational damage highly visible

Traits derived from these loci amplify small differences in replication fidelity into large differences in reproductive success. I call these screening phenotypes Everest tests.

Figure 2A shows how these classes might be arranged conceptually: 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 2B shows the consequence: error-prone replisomes generate mutations across the genome, degrading the complex traits underlying mate choice and reducing mating success.

Figure 3 depicts the predicted dynamics across multiple scales. Panel A presents an individual-level schematic: parents who pass a demanding test serve as a baseline, and offspring that accumulate excessive mutational damage are more likely to fail the same test. Panel B shows the accumulation of mutations over time at the population level, and Panel C maps the same scenario into a two-dimensional adaptation–fidelity space. In both representations, strong selection can initially favor mutator lineages that adapt rapidly but accumulate mutations unsustainably; recombination with residual high-fidelity genotypes can later generate well-adapted lineages with restored fidelity.

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*.



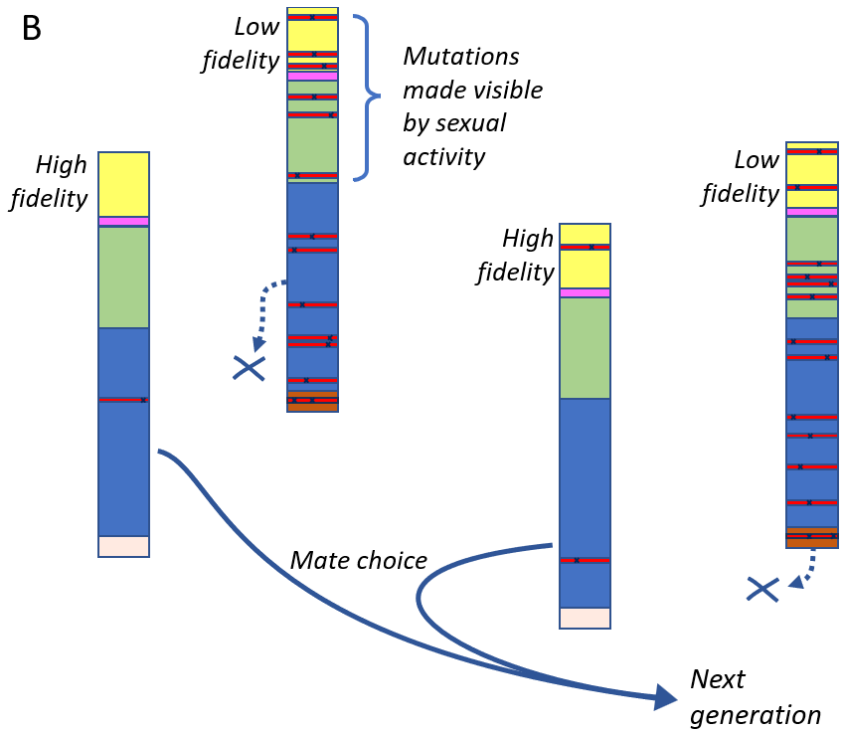
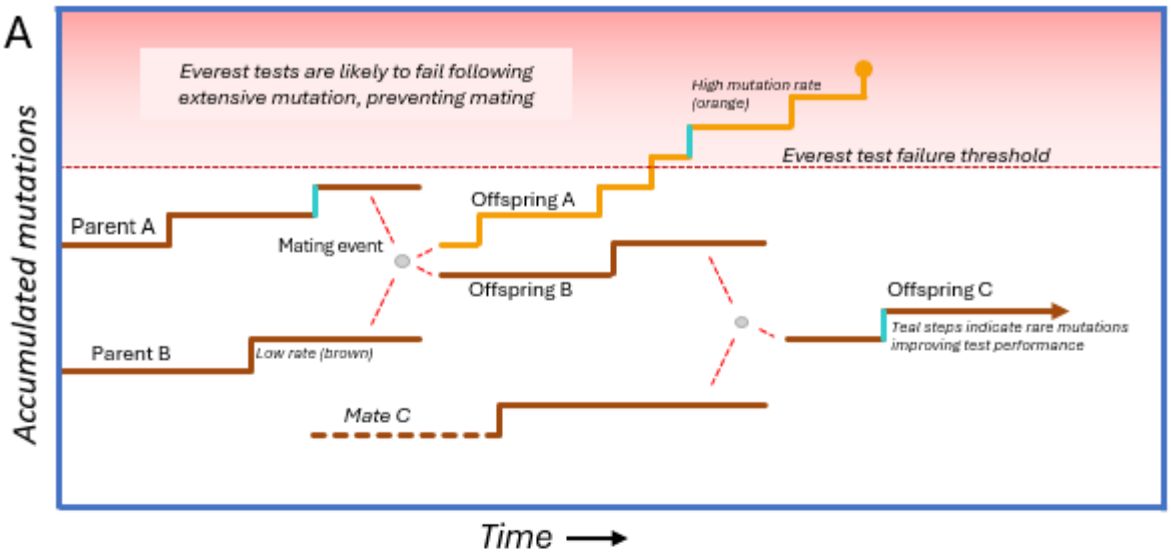
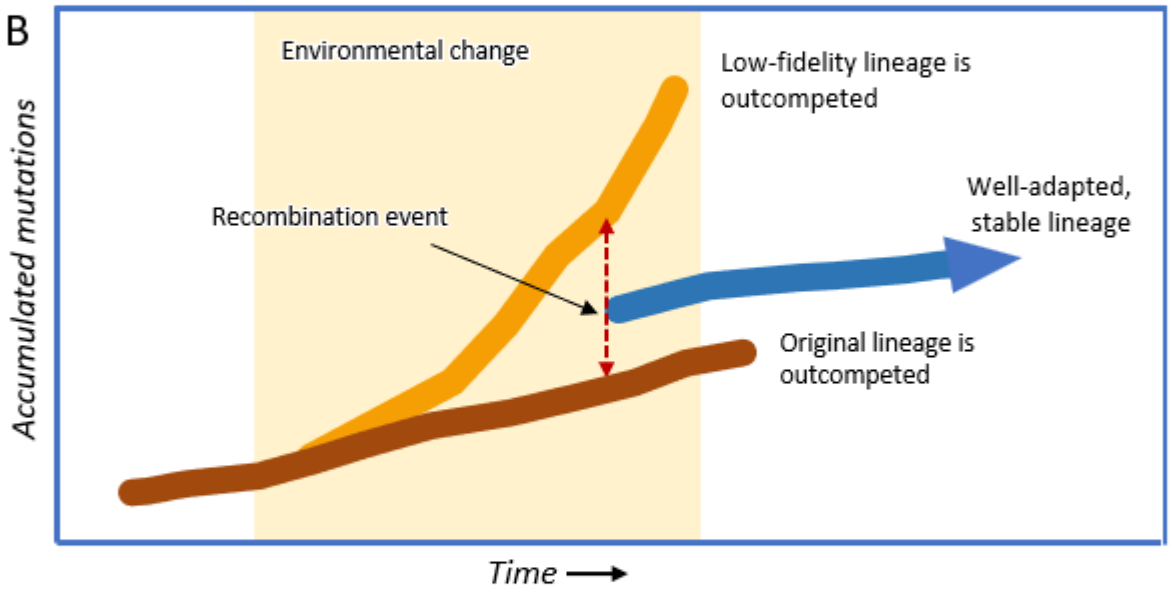


Figure 2. Genomic loci and mate choice under the Everest hypothesis. Everest predicts that mate choice can act as a mutation-rate filter by coupling mating success to genome-wide genetic integrity. (A) Conceptual arrangement of loci by their biological role. Yellow: Everest loci—affecting mate choice but not survival, elaborated to expose mutational damage. Green: signal–survival loci—influencing both mating success and survival. Blue: housekeeping loci—core functions, often hidden from mates. Brown: replisome loci—encoding replication and repair proteins. Mutations in replisome loci (brown arrows) increase mutation rates genome-wide, including at mate-choice loci where effects become visible. Red Xs indicate mutations. (B) Individuals with error-prone replisomes accumulate more mutations in mate-choice loci, reducing mating success. High-fidelity individuals are more likely to mate and pass on accurate replication machinery.


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


B
Accumulated mutations
Environmental change
Low-fidelity lineage is outcompeted
Well-adapted, stable lineage
Recombination event
Original lineage is outcompeted
Time

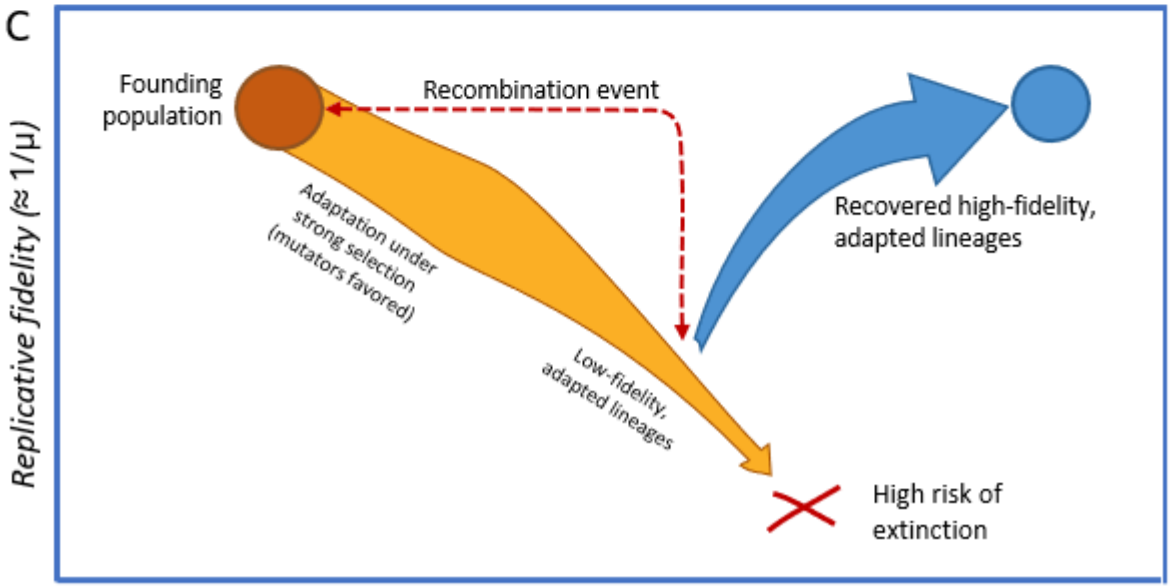

C
Replicative fidelity (≈ 1/μ)
Founding population
Recombination event
Adaptation under strong selection (mutators favored)
Low-fidelity, adapted lineages
Recovered high-fidelity, adapted lineages
High risk of extinction
Adaptation to a new environment (relative fitness)

Figure 3. Everest predicts that mate choice can act as a mutation-rate filter: error-prone lineages accumulate genome-wide damage and fail mating tests, while recombination can restore high fidelity without erasing adaptation. (A) Individual-level schematic (accumulated mutations vs time). Two low-mutation-rate parents (brown) produce two offspring: one becomes a mutator (orange), crosses the test-failure threshold (red dashed line), and is unlikely to mate; the other remains low mutation rate (brown) and produces a low-mutation-rate descendant. Teal steps denote rare mutations that improve test performance; dashed connectors show parentage schematically. (B) Population-level mutation–time dynamics. During rapid environmental change (shading), a mutator lineage (orange) adapts quickly but accumulates mutations unsustainably and risks extinction; a low-mutation-rate lineage (brown) adapts more slowly. Everest recombination between them (red dashed double-arrow) yields a lineage (blue) that combines adaptation with restored high fidelity. (C) The same scenario shown in adaptation–fidelity space (fidelity ≈ 1/μ). Mutators move toward higher adaptation but lower fidelity (orange, red X), whereas Everest recombination produces recovered lineages that regain high fidelity while retaining adaptation (blue). Curves are qualitative, not fitted data.

The population dynamics that underlie the recovery shown in figure 3 deserve emphasis. During rapid environmental change, low-fidelity (mutator) lineages adapt quickly and rise to high frequency, temporarily outcompeting others. Their ongoing mutational load, however, erodes their fitness even in the new environment, causing their populations to decline. This reduces competitive pressure on residual high-fidelity lineages, allowing them to rebound in frequency. This demographic shift creates favorable conditions for Everest recombination. In colonization events or metapopulations, immigration from source populations can replenish high-fidelity genotypes, providing additional opportunities for Everest recombination without sacrificing local adaptation.

Everest loci serve two functions. First, they expand the mutational target: adding non-essential components to reproductive traits increases the target DNA regions where mutations affect mate choice. Second, they interact with signal–survival loci to create additional monitored features, further enlarging the target DNA. In migratory birds (including partially migratory birds such as European robins), for instance, loci promoting migration on demanding routes magnify the effects of the signal–survival loci that underlie endurance, orientation, and energy balance, such that modest defects can cause failure to complete migration. Multiple mate-choice loci can also be "stacked" into highly integrated traits with a single readout. Where viability selection should favor robustness and redundancy, sexual selection may favor tests where this fragility cannot be avoided, precisely because it yields more discriminating tests of genetic integrity.

Ordinary viability selection typically acts on small fitness differences: a bird with a mildly defective heart valve might lose 20–30% of its lifetime reproduction. By contrast, mate choice based on a demanding Everest test can amplify modest genetic differences into large differences in reproductive success—some individuals may mate many times, while others fail entirely. Sexual selection acting through Everest tests thus purges mutator alleles far more strongly than viability selection alone.

High-fidelity individuals may choose very fit partners with lower fidelity, provided mechanisms exist to restore high fidelity in their descendants; conversely, very fit individuals may prefer high-fidelity partners to ensure accurate transmission of their beneficial alleles. In some species, the sexes may contribute asymmetrically: in peafowl, males may play a disproportionate role in conserving fidelity through their elaborate visual displays, whereas viability selection acts on both sexes. In traditional human societies, mate preferences may show a different asymmetry: men may more often emphasize physical attractiveness, whereas women may more often prioritize indicators of success or resource acquisition—proxies for current fitness.

The hypothesis makes at least three testable predictions. First, under strong selection in a new environment, sexual populations should evolve well-adapted but higher-mutation-rate lineages, as mutator alleles are temporarily favored during adaptation. Second, Everest recombination should restore low mutation rates without sacrificing adaptation. Third, across taxa, greater sexual and reproductive complexity should correlate with lower mutation rates, reduced mutational load, or greater lineage persistence relative to otherwise similar groups lacking such complexity. A simple two-locus population genetic model, presented in the Supplementary Information (figure S1), confirms that this logic is mathematically sound: under plausible parameters, Everest-style mate choice rapidly purges mutator alleles, and populations can recover high fidelity even after mutators are transiently favored during environmental change.

It should be noted that the mate-selection strategies envisaged here can also reveal genetic defects beyond those that directly affect DNA replication. Complex behaviors and displays may be very sensitive to rare mutations in essential housekeeping genes—those involved in ribosomal function, cell cycle control, mitochondrial metabolism, transcription, and translation—as well as in genes encoding the somatic DNA replication machinery that contribute to individual performance.

Relations to previous work

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

Everest aligns closely with good-genes and condition-dependent signaling models (e.g., Hamilton & Zuk 1982; Pomiankowski et al. 1991), which view reproductive traits as indicators of genome-wide quality. However, it makes three distinct claims: (1) replication fidelity (mutation rate) is a separable target of selection, not merely folded into "condition" or short-term fitness; (2) adaptation and fidelity can trade off during environmental change, with mutators transiently favored before being purged; and (3) non-essential complexity evolves specifically to amplify subtle fidelity differences into detectable reproductive failures, promoting population-wide convergence on shared, multigenic screens. Everest thus complements existing frameworks on sexual selection, recombination benefits, and genomic integrity (e.g., drift-barrier theory; Kondrashov 1988; Lynch et al. 2016) by providing a concrete phenotypic mechanism—demanding reproductive tests—that couples mating success to mutation rate and enables recombination to restore fidelity while retaining adaptations. Table 1 summarizes these relationships; a detailed discussion appears in Supplementary Information SI.2.

Natural phenomena that the Everest hypothesis can explain

Many traits and behaviors in nature appear excessively costly, risky, or intricate yet persist and often become highly stereotyped. Everest interprets some of these as integrity-sensitive

screens: complex, multigenic traits whose performance is unusually vulnerable to mutational disruption, thereby making small declines in replication fidelity visible at mate choice or fertilization. Several of the examples below are long-standing biological puzzles—traits whose costs, precision, or stereotypy seem to demand explanation even under familiar frameworks. The examples are not a survey and are not meant to imply that any single trait or test is universal; they were selected from various species and life cycles to show that the same logic can apply and to help create specific, testable predictions.

Long-distance migration. Arctic terns, Atlantic salmon, and Monarch butterflies undertake demanding journeys that require tight coordination of navigation, physiology, and timing (Fig. 4). 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 with subtly elevated mutation rates. Arctic terns complete annual round-trip journeys exceeding 70,000 km yet maintain large, long-lived populations, consistent with strong filtering. Monarch butterflies present a further challenge: North American populations complete migration as a multi-generation relay, so insects breeding in northerly regions must faithfully transmit alleles controlling overwintering behavior that they never experience themselves. Everest therefore predicts that lineages with slightly higher mutation rates will be under-represented among successful migrants, because failure can occur at many steps.

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 makes small losses in replication fidelity visible before mating. The peacock's tail (figure 4) illustrates how complex traits can make underlying mutations visible: its development depends on the coordinated action of many genes, and small defects in any of them might visibly degrade the result.

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 tails, more ocelli, or longer tails, and noted that male variation was low [Takahashi et al., 2008]. This suggests that past selection may already have removed low-fidelity individuals, so peahens now treat a well-formed tail as an “entrance exam” and base final choice on additional traits. This pattern is consistent with multifactorial mate selection that combines an effective filter for genetic integrity with selection for current fitness.

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 based on a multigenic template of acceptable appearance, with individuals whose development is perturbed by mutational damage more likely to deviate and suffer reduced mating success.

Symmetry. Bilateral and radial symmetry are widespread targets of mate choice across taxa, as illustrated in figure 4. Producing a symmetrical body requires the same developmental program to be executed with equal precision on both sides—a demanding test sensitive to mutational noise. Partially defective proteins can cause inconsistent developmental outcomes; defective immunity increases susceptibility to infections that may affect one side more than the other; and inappropriate behavior can cause asymmetric physical damage. Preferences for symmetry have been documented in pollinators choosing among flowers, in female guppies, moths [Koshio, 2007], and barn swallows choosing among males, and in humans evaluating potential partners. The Everest hypothesis interprets these preferences as mechanisms for detecting

elevated mutation rates: symmetry indicates that the many genes underlying bilateral development are intact and functioning reliably.

Figure 4. Illustrative examples of complex or highly stereotyped traits that could serve as integrity-sensitive screens under the Everest hypothesis. Top row: long-distance migrants—Arctic tern, Atlantic salmon, monarch butterfly—undertake demanding journeys that require coordinated navigation, physiology, and timing; failure can occur at many steps, so successful migrants may be biased toward higher-fidelity lineages. Middle row: the peacock's tail depends on coordinated expression of many developmental genes; bee orchid mimicry requires high precision in 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]; the moth *Macrocilix maia* displays wing patterns resembling flies near a bird dropping, yet retains near-perfect bilateral symmetry despite predation pressure that might favor asymmetry [Merilaita and Lind, 2006]; pollinators prefer symmetrical flowers such as arugula (*Eruca vesicaria*) [Møller and Eriksson, 1995]. In each case, Everest interprets performance, precision, or symmetry as a potential filter: successful execution or well-formed ornaments suggest that many underlying loci are intact, consistent with lower germline mutation rates. These examples are included to illustrate Everest's logic and suggest testable predictions, not to provide a systematic survey.

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.

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 built by many genes, functioning as real-time assays of genomic integrity.

Molecular compatibility in sessile organisms. Corals and marine broadcast spawners rely on synchronized spawning and intricate gamete-recognition systems. Flowering plants use complex pollen–pistil signaling mechanisms and multilayered signaling pathways to guide pollen tubes (figure 5). Fungi and some protists have multigenic mating-type and compatibility systems. These mechanisms involve many interacting receptors, ligands, and regulatory genes—tests of fidelity expressed through fertilization success rather than behavioral choice.

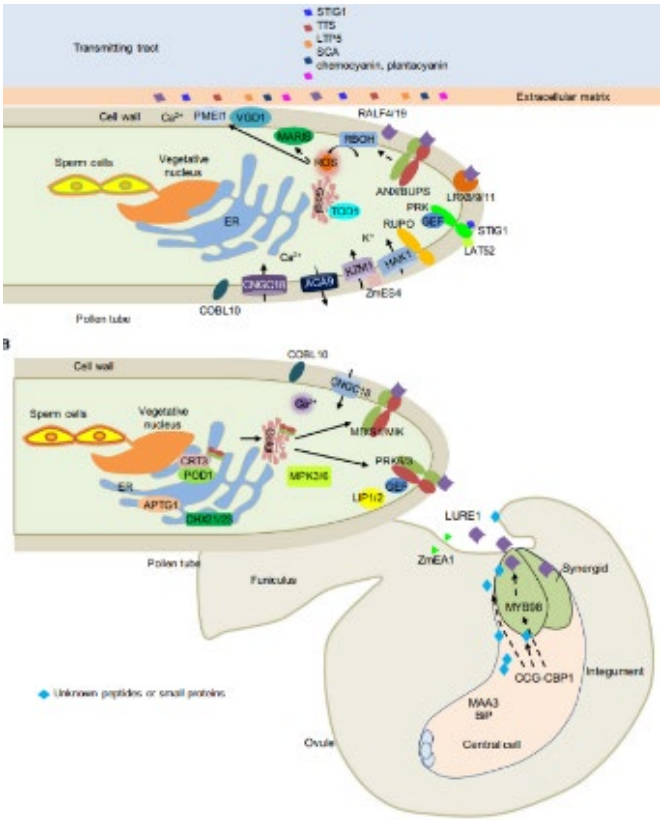


Figure 5. Pollen tube guidance as a molecular Everest test. Fertilization in flowering plants requires pollen tube elongation through maternal tissue and precise navigation to the ovule, mediated by successive cell–cell interactions and multilayered biochemical signaling [Li et al., 2018]. Pollen grains function as mobile haploid organisms whose success depends on many

gene products. This complexity filters out error-prone lineages, supporting the Everest hypothesis that elaborate fertilization systems act as tests of genetic fidelity.

Floral complexity in angiosperms. Flowers add an additional layer of testing in plants. Many species have structurally complex blooms whose shapes, colors, scents, and pollination routines are learned by particular pollinators. Successful visitation depends on coordinated features: petal number and symmetry, landing platforms, nectar guides, and mechanical fit. Plants with flowers slightly deformed by mutation are likely to receive fewer visits, so pollinators tend to move pollen between plants with well-formed flowers. Because most angiosperms lack an early-segregated germline, the tissues producing petals, pollen, and ovules share a recent developmental history—low mutational load in floral tissues likely correlates with low load in gametes. Floral complexity thus acts as a pollinator-mediated test, biasing mating toward high-fidelity lineages.

Human mate choice. Humans show strong, though culturally modulated, preferences for partners with facial and bodily symmetry, clear skin, good teeth, athleticism, intelligence, humor, etc.—traits that depend on complex developmental pathways encoded by many genes. While social and cultural factors clearly shape preferences, some sensitivity to physical and behavioral cues may point back to an ancient role for mate choice in gauging replication fidelity.

Sperm competition. In many animals, sperm from different males compete within the female tract, imposing strong selection on sperm number, form, and performance [Møller, 2003] . Genomes with elevated mutation rates are more likely to produce defective sperm that fail, so high-fidelity lineages contribute disproportionately to fertilizations.

Everest loci versus signal–survival loci in nature

These examples also illustrate the varying contributions of Everest and signal–survival loci. In long-distance migrants such as Arctic terns, most underlying traits—such as flight performance and endurance—are built on signal–survival loci that directly enhance survival, while a smaller number of Everest loci generate the migration instinct and fine-tune navigation and timing. Birds-of-paradise and peafowl sit at the opposite extreme: many loci underlying plumage and display choreography are Everest loci—costly elaborations whose main function is to make mutational damage conspicuous—while only a subset contributes directly to survival.

In ephemeral-winged insects, most growth occurs during the juvenile stages, so adult wings and mating flights are close to pure Everest traits. In molecular systems such as fungal mating types or plant pollen–pistil interactions, core compatibility pathways are plausibly built from signal–survival loci, whereas additional recognition steps and biochemical refinements may function as Everest loci that impose extra testing without being strictly required for fertilization.

Genomic signatures of Everest loci

A natural expectation is that Everest loci should be structurally larger than housekeeping loci because their role is to provide mutational targets. One might hope to test the hypothesis by exploiting this difference. However, this overlooks a tension in the evolutionary forces acting on these loci. From the individual's perspective, what matters is passing the test, not providing a large target—so once an ornament or fertilization system satisfies choosy mates, selection should favor minimizing the size and fragility of the underlying loci, just as it does for essential housekeeping genes.

The key distinction is therefore not "mate-choice loci versus housekeeping loci" but "highly important versus less important functions." Both Everest loci and critical housekeeping loci (core cell cycle or replication proteins, for example) likely experience strong purifying selection to reduce their effective target size, whereas less important housekeeping functions may remain more extensive. Moreover, Everest loci may often be relatively recent elaborations, and newly evolved genes may start large and only gradually be streamlined.

These opposing forces—selection to minimize individual fragility versus recent evolutionary origin—make it difficult to derive simple predictions about gene length or intron number. Any genomic test of Everest, such as comparing pollen tube recognition genes in plants with core metabolic genes, would need to control for gene age, essentiality, and expression breadth and should seek statistical trends rather than definitive proof.

The evolution of sexual reproduction

Ancient asexual lineages likely existed as quasispecies—clouds of related, mutating genotypes, much like modern viruses. The earliest lineages formed diverging trees with no mechanism for recombination and regeneration of high-quality genomes. Simple recombination mechanisms probably evolved early because they allow advantageous mutations from different branches to collect within a single lineage [Muller, 1932]. As entities become well-adapted in stable environments, the proportion of advantageous mutations falls, and most new mutations become harmful—favoring high-fidelity replicators and driving the elimination of mutator alleles, which are linked to the deleterious variants they produce [Kimura, 1967].

The persistence of simple viruses shows that such systems can be stable. Early life may have thrived in stable environments, but lineages spreading into variable environments would tend to lose fidelity and have limited longevity. The emergence of complex recombination mechanisms—incorporating biochemical lock-and-key mechanisms and mate selection—may have enabled ancient life to colonize unstable environments without catastrophic loss of fidelity. Sexual reproduction can thus be viewed as a highly sophisticated adaptation to environmental instability, counteracting the natural loss of fidelity predicted under such conditions [Duffy, 2018]. This helps explain why sexual reproduction predominates among complex modern organisms: they experience transient, strong selection and must periodically restore fidelity in otherwise well-adapted lineages.

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, birds, and captive mammals. The design in figure 6A and Box 1 asks, first, whether strong selection affects replication fidelity (series S2 versus control S1), and second, whether recombination with wild-type strains can restore fidelity (series S4 versus S3), with sequencing of replisome genes or whole genomes likely needed for unambiguous interpretation; similar approaches could be extended to zoo populations. Figure 6B maps the predicted trajectories of each series onto adaptation–fidelity space, showing how comparison of S3 and S4 directly tests whether recombination can restore fidelity.

2. **Experimental manipulation of cricket song.** A manageable 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—where a key Everest test is removed—will accumulate higher mutation rates or a greater mutational load in replication-associated genes than the control line.

3. **Conceptual extension to peafowl.** It would be valuable to extend experimentation to birds and mammals. Conceptually similar work in peafowl could shorten males' trains and trim the "eye" spots, preventing females from using full tail elaboration in mate choice. Such a study should proceed only if tail trimming can be shown to have minimal long-term effects on health and well-being and is approved by ethical review.

4. **Experimental manipulation of pollination in flowering plants.** A parallel test could be carried out in a model plant such as Arabidopsis thaliana. 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 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 growing both plant lines in a common garden for several generations, the lines would be compared using genome sequencing and analysis of replication and repair genes. The Everest hypothesis predicts that removing pollen competition and pistil-level screening will allow greater accumulation of deleterious mutations (and possibly reduce vigor) in the restricted line.

5. **Phylogenetic investigations of migration and mutator prevalence.** A more challenging observational approach would use phylogenetic and population-genetic data to ask whether migratory lineages contribute more gene flow to non-migratory populations than the reverse. After controlling for population size and demography, Everest predicts that high-fidelity migratory lineages should more often infiltrate non-migratory populations than vice versa.

6. **Comparative tests of migration and longevity.** Another approach is to examine whether species or populations that undertake long migrations tend to have greater longevity than their closely related non-migratory counterparts. In birds, this could be tested using existing ringing and mark–recapture survival data, combined with phylogenetic comparisons of migratory and resident lineages.

7. **Direct comparisons of mutation rates in migratory versus non-migratory lineages.** The hypothesis predicts that long-distance migrants should have lower mutation rates than comparable non-migratory relatives. Partially migratory species such as European robins, in which migratory and non-migratory populations can be compared directly, offer a natural test: sequencing nestlings from both populations could reveal whether migratory lineages maintain lower germline mutation rates.

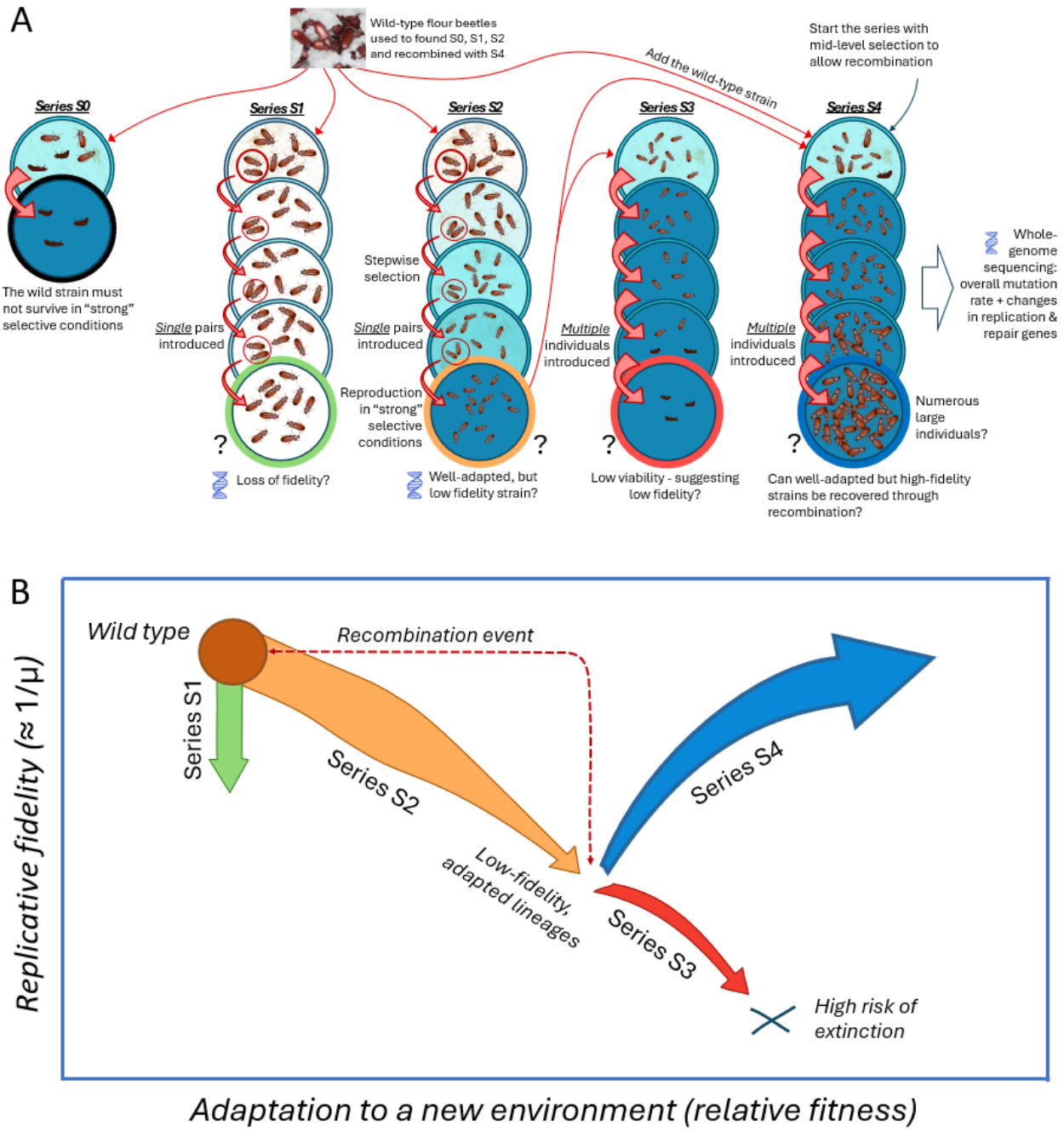


Figure 6. Experimental test of the Everest hypothesis in a sexual model system (e.g., flour beetles). *(A)* Experimental design. Populations are exposed to increasingly severe selective environments (darker blue circles). Series S0 identifies conditions that just prevent wild-type reproduction. In S1 and S2, single pairs found each generation to minimize recombination: S1 is a bottleneck control without strong selection, whereas S2 experiences stepwise increasing selection and is predicted to favor mutator genotypes (reduced replicative fidelity). From S2, two series are derived. S3 continues strong selection without backcrossing, predicted to show further fidelity loss and elevated extinction risk. S4 introduces wild-type individuals to allow recombination (backcrossing) while maintaining strong selection, predicted to restore high fidelity while retaining adaptation. Whole-genome sequencing can estimate the overall mutation rate and identify changes in replication/repair genes. *(B)* Predicted trajectories in adaptation–fidelity space corresponding to the design in (A) (axes as in Fig. 3C). The wild-type population (brown circle) begins with high fidelity but low adaptation. S1 (green) represents bottlenecking without strong selection. S2 (orange) represents adaptation under stepwise selection with restricted recombination, yielding well-adapted but lower-fidelity lineages. S3 (red) represents continued strong selection without backcrossing, predicting further fidelity

loss and high extinction risk. S4 (blue) represents recombination with wild type followed by strong selection, predicted to restore high fidelity while maintaining adaptation. The key test is whether S4 restores fidelity relative to S3 while preserving adaptation.

Box 1. Experimental readouts and predictions for the design shown in figure 6

Measurements

1. Per-site mutation rate in individuals from each series (S0–S4), 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 Everest mechanism)

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

- Wild-type and S1 (bottleneck control) should show similar mutation rates and no consistent changes in replication genes beyond drift
- S2, S3, and S4 may become fitter but should not differ systematically in mutation rate or replication-gene profiles
- Recombination with wild-type in S4 should not restore mutation rates lower than those in S2 and S3

Patterns supporting the Everest hypothesis

- S2 has higher mutation rates and more changes in replication or repair genes than S0 and S1
- S3 shows continued elevation of the mutation rate, increased mutational load, and reduced viability
- S4 shows lower mutation rates, fewer mutator alleles, and higher viability than S2 and S3, indicating the recovery of high-fidelity lineages through recombination

Patterns that would challenge the hypothesis

- S2 and S3 show no consistent increase in mutation rate or changes in replication genes relative to controls
- S4 fails to recover lower mutation rates or improved viability despite the opportunity for recombination with high-fidelity genotypes

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A speculative extension: asexual organisms and the adaptation-fidelity tradeoff

The fundamental tension between adaptation and fidelity illustrated in figure 3, panels B and C, should not be unique to complex eukaryotes—it arises from basic molecular constraints on replication and applies to any entity that must both adapt to changing conditions and maintain

a functional genome. If this tension is real, we should expect to find mechanisms that address it across all domains of life, not just in sexually reproducing organisms. The existence of such mechanisms in bacteria and viruses, therefore, provides indirect support for the Everest hypothesis by confirming that the underlying problem is universal. Unlike canonical sexual systems, asexual lineages typically lack regular, genome-wide recombination in every generation; however, bacterial conjugation and viral recombination can still provide powerful, episodic reshuffling that allows high-fidelity genomes to re-emerge after mutator-driven bursts of adaptation.

For bacteria, conjugative plasmids such as the F factor in Escherichia coli offer a partial analogy to Everest tests. F-like plasmids carry their own origins of replication and transfer and a suite of regulatory genes, but they crucially rely on host replication machinery. Successful conjugation and stable plasmid maintenance depend on the correct expression of many plasmid-borne functions, so hosts with impaired fidelity might accumulate disruptive mutations and fail at transfer or maintenance, biasing plasmid spread toward higher-fidelity lineages. Additionally, mechanisms such as stress-induced mutagenesis allow bacteria to temporarily elevate mutation rates and then restore baseline fidelity—a reversible strategy unavailable to complex multicellular organisms.

Viruses present a particularly interesting case. RNA viruses typically have high mutation rates and exist as quasispecies—clouds of related variants. When adapting to new hosts or evading immunity, elevated mutation rates can accelerate adaptation, but without mechanisms to restore fidelity, viral lineages might be expected to follow the orange trajectory in figure 3C indefinitely. However, many viruses can recombine between co-infecting strains, as documented in phylogenetic analyses of coronaviruses and influenza viruses [Jackson et al., 2021; Lowen, 2017]. This recombination could, in principle, allow the recovery of high-fidelity lineages, paralleling the green trajectory in figure 3. The evolutionary dynamics of SARS-CoV-2—including the extraordinarily sharp epidemic peaks observed in countries such as India and South Africa—may offer a natural experiment in adaptation-fidelity dynamics, though disentangling viral evolution from other epidemiological factors would require careful analysis.

The diversity of these mechanisms—stress-induced mutagenesis, conjugation, viral recombination, and segmented genomes that facilitate reassortment (as in influenza)—suggests that the adaptation-fidelity tradeoff has driven the evolution of multiple independent solutions across the tree of life. Sexual reproduction, with its combination of regular recombination and complex mate-choice mechanisms, may represent the most sophisticated solution—one that allows large, complex genomes to navigate environmental instability without sacrificing long-term integrity.

Conclusions

Several lines of biological evidence are consistent with the Everest hypothesis. Many sexually reproducing species show elaborate traits and risky behaviors that are difficult to explain solely by short-term survival benefits, including extreme migrations, intricate plumage and courtship displays, complex birdsong, biochemical "lock-and-key" systems in plant and fungal fertilization, and strong preferences for symmetry and coordinated performance. Under the Everest framework, such traits naturally function as multigenic tests of fidelity. Even small increases in mutation rate are expected to degrade the precision or reliability of these traits and thereby reduce mating or fertilization success. The experimental and observational approaches outlined here—laboratory evolution under strong selection with and without recombination, manipulations of key traits such as cricket song, phylogenetic contrasts between migratory and

non-migratory lineages, and comparative studies of mutation rate and longevity—provide concrete ways to test whether such traits systematically help maintain low mutation rates.

A strength of the Everest hypothesis is that it is explicitly testable and potentially falsifiable. It would be seriously challenged if experimental lines exposed to strong selection without recombination do not evolve higher mutation rates or show any excess of changes in replication and repair genes, or if lines given opportunities for recombination with high-fidelity genotypes fail to show a restoration of lower mutation rates despite achieving similar levels of adaptation. The hypothesis would also be weakened if migratory species do not differ from close non-migratory relatives in mutation rate, mutational load, longevity, or net gene flow, or if the complexity of sexual and reproductive traits shows no consistent negative association with mutation rate across taxa.

The Everest hypothesis is not intended to displace Fisherian runaway, Zahavi's handicap, or good-genes models, but to make a different claim about the function of reproductive complexity [Fisher 1930; Zahavi 1975; Hamilton and Zuk 1982]. Those frameworks ask how preferences and costly traits evolve and persist, treating sexual selection as acting on a single composite quantity—fitness or condition—with ornaments signaling this one-dimensional "quality." Everest begins instead with a two-dimensional view in which short-term fitness and replication fidelity are distinct properties that can be traded off against one another. It treats fidelity as a long-term target of selection and assigns a specific functional role to otherwise "non-essential" reproductive complexity: not merely to advertise condition, but to reveal how faithfully the genome has been replicated. On this view, mate-choice and fertilization traits act as multigenic stress tests that magnify the phenotypic consequences of elevated mutation rates, making high-mutation lineages more likely to fail in courtship, gamete competition, or biochemical compatibility.

For such tests to work well, mate evaluation mechanisms must be shared widely enough within an interbreeding population to establish a common standard; if criteria are too fragmented, the power to distinguish higher- and lower-fidelity lineages is reduced. This introduces a Fisherian element of convergence on shared preferences, but with a key constraint: the standards that spread are predicted to be complex and multigenic, providing large mutational targets, rather than arbitrary ornaments whose appeal is self-perpetuating; conversely, where standards mix across contact zones, fidelity filtering is expected to be weaker.

The hypothesis's logic extends well beyond animals with behavioral "choice" to protists, corals, shellfish, fungi, and plants, in which choice is expressed primarily through fertilization success and molecular matching. The Everest hypothesis therefore predicts that in virtually all sexually reproducing species, some degree of genetic-fidelity testing—based on at least a handful of independently encoded functions—occurs before mating or fertilization. The elaborate and sometimes puzzling accompaniments of sex are thus not arbitrary ornaments or generic condition signals but evolved instruments that allow lineages to benefit from short-term increases in mutation in changeable habitats without permanently forfeiting the high-fidelity replication required for long-term persistence.

References

Bańbura, Jerzy. "Sexual selection in the Swallow Hirundo rustica-A review." (2005). http://hdl.handle.net/11089/12107

Bank C. Epistasis and adaptation on fitness landscapes. *Annual Review of Ecology, Evolution, and Systematics.* 2022 Nov 2;53(1):457-79. https://doi.org/10.1146/annurev-ecolsys-102320-112153

Duffy, S. "Why are RNA virus mutation rates so damn high?" *PLoS biology* 16.8 (2018): e3000003. https://doi.org/10.1371/journal.pbio.3000003

Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Clarendon Press, Oxford. https://www.google.de/books/edition/The_Genetical_Theory_of_Natural_Selectio/sT4lIDk5no4C?hl=en&gbpv=0

Galhardo RS, Hastings PJ, Rosenberg SM. Mutation as a stress response and the regulation of evolvability. Critical reviews in biochemistry and molecular biology. 2007 Jan 1;42(5):399-435. https://doi.org/10.1080/10409230701648502

Hamilton WD, Zuk M. Heritable true fitness and bright birds: a role for parasites? Science. 1982 Oct 22;218(4570):384-7. https://doi.org/10.1126/science.7123238

Jackson B, Boni MF, Bull MJ, Colquhoun RM, Finber AM, Goater R, Golubchik T, Li K, Sheridan E, del Amo J, Mayoral T, Sherwood E, Robertson DL, Connor TR, Volz EM, Rambaut A. Generation and transmission of interlineage recombinants in the SARS-CoV-2 pandemic. Cell. 2021 Oct 14;184(20):5179-5188.e8. https://doi.org/10.1016/j.cell.2021.08.014

Kimura, Motoo. "On the evolutionary adjustment of spontaneous mutation rates." Genetics Research 9.1 (1967): 23-34. https://doi.org/10.1017/S0016672300010284

Koshio C, Muraji M, Tatsuta H, Kudo SI. Sexual selection in a moth: effect of symmetry on male mating success in the wild. Behavioral Ecology. 2007 May 1;18(3):571-8. https://doi.org/10.1093/beheco/arm017

Lanfear R, Ho SY, Love D, Bromham L. Mutation rate is linked to diversification in birds. Proceedings of the National Academy of Sciences. 2010 Nov 23;107(47):20423-8. https://doi.org/10.1073/pnas.1007888107

Li, Hong-Ju, Jiang-Guo Meng, and Wei-Cai Yang. "Multilayered signaling pathways for pollen tube growth and guidance." *Plant reproduction* 31 (2018): 31-41. https://doi.org/10.1007/s00497-018-0324-7

Lowen AC. Constraints, drivers, and implications of influenza A virus reassortment. Annual Review of Virology. 2017;4(1):105-121. https://doi.org/10.1146/annurev-virology-101416-041726

Lynch M, Ackerman MS, Gout JF, Long H, Sung W, Thomas WK, Foster PL. "Genetic drift, selection and the evolution of the mutation rate." *Nature Reviews Genetics.* 2016 Nov;17(11):704-14. https://doi.org/10.1038/nrg.2016.104

Merilaita S, Lind J. Great tits (Parus major) searching for artificial prey: implications for cryptic coloration and symmetry. Behavioral Ecology. 2006 Jan 1;17(1):84-7. https://doi.org/10.1093/beheco/arj007

Møller AP, Cuervo JJ. Sexual selection, germline mutation rate and sperm competition. BMC Evolutionary Biology. 2003 Apr 18;3(1):6. https://doi.org/10.1186/1471-2148-3-6

Møller AP, Eriksson M. Pollinator preference for symmetrical flowers and sexual selection in plants. Oikos. 1995 May 1:15-22. https://doi.org/10.2307/3545720

Stephenson JF, Stevens M, Troscianko J, Jokela J. The size, symmetry, and color saturation of a male guppy's ornaments forecast his resistance to parasites. The American Naturalist. 2020 Nov 1;196(5):597-608. https://doi.org/10.1086/711033

Takahashi M, Arita H, Hiraiwa-Hasegawa M, Hasegawa T. Peahens do not prefer peacocks with more elaborate trains. *Animal Behaviour*. 2008 Apr 1;75(4):1209-19. https://doi.org/10.1016/j.anbehav.2007.10.004

Zahavi A. Mate selection—a selection for a handicap. *Journal of Theoretical Biology.* 1975 Sep 1;53(1):205-14. https://doi.org/10.1016/0022-5193(75)90111-3

The Everest hypothesis: Supplementary Information

This Supplementary Information contains (SI.1) a toy model illustrating the Everest mechanism and (SI.2) an expanded discussion of relations to previous work.

SI.1. A Toy Model of the Everest Mechanism

Overview

This supplementary material presents a simple mathematical model illustrating how complex mate-choice traits ("Everest traits") can purge mutator alleles from sexual populations. Here, the Everest trait is a simple stand-in for any complex Everest test constructed from multiple Everest loci as described in the main text. The model demonstrates that when mating success depends on the integrity of traits sensitive to mutational damage, mutator genotypes are indirectly selected against—even when the mutator alleles themselves impose no direct fitness cost. This selection can be particularly effective when it acts through sexual selection. An interactive Excel spreadsheet implementing this model is available as Supplementary File S2, allowing readers to explore the effects of different parameter values.

Model Description

The model tracks a haploid population with two biallelic loci:

1. Polymerase locus (P/p): Determines replication fidelity. P = high-fidelity polymerase (low mutation rate); p = mutator polymerase (high mutation rate).

2. Everest trait locus (E/e): Determines mating success. E = intact Everest trait (high mating success); e = degraded Everest trait (low mating success).

This gives four genotypes: PE, Pe, pE, and pe. The critical feature is that the mutation rate from E → e depends on which polymerase allele is carried: non-mutators (P genotypes) experience E → e at rate μ_{wt} = 0.002 per generation, while mutators (p genotypes) experience E → e at rate μ_{mut} = 0.10 per generation (50 × higher). Mutations at the polymerase locus (P ↔ p) are ignored for simplicity. Thus, mutator genotypes are more likely to acquire degraded Everest traits, thereby reducing their mating success and creating indirect selection against mutators.

In the preprint version of this paper, the Excel file may not be available for download. However, it is freely available - please send requests to patrick.ss.home@gmail.com .

Dynamics

Each generation proceeds in two steps:

Step 1 – Selection: Genotype frequencies are weighted by mating success and then normalized so that the sum of genotypes always equals 100%.

Step 2 – Mutation: The Everest trait mutates from E → e at rate μ_{wt} (for P genotypes) or μ_{mut} (for p genotypes). This transfers individuals from PE → Pe and from pE → pe at the appropriate rates.

Parameters

Parameter	Symbol	Value	Description

Non-mutator mutation rate	μ_wt	0.002	E → e rate for P genotypes
Mutator mutation rate	μ_mut	0.10	E → e rate for p genotypes
Mating success (intact)	w_E	1.5	Relative mating success with E
Mating success (degraded)	w_e	1.0	Relative mating success with e
Mutator advantage	—	1.4	Fitness multiplier of p genotype (mutator) during environmental change
Adaptation phase	—	Gen 20-55	Period when mutators are favored

The parameter values are illustrative rather than empirically derived. They are chosen so that mutator effects on the Everest trait are strong (50 × higher E → e rate), while the mating advantage of intact traits (50% higher mating success) is modest. This demonstrates that even moderate mate-choice differentials can effectively purge mutators when those mutators damage the traits under selection.

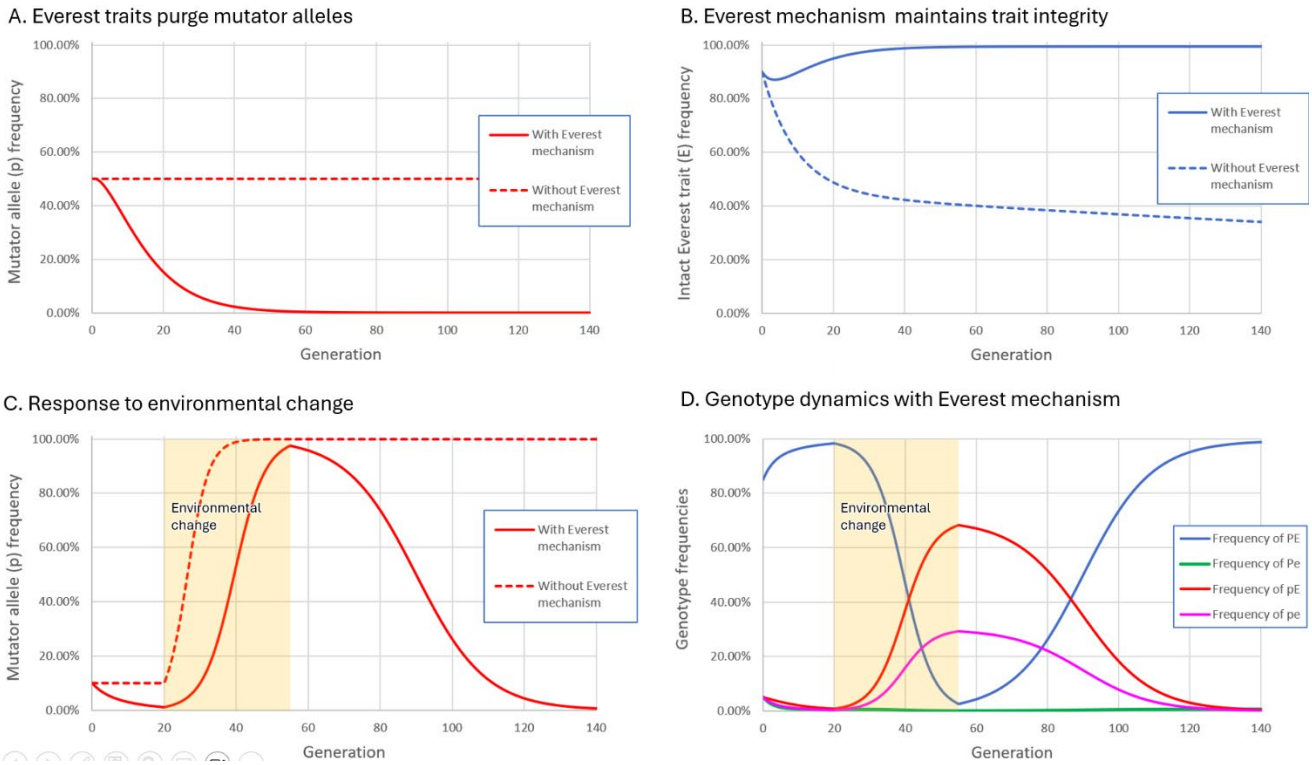

Figure S1. A toy model of the Everest mechanism. (A) In a stable environment, the Everest mechanism rapidly purges mutator alleles (red curve), whereas in the absence of the Everest mechanism (red dashed), mutators persist at their initial frequency. "With Everest mechanism" uses $w_E = 1.5$, $w_e = 1.0$; "without Everest mechanism" sets $w_E = 1.0$, $w_e = 1.0$ (i.e. mating success does not depend on the Everest trait). (B) The Everest mechanism maintains (blue) the integrity of mate-choice traits by purging the mutators that would otherwise degrade them (blue dashed). (C) During an environmental change (orange shading, generations 20-55), mutators are transiently favored (red solid and dashed) in both scenarios

because elevated mutation rates accelerate adaptation; however, only the Everest mechanism allows subsequent recovery (red) of low mutation rates. (D) Detailed genotype dynamics show that mutators with intact Everest traits (pE, solid red) initially rise during adaptation but are eventually converted to pe genotypes (purple) by their own elevated mutation rate, leading to their elimination through reduced mating success.

Model Limitations

This toy model deliberately simplifies several aspects of real biological systems: (1) only two loci are modeled, whereas real Everest traits likely involve many loci, amplifying the effect; (2) haploid genetics are assumed; (3) no recombination occurs between loci; (4) mutations at the polymerase locus are ignored; (5) dynamics are deterministic, with no genetic drift; (6) population size is infinite. Despite these simplifications, the model captures the essential logic: traits that are both important for mating success and sensitive to mutational damage create indirect selection against mutator alleles.

Reproducing Figure S1 with the Excel Spreadsheet

The interactive Excel spreadsheet (Supplementary File S2) can be used to reproduce each panel of Figure S1. The table below lists the parameter settings for each scenario. To simulate the "without Everest mechanism" control, set wE = 1.0 and we = 1.0. To disable the environmental change phase, set the start generation to a value greater than 120.

Panel	A	A	B	B	C	C	D
Scenario	With Everest	Without Everest	With Everest	Without Everest	With Everest	Without Everest	With Everest
μ_wt (non-mutator mutation rate)	0.002	0.002	0.002	0.002	0.002	0.002	0.002
M_mut (mutator mutation rate)	0.1	0.1	0.1	0.1	0.1	0.1	0.1
wE	1.5	1	1.5	1	1.5	1	1.5
we	1	1	1	1	1	1	1
Mutator advantage during environmental change	1.4	1.4	1.4	1.4	1.4	1.4	1.4
Environmental change start	141	141	141	141	20	20	20
Environmental change end	141	141	141	141	55	55	55
Initial frequency PE	45%	45%	45%	45%	85%	85%	85%
Initial frequency Pe	5%	5%	5%	5%	5%	5%	5%
Initial frequency pE	45%	45%	45%	45%	5%	5%	5%
Initial frequency pe	5%	5%	5%	5%	5%	5%	5%
Genotypes displayed	Mutators	Mutators	E intact	E intact	Mutators	Mutators	All four genotypes
Columns to be plotted	G	G	H	H	G	G	B-E

In the spreadsheet, column G shows the mutator allele frequency (for panels A and C), and column H shows the intact Everest trait frequency (for panel B). For panel D, columns B–E show the frequencies of all four genotypes (PE, Pe, pE, pe).

SI.2. Relations 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, differences, and the key points of contrast.

SI.2.1 Sexual selection frameworks

Fisherian runaway

Ronald Fisher's "sexy sons" idea explains exaggerated ornaments through positive feedback between preference and display [Fisher, 1930]. Everest shares the idea that mating preferences can converge within populations, but differs in its target: Everest treats the relevant "quality" as replication fidelity (mutation rate) and predicts that screens are favored when they are complex and mutationally fragile rather than arbitrary ornaments.

Handicap and indicator models

Amotz Zahavi's handicap principle and related indicator models treat costly traits as honest signals of condition or quality [Zahavi, 1975]. Everest is compatible with the general idea that mate choice can "see" quality, but proposes a specific reason why some reproductive traits may become *more complex than strictly necessary*: added complexity can increase sensitivity to mutational disruption, strengthening filtering 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, since recurrent deleterious mutations reduce condition and condition-dependent ornaments are expected to be less well formed when load is high [Hamilton and Zuk, 1982; Pomiankowski et al., 1991]. Everest is close to these models in spirit, but it shifts the emphasis in four ways. First, it treats replication fidelity (mutation rate) as a distinct target of selection, rather than folding everything into "condition" or short-term fitness. Second, it allows adaptation and fidelity to move in opposite directions during environmental change, and it proposes that some reproductive "excess" complexity can evolve because it makes small fidelity defects easier to detect. Third, it expects convergence on one or a few dominant screens within interbreeding populations, because overly fragmented criteria weaken filtering. Fourth, it is intended to apply broadly, including cases where "choice" is expressed through fertilization success and biochemical compatibility rather than overt courtship.

A related contrast concerns the long-term consequences of extreme tests. Under Fisherian runaway or handicap interpretations, increasingly costly ornaments or behaviors are typically expected to impose net viability costs, so populations (or species) that evolve more extreme displays or more demanding behaviors would often be at a disadvantage relative to less extreme ones. Everest predicts an additional, longer-term effect: sufficiently demanding tests can improve population persistence by filtering out mild mutators more efficiently, so extreme tests may sometimes be favored despite their short-term costs if they help maintain genomic integrity over time.

Composite explanations. Some traits may be more naturally interpreted as mixtures of mechanisms rather than purely Everest screens. For example, the peacock's tail could function as a multigenic test, even if modest in size, yet it is extremely large and costly, suggesting a runaway or handicap component that pushes elaboration beyond what complexity alone requires. A similar possibility may apply to some human sexual traits: breast tissue is required for lactation, but permanent enlargement outside pregnancy may also have been shaped by sexual selection. Such traits may reflect Fisherian or handicap dynamics layered on top of any screening role. Everest is therefore framed as complementary to these frameworks rather than a replacement.

SI.2.2 Red Queen and fluctuating selection

Red Queen models emphasize ongoing coevolution (often with parasites) as a driver of sex and recombination [Hamilton and Zuk, 1982]. Everest is compatible with the general idea that fluctuating selection matters, but it makes a different claim about what is being protected: it treats replication fidelity (mutation rate) as a distinct dimension that can be eroded during episodes of strong selection and then restored.

SI.2.3 Mutation rate evolution

A separate literature focuses on the evolution of mutation rates and on the conditions under which mutator alleles spread or are removed. Everest aligns with the premise that selection can, in principle, act on mutation rate, but proposes a phenotypic mechanism for doing so: complex reproductive screens can translate very small differences in replication fidelity into differences in reproductive success, strengthening selection against mild mutators.

Recent work by Roberts and Petrie argues that mate choice for beneficial mutations—rather than simply against high mutational load—can improve genetic quality and may help explain the prevalence of sex [Roberts and Petrie, 2022]. Their model relaxes the assumption of a fixed mutation rate and shows that mate choice could, in some settings, support higher mutation rates when beneficial mutations are available. Everest makes the opposing prediction that demanding reproductive screens will often maintain low mutation rates by purging mutator alleles, especially once short-term adaptation has been achieved.

SI.2.4 Mutation load and classic benefits of recombination

Kondrashov's "hatchet"

Kondrashov's "hatchet" emphasizes purging deleterious mutation load under strong selection (often discussed in the context of synergistic effects among mutations) [Kondrashov, 1988; Kondrashov, 1993]. Everest overlaps with this tradition in treating mutation load as important, but differs in emphasis: it focuses on how selection can act on small differences in mutation rate by routing them through complex reproductive 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 as a general driver of

recombination, but it also predicts a specific role for reproductive complexity: screens can make fidelity differences visible, shaping which lineages contribute disproportionately to future generations.

Müller's ratchet and lineage-level decay

Müller's ratchet emphasizes the irreversible accumulation of deleterious mutations in finite asexual populations and the lineage-level consequences of mutational decay [Müller, 1964; Felsenstein, 1974]. Everest aligns with the broader theme that sex and recombination help maintain long-term genomic integrity, but it highlights a particular route by which selection can act: mate choice and fertilization filters can "see" mutational disruption in complex traits and thereby disproportionately remove lineages with reduced fidelity.

Related ideas appear in classic work on recombination and mutation load. For example, Hermann Müller emphasized that recombination can bring together advantageous mutations arising in different individuals [Müller, 1932], and Motoo Kimura showed that mutator alleles can be indirectly selected against through linkage to the deleterious mutations they generate [Kimura, 1967]. Everest agrees that linkage and indirect selection matter, but stresses that reproductive screening can amplify these effects by converting small differences in fidelity into large differences in reproductive success.

SI.2.5 Genomic integrity and the drift barrier

A further line of work treats sex as a maintainer of genomic integrity. Elisabeth 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 and Hadacek, 2013]. In parallel, drift-barrier theory asks why mutation rates evolve downward only to a limit, proposing that beyond this bound further improvements are too weakly favored to overcome drift [Lynch et al., 2016]. These frameworks align with Everest in treating genomic integrity as a central long-term benefit of sex, but they are often agnostic about which phenotypes allow selection to discriminate among slightly different mutation rates.

In summary, Everest 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 makes small fidelity differences visible to selection. For a concise summary of how conventional frameworks relate to Everest (and of the main diagnostic tests), see table 1 in the main text.

References

Felsenstein J. The evolutionary advantage of recombination. Genetics. 1974 Oct 1;78(2):737-56. https://doi.org/10.1093/genetics/78.2.737

Fisher, R.A. The genetical theory of natural selection: a complete variorum edition. Oxford University Press; 1930. https://www.google.de/books/edition/The_Genetical_Theory_of_Natural_Selectio/sT4lIDk5no4C?hl=en&gbpv=0

Hamilton WD, Zuk M. Heritable true fitness and bright birds: a role for parasites?. Science. 1982 Oct 22;218(4570):384-7. https://doi.org/10.1126/science.7123238

Hill WG, Robertson A. The effect of linkage on limits to artificial selection. Genetics Research. 1966 Dec;8(3):269-94. https://doi.org/10.1017/S0016672300010156

Hörandl E. A combinational theory for maintenance of sex. Heredity. 2009 Dec;103(6):445-57. https://doi.org/10.1038/hdy.2009.85

Hörandl E, Hadacek F. The oxidative damage initiation hypothesis for meiosis. Plant reproduction. 2013 Dec;26(4):351-67. https://doi.org/10.1007/s00497-013-0234-7

Kimura M. On the evolutionary adjustment of spontaneous mutation rates. Genetics Research. 1967 Feb;9(1):23-34. https://doi.org/10.1017/S0016672300010284

Kondrashov AS. Deleterious mutations and the evolution of sexual reproduction. Nature. 1988 Dec 1;336(6198):435-40. https://doi.org/10.1038/336435a0

Kondrashov AS. Classification of hypotheses on the advantage of amphimixis. Journal of Heredity. 1993 Sep 1;84(5):372-87. https://doi.org/10.1093/oxfordjournals.jhered.a111358

Lynch M, Ackerman MS, Gout JF, Long H, Sung W, Thomas WK, Foster PL. "Genetic drift, selection and the evolution of the mutation rate." *Nature Reviews Genetics.* 2016 Nov;17(11):704-14. https://doi.org/10.1038/nrg.2016.104

Müller HJ. Some genetic aspects of sex. The American Naturalist. 1932 Mar 1;66(703):118-38. https://doi.org/10.1086/280418

Müller HJ. The relation of recombination to mutational advance. Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis. 1964 May 1;1(1):2-9. https://doi.org/10.1016/0027-5107(64)90047-8

Pomiankowski A, Iwasa Y, Nee S. The evolution of costly mate preferences I. Fisher and biased mutation. Evolution. 1991 Sep;45(6):1422-30. https://doi.org/10.1111/j.1558-5646.1991.tb02645.x

Roberts G, Petrie M. "Sexual selection for males with beneficial mutations." *Scientific Reports*. 2022 Jul 23;12(1):12613. https://doi.org/10.1038/s41598-022-16002-y

Zahavi A. Mate selection—a selection for a handicap. *Journal of Theoretical Biology.* 1975 Sep 1;53(1):205-14. https://doi.org/10.1016/0022-5193(75)90111-3