Resetting the rules: Sex chromosome turnover and mitonuclear conflict as engines of biodiversity

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Abstract

Life's diversity depends on both the stability and flexibility of inheritance systems. Mitochondrial and nuclear genomes must cooperate to sustain oxidative phosphorylation and cellular metabolism, yet their distinct inheritance routes often create conflict. Sex chromosomes modulate these conflicts by biasing transmission of thousands of nuclear genes toward one sex. Here I synthesize evidence that turnover in sex-determining systems, i.e., the gain, loss, or replacement of X, Y, Z, or W chromosomes, acts as an evolutionary "escape hatch'' from persistent mitonuclear conflict. When nuclear-encoded mitochondrial (N-mt) genes become trapped in non-recombining sex-linked regions, coadaptation with maternally inherited mitochondria is constrained, producing sex-biased incompatibilities. Subsequent turnover can release these loci, resetting conflict intensity and creating pulses of hybrid breakdown and relief. Comparative data from plants, invertebrates, fishes, amphibians, birds, and mammals reveal predictable signatures of such cycles in genomes and hybrid zones. Viewing sex chromosome turnover through the lens of mitonuclear ecology links molecular evolution to macro-biodiversity: the same genomic resets that resolve conflict can also accelerate diversification.

Keywords

Sex chromosome turnover, Mitonuclear conflict, Inheritance asymmetry, Genomic conflict, Hybridization and reproductive isolation, Diversification and macroevolution, Mitochondrial sweeps

Key Points

- **Inheritance architectures evolve, and these changes reshape biodiversity.** Sex chromosome turnover rewires patterns of nuclear–cytoplasmic co-transmission, altering how mitonuclear coadaptation proceeds and how quickly reproductive barriers form or erode.
- **Mitonuclear conflict is not static but cyclical.** When nuclear-encoded mitochondrial (N-mt) genes become trapped in young non-recombining sex-linked strata, conflict intensifies ("entrapment"). Subsequent turnover or recombination restoration releases these loci, relieving conflict and generating predictable pulses of hybrid dysfunction and recovery.
- **Turnover history predicts the strength and sex-bias of hybrid incompatibilities.** Stable XY and ZW systems show entrenched, sex-specific mitonuclear signatures, whereas labile systems (e.g. fishes, frogs, willows, insects) exhibit episodic, lineage-specific amplification–relief cycles tied to the timing and direction of turnover (XY→ZW, ZW→XY).
- **Comparative genomics now reveals repeated capture and release of N-mt loci across the tree of life.** Plants with CMS–Rf cycles, crustaceans with endosymbiont-driven feminization, DUI bivalves, and rapidly diversifying fishes and frogs all show turnover-driven restructuring of mitonuclear interactions.
- **Hybrid zones offer powerful tests of localized conflict versus genome-wide sweeps.** Mito-aware genomic clines, sex-differentiated coverage, and temporal dynamics distinguish N-mt entrapment from W-driven maternal sweeps or autosomal DMIs, enabling falsifiable tests of the escape-hatch model.
- **Turnover links microevolutionary conflict to macroevolutionary diversification.** Clades with frequent turnovers (cichlids, darters, ranid frogs, willows) show elevated species richness even after accounting for ecology, suggesting that genomic "resets" repeatedly open new adaptive pathways and shape global biodiversity patterns.

Introduction

Biodiversity reflects not only ecological opportunity and environmental context, but also the genomic rules of inheritance that govern how organisms evolve (Hill 2019, Rand & Mossman 2020). Every lineage inherits a distinct framework for how genes are transmitted through recombination, cytoplasmic inheritance, and sex determination, and those rules govern how quickly adaptation, conflict, and speciation unfold (Payseur et al. 2018). Shifts in these inheritance architectures, from the origin of sex chromosomes to their repeated turnover, rewrite the map of genomic interaction and coevolution (Vicoso 2019). Understanding how these systems evolve can reveal the ways in which inheritance itself generates, constrains, and reshapes life's diversity. Because inheritance systems set the tempo of adaptation, changes

in those systems can resonate from genes to ecosystems. Recognizing inheritance architecture as a driver of biodiversity helps explain why some lineages diversify explosively while others remain static, even under similar ecological conditions (El Taher et al. 2021).

Ecological opportunity, sexual selection, and developmental constraint have long been invoked to explain uneven patterns of diversification across clades (e.g., Schluter 2000, Brakefield 2006, Seehausen 2006). Yet, variation in biodiversity also reflects a less examined factor: the *genetic architecture* of inheritance. Here, genetic architecture refers to the structure of recombination, cytoplasmic transmission, and sex determination that governs how genes are co-inherited across generations (Barton & Charlesworth 1998, Rice 2013, Bachtrog et al. 2014, Greiner et al. 2015). By reshaping linkage and transmission, these systems govern the pace at which conflicts arise and are resolved between genomes. Over evolutionary timescales, inheritance architectures are not static. Polyploidy, endosymbiosis, and horizontal transfer have repeatedly rewritten the rules of genomic cooperation. In animals and many plants, sex chromosome turnover provides a recurrent, lineage-specific mechanism for doing so. Each replacement of a sex-determining region (SDR) redraws the genetic map of sex-biased transmission, altering how nuclear and cytoplasmic genomes interact. The increasing availability of genomic data across diverse taxa has revealed that these events occur far more frequently than once assumed (sometimes every few million years in fishes and frogs; Kitano & Peichel 2012, Gammerdinger & Kocher 2018, Jeffries et al. 2018, Evans et al. 2024), and their cumulative effects on genomic conflict, reproductive isolation, and lineage diversification are only now becoming clear.

Because inheritance asymmetry determines which genes share evolutionary fate, it also defines where conflict can arise between genomes. Whenever cytoplasmic and nuclear genes follow different transmission routes, their evolutionary interests may diverge, generating tension that manifests in physiological dysfunction or reduced fertility (Rice 2013, Havird et al. 2019, Rand & Mossman 2020). Such conflicts are especially pronounced when they interact with the asymmetric transmission of sex chromosomes, which bias large fractions of the genome toward one sex and therefore toward one cytoplasmic lineage (Burton & Barreto 2012, Hill 2019).

In this Review, I argue that the strength and genomic location of mitonuclear conflict through time is shaped by turnover in the identity of the sex determining chromosome (i.e., whether a lineage maintains a long-standing XY or ZW pair or frequently shifts among different SDRs). Building on theory that mitonuclear conflict selects on recombination, sex linkage, and, in some contexts, biparental or sperm-line mitochondrial transmission within fixed systems (Rice 2013, Connallon et al. 2018, Havird et al.

2019, Radzvilavicius et al. 2021), I propose an "escape-hatch'' framework in which new sex determining regions capture or release nuclear-encoded mitochondrial (N-mt) loci, generating pulses of conflict that leave detectable genomic scars. Because turnover reshapes which N-mt loci reside on the sex chromosomes, the magnitude and even the direction of classic speciation patterns such as Haldane's rule and the large-X/Z effect should vary with sex chromosome age and identity. In highlighting these links, the escape-hatch model integrates inheritance architecture into the broader study of biodiversity, reframing sex chromosome evolution not as a static consequence of genomic conflict but as a dynamic engine of diversification.

Mitonuclear coadaptation, conflict, and sex chromosomes

Mitochondria are central not only to ATP production through oxidative phosphorylation (OXPHOS) but also to heat generation, apoptosis, calcium homeostasis, steroid synthesis, and production of reactive oxygen species (Rand et al. 2004, Ballard & Pichaud 2014). These functions depend on tight coordination between maternally inherited mtDNA and hundreds of nuclear-encoded mitochondrial (N-mt) genes (Hill 2019). This cooperation persists despite distinct inheritance routes: mtDNA is transmitted only through the cytoplasmic lineage, whereas nuclear loci recombine biparentally. Such asymmetry can generate conflict whenever selection on mtDNA diverges from that on the nuclear background or when hybridization brings together incompatible mito-nuclear combinations (Burton & Barreto 2012, Havird et al. 2019, Rand & Mossman 2020). Mutations advantageous to the maternally inherited mtDNA may spread even if deleterious to males ("Mother's Curse"), while compensatory mutations must arise in nuclear genes to restore coadaptation (Gemmell et al. 2004). Even under a fixed inheritance architecture, such mitonuclear coadaptation cycles impose a sizable, male-biased mitochondrial load that is strongly dependent on effective population size (Ne), providing a quantitative baseline for transient male fitness costs against which the turnover-driven pulses proposed here can be evaluated (Connallon et al. 2018). Hybridization between divergent lineages can further disrupt these pairs, producing reduced fitness, sex-biased sterility, or energetic inefficiency, phenomena documented in copepods, mussels, *Drosophila*, and fishes (Burton et al. 2006, Ellison & Burton 2008, Saunier et al. 2014, Hill 2019).

Sex chromosomes intersect with these dynamics because they bias the co-transmission of nuclear loci with mtDNA. Z chromosomes spend two-thirds of their evolutionary history in males, X chromosomes two-thirds in females, while W and Y are confined to the heterogametic sex, with the W co-inherited with mtDNA and the Y entirely decoupled from it (Mank 2012). When N-mt genes become trapped in low-recombination, sex-linked strata, their inheritance patterns shift in ways that can generate maladaptive combinations, segregation distortion, or hybrid breakdown. These interactions underlie classic

macroevolutionary patterns such as Haldane's rule (Haldane 1922, Orr 1997) and the large-X/Z effect (Ellegren 2009, Llopart 2012), and they set the stage for turnover to modulate both the intensity and the sex bias of mitonuclear conflict. Because each turnover event changes which genomic regions share transmission with mitochondria, it effectively resets the evolutionary context in which mito-nuclear coadaptation evolves.

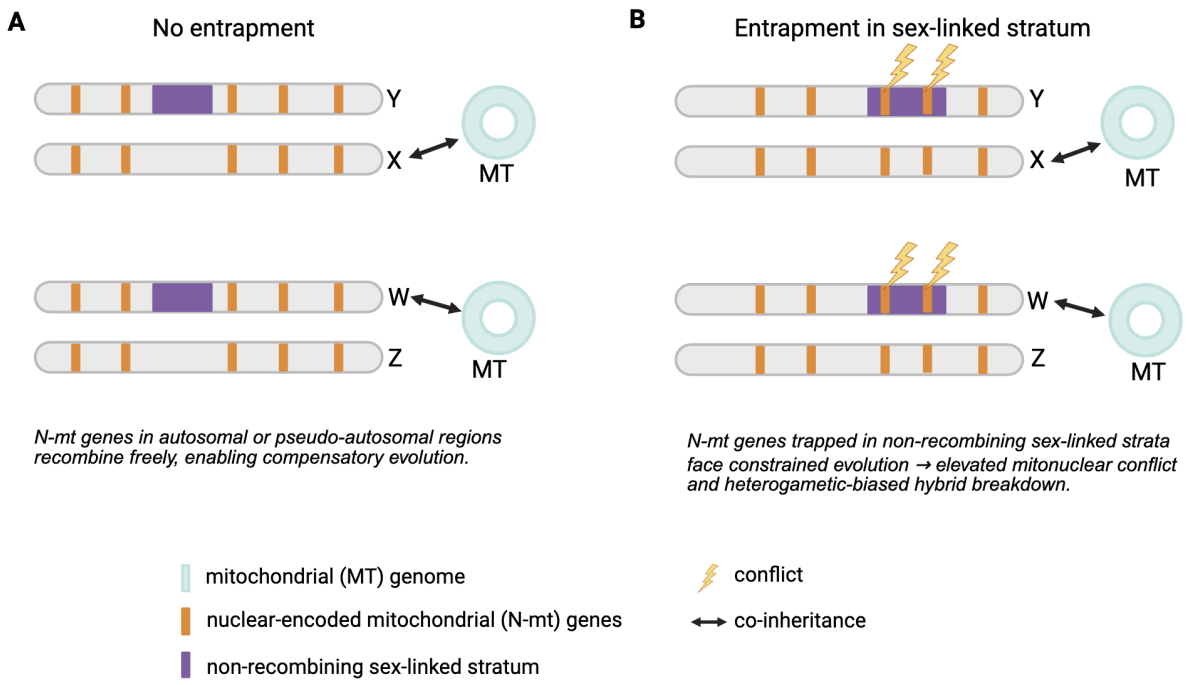

Figure 1. Entrapment, not enrichment: how sex-linked strata bias mitonuclear loci. Schematic chromosomes illustrate two genomic contexts for nuclear-encoded mitochondrial (N-mt) genes (orange). (A) N-mt genes within autosomal or pseudo-autosomal regions recombine freely and can track mitochondrial evolution, minimizing mitonuclear conflict. (B) Local capture of N-mt genes in a non-recombining sex-linked stratum (purple) constrains compensatory evolution (Princepe & de Aguiar 2024), intensifying mitonuclear conflict (lightning bolts) and can contribute to heterogametic-biased hybrid breakdown when mtDNA-conditional effects are demonstrated. MT = mitochondrial genome (light blue).

Insights from stable XY and ZW systems

Across the tree of life, certain lineages have maintained the same sex chromosome systems for tens to hundreds of millions of years (Graves 2016, Vicoso 2019). These *stable* architectures offer a view of how

long-term genomic arrangements shape mitonuclear dynamics once turnover has ceased. In such systems, the signatures of conflict are often deeply embedded: sex-linked strata accumulate degeneration, meiotic drive elements, and dosage-compensatory changes that influence how nuclear and cytoplasmic genomes coevolve (Charlesworth 1991, Bachtrog 2013). Although mammals and birds are the best-studied examples, similar principles emerge in insects, nematodes, and flowering plants where heterogamety has remained constant over long evolutionary timescales (Bull 1983, Charlesworth 2017, Renner 2014).

Stable XY systems

In taxa with stable XY systems, the X is largely recombining in females and hemizygous; the Y is gene-poor and decoupled from mtDNA (Bachtrog 2013). Across mammals, N-mt genes are often depleted from the X (consistent with selection to relocate conflict-prone loci to autosomes; Drown et al. 2012, Dean et al. 2014), reducing persistent sex-linked mitonuclear conflict even as heterogametic males often suffer hybrid costs (**Table 1**). The observed paucity of N-mt genes on the mammalian X fits models in which mitonuclear conflict favors sex-independent segregation or relocation off sex chromosomes; related models predict sex-specific selection on recombination and, context-dependently, paternal leakage (i.e., the rare transmission of mitochondria through sperm, which can transiently increase co-transmission between male nuclear alleles and mtDNA; Radzvilavicius et al. 2021). Comparable patterns occur in long-conserved XY systems of *Drosophila*, where mito-sensitive genes are preferentially autosomal and male-biased sterility is pervasive (Gallach et al. 2010, Lindsley et al. 2013, Rogell et al. 2014, Ågren et al. 2020). In nematodes, where X-autosome balance governs sex, N-mt loci are similarly under-represented on the X, hinting at parallel selective pressures to decouple mitochondria from sex-linked inheritance (Dean et al. 2014).

Stable ZW systems

In taxa with stable ZW systems such as birds, the Z spends two-thirds of its time in males, while the W is maternally co-inherited with mtDNA. The avian Z retains many functional genes, including some N-mt loci, and female-biased dysfunction is common (Ottenburghs 2022). Several avian hybrid zones show coincident clines of mtDNA and Z-linked alleles (Lopez et al. 2021, Del-Rio et al. 2022), patterns consistent with (i) mitonuclear entrapment of N-mt loci in Z-linked low-recombination strata or (ii) W-linked meiotic drive causing mitochondrial sweeps (Irwin 2025) (**Table 1**). Because female-biased hybrid breakdown could also arise from intrinsic W–Z genic incompatibilities unrelated to mitochondria, we

182 treat "entrapment'' here as strictly mtDNA-conditional and recommend, for example, testing fitness of F_1
183 females vs. F_1 males, applying mito-aware genomic-cline analyses (e.g., local ancestry near SDR vs.
184 genome-wide), and assaying OXPHOS performance in reciprocal hybrids to separate these mechanisms
185 (see *Testing the escape-hatch hypothesis across scales* below).

186 Stable ZW systems outside vertebrates reveal similar principles. In Lepidoptera, the maternally inherited
187 W chromosome is often enriched in repetitive elements and may carry sequences derived from
188 cytoplasmic endosymbionts or meiotic-drive elements (Fraïsse et al. 2017, Dalíková et al. 2017). Many
189 butterflies and moths exhibit female-biased hybrid sterility and tight mito-W associations consistent with
190 historical drive or entrapment. In some crustaceans (e.g., *Armadillidium*), bacterial endosymbionts such
191 as *Wolbachia* can feminize genetic males, effectively replacing the W and altering mitochondrial
192 inheritance patterns - an endosymbiotic analogue of sex chromosome turnover that reshapes mito-nuclear
193 coevolution (Cordaux et al. 2011). Even flowering plants with long-standing dioecy, such as *Silene*, show
194 analogous mt-linked sex-determinant systems that maintain stable yet conflict-prone inheritance
195 architectures (Charlesworth 2016).
196
197 *Takeaways*

198 Stable systems show that mitonuclear conflict can leave persistent, sex-linked signatures, but their ancient
199 architectures limit leverage to ask how *changes* in linkage reshape conflict dynamics. They establish the
200 baseline expectation - entrapment, degeneration, and dosage compensation - against which more labile
201 systems can be compared.

202

203 **Table 1. Comparative predictions for mitonuclear conflict across sex chromosome systems**.
204 Predictions for the strength, persistence, and sex-bias of mitonuclear conflict across stable versus labile
205 sex-determination lineages and heterogametic systems (XY vs ZW). Rows highlight representative taxa,
206 characteristic genomic context (e.g. age of the SDR, degree of N-mt linkage), predicted amplification vs
207 relief phases of conflict, expected patterns of hybrid dysfunction, and genomic signatures of barriers.
208 Conflict tends to be most entrenched in ancient, stable X/Z systems, whereas labile lineages with frequent
209 turnover experience more episodic, lineage-specific conflict. ZW systems (purple) often show stronger
210 maternal-haplotype asymmetries because the W chromosome co-inherits with mitochondria, while XY
211 systems (blue) typically display male-biased breakdown during early phases when newly formed strata
212 capture N-mt loci.

Comparative predictions for mitonuclear conflict across sex-chromosome systems				
Axis	**Labile XY**	**Labile ZW**	**Stable XY**	**Stable ZW**
Exemplars	Fishes, amphibians, some insects, and plants with frequent SDR turnover	Fishes, amphibians, crustaceans, and plants with W-linked strata (e.g., *Silene*, *Salix*)	Mammals, *Drosophila*, nematodes	Birds, Lepidoptera, some reptiles and flowering plants (e.g., *Fragaria*)
State / Age	Young / neo-XY; patchy suppression	Frequent XY↔ZW switches; homomorphy common	Ancient, conserved XY; long-term suppression	Ancient, conserved ZW
N-mt context	Recent capture of N-mt loci possible	Episodic capture/release of N-mt as SDR moves	X depleted of N-mt; Y decoupled from mtDNA	Z retains many genes; W co-inherited with mtDNA
Conflict expectation	Amplification soon after capture; relief after turnover/expansion	Transient female-biased conflict when W-linked strata trap N-mt	Moderate, persistent (few N-mt loci involved)	Persistent, female-biased; possible W-drive
Hybrid dysfunction	Episodic **male-biased** (heterogametic) breakdown	Lineage-specific; often short-lived	**Male-biased**; large-X effect	**Female-biased**; large-Z effect
Turnover dynamics	Frequent XY↔polygenic transitions	Frequent XY↔ZW; sex-reversal 'fountain-of-youth' recombination	Rare/absent	Rare/absent
Genomic signatures	Sharp local clines at SDR; transient barriers; limited strata	Moving SDR hotspot; test entrapment vs W-drive	Entrenched X-linked barriers; weak mito–X coupling	Mito–Z cline coincidence; entrenched Z effect

Note: Female-biased dysfunction in ZW clades can arise from Z-W genic incompatibilities independent of mitochondria. "Entrapment" specifically predicts mtDNA-conditional effects (see Comparative genomics and new opportunities for diagnostics).

Labile systems as natural experiments

In contrast to the long-entrenched XY and ZW systems of mammals and birds, many lineages spanning fishes, amphibians, insects, crustaceans, and plants display extraordinary fluidity in sex determination. Frequent turnovers, fusions, and XY↔ZW switches continually rewrite the genomic landscape of inheritance, generating powerful natural experiments for understanding how shifting linkage relationships modulate mitonuclear interactions. This lability reveals that sex chromosome turnover is not a vertebrate peculiarity but a recurrent evolutionary strategy for reconfiguring inheritance across eukaryotes.

Rapid turnover and compound systems in fishes and insects

Teleost fishes exemplify the fastest known rates of turnover. Multiple lineages (cichlids, sticklebacks, salmonids, Medaka, darters) exhibit rapid and repeated transitions, often accompanied by fusions and even XY↔ZW switches within <15–20 Myr (Kitano & Peichel 2012, Lubieniecki et al. 2015, Myosho et al. 2015, El Taher et al. 2021, Radcliffe et al. 2025). Cichlids are among the most dynamic: over a dozen independent XY/ZW systems occur across at least ten linkage groups in East African radiations (<2 Myr

old), including several compound (W + Y + B-chromosome) systems (Gammerdinger & Kocher 2018). B-chromosomes are supernumerary chromosomes, typically dispensable, that can sometimes carry sex-determining or meiotic-drive loci, thereby interacting with primary sex chromosomes. This "hot-potato'' pattern where new sex-determining alleles repeatedly invade, degenerate, and are replaced produces cycles of transient conflict and resolution (Blaser et al. 2014).

Comparable tempos occur in non-vertebrate taxa. Drosophilids repeatedly lose the Y and form neo-X systems on similar timescales (Vicoso & Bachtrog 2015), and many beetles and true bugs undergo autosome–sex fusions or X_1X_2 rearrangements (Blackmon & Demuth 2014, Blackmon et al. 2017). Such frequent reorganization suggests that sex chromosome lability, and the resulting reshuffling of N-mt linkage, is a general feature of rapidly diversifying clades.

Homomorphy and fountain-of-youth dynamics in frogs and plants

Anurans provide an intermediate pattern: many species retain homomorphic, recombining sex chromosomes that limit long-term entrapment of N-mt loci, whereas others (e.g., *Xenopus*, brown-frog complexes) show recurrent strata formation and XY↔ZW transitions (Evans et al. 2024, Miura et al. 2024, Uno & Matsubara 2024). In *Xenopus*, seven turnovers across ~321 Myr imply rates of ≈0.02 events Myr^{-1} (Evans et al. 2024); *Ranidae* frogs show comparable estimates (~1 per 50 Myr; Jeffries et al. 2018).

These dynamics mirror the "fountain-of-youth'' effects observed in plants such as *Silene*, *Rumex*, and *Populus*, where occasional recombination between sex chromosomes rejuvenates homomorphy and delays degeneration (Renner 2014, Muyle et al. 2017). In many dioecious angiosperms, mitochondrial mutations causing cytoplasmic male sterility (CMS) drive the evolution of nuclear restorer (Rf) genes that often become sex-linked, establishing direct cytoplasmic-nuclear feedback similar to mitonuclear conflict (Touzet & Meyer 2014, Renner 2014). Independent origins of sex chromosomes in *Silene*, *Fragaria*, *Salix*, and *Populus* repeatedly capture or release these Rf loci, producing recurrent cycles of entrapment and relief that parallel the escape hatch dynamic described here (Bergero & Charlesworth 2011, Muyle et al. 2017). Both groups illustrate how partial recombination can maintain flexibility in linkage and periodically reset opportunities for mitonuclear realignment.

Endosymbiont-driven transitions in crustaceans and other invertebrates

In crustaceans and other invertebrates, sex determination is further destabilized by cytoplasmic endosymbionts. Isopods and copepods frequently experience feminization or sex-ratio distortion induced

by *Wolbachia* or *Cardinium*, which can precipitate turnover by shifting the effective heterogametic sex (Rigaud et al. 1997, Bouchon 1998, Cordaux et al. 2011). Because these bacteria are maternally inherited, they couple cytoplasmic and nuclear interests in unique ways, amplifying mitonuclear conflict and sometimes driving fixation of new sex-determining alleles (Charlat et al. 2007, Engelstädter & Hurst 2009). Similar cyto-nuclear interactions likely underlie recurrent transitions in other arthropods with complex maternal symbiont transmission, including Drosophila, butterflies, and mites (Hurst & Frost 2015, Kageyama et al. 2017).

Takeaways

Across fishes, amphibians, insects, crustaceans, and plants, turnovers often occur on timescales of 10^3-10^6 years (i.e., within the span of population divergence rather than deep phylogenetic splits) and are typically accompanied by localized recombination suppression around newly arising sex-determining loci (Saunders 2019, Palmer et al. 2019). These "resets'' repeatedly alter the linkage context for nearby N-mt genes, creating natural experiments: some turnovers generate sharp but transient mitonuclear conflicts when N-mt loci become newly sex-linked, whereas others erase conflict when recombination is restored or the SDR shifts to a different chromosome.

Classic models of sex chromosome turnover highlight a recurring set of underlying mechanistic factors, including sexually antagonistic selection, mutational load on non-recombining Y/W regions, genetic drift, heterochiasmy, and sex reversal (reviewed in Vicoso 2019), that jointly influence when new SDRs arise, when recombination is suppressed, and whether existing systems become evolutionarily "trapped'' or replaced. These same factors are expected to modulate the likelihood and strength of mitonuclear entrapment, with strongest effects in young non-recombining strata and weaker effects under homomorphy or when sex-reversed recombination ("fountain-of-youth'' effects) (Perrin 2009) maintains gene exchange between the sex chromosomes (see **Table 2**). Together, these diverse systems demonstrate that the genomic architecture of inheritance, and the conflicts it mediates, can be repeatedly rewritten on surprisingly short timescales.

Table 2. Mechanistic drivers of sex chromosome turnover and predicted mitonuclear outcomes. Primary evolutionary mechanisms that generate or resolve sex chromosome turnover, grouped by how they restructure inheritance between nuclear and cytoplasmic genomes. Each mechanism alters the

linkage of nuclear-encoded mitochondrial (N-mt) genes, thereby modulating the strength and direction of mitonuclear conflict. Drivers such as sexually antagonistic selection and chromosomal fusion amplify conflict by trapping N-mt loci in newly non-recombining regions, whereas mutation load, sex reversal, and polyploidy act as release valves that restore recombination or duplicate interacting partners. Example systems and key references illustrate the tempo and diversity of turnover processes across vertebrates and plants.

Driver	Mechanistic basis	Predicted impact on conflict	Example systems	Key references
Sexually antagonistic selection	New SDR invades linked to sex-beneficial allele	Captures nearby N-mt loci → conflict ↑	Cichlids, guppies	Kitano & Peichel 2012; Blaser et al. 2014
Mutation load ("hot-potato")	Degeneration of Y/W favors SDR replacement	Releases trapped N-mt loci → conflict ↓	Frogs, salmonids	Charlesworth & Wall 1999; Jeffries et al. 2018
Heterochiasmy	Recombination suppressed in one sex	Maintains moderate entrapment	Birds, mammals	Lenormand 2003; Vicoso 2019
Sex reversal / ESD	Recombination restored through sex-reversal or environmental triggers	Resets linkage, conflict decays	Ranid frogs, reptiles	Perrin 2009; Miura et al. 2024
Chromosomal fusion	Expansion of non-recombining region via fusion	Traps new N-mt loci → transient conflict ↑	Sticklebacks, willows	Kitano et al. 2009; Pucholt et al. 2017
Polyploidy / WGD	Duplicates SDRs and N-mt partners	May buffer or restructure conflict	Salmonids, angiosperms	Allendorf & Thorgaard 1984; Muyle et al. 2017

Turnover as an evolutionary "escape hatch"

Sex-chromosome turnover may function as an evolutionary escape hatch, i.e., a mechanism by which lineages reset inherited conflicts between nuclear and cytoplasmic genomes. When deleterious mitonuclear interactions are amplified because an N-mt gene becomes entrapped within a Y- or W-linked non-recombining stratum, a subsequent turnover (either restoring recombination or moving the sex-

determining locus to a new chromosome) can release that locus from persistent sex-biased inheritance (Fig. 2). Similar "reset" processes have been proposed in other genomic contexts, such as translocation of selfish elements (Hurst 1995, Hurst & Werren 2001) or recombination restoration following neo-sex fusion decay (Perrin 2009), but here I extend the concept specifically to the resolution of cyto-nuclear conflict. This framework extends baseline expectations from coadaptation theory, where male load accrues under fixed linkage, by showing how turnover restructures linkage to first amplify and then relieve that load in predictable phases (Connallon et al. 2018).

Predictions

1. **Amplification phase:** Nascent non-recombining strata that capture N-mt loci produce intensified, often heterogametic-biased conflict, segregation distortion, and steep genomic clines centered on the SDR (Fig. 2).
2. **Relief phase:** Later turnover or recombination expansion decouples the locus from tight sex linkage, lowering conflict and eroding barriers (Fig. 2); this leaves a genomic "scar" characterized by attenuating introgression deficit/discordant ancestry through time.
3. **XY vs. ZW asymmetries:** Because the W co-transmits with mtDNA whereas the Y does not, ZW systems should more often exhibit persistent female-biased breakdown and mt sweeps via W-drive; XY systems may show more frequent relief via turnover if N-mt loci are purged from tight X-linkage (Table 1).
4. **Demography dependence:** Transient costs should scale with effective population size, i.e., large costs in very small and very large Ne, minimized at intermediate Ne per coadaptation theory; turnover modulates the duration and visibility of these costs by breaking or restoring tight sex-linked N-mt inheritance (Connallon et al. 2018).

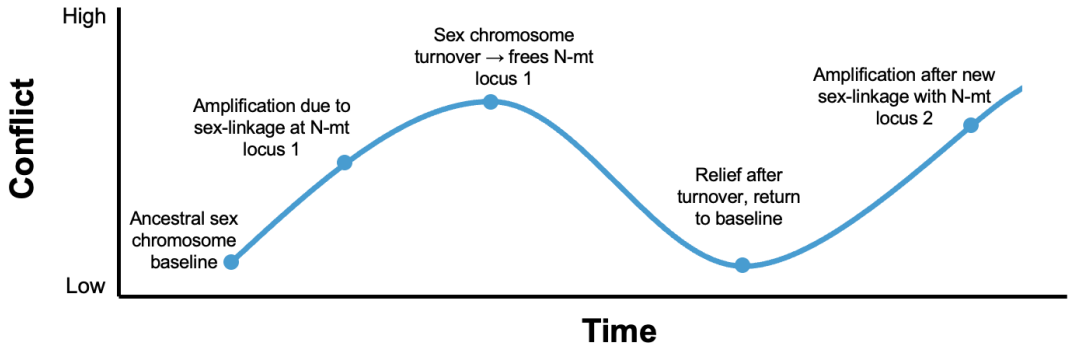

Figure 2. Turnover as an evolutionary "escape hatch." Conceptual timeline showing how cycles of

sex chromosome turnover modulate the intensity of mitonuclear conflict through time. Conflict is low under the ancestral sex-determining region (SDR). When a turnover event establishes a new SDR that captures an N-mt locus, conflict is amplified (upslope) as compensatory evolution is constrained in the non-recombining stratum. A subsequent turnover that frees that locus from the SDR relieves conflict (downslope). If a later turnover again captures a different N-mt locus, conflict rises anew. This model predicts repeated amplification–relief cycles that may generate detectable pulses and localized ancestry deficits; time-calibrated tests are now needed to validate this dynamic.

From hybrid breakdown to biodiversity: turnover as an engine of diversification

Hybrid zones provide natural experiments for testing how sex-linked mitonuclear entrapment and its subsequent release shape reproductive isolation and, ultimately, diversification. In taxa with stable XY systems such as mammals and *Drosophila*, hybrid breakdown typically affects the heterogametic sex (males), largely through hemizygous exposure and dosage-compensation limits rather than active mitonuclear conflict, as most nuclear-encoded mitochondrial (N-mt) loci have relocated to autosomes (Drown et al. 2012, Gallach et al. 2010; Table 1). In contrast, stable ZW systems such as birds, snakes, and butterflies often exhibit coincident mitochondrial and Z-linked clines and female-biased hybrid sterility or inviability (Lopez et al. 2021, Del-Rio et al. 2022). Whether such patterns arise from entrapment of N-mt genes in low-recombination strata or from W-linked drive can be distinguished by spatial scope and temporal dynamics, i.e., local versus genome-wide mitochondrial sweeps, transient sex-ratio skews, and W-centric centromeric signatures (Irwin 2025; **Fig. 3**).

Turnover-prone lineages, including many fishes, amphibians, and plants, are predicted to follow a two-phase cycle. When new sex-determining regions capture N-mt loci, conflict intensifies and hybrid incompatibilities peak (the amplification phase; Fig. 2). Subsequent turnover or recombination restoration decouples these loci from sex linkage, relieving conflict and eroding barriers (the relief phase; Fig. 2). These cycles predict temporal heterogeneity in the strength and sex-bias of reproductive isolation among closely related taxa (Radcliffe et al. 2025). Homomorphic systems such as many frogs exhibit only transient entrapment because recombination and sex reversal recurrently reset linkage (Dufresnes & Crochet 2022). The tempo and mode of postzygotic isolation therefore depend not only on sequence divergence but on sex chromosome age and turnover history.

At macroevolutionary scales, these repeated genomic resets appear to catalyze diversification. Clades with rapid turnover, including African cichlids, darters, ranid frogs, and willows, are among the most species-rich vertebrates and plants (Kitano & Peichel 2012, Jeffries et al. 2018, Muyle et al. 2017, Radcliffe et al. 2025). Each turnover can reconfigure recombination landscapes and release formerly trapped genetic variation, analogous to the repatterning events following whole-genome duplication, and may promote diversification even after accounting for ecological context (e.g., lake vs. river, island vs. mainland), life history, and range size. Comparative phylogenetic analyses already suggest a positive association between turnover rate and net diversification in fishes and flowering plants, even after accounting for ecological opportunity (Palmer et al. 2019, El Taher et al. 2021). Recent work in willows reveals rapid sex chromosome turnover coinciding with reduced introgression and lineage divergence (see Xue et al. 2024). By altering which genes are sex-linked, turnover also modulates the targets of sexual selection, redirecting inheritance of traits involved in mate choice, signaling, and parental care, and thereby influencing how new species form.

Turnover extends its influence beyond speciation to ecology and conservation. Because temperature and stress can induce sex reversal, environmental change may accelerate turnover in ectotherms, coupling climate sensitivity with genomic conflict (Miura et al. 2024). Lineages with flexible sex determination may track shifting environments more effectively but risk demographic collapse under extreme sex-ratio skew. In plants, cytoplasmic male sterility (CMS)-linked turnovers reshape pollination networks and seed set by altering sex ratios and mating systems (Touzet & Meyer 2014, McCauley et al. 2000, Dufaÿ et al. 2014), while in animals, environmentally induced sex-ratio shifts, from climate-driven sex reversal to density-dependent bias, can reverberate through population persistence and predator–prey dynamics (Wedekind 2017, Schwanz & Georges 2021). Recognizing turnover as a dynamic trait therefore adds a genomic dimension to ecosystem resilience and conservation management: misidentifying sex systems can mislead demographic inference, breeding design, or population viability forecasts. Through these lenses, from hybrid dysfunction to ecological adaptation, sex chromosome turnover emerges not merely as a by-product of conflict but as a recurrent driver linking genomic architecture to biodiversity.

Ecological, community, and conservation consequences of inheritance restructuring

By altering how genes are inherited, turnover cycles influence not only the tempo of speciation but also the ecological and evolutionary flexibility of lineages. Genomic frameworks for biodiversity resilience emphasize the need to integrate evolving inheritance architectures into monitoring (see Buzan et al.

2025). When sex chromosome architectures reset, they reshape the recombination landscape, effective population size, and linkage of adaptive loci. These effects scale upward, from individual fitness to ecosystem dynamics, linking genomic conflict to biodiversity resilience.

Adaptive landscapes and phenotypic evolution

Sex chromosomes often house genes regulating sensory, endocrine, and behavioral pathways; thus, their turnover can rewire the genotype–phenotype map underlying mate choice, parental care, or communication. Shifts in sex linkage may change how sexually selected traits evolve or which sex experiences selection on signaling and perception. In darters, independent losses and gains of acoustic signaling correspond to distinct sex-system histories, suggesting that inheritance structure itself can tilt adaptive landscapes and channel phenotypic evolution. Comparable patterns in cichlids and ranid frogs imply that rapid turnover may repeatedly open or close pathways for sexual selection, influencing both the direction and pace of diversification.

Community and ecosystem effects

Genomic conflicts scale up to shape population and community processes. In plants, cycles of cytoplasmic male sterility and nuclear restorers (Rf) alter population sex ratios, pollen flow, and mating networks, reshaping pollinator interactions and even local species composition. In animals, temperature-sensitive sex determination interacting with turnover can drive sex-ratio skews, affecting population persistence, predator–prey dynamics, and trophic balance. Across taxa, such genomic restructuring acts as a hidden axis of ecological variation, influencing which species persist, adapt, or collapse under changing environments.

Conservation and global change

Recognizing sex chromosome turnover as a dynamic trait has direct implications for conservation and management. Climate-driven sex reversal in ectotherms can generate *de facto* turnover, altering sex determination mechanisms and destabilizing population sex ratios (Holleley et al. 2015, Bókony et al 2017). Such shifts reduce effective population size (N_e), increase inbreeding risk, and can even trigger demographic collapse under sustained environmental stress (Miura et al. 2024). Because sex-determining regions (SDRs) can change identity, location, and linkage relationships on ecological timescales, management plans should treat sex-system identity as fluid rather than fixed. Genomic monitoring of SDR location, age, and turnover frequency will be essential for predicting population responses to environmental change.

Artificial translocations and captive breeding programs must also consider mitonuclear compatibility. Mismatches between mitochondrial haplotypes and local nuclear backgrounds represent avoidable incompatibilities that can depress fitness (Burton & Barreto 2012, Iverson 2024), especially when sex-linked inheritance differs between source and recipient populations. “Mito-aware” pairing, i.e., matching mitochondrial haplotype with SDR background, offers a practical framework for minimizing cryptic fitness costs in managed populations. Finally, localized ancestry deficits near SDRs in natural hybrid zones can serve as early-warning indicators of evolving reproductive barriers or impending mitonuclear sweeps. Incorporating such genomic diagnostics into long-term monitoring will improve detection of hidden risks before they manifest as declines in viability or resilience.

Testing the escape-hatch hypothesis across scales

The mechanistic diversity of sex chromosome turnover reflects multiple evolutionary drivers that alternately amplify or relieve mitonuclear conflict. Turnovers can arise from sexually antagonistic selection, mutation load in nonrecombining regions, heterochiasmy, sex reversal, chromosomal fusion, or polyploidy, each restructuring how nuclear and cytoplasmic genomes are linked and inherited (**Table 2**). Identifying which mechanisms dominate in different clades provides testable predictions for the intensity, sex bias, and persistence of conflict cycles.

With the growing availability of chromosome-scale assemblies, population resequencing, and hybrid-zone datasets, it is now possible to move beyond qualitative models and empirically test these predictions through mito-aware comparative genomics, functional experiments, and macroevolutionary synthesis (**Table 3**).

Table 3. Diagnostic genomic and phenotypic signatures of entrapment versus relief phases.
Predicted contrasts between the amplification (“entrapment”) and resolution (“relief”) phases of mitonuclear conflict under the escape-hatch model. Entrapment is predicted to be associated with sex-biased sterility, mtDNA-coincident clines, and elevated dN/dS, but these signals are not unique; they gain specificity only when combined across multiple independent lines of evidence. Relief follows turnover or recombination restoration, producing relaxed dN/dS, re-established dosage compensation, and reduced sex-differentiated coverage around the sex-determining region (SDR). Example empirical tests link each predicted signature to corresponding genomic or physiological assays.

Signature	Entrapment phase	Relief phase	Example test
Sex-biased hybrid sterility	Strong	Weak or absent	Reciprocal crosses, fertility assays
Ancestry cline steepness near SDR	Sharp, mtDNA-coincident	Flattened	Mito-aware genomic clines
dN/dS of N-mt loci	Elevated	Relaxed	Phylogenetic dN/dS tests
GC bias / codon usage	Strong	Moderate	Comparative codon-usage bias
Dosage compensation	Partial	Re-established	RNA-seq expression ratios
SDR coverage difference	High	Reduced	Sex-differentiated coverage scans

Comparative genomics

High-quality chromosome assemblies allow every nuclear-encoded mitochondrial (N-mt) gene to be placed in its physical context relative to the sex-determining region (SDR). Comparative datasets across related taxa can date when each SDR arose, when recombination suppression expanded, and whether N-mt genes were repeatedly captured or released during turnover (Wright et al. 2017, Palmer et al. 2019, Vicoso 2019).

Different tools apply at different strata ages: in young regions, search for sex-biased heterozygosity, coverage shifts, and peaks in sex-differentiated Fst; in older strata, gene-tree monophyly and topology weighting reveal ancient nonrecombining blocks (Bachtrog 2013, Charlesworth 2017, Darolti et al. 2019). Hybrid zones add a powerful population-level test: *mito-aware genomic clines* treat mtDNA ancestry as a covariate to identify sex-linked N-mt loci showing steeper, mtDNA-coincident barriers than neutral loci (Gompert & Buerkle 2011, Li et al. 2022, Heidbreder et al. 2025). Combining ABBA-BABA, local-ancestry barrier scans, and expression assays can reveal selection acting on mitonuclear combinations (Radcliffe et al. 2025). Diagnostic genomic and phenotypic patterns distinguishing entrapment from relief phases are summarized in **Table 3.** These signatures are *not* unique to mitonuclear entrapment; comparable patterns can arise from autosomal Dobzhansky-Muller incompatibilities, ecological selection, demographic structure, or endosymbiont-mediated distortions.

Distinguishing a localized mitonuclear barrier caused by entrapment from genome-wide maternal sweeps or other confounders (e.g., W-drive, autosomal DMIs, demographic structure) requires integrating multiple diagnostics and seeking convergent evidence across genomic and physiological signatures. Recent simulations and theoretical work (Irwin 2025) indicate that W-drive (i.e., a genome-wide maternal sweep driven by a W-linked meiotic-drive allele) can rapidly displace mitochondrial haplotypes across hybrid zones even in the absence of strong mitonuclear incompatibilities. Spatially, entrapment produces localized barriers near the SDR, whereas W-drive produces genome-wide displacement of the mitochondrial cline (**Fig. 3**). Temporally, a W-driven sweep is expected to leave a step-like contraction in Bayesian skyline plots of mtDNA diversity that reflects a recent selective sweep of one haplotype. Additional evidence can come from sex-ratio distortions through time, distinctive W-linked centromere features, and screens for endosymbionts that sometimes mediate female-biased transmission distortions (Irwin 2025). Distinguishing localized entrapment from genome-wide maternal sweeps or demographic artifacts will ultimately require simulation-based calibration. Power and false-discovery rates for the composite **Table 3** signatures have not yet been benchmarked, and future work should quantify their sensitivity and specificity across realistic demographic and recombination parameters.

Functional and physiological validation

Functional assays provide the crucial causal bridge between genomic signatures and organismal fitness. Transcriptomic approaches can reveal whether N-mt loci within sex-linked strata show altered or sex-biased expression, incomplete dosage compensation, or disrupted coordination with mtDNA-encoded OXPHOS genes. Physiological assays, including measurements of mitochondrial respiration rate, ATP output, and oxidative stress, can then test whether these transcriptional changes translate into energetic deficits (Hill 2019, Mossman et al. 2019, Moran et al. 2024). Reciprocal hybrid crosses between lineages differing in SDR identity or turnover history can determine whether hybrid dysfunction aligns with predicted entrapment or relief phases. Emerging genome-editing tools (e.g., CRISPR-mediated relocation of N-mt genes or engineered recombination windows) now make it possible to manipulate linkage explicitly, providing the first direct tests of causality. Linking differential expression, mitochondrial physiology, and fitness across turnover states will decisively establish whether mitonuclear entrapment is a primary driver of sex-biased hybrid breakdown.

Macro-evolutionary synthesis

At the broadest scale, dated phylogenies enable tests linking turnover rate, hybrid-zone width, and diversification. If turnover periodically resets recombination, then diversification should correlate with turnover frequency after controlling for ecology, life history, and range size (Palmer et al. 2019, El Taher et al. 2021). Environmental factors such as temperature or latitude can further modulate turnover, predicting clade-specific differences in the tempo of conflict and speciation.

Integration across biological scales

The escape-hatch framework unites molecular, population, and macroevolutionary perspectives. At the *molecular level*, conflict arises from mispaired OXPHOS subunits. At the *population level*, it manifests as sex-biased hybrid dysfunction and segregation distortion. At the *macroevolutionary level*, repeated turnover reshapes diversification rates. Together, these processes illustrate how *inheritance restructuring* links cellular conflict to global biodiversity patterns.

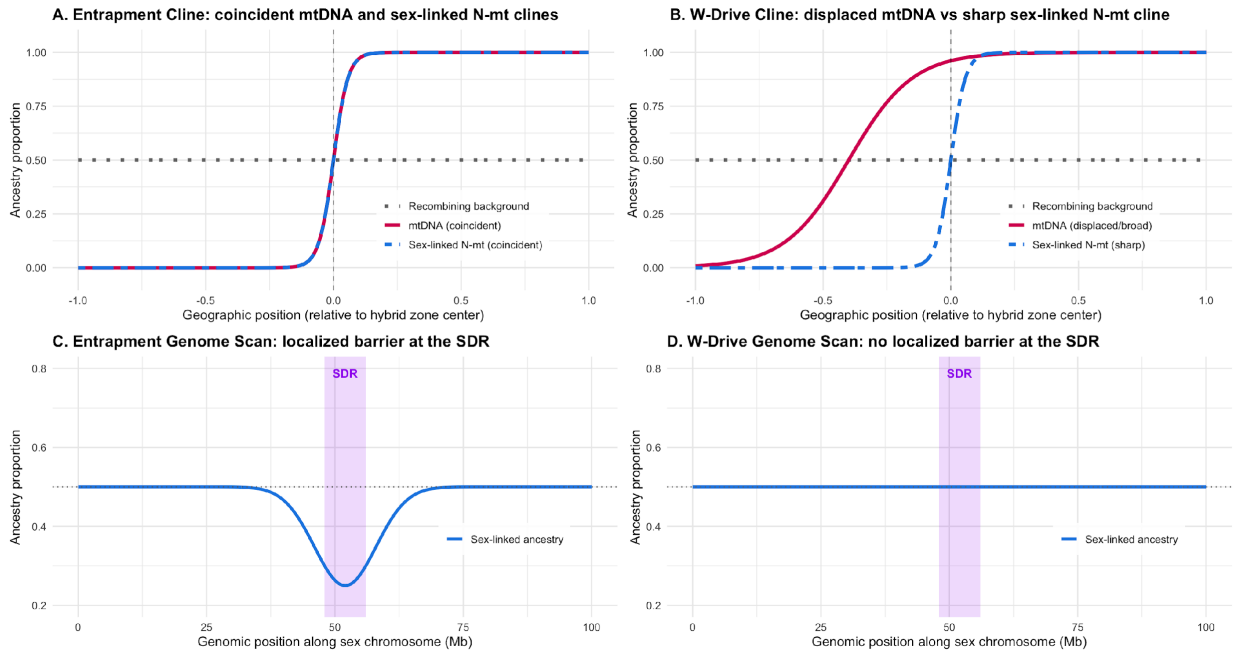

Figure 3. Hybrid-zone and genome-scan signatures distinguishing sex-linked mitonuclear entrapment from W-driven mitochondrial sweeps.

(A) Entrapment cline: In a hybrid zone, a nuclear-encoded mitochondrial (N-mt) locus captured in a newly non-recombining sex-linked stratum shows a steep ancestry cline that coincides with the mitochondrial cline at the hybrid-zone center, reflecting mito-aware selection against mismatched combinations. Recombining background loci remain near-neutral (have mean ancestry ~0.5). (B) W-drive cline: A W-driven sweep moves the mitochondrial ancestry far across the contact zone, capturing much of the range of the other species. As a result, the mtDNA cline no longer sits at the center of the zone (x = 0); it becomes shifted (displaced) toward one side and broadened, because the sweep acts genome-wide on mitochondria, not just locally at the sex-linked stratum. The positional and width mismatch between mtDNA and the sex-linked N-mt cline are indicative of a maternal sweep rather than local entrapment. (C) Entrapment genome scan: Along the sex chromosome, introgression is near-neutral (~0.5 ancestry proportion) in recombining regions but shows a localized dip at the sex-determining region (SDR), a persistent "genomic scar'' where the N-mt locus was entrapped. (D) W-drive genome scan: Introgression along the sex chromosome remains flat (mean ancestry ~0.5) across both recombining and SDR regions because a W-linked sweep is predicted to displace mitochondrial haplotypes genome-wide rather than creating a local incompatibility. Accordingly, no localized barrier appears at the SDR.

Broadening the escape-hatch framework beyond vertebrates

Plants

In dioecious angiosperms, mitochondrial mutations causing cytoplasmic male sterility (CMS) trigger nuclear restorers (Rf genes) that often become sex-linked. Repeated shifts between male and female heterogamety in *Silene*, *Fragaria*, and *Salix* demonstrate how cyto-nuclear conflict drives sex-system turnover (Renner 2014, Muyle et al. 2017, Sanderson et al. 2021). Comparative genomics across these lineages reveals parallel capture and release of restorer loci, the botanical counterpart of the escape-hatch dynamic.

Insects and other invertebrates

Turnover and mitonuclear dynamics are pervasive in arthropods. Across Lepidoptera (ZW), sex chromosome–autosome fusions are documented, including a neo-W formed by W–autosome fusion in *Heliconius* (Smith et al. 2016, Rueda-M 2024), and female-biased hybrid sterility consistent with Haldane's rule is well supported (Rosser et al. 2022; but see Smith et al. 2016). In parallel, maternally

inherited endosymbionts can drive mtDNA selective sweeps, generating mito–nuclear discordance in several butterfly lineages (Hurst & Jiggins 2005). *Hymenoptera* (haplodiploidy) present a distinct inheritance architecture, as males transmit only nuclear DNA (Blackmon et al. 2015), yet parasitic wasps reveal mito-conditioned hybrid fitness effects (Niehuis et al. 2008). *Drosophila* repeatedly shows large-X effects; mito-aware mapping in *D. yakuba–D. santomea* and *D. simulans–D. mauritiana* indicates X-linked N-mt compensation during early divergence (Rogell et al. 2014, Ågren et al. 2020).

Molluscs and doubly uniparental inheritance (DUI)

Several bivalves transmit distinct male and female mitochondrial lineages, termed doubly uniparental inheritance (DUI; Breton et al. 2007). Nuclear loci modulating mitochondrial segregation often show sex-biased or sex-linked expression (Kenchington et al. 2020), and hybrid zones in *Mytilus* display alternating mito–nuclear discordance with contact history (Stuckas et al. 2009). Though not classical XY/ZW turnover, DUI demonstrates how shifts in cytoplasmic–nuclear transmission modulate conflict intensity, functionally analogous to escape and entrapment.

Fungi and protists

In fungi (e.g., *Neurospora*), large non-recombining mating-type chromosomes show introgression dynamics that hint at genomic conflict and linkage reshaping (Sun et al. 2012, Meunier et al. 2022). In algae such as *Chlamydomonas*, nuclear–organelle gene families show rapid evolution and tight coordination, indicating that shifts in cytoplasmic–nuclear transmission may modulate genomic conflict even outside classic sex chromosome systems (Craig et al. 2021)

Outlook

Sex-chromosome turnover reframes long-standing patterns of postzygotic isolation as the moving outcome of inheritance architecture rather than fixed “rules.” The escape-hatch model makes this explicit: conflict intensifies when N-mt loci are entrapped in young, non-recombining strata and relaxes when turnover restores recombination or relocates the SDR. What varies among clades is not whether conflict exists, but where it sits in the genome, which sex bears the cost, and how long it persists. That variability is predictable from turnover history, SDR age, and the direction of cytoplasmic co-transmission (XY vs ZW).

In the coming years, improved chromosome-scale assemblies and long-read W/Y reconstructions will let us place every N-mt gene relative to the SDR, date strata, and test for repeated capture/release (**Table 2**). Mito-aware hybrid-zone toolkits, including genomic clines with mt ancestry as a covariate, local ancestry barrier scans, and topology-weighting, tie these histories to contemporary selection (**Table 3**; **Figs. 2,3**). Physiological assays (OXPHOS rate, ATP output) and single-cell expression will close the loop from genotype to fitness, while targeted crosses among lineages differing only in SDR identity can isolate turnover's causal role. Where feasible, manipulative tests (e.g., gene relocation or engineered recombination windows) can provide decisive falsification of entrapment predictions. Doing so will reveal whether the genomic architecture of speciation is predictable from the dynamics of inheritance asymmetry and conflict resolution. Key next steps are outlined in **Box 1**, which highlights falsifiable predictions spanning molecular to macroevolutionary scales, from decay of the large X/Z effect with SDR age to environment-coupled turnover and diversification.

A broader synthesis will come from bringing plants, invertebrates, and endosymbiont-mediated systems under the same umbrella. CMS-Rf cycles in angiosperms, DUI in bivalves, and Wolbachia-driven distortions in arthropods offer natural experiments in cyto-nuclear co-transmission. Comparing these to vertebrate XY/ZW clades will reveal whether conflict pulses follow shared rules whenever inheritance paths are rewired, irrespective of whether the mechanism is an SDR shift, recombination restoration, or cytoplasmic drive.

Finally, the escape-hatch framework has tangible value for biodiversity conservation. Climate-linked sex reversal and shifting SDRs can change demographic trajectories on management timescales. "Mito-aware" conservation - jointly monitoring mt haplotypes, SDR location/age, and sex ratios - can anticipate cryptic load, guide translocations, and reduce avoidable fitness costs in captive breeding. Because entrapment leaves localized genomic scars while W-linked sweeps displace mitochondria genome-wide, these diagnostics offer early-warning indicators before viability declines are evident (**Table 3**; **Fig. 3**). Together, these paths make the escape-hatch framework not only conceptually unifying but empirically tractable (see **Box 1**). The current diagnostics provide qualitative expectations and a framework for hypothesis testing; quantitative calibration via forward simulations and controlled hybrid-zone benchmarks will be essential to estimate error rates, establish thresholds, and generalize across taxa.

Box 1. Outstanding questions and falsifiable predictions.

Molecular and population scales

1. *Clock of conflict.* Does the large X/Z effect decay with SDR age within clades, as predicted by turnover relief?
2. *Direction flips.* In lineages with XY↔ZW switches, does the sex bias of hybrid breakdown invert across sister taxa?
3. *Pathway reuse vs gene reuse.* Are convergent conflicts driven by shared OXPHOS pathways rather than identical genes?

Ecological and environmental scales

4. *Environment coupling.* Do thermal regimes that favor sex reversal increase turnover frequency?
5. *Predictive conservation.* Can early detection of SDR shifts improve population viability forecasts under climate change?

Macro-evolutionary scales

6. *Turnover–diversification correlation.* Does net diversification scale with turnover rate across taxa after controlling for ecology?
7. *Epigenetic resets.* Do methylation or small-RNA mechanisms mediate recombination restoration after turnover?

Conclusions

Viewed through inheritance architecture, the genome is not a static blueprint but a self-modifying system. Each sex chromosome turnover rewrites the rules that tie nuclear alleles to a single cytoplasmic lineage, sometimes amplifying conflict (entrapment), sometimes dissolving it (relief). This cycle predicts when and where hybrid dysfunction will concentrate, how its sex bias will flip across XY↔ZW transitions, and why the large X/Z effect is strong in entrenched systems yet weak or transient in labile clades. It also explains how genomic "resets" can release standing variation and open new adaptive paths, helping to connect microevolutionary coadaptation to macroevolutionary diversification. The agenda is clear: place N-mt genes on chromosome-scale maps relative to dated SDRs; test for localized, mt-coincident barriers in hybrid zones; measure energetic performance in reciprocal hybrids; and distinguish entrapment from maternal sweeps using spatial and temporal diagnostics. Extending the same logic to plant CMS–Rf cycles, DUI in molluscs, and symbiont-mediated sex-ratio distortions will reveal whether common rules govern conflict resolution whenever co-transmission changes. By treating sex chromosome turnover as

both symptom and engine of mitonuclear evolution, we gain a predictive handle on how genomic architecture shapes the origin of species and the resilience of biodiversity.

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