Soma as transmission control in multicellular evolution: a population-genetic framework for germline restriction and cellular altruism

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Abstract

A central question in the evolution of multicellularity is why lineages repeatedly transition from germ-dominated unicellular states to organizations with extensive somatic investment. If somatic cells are largely excluded from future generations, why are they produced at all, and why do soma-free multicellular alternatives appear limited in stability, persistence, or attainable complexity? Here, we propose a population-genetic framework that distinguishes germline and soma by their differential capacity to transmit genetic variation across generations. Cellular altruism emerges when replication within an individual is decoupled from heritable contribution, allowing cells to adopt transient or permanent somatic roles. This review presents an updated evolutionary synthesis incorporating recent discoveries that expand our understanding of germ-soma separation archetypes. We argue that somatic organization may repeatedly evolve because it mediates cellular conflict by allocating labour, buffering mutational damage, and restricting the lineages through which mutations reach future generations. Facultative altruism can serve as an evolutionary bridge to obligate soma, while creating asymmetries in mutation propagation that favour progressively tighter restrictions on heritable transmission. Under this view, soma is not merely sterile tissue but a recurrent evolutionary filter that governs the amount and composition of genetic variation reaching subsequent generations.

Two kinds of competing cells

Multicellular organisms are cooperative assemblies of cells, but not all cells contribute equally to reproduction. Some lineages retain the capacity to generate new individuals and thereby transmit genetic information across generations, while others are restricted to maintaining the current organism (Weismann, 1893). The latter constitute the *soma*: cells that can proliferate within an individual but whose lineages terminate with it. In contrast, the *germline* comprises those cells, or more generally those heritable genetic lineages, whose genetic material can contribute to future generations, providing continuity across evolutionary time (Weismann, 1893; Jaenisch, 1976).

The distinction reflects a fundamental division of labour. Somatic cells sustain organismal function, whereas germline cells ensure the propagation of genetic information (Michod, 2007). Most cellular specialization, such as neuronal, muscular, or epithelial cells, falls within the somatic lineage. Only at a higher level does the key distinction emerge: between maintaining the individual and reproducing it. From an evolutionary perspective, somatic cells forgo direct transmission and instead act altruistically by supporting the reproductive success of the germline.

This division raises an apparent paradox: if only germ cells contribute to future generations, why produce large numbers of non-reproductive cells? Early life provides a baseline (Moody et al., 2024). In ancestral single-celled organisms, each cell was both an individual and a reproductive unit, so reproduction of any individual directly contributed to the next generation. In this context, all cells effectively belonged to the germline, because cellular replication and heredity were not yet partitioned into separate lineages. The emergence of multicellularity altered this condition by introducing organisms composed of more cells than required for reproduction, thereby creating a new level of conflict among cells sharing the same body (Folse III and Roughgarden, 2010; Herron et al., 2022).

Within multicellular organisms, cells compete for resources and replication opportunities, yet are constrained within a shared structure. Unchecked competition would destabilize the organism, as proliferative lineages could outcompete others at the expense of collective function(Bentley et al., 2022). A stable solution is the evolution of cooperation through a division between reproductive and non-reproductive roles (Tverskoi and Gavrilets, 2022). This division, however, need not appear in its modern obligate form from the outset. It can range from facultative cellular altruism, in which some cells temporarily lose reproductive potential under particular ecological or developmental conditions, to obligate somatic differentiation, in which entire lineages are permanently excluded from future generations. Thus, somatic cells suppress their own reproductive potential to promote individual fitness, that is, the success of the heritable lineage.

Despite the apparent inefficiency of producing large numbers of non-reproductive cells, this seemingly "wasteful" strategy has proven remarkably persistent across diverse lineages.

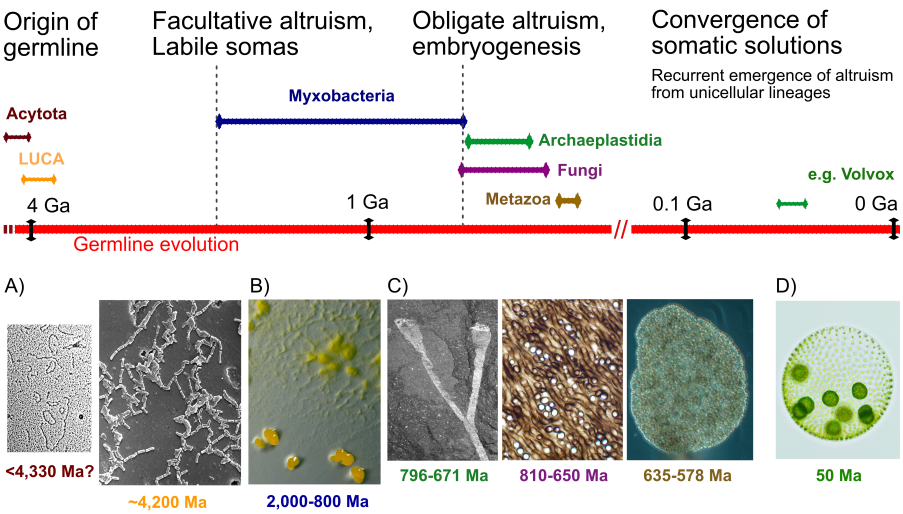

Figure 1. Conservative ranges for the emergence of germline continuity and major somatic regimes (Bonner, 2003; Kirk, 2004; Lucking et al., 2009; Forterre and Prangishvili, 2009; Butterfield, 2009; Arias Del Angel et al., 2017; Valentine and Marshall, 2015; Nagy et al., 2018; Bonneville et al., 2020; Bowles et al., 2024; Nguyen and Farge, 2024). The timeline summarizes conservative age ranges for the origin of hereditary continuity and for major transitions in somatic organization. Horizontal bars indicate approximate temporal ranges for selected clades or examples; dashed vertical lines mark broad conceptual transitions rather than sharp evolutionary boundaries. A) The origin of germline-like continuity corresponds to the emergence of lineages capable of transmitting genetic information across generations. This predates LUCA and remains uncertain. A conservative range for acytotal hereditary systems is shown before 4.3 Ga, with LUCA also placed in the early history of life; images illustrate free DNA and a bacterial representative, respectively. B) Facultative altruism and labile soma are represented by transient multicellular systems in which cells retain reproductive potential under some conditions but differentiate into reproductive and non-reproductive roles under others. Myxobacteria provide a canonical example: under starvation, cells aggregate into fruiting bodies in which some cells form spores while others perform somatic functions or die. C) Obligate altruism and embryogenesis are associated with independently evolved complex multicellular lineages in which somatic differentiation becomes developmentally integrated. Conservative fossil and molecular ranges are shown for Archaeplastida, Fungi, and Metazoa, illustrated by early plant, fungal, and animal examples. D) Somatic solutions have also evolved convergently and more recently from unicellular ancestors. Volvocine green algae, illustrated here by *Volvox aureus*, provide a well-studied case in which soma-germline differentiation evolved from a *Chlamydomonas*-like ancestor.

Germ-soma differentiation, broadly construed, has evolved repeatedly across the tree of life, but not always in the same form (Grosberg and Strathmann, 2007; Rokas, 2008; Butterfield, 2009). Conservative evolutionary ranges suggest a sequence from the earliest germline at the origin of life, through facultative somatic rise in transient multicellular bodies, to obligate cellular altruism in several complex multicellular lineages, and finally to more recent expansions or refinements of somatic organization (Figure 1). However, somas also emerge *ex novo* from single-celled ancestors. The recurrent acquisition of soma suggests that restricting heritable contribution represents a robust evolutionary solution to the challenges posed by multicellularity. Explaining why such organisation evolves and persists requires a framework linking cellular roles to evolutionary outcomes, motivating a population genetic formalism to cellular altruism.

In the following sections, we build on this tension by reconsidering what counts as germline. We introduce a definition based not on cell type, anatomical location, developmental origin, or reproductive function, but solely on the capacity to transmit genetic information to subsequent generations. This population-genetic interpretation clarifies the distinction between germline and soma, including recently described and/or atypical forms of germ–soma separation. We then use this framework to show that the distinction between soma and germline is not fixed, but can vary across life cycles and lineages, giving rise to different germ-to-soma archetypes. Finally, we discuss several evolutionary explanations for the emergence of somatic organization: the mediation of cellular conflict through altruistic role differentiation, the sequestration of mutational "dirty work" in disposable cell lineages, mutation-selection filtering before organism formation, resource-allocation tradeoffs between somatic maintenance and reproduction, and the broader decoupling of mutation exposure from heritable contribution. Together, these considerations suggest that soma is not merely non-reproductive tissue, but rather an evolutionary solution to the challenges of transmission fidelity, genetic conflict, mutational exposure, and organismal persistence.

A unifying definition of the germline

The distinction between somatic and germ cells is not always clear-cut across the diversity of life. In many organisms, cells cannot be unambiguously classified by morphology, developmental origin, or fate restriction alone (Ishikawa and Schumacher, 2025). A more general criterion is therefore required.

From a population-genetic perspective, the germline can be defined as the set of cellular lineages, or heritable genetic materials, with the capacity to *transmit genetic information* to subsequent generations of complete individuals. Somatic lineages, by contrast, are those whose genetic changes remain confined to the lifespan of the individual. Under this definition, germline identity is not determined by what a cell is, where it is located, or what function it performs, but by whether its genetic information can contribute to the germline of the next generation (Ratcliff and Burnetti, 2026).

This interpretation differs from stricter developmental definitions. Cells with broad developmental potential, such as embryonic stem cells or early blastomeres, can generate both somatic and germline tissues and must therefore be included within the germline under an *inclusive* transmission-based definition (Driesch, 1892). This contrasts with exclusive, or Wilsonian, definitions, which restrict the germline to lineages committed solely to gamete production and exclude multipotent cells (Wilson, 1928); see also recent treatment in (Schoen and Schultz, 2019).

The distinction is therefore probabilistic rather than absolute. Not all mutations arising in germ cells are ultimately represented in populations: many are lost through selection, developmental deficiencies, drift, or failure of reproduction (Lynch et al., 2016; Haig, 2025b).

Table 1. Representative features of germline propagation across biological systems. Row values indicate prevalent or illustrative states rather than exhaustive categories.

Feature	Acytota	Prokaryota	Simpler eukaryotes	Complex euk.
Germ	Partitioned	Unitary/partitioned	Mostly unitary	Unitary, but prone to hijacking
Soma (if existent)	–	Facultative	Mostly obligate	Obligate
Ploidy	Haploid	Haploid	Variable	Diploid/polyploid
Reproduction	Clonal	Clonal	Variable	Apomictic/sexual
Gamete type	–	Hologamous	Mostly isogamous	Anisogamous
Embryogenesis	–	–	–	+

Conversely, mutations arising outside anatomically defined germ cells may influence evolution indirectly by altering individual fitness (Lynch, 2010), and in some organisms may enter into the reproductive lineages (Schoen and Schultz, 2019; Ishikawa and Schumacher, 2025). Thus, the relevant distinction is not simply where a mutation arises, but whether it contributes to realised heritable variation.

Germline architecture also varies across organisms (Table 1). Differences in ploidy, reproductive mode, life cycle structure, and gamete biology generate asymmetries in how mutations are produced and transmitted. In anisogamous species, for example, paternal and maternal germlines often differ in mutation rate because they differ in cell division dynamics and gene expression (Campbell and Eichler, 2013; Kong et al., 2012). In prokaryotes, clonal reproduction can be supplemented by horizontal gene transfer (HGT), allowing genetic material to move between lineages that do not share conventional gamete identity (Jain et al., 1999; Koonin, 2016; Hall et al., 2020). To distinguish this form of genetic exchange from isogamy or anisogamy, we refer to it here as *hologamy*.

Despite this diversity, a unifying principle remains: the capacity to transmit genetic information to the germline of subsequent generations of complete individuals. Cells exhibiting this property have recently been termed the "germ stem" (Haig, 2025a). We argue here that this transmission criterion provides a quantitative basis for distinguishing germline from soma across highly heterogeneous biological systems.

Extending the germline beyond cellular lineages

Taken strictly, a transmission-based definition has an important consequence: the germline need not be exclusively cellular. Genetic elements that are not themselves cells, including viruses, transposable elements, and other mobile DNA, can satisfy the definition whenever they enter heritable lineages and are transmitted across generations (Lynch et al., 2016; Ratcliff and Burnetti, 2026).

At first glance, this appears problematic. Cells are autonomous units capable of sensing,

metabolism, and regulated replication, whereas viruses and mobile genetic elements depend on cellular machinery for propagation. Classical views of germ-soma separation also assume that inheritance is mediated through a dedicated cellular lineage protected by the Weismann barrier (Weismann, 1893). Under this view, non-cellular agents are external to the germline rather than part of it.

However, this separation is permeable in practice. Viral sequences, transposable elements, and other selfish genetic elements can integrate into host genomes and become *endogenised* (Aziz et al., 2010; Zeng et al., 2013; Blinov et al., 2017; Ritsch et al., 2024). Once incorporated into reproductive material without preventing transmission, these sequences may be replicated alongside host DNA and inherited across generations. In this sense, germlines are not isolated repositories of lineage information, but *composite* hereditary systems containing genetic elements with different origins and evolutionary interests (Lynch and Walsh, 2007; Monaghan and Metcalfe, 2019).

This observation and recent theoretical developments call for an extended conception of the germline (Monaghan and Metcalfe, 2019; Majic et al., 2025; Haig, 2025b,a; Padilla-Iglesias and Majic, 2026; Ratcliff and Burnetti, 2026). If the goal is to trace the origin and propagation of heritable variation, then all genetic elements that successfully enter and persist in reproductive lineages must be considered. This includes viruses, transposable elements, horizontally transferred genes (Lynch and Conery, 2003; Lynch and Walsh, 2007), and environmental DNA incorporated through transformation or conjugation (Harrison et al., 2019). By contrast, processes such as transmissible cancer (Epstein et al., 2016) or soma-to-germ communication via RNA (Conine and Rando, 2022) may affect fitness or inheritance-like phenotypes, but they do not constitute germline transmission unless genetic material itself reaches the germline of the next generation.

Under this extended perspective, it is useful to distinguish germ cells from germline information. Germ cells are cellular vehicles that generate new complete individuals and establish the next generation's germline. Germline information comprises the set of genetic elements transmitted germ-to-germ, regardless of whether those elements originated in the host genome, an endosymbiont, a virus, or another lineage. In this sense, the germline is not always a single unified genome but a hereditary assemblage. A striking example is the proposed spatio-temporal segregation of somatic DNA from germline material within a single nucleus by extensive endoreplication in *Allogromia laticollaris* (Timmons et al., 2024).

Unitary and partitioned germline propagation

The extended germline need not propagate as a single, indivisible hereditary unit. Multiple genetic lineages may coexist within the same cellular context, and different components of the germline can have different transmission routes. Endosymbiotic elements such as mitochondria or plastids possess their own genomes and are transmitted alongside nuclear DNA, often with coordinated but distinct evolutionary dynamics (Hurst and Hamilton, 1992).

Whether such elements constitute independent germlines remains debated, because their replication is tightly coupled to host cell division (Bennett et al., 2024), they may exhibit heteroplasmy (different copies of organellar DNA) (Khachaturyan et al., 2024), and mitochondrial small RNAs may contribute to gonad formation (Pozzi et al., 2017). Nonetheless, their presence reinforces the view that heritable material is not always confined to a single unified lineage.

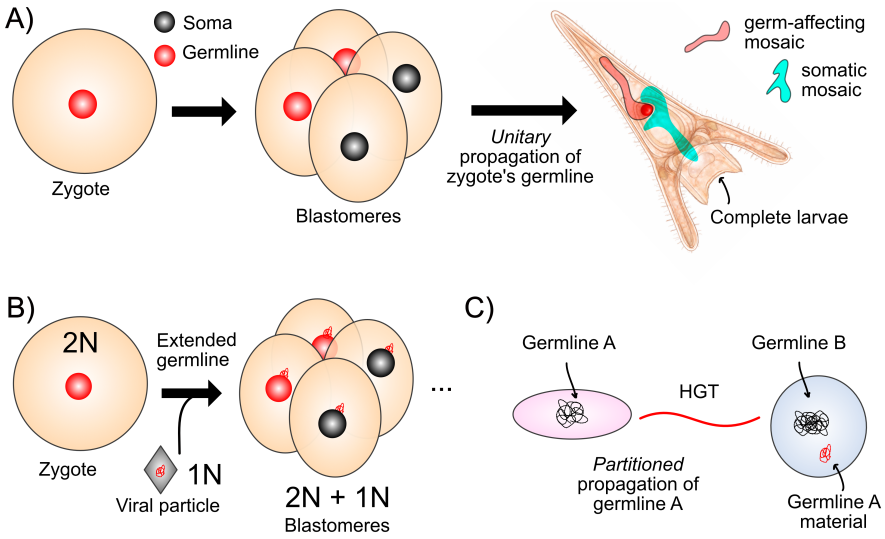

Figure 2. Unitary and partitioned propagation in an extended germline framework. A) Unitary propagation occurs when the organism-generating genetic material of a zygote is transmitted through development as a single reproductive lineage. Red nuclei indicate germline lineages and black nuclei indicate somatic lineages. In canonical development, mutations arising in somatic lineages are excluded from inheritance, but somatically acquired genetic variants can sometimes cross the Weismann barrier and enter reproductive lineages. Such variants are germline *de facto* if they contribute to the genetic material transmitted to offspring. The larval schematic illustrates this distinction: red shading marks germ-affecting mosaicism, whereas turquoise shading marks somatic mosaicism that does not contribute directly to inheritance. B) Partitioned propagation expands this framework by allowing genetic material from different sources to contribute to the same future germline. A viral particle carrying additional genetic material enters a zygote and integrates into the host genome without preventing development. The resulting blastomeres contain both host-derived genetic material ($2N$) and integrated exogenous material ($1N$), so the future germline contains a composite set of heritable elements rather than only the original zygotic genome. C) Partitioned germline propagation can also occur through horizontal gene transfer (HGT). In clonal prokaryotic reproduction, most genetic material is transmitted unitarily within a lineage, but HGT can move a subset of material from germline A into germline B. If the recipient lineage propagates, the transferred material becomes part of another germline despite not having been transmitted as a complete organism-generating genome.

Figure 2 illustrates two modes of germline propagation under this extended definition. In unitary propagation, the transmitted material is carried by a cellular vehicle capable of producing a complete individual and establishing the next generation's germline (Fig. 2A). Germ's unitary propagation can occur even from *a priori* somatic lineages if they successfully integrate into the germline. In partitioned propagation, only a subset of genetic material enters another germline, as in viral endogenisation or HGT (Fig. 2B–C). The distinction

is therefore not between endogenous and exogenous DNA, but between genetic material transmitted as part of an organism-generating germline and genetic material transmitted as a partial contribution to another germline.

Using this extended definition, germline propagation can therefore occur in two broad modes. In *unitary* propagation, germline material is transmitted through a vehicle capable of generating a complete individual and establishing the next generation's germline. This is the prevalent mode of embryogenic germlines. In *partitioned* propagation, genetic material is transmitted into another germline without carrying sufficient information to reconstruct the complete genetic content of the donor lineage. HGT is the clearest example: a fragment of one germline can enter another and persist if the recipient lineage survives and reproduces (Fig. 2C).

Partitioned propagation can generate genomic conflict, but conflict is not restricted to partitioned systems. Even under unitary propagation, different components of the germline may have different evolutionary interests. Germline-restricted chromosomes provide a clear example: they are transmitted through germ cells but eliminated from somatic lineages in several animals (Pigozzi and Solari, 1998; Hodson et al., 2022; Sotelo-Muñoz et al., 2022; Dedukh et al., 2025). These chromosomes appear functionally linked to germ-cell fitness (Mueller et al., 2023). Similar forms of programmed somatic DNA elimination occur in lampreys and nematodes (Boveri, 1887; Wang and Davis, 2014; Smith, 2018). Such cases show that even a unitary organismal germline can contain internally partitioned hereditary interests.

Somatic archetypes across the tree of life

The extended germline concept reframes soma and germline as transmission states rather than fixed anatomical categories. This reframing allows somatic organization to be compared across systems that differ radically in development, reproduction, and life cycle structure. The relevant question is no longer whether a lineage resembles a canonical germ cell, but how much access it has to future generations. We can therefore classify somatic organization by the degree to which heritable contribution is restricted: from labile systems in which cells switch between reproductive and somatic roles, to obligate systems in which somatic lineages are recurrently or permanently excluded from transmission, to embryogenic systems in which this exclusion is embedded within development. We refer to these recurrent arrangements as somatic *archetypes*.

Facultative altruism

We can now recognise cases in which the same cell type can behave as germline under some conditions and soma under others. We refer to this regime as *facultative* altruism: a labile form of somatic organization in which reproductive and non-reproductive roles are conditionally assigned by environmental or developmental context.

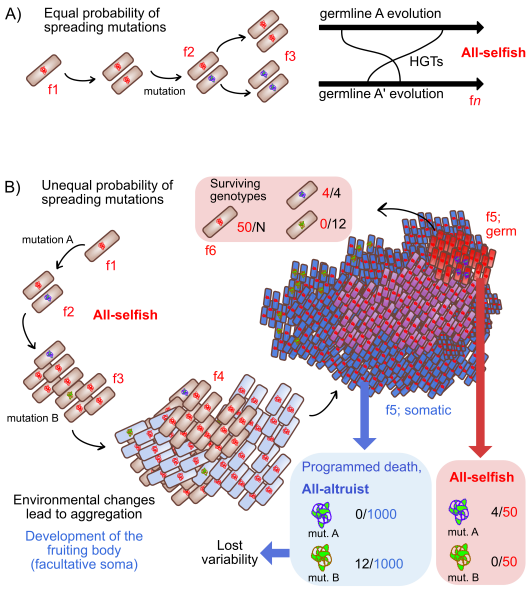

Figure 3. Labile soma and unequal mutation transmission. A) When all cells retain reproductive potential, mutations arising in any lineage can, in principle, be transmitted to future generations. In this all-reproductive state, mutant and wild-type copies have the same expected heritable contribution in the absence of fitness effects, so allele-frequency change reflects drift. Horizontal gene transfer (HGT) can redistribute genetic variants among reproductive lineages without producing systematic differences in transmission probability. B) Environmental change induces aggregation and fruiting-body development, producing a facultative soma. Only a subset of cells contributes to the next generation, while others enter transient somatic roles and undergo programmed cell death. Mutation A, present in cells that survive as reproductive propagules, remains heritable. Mutation B, confined to labile somatic cells, is lost even if it affects cellular performance. Facultative soma therefore converts an initially broad distribution of potential heritable contribution into a skewed distribution in which hereditary consequence is concentrated in the surviving reproductive subset. Across panels, coloured DNA represents genetic state: red DNA denotes the ancestral genotype, whereas blue and green DNA denote independently arising mutations. Cell shading represents developmental fate: blue cells are transient somatic cells destined for programmed cell death, whereas red cells constitute the surviving reproductive subset. Labels f_1–f_n indicate successive stages in the life cycle. Fractions indicate realised representation among reproductive lineages or next-generation descendants: $4/4$ denotes representation in all depicted reproductive lineages, whereas $50/N$ denotes 50 transmitted copies in a next-generation population of size N.

In facultative altruism, soma formation is transient and environmentally induced. Mutation transmission probabilities therefore shift from a broadly distributed state, in which most cells retain reproductive potential (Fig. 3A), to a skewed state in which only a subset of lineages contributes to the next generation (Fig. 3B). A canonical example is provided by *Myxobacteria*. Under favourable conditions, cells retain reproductive potential and behave as germline. Under starvation, however, they aggregate into multicellular fruiting bodies in which an apical subset differentiates into spores, while many remaining cells adopt somatic roles or undergo programmed death (Muñoz-Dorado et al., 2016). This absence of permanent developmental commitment defines a *labile* soma. During fruiting body formation, mutation transmission becomes restricted to a subset of lineages: a minority differentiates into myxospores, on the order of 10^5 per fruiting body (Zusman et al., 2007), or persists as

peripheral rods, while the majority undergoes programmed death (Søgaard-Andersen and Yang, 2008), likely supplying nutrients to developing spores (Muñoz-Dorado et al., 2016).

Further examples of facultative altruism in single-celled species (Ratcliff et al., 2012) underline its general features: (1) absence of embryogenesis or stable developmental programs; (2) environmentally induced aggregation; and (3) unequal transmission probabilities among participating cells. As a result, the equal transmission of cellular lineages is broken even in initially clonal populations (Yang et al., 2025).

This provides an empirical model of labile soma formation, but aggregation alone is insufficient. A multicellular association qualifies as labile soma only when some cells experience reduced or null probability of contributing genetic material to subsequent generations. Microbial mats, for example, do not constitute labile somas if all cells retain comparable reproductive potential (Grosberg and Strathmann, 2007). Conversely, systems such as *Bacillus subtilis*, in which aggregation generates cell types with distinct survival, reproductive, or transmission probabilities, approach the labile soma regime (Aguilar et al., 2007; Lopez et al., 2008; Shank and Kolter, 2011). HGT can blur the boundary between soma and germline, but under the extended definition this ambiguity is resolved operationally: genetic material belongs to the germline only when it reaches a lineage that contributes to subsequent generations.

Obligate altruism

In contrast to labile systems, obligate altruism arises when soma formation is developmentally recurrent and irreversible within the life cycle. Here, the germ-soma distinction is stabilized: individuals reliably produce somatic lineages whose genetic variation has little or no probability of contributing to subsequent generations. This organization represents an altruistic consortium, in which reproductive success depends on coordinated interactions between germline and soma. The key population-genetic feature is a stable asymmetry in transmission probabilities across cell lineages.

Simple obligate altruism. Simple obligate altruism is characterised by stable germ-soma differentiation in the absence of embryogenesis. Multicellularity may arise through growth or aggregation rather than through a deterministic embryogenic program. Nevertheless, heritable contribution is restricted: only a subset of lineages contributes genetic material to the next generation. Under the extended germline definition, sequestration is achieved whenever transmission becomes restricted to particular cell lineages, regardless of whether those lineages were developmentally specified from the beginning.

Fungal systems illustrate both this structure and its ambiguities. In *Basidiomycota*, mutation rates are lower in spore-forming lineages than in the surrounding soma (Thorén et al., 2025), and this control arises prior to fruiting body formation (Thorén et al., 2025). However, several processes complicate lineage assignment: clonal growth generates mosaicism (Fis-

cher and Glass, 2019); coenocytic organisation –a cell contains multiple nuclei without the division of the cytoplasm– allows nuclear mixing (Daskalov et al., 2017); somatic cell fusion (anastomosis) may produce heterokaryotic hyphae (Giovannetti et al., 1999; Daskalov et al., 2017; Fischer and Glass, 2019; Clark-Cotton et al., 2022); and fragmentation can reassign somatic lineages to reproductive roles. All these ambiguities are resolved operationally: any lineage that contributes genetic material to sporulating structures is considered germline (Figure 4).

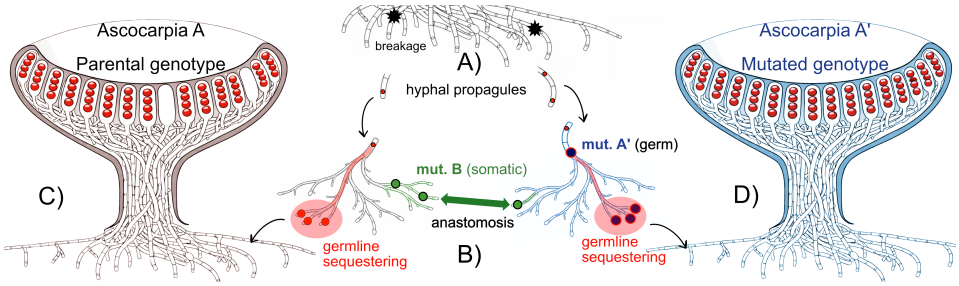

Figure 4. Tracing the extended germline in a simple obligate soma. A) Hyphal propagules can originate from vegetative mycelium after fragmentation or breakage and can and generate independent fungal individuals (Nicholson, 1996; Masuya et al., 2009). Under a transmission-based definition, such propagules are germline because they establish new reproductive lineages. B) During mycelial growth, mutations may arise in different hyphal regions. Mutation A′ reaches the lineage that later contributes to *ascogenous hyphae* and sporulating structures, whereas mutation B spreads through vegetative hyphae but remains excluded from reproduction. C) A propagule lacking mutation A′ develops into an ascocarp with the parental genotype and produces parental spores. D) A propagule carrying mutation A′ transmits this mutation through ascocarp development and into the resulting ascospores. Thus, in simple obligate systems, germline identity is assigned by realised transmission rather than by whether a lineage originated in vegetative or reproductive tissue. Across panels, red circles indicate nuclei or spores carrying the parental genotype, blue marks mutation A′, and green marks mutation B. Blue hyphae and blue ascocarp shading indicate lineages carrying mutation A′, whereas green hyphal segments indicate lineages carrying mutation B. Red shaded regions mark germline-sequestering regions that contribute to reproductive structures.

Slime molds, and particularly the amoeba *Dictyostelium discoideum*, illustrate the boundary between facultative and simple obligate altruism (Bonner, 2003). Although these are free-living cells at the beginning of their life cycle, they retain the ability to seek out a suitable location to feed on bacteria and, once nutrients are depleted from the area, they cluster together to form spore-producing fruiting bodies. While some apical amoebae undergo sporulation, the fragile cellulose stalks remain as vacuolated dead cell parenchyma. This altruistic migration of cells is tightly regulated by molecular mechanisms (Kim et al., 2022), moving "in a remarkably directed fashion" toward an optimal point for spore dispersal into the air (Bonner, 2003). Aggregative fruiting has evolved convergently in other eukaryotic clades, mainly Rhizaria, Excavata, Fonticula, and Sorodiplophrys, although some of them retain non-deterministic facultative somas (Broersma and Ostrowski, 2022). These systems are inherently prone to chimerism and internal conflict (Strassmann and Queller, 2011).

Volvocine algae provide a widely-known complementary example. Across the clade,

somatic investment varies widely, whereas only a small number of gonidia transmit mutations (Shelton et al., 2012). Somatic roles are obligate, although partial reversibility persists in simpler taxa (Kochert, 1968). Genetic regulation of this division, e.g., via *regA* (Huskey and Griffin, 1979), and structural constraints, e.g., daughter-cell retention (Koufopanou, 1994), stabilise this asymmetry. Functionally, soma enables division of labour, such as maintaining buoyancy while reproductive cells divide (Koufopanou, 1994).

Complex obligate altruism (embryogenic somas). Complex obligate altruism is defined by deterministic embryogenesis. Embryogenesis generates soma through a developmental program that controls lineage fate and canalizes transmission through specific reproductive lineages (Cridge et al., 2016). This transition has several population-genetic consequences. First, embryogenesis structures mutation accumulation, because the number and timing of cell divisions influence both germline mutation load and somatic mosaicism (Kunkel and Erie, 2015; Manhart and Alani, 2017). Second, early germline sequestration reduces the probability that somatic mutations reach subsequent generations. Third, increased somatic complexity alters lifespan, ecological interaction, and the opportunity for somatic mutation accumulation (Cagan et al., 2022). Reproductive mode then further modulates these dynamics by determining how germline material is packaged, recombined, or clonally propagated.

Asexual reproduction encompasses several processes with different consequences for germline transmission. Some forms, such as apomixis, can preserve clonality by avoiding meiosis and fertilisation, whereas others, such as automixis, involve meiotic processes and may reduce heterozygosity (Klemp et al., 2026). Parthenogenesis is therefore not necessarily equivalent to strict clonality, and selfing is not asexual reproduction because it involves gamete fusion, even when both gametes derive from the same individual (Klemp et al., 2026). These distinctions matter because clonal propagation of somatic material can blur the soma–germline boundary. When propagation occurs without embryogenesis, as in fissiparity (Goldschmidt, 2014), mutation transmission may resemble simple obligate altruism (Lanfear, 2018). Under a transmission-based definition, any somatic lineage that acquires the capacity to generate a new individual becomes an *ex novo* germline.

On the other hand, gametes from sexual individuals suffer much higher degrees of specialisation, and are virtually excluded from the environment, often dimorphic (anisogamous), where two haploid nuclei fuse to form a zygote, following meiotic recombination. Gamete specialisation likewise entails advanced mechanisms of germline recognition (e.g. sperm-oocyte recognition, chromosome compatibility, adjacent tissue nourishment) that are absent in simple obligate altruist and some apomictic organisms.

The evolution of anisogamy in sexual individuals has been interpreted as a response to parasite pressure, where asymmetry in gamete size contributes to limiting parasite transmission and diversifying offspring genotypes (Hurst, 1990). Whether these hijacking-avoidance

mechanisms are causes or consequences of soma acquisition is one question that would require a complicated experimental falsification.

Ontogenetic determination of germline and the time of germline definition greatly determine the amount of inherited mutations in any somatic organism (Buss, 1988). However, in sexual organisms, gametes often suffer different mutation loads because of differential division and a distinctive extension of DNA repair (Qin et al., 2025).

Why does somatic organization emerge convergently?

We have argued that the soma-germline distinction is best defined not by cell type, anatomical position, or developmental origin, but by hereditary consequence: germline lineages are those whose genetic variation can contribute to future generations, whereas somatic lineages are those whose variation is largely confined to the current individual. This framing sharpens the *central* evolutionary question. Why does multicellular evolution so often restrict heritable contribution to a subset of cells, rather than preserving reproductive equivalence among all cellular lineages?

Across diverse multicellular lineages, reproductive cells tend to be numerically limited, spatially restricted, or developmentally protected, whereas non-reproductive compartments expand and diversify. By contrast, systems in which heritable contribution remains broadly distributed across many cells appear to face recurrent limits on stability, persistence, or attainable complexity. This asymmetry suggests that somatic organization is not merely a historical accident, but may reflect a common solution to the problems created by multicellularity: conflict among cells, exposure to mutation, and the need to regulate which lineages transmit variation across generations.

Hypotheses of soma acquisition in the light of population genetics

At the most general level, soma can be understood as a mechanism for limiting competition among cellular lineages. In multicellular groups, cells compete for resources, survival, and replication opportunities. If all cells retain equivalent reproductive potential, mutations that increase cellular propagation may spread even when they reduce the fitness of the collective. Somatic differentiation provides one route out of this conflict by separating cellular functions that support organismal performance from lineages that transmit genetic information to future generations. This mode of *cellular* altruism can be understood as a conflict-mediated transfer of fitness from lower-level units to the reproductive lineage of the collective (Michod, 2007).

Volvocine algae provide a useful illustration of this principle. Across the Volvocine clade, species vary widely in both somatic and reproductive cell number, indicating repeated shifts in the degree of germ-soma differentiation (Shelton et al., 2012). In this system, somatic cells perform functions that can be incompatible with immediate reproduction. Green algal colonies face a problem of negative buoyancy: if all cells divide simultaneously, flagellar func-

tion is lost and the colony may sink, compromising photosynthesis. By suppressing reproduction in some cells, a colony can maintain motility while other cells divide (Koufopanou, 1994). Thus, apparently "wasteful" somatic cells can increase the reproductive success of the germline by performing physiological work that reproductive cells cannot perform at the same time. Perturbations of genes such as *regA*, which maintains somatic cell identity by suppressing reproductive functions, further illustrate how relatively simple developmental changes can alter the balance between reproductive and non-reproductive roles (Huskey and Griffin, 1979; Nedelcu, 2009).

This conflict-mediated account explains why cellular altruism can be favoured, but it does not fully explain why multicellular evolution so often produces durable restrictions on heritable contribution. Once mutation is considered, soma is not only a cooperative compartment; it is also a way of controlling which cellular lineages are allowed to persist as *carriers* of evolutionary change.

One possibility is captured by the "dirty work" hypothesis, according to which somatic cells perform metabolically costly or mutagenic functions and thereby shield reproductive lineages from damage (Goldsby et al., 2014). On this view, soma acts as a mutation buffer: it localizes physiological risk to lineages with little or no heritable future. A related but broader interpretation is that somatic organization decouples mutation exposure from heritable contribution. Cells may divide, differentiate, metabolize, repair, or interact with the environment in ways that generate mutations, but only a restricted subset of those mutations can reach the next generation.

A distinct mechanism is emphasized by *mutational selection models*, in which filtering occurs primarily before organism formation through selection among gametes, germ cells, or zygotes (Haig, 2025b,a). Under this view, many mutations are eliminated before they are ever realized as organismal phenotypes, while those that pass this filter constitute genetic "innovations" available to individual-level selection. For example, spermatogonial mutations affecting pathways such as RAS-MAPK may expand within the germline even when they are deleterious after fertilization (Goriely and Wilkie, 2012). This illustrates that selection within reproductive tissues is not necessarily aligned with the fitness of future individuals.

The importance of pre-zygotic filtering depends on life cycle structure. In organisms with strong germline sequestration and a narrow zygotic bottleneck, many mutations can be filtered before contributing to organismal variation. In multicellular systems lacking such bottlenecks, or in systems where somatic mutations can enter reproductive lineages, this distinction becomes less sharp (Fig. 3). In these cases, somatic partitioning may reduce the cost of deleterious variation by restricting which cellular lineages are permitted to contribute to future generations.

Other hypotheses emphasize complementary constraints. The *disposable soma* theory

treats somatic maintenance as a resource-allocation problem: investment in repair and survival of the body competes with investment in reproduction, thereby contributing to senescence (Kirkwood, 2017). Ecological models of *de novo* multicellularity emphasize external pressures, such as predation, as drivers of aggregation, increased size, and subsequent differentiation (Herron et al., 2019). These hypotheses differ in mechanism and explanatory level, but they converge on a shared structural outcome: the separation of physiological function, mutation exposure, and heritable transmission.

A key empirical prediction of this view is that somatic and germline compartments should often exhibit different mutation-rate regimes. Indeed, mutation rates can differ systematically between somatic and germline cells (Chen et al., 2017; Milholland et al., 2017). This asymmetry is consistent with selection for higher fidelity in lineages that contribute to future generations, although its magnitude and direction will depend on cell depth, tissue type, life history, and effective population size. If the efficacy of selection on replication fidelity is limited by the drift barrier (Sung et al., 2012), then the marginal benefit of further reducing mutation rate should eventually become too small for selection to act on efficiently. Differences between somatic and germline fidelity regimes may therefore help distinguish among explanations for soma acquisition. If pre-zygotic filtering is the dominant mechanism, reductions in germline mutation rate may yield diminishing returns once filtering is already effective. If soma functions primarily as a mutation buffer, selection should instead favour stronger contrasts between high-fidelity heritable lineages and lower-fidelity disposable tissues.

The triple ecology of realised heritable variation

The population-genetic definition of germline implies that realised heritable variation is shaped by three nested selective stages (Fig. 5). First, mutations are produced and filtered within the ecology of germ cells themselves. Second, mutations arise, expand, and are eliminated within somatic tissues, where their fate depends on cell division rate, repair activity, clonal competition, tissue architecture, and the probability of entering reproductive lineages. Third, organismal phenotypes compete through their effects on survival and reproduction. The realised germline mutation rate is therefore not simply the mutation rate of anatomically defined germ cells, but the subset of genetic changes that pass through these filters and contribute to future generations.

This distinction matters because somatic organization is highly variable across lineages. Even closely related groups may differ in the architecture of development, the timing of germline segregation, the number and persistence of stem cells, and the permeability of the soma-germline boundary. In plants, for example, meristems can generate structured mosaics in which somatic mutations spread through growth and may eventually contribute to reproduction. Fern meristems with persistent initials differ from many spermatophyte meristems, where multipotent stem-cell layers and inter-layer genetic exchange create different opportunities for intraorganismal selection (Schoen and Schultz, 2019). Such diversity

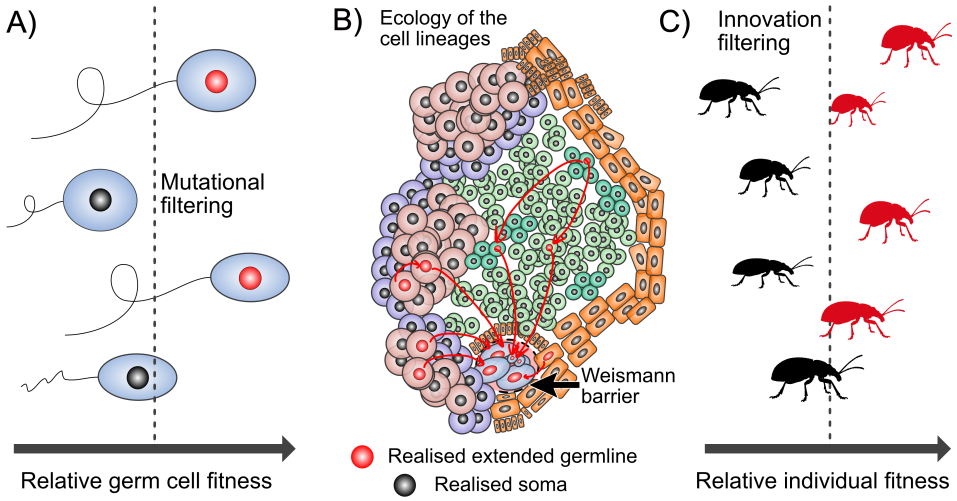

Figure 5. Realised heritable variation depends on a triple germ-cell, tissue, and organismal ecology. A) Germ-cell ecology filters genetic variation before organismal reproduction. Mutations that alter gamete competence can affect transmission through the germ line even when their effects are partly or wholly disconnected from individual-level fitness (Haig, 2025b). The dashed line represents mutational filtering, separating germ cells with low realised transmission from those that contribute to the next generation. B) Somatic and tissue ecology shape the production, expansion, and possible reproductive access of mutations during development and adult life. Cell division rate, repair activity, clonal selection, mutation rate, and probability of entry into reproductive lineages are tissue-dependent parameters (Martincorena et al., 2015, 2017; Cagan et al., 2022; Lawson et al., 2025). The Weismann barrier therefore acts as an additional filter on genetic variation by separating most somatic lineages from the realised germ line. C) Individual-level ecology filters heritable innovations according to their effects on organismal reproduction. Phenotypes that increase relative individual fitness spread their reproductive lineages into the next generation, whereas less successful phenotypes are lost. These phenotypes may reflect inherited genetic variants (Haig, 2025b) and, in some cases, somatic mutations that alter organismal fitness, such as cancer. Across panels, red denotes realised extended germline material or lineages that contribute to future generations, whereas black denotes realised soma or lineages excluded from direct hereditary transmission. In panel B, coloured cell populations represent distinct tissue lineages with different ecological conditions for mutation, expansion, competition, and access to the germ line.

makes it misleading to treat "somatic" and "germline" mutation rates as fixed organism-wide properties.

In practice, estimates of germline mutation may include mutations that arose in somatic or tissue-specific contexts but later entered reproductive lineages. Conversely, many mutations arising in reproductive cells are never transmitted and are therefore effectively somatic in the population-genetic sense (Lynch et al., 2016). For this reason, tissue-specific mutation rates and transmission probabilities may provide a more precise framework than a strict qualitative distinction between somatic and germline mutation rates. Mutation rates differ across tissues and organs, and these differences can contribute to variation in evolutionary rates among organs and cell lineages (Li et al., 2021; Moore et al., 2021; Cardoso-Moreira, 2026). What matters evolutionarily is not only where a mutation arises, but its probability of reaching the next generation.

This view also clarifies why selection within germline tissues can differ from selection across generations. Human male germ cells, for instance, can experience positive selection for particular mutations within the tissue, even when those mutations are constrained or deleterious at the organismal level (Ward and Kellis, 2012; Neville et al., 2025). Such cases reinforce the need to distinguish anatomical germline, cellular competition within reproductive tissues, and realised heritable contribution.

Taken together, these considerations support a transmission-control view of somatic evolution. The recurrent feature of complex multicellularity is not necessarily soma in the narrow anatomical sense, but the evolution of mechanisms that *decouple* mutation exposure from heritable contribution. Dedicated soma is one conspicuous realization of this principle. More generally, multicellular systems repeatedly evolve ways to control which cellular lineages perform physiological work, which lineages absorb mutational risk, and which lineages are permitted to transmit variation to subsequent generations. We conclude that a general theory of soma evolution requires a germline defined by realised heritable variation.

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Data availability

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