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Collective mitochondrial dynamics resolve conflicting cellular tensions: From plants to general principles

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ABSTRACT

Mitochondria play diverse and essential roles in eukaryotic cells, and plants are no exception. Plant mitochondria have several differences from their metazoan and fungal cousins: they often exist in a fragmented state, move rapidly on actin rather than microtubules, have many plant-specific metabolic features and roles, and usually contain only a subset of the complete mtDNA genome, which itself undergoes frequent recombination. This arrangement means that exchange and complementation is essential for plant mitochondria, and recent work has begun to reveal how their collective dynamics and resultant "social networks" of encounters support this exchange, connecting plant mitochondria in time rather than in space. This review will argue that this social network perspective can be extended to a "societal network", where mitochondrial dynamics are an essential part of the interacting cellular society of organelles and biomolecules. Evidence is emerging that mitochondrial dynamics allow optimal resolutions to competing cellular priorities; we will survey this evidence and review potential future research directions, highlighting that plant mitochondria can help reveal and test principles that apply across other kingdoms of life. In parallel with this fundamental cell biology, we also highlight the translational "One Health" importance of plant mitochondrial behaviour – which is exploited in the production of a vast amount of crops consumed worldwide – and the potential for multi-objective optimisation to understand and rationally re-engineer the evolved resolutions to these tensions.

1. Introduction

Plant mitochondria are responsible for plant respiration, and consequently all human life. Universally regarded as the "powerhouse" of the cell due to their housing of oxidative phosphorylation, mitochondria also play central roles in vital metabolic and signalling processes in plant cells [112,125,158]. In tandem, mitochondria undergo striking and heterogeneous physical dynamics in plant cells, moving rapidly on the cytoskeleton and adopting different placements in different cell types and conditions [4,76,96,98,99,143]. The reasons why plant mitochondria move in these complex dynamics is a long-standing mystery – although the disruption of this motion is highly detrimental to cell and plant performance [36]. Recent work has begun to suggest that their motion may offer a resolution to competing cellular priorities that would be impossible to achieve with static organelles [27,26,48,133]. This article will explore and attempt to extend this general picture of dynamic resolutions to cellular tensions [76]. We will survey different cellular pressures that may shape mitochondrial behaviour in plant cells, and discuss plant-specific features that may be responsible for the dramatic mitochondrial differences observed between plants and other kingdoms. We will summarise classical and emerging ways of characterising these dynamics and highlight some routes for future investigation.

We will take a perspective from systems biology. Although sometimes forgotten in the era of ever-expanding omics data, genes, interactions, and pathways together only constitute a small proportion of the "pillars" of systems biology. An influential early paper [84] has such "system structures" as only one of four key properties, the others being "system dynamics", the "control method", and the "design method". We will focus on the second and fourth of these – how the "design" of the dynamic physical behaviour of organelles can potentially fulfil cellular criteria. We will not focus on the specific molecular players involved in controlling this behaviour (some are catalogued in [143,76]), but rather its implications for the wider cellular system.

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The emerging perspective of an organellar 'interactome' is tightly linked to this picture, as inter-organellar interactions allow the coordination of spatially and temporally separated biological processes [28, 158,167,170,178]. It has been proposed that mitochondrial behaviour may be viewed through a social lens, as a system of interacting, and sometimes specialized, individuals that are functionally interdependent [27,123, 181]. In plants, mitochondria typically exist as separate individuals which transiently meet and exchange biomolecules, making the metaphor of a "social network" (where nodes are individual mitochondria and edges are interactions between two mitochondria) particularly apt. Indeed, recent experimental and theoretical progress has been made exploring the properties of these dynamic social networks and their capacity for "trade" and exchange [26,27,48]. We will argue that expanding this social picture to encompass other organelle types – a "societal" network – has the potential to further our understanding of the organellar interactome and the cellular benefits it provides [74].

We must also address the elephant in the room – or rather, the botanical differences from it. Mitochondria in plants behave in several fundamentally different ways from their, perhaps more familiar, cousins in animals. They are physically fragmented by default, move on actin rather than microtubules, possess alternative electron transport chain complexes, play a role in plant-specific metabolic processes, contain fewer copies of a generally larger mtDNA genome, and undergo recombination [76]. This host of differences suggest that a survey of plant-specific mitochondrial properties may be required to help intuition.

2. Individual behavior of dynamic plant mitochondria

The physics of mitochondrial motion in plant cells has been well characterised. Historically, the visual examination of plants has been an active research area since microscopy was invented [151]. Famously, Hooke's 1665 observation of "cells" in cork gave us the word we use today. But microscopy of plant mitochondria required several further technological advances, with groundbreaking work in 1955 [153,152] using phase contrast microscopy to characterise lettuce mitochondria, cytoplasmic streaming, colocalisation with other organelles, and more features that remain of interest today. Recent and ongoing developments of fluorescent reporter lines and microscopy tools and techniques have revealed the intricacy and dynamism of organelles within cells [172,98]. Using fluorescently labelled plant lines, live imaging and staining, experiments have demonstrated the physical heterogeneity of the mitochondrial population in individual cells [142,143,4,6,96,99]. These organelles show variation in shape, speed, size and distribution within the cell, across developmental stages, and between cell types [26,43,99]. Mitochondrial structure and dynamics vary across specific cell types, seen particularly in the streaming motion of mitochondria in root hairs and pollen tubes, their reticulated structures in apical meristems, and close chloroplastic colocalisations in leaf epidermal cells [14,144,190, 66].

In plant cells, mitochondria usually exist as punctate, rod- or sphere-shaped individuals, with a characteristic diameter around 1 μm (Fig. 1A-B). The physical network structures seen often in yeast and mammalian cells are the exception rather than the rule in plants – only observed under certain circumstances, like pre-germination wetting and shoot apical meristem development [121,144,76], described further in the next section. The number of mitochondria in the plant cell vary by tissue and species. Onion epidermal cells can have > 10,000 mitochondria [6], melon leaf cells may have ~450 [149], *Arabidopsis* mesophyll cells contain around 200–300 individual mitochondria, and tobacco protoplasts may contain 500–600 [148]. Individual mitochondria are typically on the 1 μm length scale in plants, and have a width around 0.5 μm [111]. In spinach, mitochondria have an area of 0.5–0.8 μm^2, and a volume of 0.1–1.6 μm^3 [189]. Given their endosymbiotic origins [134], it is not surprising that these mitochondria share a similar size and shape with many bacteria [111]. This stands in contrast to the extended, reticulated form common in some metazoan and fungal cells [128].

The populations of fragmented mitochondria are highly dynamic, with motile individuals interacting through colocalisations with each other and other organelles (Figs. 1, 2) [107,14,146,26,67,68,70,86]. Plant mitochondrial motion is quite heterogeneous- individual mitochondria move with a range of speeds and behaviours. Heterogeneity in angle of motion, distance between individuals and area covered by

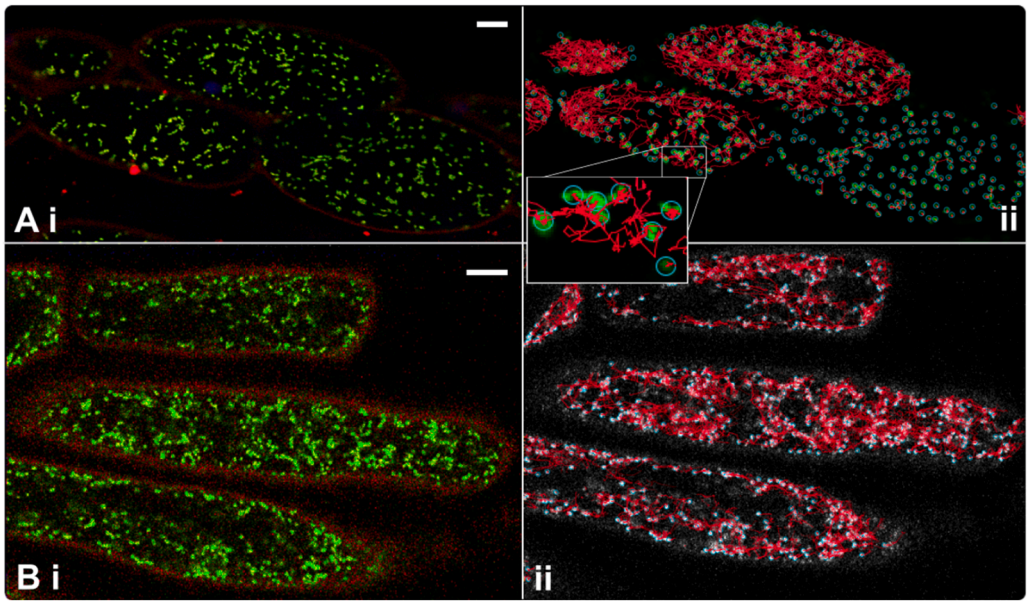

Fig. 1. Dynamic mitochondrial populations within plant cells. (A) Confocal microscopy of *Arabidopsis* hypocotyl cells with GFP localised to mitochondria (green), also stained with propidum iodide to highlight cell wall (red); (i) snapshot; (ii) dynamics, with trajectories in red: each individual mitochondrion is motion tracked over 4 min video time using Trackmate [162]. One cell (lower right) has close to static mitochondria, demonstrated by lack of motion tracks; such cells make up around 1–5% of our observations in *Arabidopsis* hypocotyl. MitoGFP plant lines are from [99]. (B) Root epidermis in wheat *Triticum aestivum*, with MitoTracker Green staining mitochondria (green) and propidium iodide cell wall (red): (i) snapshot; (ii) dynamics analysed and visualised as for (A) over a 2 min video. Scale bars are 10 μm.

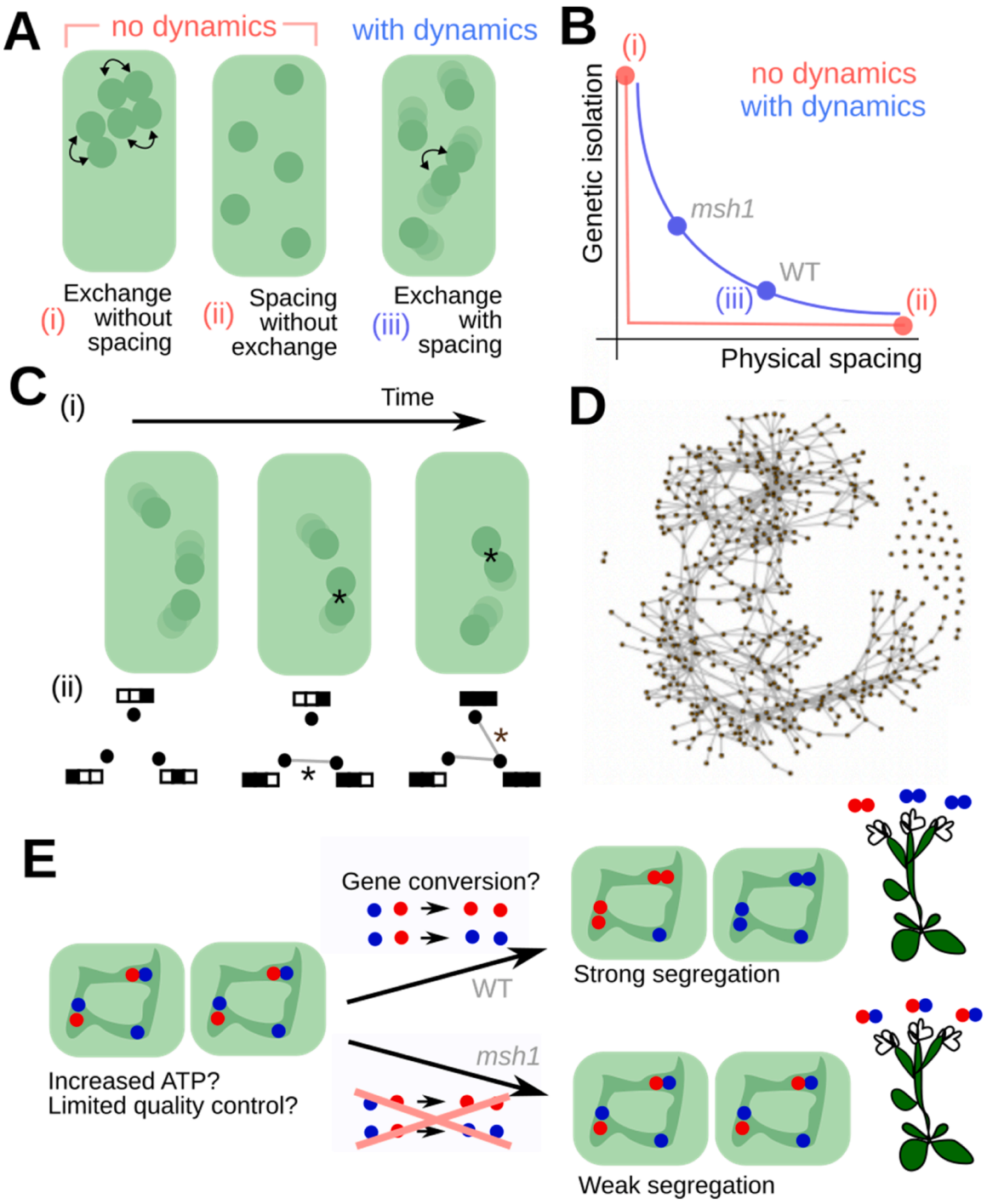

Fig. 2. Hypothesised links between physical, genetic, and biochemical mitochondrial dynamics in plants. (A) A tradeoff between mitochondria retaining physical spacing and supporting exchange of DNA and gene products [27,26,48]. Without dynamics, mitochondria can colocalize and exchange contents (i) or remain spaced (ii), but dynamics are required to support both together (iii). A Pareto front (B) captures this tradeoff; dynamics allow some resolution to these conflicting priorities, and wildtype and *msh1* (and *friendly*) mutant plants adopt different resolutions on this front [27]. (C) Physical encounters between mitochondria over time (i) allow complementation of gene products. This can be pictured as "trade" on a "social network" (ii) where mitochondria are nodes and physical encounters are edges (asterisks). As each mitochondrion only carries a subset of the full genome (black squares in (ii)) [126], exchanges allow mitochondria to share subsets and build up a complete "effective genome" over time. (D) An experimentally observed encounter network from *Arabidopsis* hypocotyl, demonstrating high but diverse connectivity and capacity for exchange [48]. (E) Mitochondrial fusion supports increased colocalization of mtDNA molecules, in turn allowing recombination [144,33]. If a cell's mtDNA population is heteroplasmic (red and blue mtDNA types), the presence of MSH1 supports gene conversion to generate cell-to-cell variability in heteroplasmy (here, proportion of the red mitotype), sorting heteroplasmy and allowing substoichiometric shifting between generations [19,20]. Without MSH1, this segregation is more limited and heteroplasmy is not sorted as rapidly.

dynamic motion has also been observed across the mitochondrial population in single plant cells [26]. As well as full fusion and fission events, plant mitochondria are able to transiently fuse, then partition and divide through 'kiss-and-run' dynamics, allowing sharing of intra-mitochondrial contents and membranes, while preserving their individual morphology [6,36,95]. In some cells (around 1–5%, based on our empirical observations in *Arabidopsis* hypocotyl; Fig. 1A-B) mitochondria are largely static. Passive motion due to physical fluctuations and hydrodynamic effects is frequently observed, and the coupled dynamics of other organelles influence plant mitochondrial motion [68], but mitochondria cover more cellular space when they are actively transported on cellular scaffolding, and this concerted movement of organelles can lead to cytoplasmic streaming [122,150,164,35].

Under this directed motion, at least an order of magnitude range of speeds is observed [26]. *Arabidopsis* leaf epidermal cell mitochondria show a range of 0.3–5.4 μm s^{-1} [32], 0.0–2.5 μm s^{-1} in *Arabidopsis* hypocotyl [26], and 0.1–0.5 μm s^{-1} in maize BY-2 cells [180]. Upper limits of 7.1 ± 1.5 μm s^{-1} are reached in *Picea wilsonii* pollen tubes [190], and up to 10 μm s^{-1} in *Arabidopsis* root hair [191]. The upper range of this motion is achieved by mitochondria moving on actin filaments [168], using plant-specific myosin motor proteins [12,11,163], particularly myosin XI family members which have high processivity. The hydrolysis of one ATP molecule will move myosin XI 35 nm along the filament, reaching a velocity of 5–7 μm s^{-1}, as demonstrated in tobacco [163]. The fastest myosin has been shown to move at 60 μm s^{-1} along actin filaments in the green algae *Chara* [57]. This is a fast, energetically expensive mode of motion, and the active movement of various particulate membrane compartments including mitochondria contribute to cytoplasmic streaming within plant cells, which in turn moves other organelles through busy cellular space [11,18,32,122,168,177].

In many cell types, plant mitochondria (and other organelles) have a physical challenge to contend with: a central vacuole that dominates cellular space in most cell types. A recent study on organelle dynamics in *Arabidopsis* and *Nicotiana* leaf epidermal cells quantifies the thin layer of cytoplasm left between the vacuolar membrane and the plasma membrane/cell wall as ~100 nm regions without organelles [37]. This extremely thin layer of cytoplasm must host many mitochondria (and other organelles) – meaning that these organelles exist in a restricted quasi-2D space, making their fast motion more remarkable, and potentially shaping the "design" of mitochondrial interactions. Transient transvacuolar strands allow mitochondria to cross the entire cell from one side to the other, which again may force colocalisation events [168,60].

Physical degradation of depolarised and unhealthy mitochondria is undertaken via mitophagy. We are beginning to understand more about this process, although much still needs to be uncovered in plant cells [91,108,115,131]. Mitophagy begins with recognition of the damaged mitochondrion by a phagophore, which eventually elongates and matures into an autophagosome, where individual mitochondria can be degraded and components recycled within the plant vacuole. The capture of a damaged, or excessively ROS-producing, whole mitochondrion and subsequent degradation in the vacuole has been demonstrated in wheat roots [110]. Recognition of damaged mitochondria is facilitated by ATG family proteins, but the mechanisms behind selective autophagy are still being elucidated [115]. Recent work has demonstrated the role of the FRIENDLY protein (FMT) in selective autophagy of depolarized mitochondria [102]. FRIENDLY is a homologue of the CLU (clustered mitochondrial) gene in yeast, mammals and *Drosophila* (where it is known as clueless) [4,98], and also influences mitochondrial fusion ([36]; see below). The rate at which selective mitophagy can occur continues to be investigated, but for example, a mitoautophagophore with ATG8 bound can selectively recognise depolarized mitochondria, and from a cup-shaped enclosure, form a 1–2 μm diameter membrane seal around the single organelle within 300 s. The entire process from an initial ATG8 puncta to a mature mitoautophagosome was estimated to take around 15 min, and massive protein reduction of outer and inner membrane as well as matrix proteins was found within four hours of depolarisation treatment [102]. Mitochondrial removal due to autophagy has so far been demonstrated in response to high UV [115], leaf senescence [91], and de-etiolation [102].

When considering the physical positioning and motion of mitochondria, their coupled metabolic and genetic properties should not be neglected [139,158,184,76]. The metabolic states of individual mitochondria are highly dynamic and responsive to stimuli, involving pulsing of membrane potential [141], responses to redox stimuli [140], ROS signalling [64] and many other degrees of freedom (recently explored in single-organelle proteomic detail [41] and reviewed, for example, in [111]. Several studies have tracked mtDNA copy number through various developmental stages in a number of plant species [45,87,88,126,160,175]. Unlike the situation in other kingdoms, individual plant mitochondria typically contain less than a full copy of the mtDNA genome, often containing a DNA molecule with a subset of genes or none at all [126,149]. This immediately suggests that interactions may be necessary within the plant mitochondria population – if an individual mitochondria does not internally encode the complete mtDNA genome, presumably complementation and sharing between individuals is necessary to acquire the full set of machinery it requires [4,48,6].

3. Collective behaviour – fission/fusion balance

The fact that plant mitochondria often exist as a fragmented population of individuals does not mean that an individual picture is sufficient to understand their behaviour. Rather, populations of plant mitochondria display a collection of "emergent" properties, whereby a set of individuals behaves differently from the sum of their parts [118,137,159,48]. First, the fragmented-population picture does not hold across all circumstances; second, the interactions within a dynamic fragmented population play important roles in the wider cellular and organismal context.

Several cell types and developmental stages have been recorded where plant mitochondria take on an extensively fused form (Fig. 2). One example is enhanced mitochondrial fusion prior to cell division in tobacco protoplast culture [148,147]. Massive mitochondrial fusion can also occur during germination, where dormant seeds have fragmented individuals, that after imbibition fuse to form a mitochondrial mesh that results in metabolic re-activation of these bioenergetic organelles [121]. The tubuloreticular mitochondrial structure is perinuclear (surrounding/adjacent to the nucleus), and this massive joining allows sharing and recombination of mtDNA, as well as other potential advantages [61].

This network case mirrors a similar situation in the shoot apical meristem (SAM) where the mitochondria form a striking cage-like structure around the nucleus [144]. Several reasons for this behaviour have been hypothesised. A physical/transport argument has suggested that general proximity to the nucleus supports optimal component supply from the nucleus for mitochondrial biogenesis, as well as optimal ATP delivery back to energy-demanding processes in the nucleus. This massive mitochondrial network has also been suggested as a mechanism for shaping the genetic population of mtDNA in the cell – particularly pertinent in the SAM, which goes on to form the aboveground germline of the plant. Seguí-Simarro and Staehelin [145] propose that the physical structure allows homogenisation of cellular mtDNA through development. As the network allows physical colocalisation of mtDNA – restricted in the somatic case of fragmented mitochondria – it may provide access to homologous template DNA, facilitating recombination between mtDNA molecules and allowing accurate repair [133,184,25,54]. We have proposed that this allows gene conversion to generate variability in mutant load across SAM cells and thus eventually between the plant's offspring, as a way to segregate mutational damage (Fig. 2) [33]. This segregation is achieved in animals by a process known as the "developmental bottleneck" or "mtDNA bottleneck" in the female

germline, involving a depletion of cellular mtDNA copy number and cell-to-cell variability induced by stochastic cell processes and divisions [79]. However, plants may not sequester a germline in the same way as animals do [33,89], and cannot rely on copy number depletion to accelerate drift. We have shown that segregation due to gene conversion does not depend on depletion[1] and that MSH1, an organelle DNA recombination factor favouring gene conversion pathways [185,54], is both highly expressed in the germline across plants [33] and dramatically accelerates segregation compared to a loss-of-function mutant [19,20]. The dynamics of the segregation through *Arabidopsis* development and between generations have been quantified with heteroplasmic profiling and stochastic modelling [20]. The rapid segregation of different mitotypes across plant offspring [105,117,19,20] supports so-called sub-stoichiometric shifting (SSS), where one mitotype – even if it is only present in a few cells in the mother plant – can rapidly amplify to dominate the mtDNA population of some offspring plants [1,73,9]. Recent experimental work has demonstrated how mitochondrial dynamics in *Arabidopsis* zygotes physically segregates the mitochondrial population in early development, with elongation of mitochondria occurring prior to asymmetric division of both the mitochondrial population and the zygote [83]; further physical investigation of plant mitochondria through development will help reveal the essential coupling between genetic and physical dynamics [76].

Fission and fusion events between these organelles are linked to quality control, mitochondrial health and stress responses. For example, mitochondrial expansion, clustering or reduced motion can be induced by stress stimuli, demonstrating the role of mitochondria as signal transducers [125]. This can interfere with the balance of mitochondrial fission, to maintain individual discrete elements, and fusion, leading to joined and elongated structures in the cell. In plants, various stimuli including but not limited to UV [44], low oxygen [130], dark [70], cold [8] or physical and cell death stimuli [142,187] can lead to this alteration in balance of fission/fusion. Mitochondrial morphology transition prior to cell death has been linked to calcium fluxes and activation of the permeability transition pore [142]. As in animals, mitochondria feature prominently in immune responses in plants, including mediating ROS signalling, cytochrome c release, and programmed cell death [142,176]. Mitochondrial dynamics appear to respond to infection and facilitate this control: for example, PEN2 controls the recruitment and accumulation of a static population of mitochondria around fungal infection sites, which exhibit a redox imbalance that may be involved in cellular signalling and infection resistance [42].

The genes controlling mitochondrial fission and fusion in *Arabidopsis* are still being revealed. Pioneering work into mitochondrial dynamics in plants was undertaken via an ethyl methanesulfonate (EMS) screen of mutant plants [100], identifying several mutants in which mitochondrial morphology, size, number and motion were impacted. These include BIGYIN, now known to be fission protein AtFIS1A, a homolog of yeast/mammalian Fis1 [4]. Nmt (Networked Mitochondria) identified due to its elongated mitochondria forms has been suggested to be allelic to Elm1 - a protein on the mitochondrial outer membrane that interacts with DRP3, although this allelism has not been tested [4,98]. Fission proteins DRP3A/B, homologous to human Drp1 have also been identified, and their influence on mitochondrial dynamics in plants is coupled to cardiolipin synthesis [120]. In yeast and mammals, mitochondrial fusion is dependent on two kinds of GTPases (Mfn1/2 and Opa1 in humans, Fzo1 and Mgm1 in yeast), but functional orthologs of these fusion factors have not been identified in plants. Indeed, the closest homologous proteins in plants – including DRP3A/3B (first published as ADL2a/2b [7,62,5]) have fission related roles, not fusion. Recently, the GTPase Miro2 has been shown to modulate mitochondrial-ER interactions, increasing mitochondrial size and decreasing mitochondrial number when constitutively activated – consistent with control of fusion [181]. A functional orthology with Mfn2 has therefore been proposed. Overall, the molecular players and mechanisms in plant mitochondrial fusion seem to differ substantially from those in animals and fungi [4], and are yet to be fully elucidated [143].

One protein that has been implicated in plant mitochondrial fusion, and identified in the EMS screen due to a clustered mitochondrial phenotype is the FRIENDLY protein (FMT). As mentioned above, FMT has been implicated in intermitochondrial associations, a prelude to mitochondrial fusion [36]. Phenotypically, disruption of FMT impacts plant growth and seedling development, including a shortened primary root, with more dead root cells, and a large increase in mitochondrial clustering, as well as more visibly depolarised mitochondria [115,129,36]. Interestingly, the differences between FMT mutants and Col-0 WT decrease as the plant ages, demonstrating the function of this protein appears to be greatest at early developmental stages. Disruption of FMT also impacts flowering time, plant size and hyponastic behavior (the raising and lowering of plant leaves in response to day/night cycles) [129]. From a cell biology perspective, FMT is cytosolic, but has been shown to form clusters in the dark, and when cells are treated with a decoupling agent [102,80]. These foci are dynamic, move along actin and sometimes microtubules, and also can colocalise with mitochondria [13]. This colocalisation has been implicated in participation of FMT in localisation and translation of mRNAs at the mitochondrial surface, and association with cytosolic ribosomes at the surface also [59]. FMT has been shown to be an RNA binding protein, and also interacts with mitophagosome marker ATG8, and translation-related proteins eIFisGI and OVA9 [13]. Finally, FMT disruption has been shown to impact the mitochondrial proteome, with 10% of mitochondrial proteins enriched (including three alternative oxidase proteins) or depleted (OXPHOS/TCA and ribosomal proteins) [59]. Overall, although FRIENDLY has been implicated in mitochondrial fusion, it also has roles in mitophagy, by preventing mitochondrial clustering and allowing autophagosome access to damaged mitochondria [13,59,80], but also translation and localisation of nuclear encoded mitochondrial-targeted proteins at the mitochondrial surface. Clearly, this protein has far reaching functions in overall plant health, mitophagy and selective translation of mRNAs, and future research will only continue to enhance our understanding.

4. Collective “social” mitochondrial dynamics resolve a tradeoff between physical spacing and genetic exchange

Large-scale fusion is not the only way that plant mitochondria can interact. The colocalisation, transient fusion, and departure of individual mitochondria in “kiss-and-run” dynamics supports exchange of contents while the population remains mainly fragmented [6,97] (Fig. 2). Such interactions between plant mitochondria facilitate the rapid sharing of contents through the fragmented population; experimental work has demonstrated that exchange of mitochondrial contents can occur within 3 s between individuals, with a period of direct adjacency of ~20 s prior to fusion [6]. In a cell with many individual mitochondria, mixing of contents could occur within 1–2hrs.

Recent work has underlined the strengths of considering the collective motion of individual mitochondria from the perspective of their encounter or “social” networks [27,26,48]. Here, individual mitochondria are “nodes” on the network (akin to individual humans, in the social network metaphor), and “edges” between nodes correspond to colocalisations (akin to relationships). Over time, more pairs of individuals experience colocalisations, so the network gains more edges. After a given period of time, the network reflects the potential interactions that could have occurred between mitochondria. Networks observed in this

[1] Specifically, the contribution of gene conversion to segregation (the increase of heteroplasmy variance) per unit time scales like $(1-f)^2$, where f is the proportion of fragmented mitochondria (i.e. high in soma, low in SAM) [33]. This contrasts with the contribution from mitophagy, which scales like f / N [10,33]– hence, physical fragmentation f (controlled by the cell), trades off genetic variance generation from mitophagy and gene conversion.

way using live-cell imaging and video analysis [162] resemble human social networks in several ways, including "long-tailed" degree distributions (some mitochondria have many encounters, many have few).

Comparing these observed networks to theoretical alternatives from physical simulation [26,48] shows that plant mitochondrial dynamics perform remarkably well in at least two tasks. The first is resolving a tradeoff between maintaining physical spacing and supporting encounters for exchange (Fig. 2). This cannot be achieved with static mitochondria (which cannot be both apart and together, Fig. 2A-B), but dynamic "kiss-and-run" behaviour provides a near-optimal resolution to this conflict [26]. The second is the sharing of genetic material and gene products (Fig. 2C-D). Exchange and complementation of mtDNA and gene products (mitochondrial proteins, tRNAs and rRNAs) has been modelled for both experimentally derived networks of connectivity between mitochondria in plant cells and a set of random, null and alternative theoretical networks [48]. The dynamics of each mitochondrion in a system accessing a full complement of genes and proteins depended on the number of genetic elements required to make a full set, but also the physical motion of these individuals within the cell. Across various parameterisations, the dynamics and connectivity seen within the plant cell chondriome offered the most efficient way of gaining a full genetic complement [48]. This collective behaviour allows a system made up of individuals to integrate an otherwise uneven distribution of genetic material.

Notably, the resolution to the spacing-exchange tradeoff is controlled under different perturbations. Challenges to mitochondrial dynamics via the *friendly* mutant [26] and to mtDNA maintenance via the *msh1* mutant [185,27] both lead to a rebalancing of the spacing-exchange poise from collective mitochondrial dynamics, towards decreased spacing and increased exchange. In the case of *friendly*, this rebalancing was time-dependent, with differences in social statistics more pronounced over shorter timescales, and converging towards WT at later time points as more connections formed in the social network [26]. In the case of *msh1*, the observed rebalancing towards increased exchange capacity is compatible with a picture where the cell attempts to compensate for decreased mtDNA integrity by amplifying mitochondrial exchange and complementation [27] (Fig. 2).[2] Hence, collective mitochondrial dynamics support a controllable, adaptable tradeoff to conflicting cellular priorities of mitochondrial spacing and biomolecular exchange [133].

5. Mitochondrial dynamics may resolve a wider set of competing cellular priorities

Jayashankar and Rafelski [74] draw attention to the integration of mitochondrial behaviour into the wider cellular architecture. We wholeheartedly endorse this line of enquiry. The aforementioned theory work has used a "social network" picture to suggest that mitochondrial dynamics optimally and adaptably resolve a tension between spacing and exchange [27,26,48]. Given the license afforded by a review paper, we can speculate further about other, possibly conflicting, priorities that may influence mitochondrial behaviour in the cell (Table 1; Fig. 3). In doing so, we will develop our proposed expansion from a between-mitochondrial "social" network to a between-organelle "societal" network picture – considering collective behaviour not just of mitochondria, but of other cellular inhabitants together in their overall environment [28,158,167,170,178].

A useful metaphor comes from the field of urban planning in human society. City planners must decide what resource to invest in power plants, factories, a transport network, communications, facilitating trade through infrastructure, avoiding pollution with decentralization,

Table 1
A non-exhaustive set of cellular priorities that mitochondrial behaviour can help address as collaborators in the cellular "societal network" of organelles. A summary description of how mitochondrial structure and/or dynamics influences each is provided (see text for more detail).

Cellular priority	Description and example references	Mitochondrial arrangement
Colocalisation of mtDNA, supporting recombination	Recombination-mediated gene conversion generates beneficial cell-to-cell variability in mutant load; recombination-mediated repair of mutational damage [33,54,81,101,145].	Together
Exchange of mtDNA and gene products	Exchange through fusion allows individual mitochondria to acquire the full set of mtDNA-encoded gene products [6,25,48,133].	Together
Even physical spacing	Reduced local buildup of damaging reactive oxygen species, even distribution of ATP supply, rapid repositioning and uniform access to other organelles [169,192,26]. Emerges spontaneously under ATP-driven random motion of ATP-producing model organelles [106].	Apart; possible emergent epiphenomenon from several of the above
Metabolic crosstalk	Colocalisation with other organelle types facilitates exchange of biomolecules and multi-compartment metabolic pathways (photorespiration, for example, requires mito-chloro-peroxisome flux) [109,114,119,15,39,53,55]. Colocalisation with the ER plays a role in fission-fusion balance [181].	Near other organelles
Oxygen and CO_2 poise	Positioning to modulate molecular gas concentrations (for example, reducing oxygen presence near the nucleus, or scavenging photorespiratory CO_2) [113,135].	Together at particular regions
Signalling	Physical localisation to convey a uniquely identifiable signal (via diffusing molecules, physical properties including membrane potential and mechanotransduction, or other media) [123,125,179].	Near the source / target for signal transduction
Quality control and inheritance of mitochondria and mtDNA	Quality control through mitophagy [102,110,115,132], classically pictured with fragmented organelles [166] but also possible (in yeast) within a network [72]; positioning relative to division plane to ensure even partitioning between daughter cells and/or generate genetic variability [33,52,71,78].	Apart or generally controlled
Chemical landscape	Positioning of mitochondria to modulate cell-wide concentration profiles of metabolites and ions, including, for example, ATP, Ca^{2+}, pH. Arguments here are by analogy with animal observations [138,21,40], but plant imaging technology is revealing ATP [31] and Ca^{2+}[173] behaviour in plants, some evidence in algae links pH and mitochondrial positioning [39], and modelling will help	Generally controlled

(*continued on next page*)

[2] At the level of individual mitochondria, we observed a slight decrease in individual mitochondrial size in hypocotyl in the *msh1* mutant, contrasting with a slight increase in size in variegated tissue from independent work [186]

Table 1 (*continued*)

Cellular priority	Description and example references	Mitochondrial arrangement
	quantify these arguments further.	
Response to environment and infection	Responses to light (possibly epiphenomenological given the response of organelle partners [186,38,66,67,69]); temperature [8,154]; and other environmental stimuli, possibly allowing physical modulation of respiration [161]. Multifaceted physical and chemical responses to infection [176].	Generally controlled
Optimising mitochondrial metabolism	Mitochondrial morphology shapes nutrient usage and metabolism [93]; fusion can improve ATP production [61].	Fused?

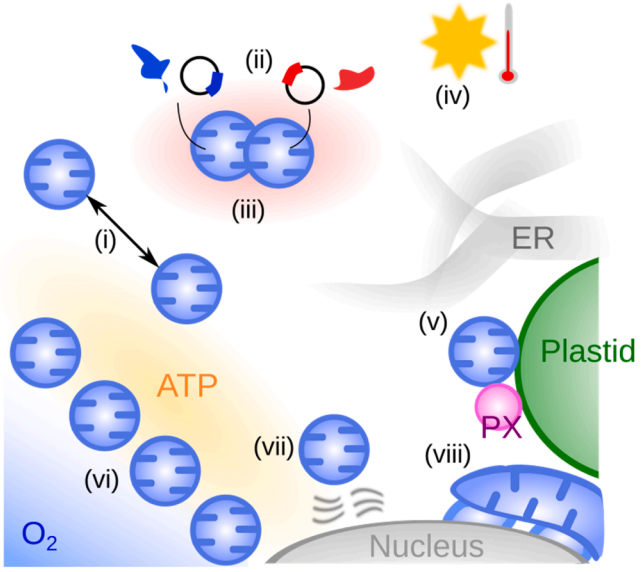

Fig. 3. Examples of possible tensions which (plant) mitochondrial dynamics resolve. (i) Even spacing of mitochondria through the cell. (ii) Exchange of mtDNA and gene products between colocalised, fused mitochondria. (iii) Local high concentrations of reactive oxygen species from colocalised mitochondria. (iv) Environmental stimuli including light and temperature shape the distribution of mitochondria and their interaction partners. (v) Colocalisation of mitochondria with other organelles including the endoplasmic reticulum (ER) for transport, calcium buffering, and metabolite exchange; plastids and peroxisomes (PX) for metabolic crosstalk including in photorespiration. (vi) Modulation of cellular gradients of oxygen, ATP, and other small molecules. (vii) Information transfer via diffusible or other biophysical signals between organelles. (viii) Fusion into a cage-like reticulum around the nucleus in the shoot apical meristem, illustrating the range of physical behaviours that may influence inheritance of mitochondria through development.

zoning to control the proportion of different functions, and so on. Feedback processes must exist to respond to changes in environment and economic supply and demand. Many tensions and tradeoffs exist, which together shape the emergence of the structure and collective dynamics of societal structure as a whole. Metaphorical connections with the plant – and general eukaryotic – cell abound. Bioenergetic organelles are power plants; the cytoskeleton is transport infrastructure; diffusing molecules and other physical factors form communication links; reactive oxygen species are under some circumstances a form of pollution; organelle DNA and gene products are tradeable goods (Table 1). Like in urban planning, ideas from operations research, systems theory, and optimisation can suggest beneficial ways of organising this complex society.

We have already discussed some "zoning" and "trade" aspects of mitochondrial behaviour in maintaining a balance between spacing and biomolecule exchange. To pursue the metaphor further, metabolite exchange with other organelles is an essential class of plant mitochondrial process. Central, essential processes like photorespiration require metabolic crosstalk between mitochondria, chloroplasts, and peroxisomes [15,34,55]. Colocalisation of these organelles will both accelerate and target metabolite exchange [109,119]. The roles (including calcium and lipid exchange and biomolecular transport) and mechanics of mitochondrion-ER colocalisation, a growing field in animals, are increasingly being uncovered in plants [92,94,114].

Metabolite exchange clearly motivates colocalisation of organelles – a diffusing molecule released by an organelle will be more rapidly taken up by a proximal organelle than a distal one. But it also suggests a more general role for mitochondrial positioning in influencing the concentration landscape of the cell. Recent results in human cell lines have demonstrated that perinuclear position of mitochondria (oxygen absorbers) can reduce the exposure of the nucleus to molecular oxygen [113]. This parallels the plant-specific that mitochondrial relocation to inner bundle-sheath cells, near the vasculature, is an early and important step in the evolution of C_4 photosynthesis [136,182] – perhaps because it supports the reuse of CO_2 efflux from mitochondria (CO_2 producers) by adjacent chloroplasts [135].

The modulation of the concentration profiles of other chemicals is another cellular priority which mitochondrial structure and dynamics can influence [40]. There is a pervasive idea that concentration gradients of small molecules are unlikely to exist, because small molecules diffuse very quickly. But rapid diffusion will only smooth out gradients in the absence of sources and sinks of molecules. If, instead, a chemical species is produced and removed at specific spatial points in the cell, it is perfectly possible for strong gradients to exist between source and sink even for rapidly diffusing molecules. Elegant experiments in mouse embryonic fibroblasts demonstrate, for example, the maintenance of an intracellular ATP gradient [138]. Perinuclear mitochondria provide high levels of ATP, demonstrated by a high ATP:ADP ratio that is diminished towards the periphery of the cell. The authors also demonstrated that in regions where mitochondria are positioned such as the leading edge of the migrating cell, mitochondria are the vital energy providers for this process, linking specific intracellular mitochondrial positioning with ATP supply for energy-intensive membrane reorganization [138]. Large tentacular cages of reticulated mitochondria in the *Arabidopsis* SAM are perinuclear, establishing a spatial relationship that would allow large quantities of ATP to be delivered to the actively dividing nucleus, energetically supporting spindle formation and chromosomal separation [144]. However, this tentacular structure is not compulsory for proliferating plant cells, as it is not seen in the root apical meristem, therefore putting more emphasis on the parallel role of allowing sharing and recombination of mtDNA. In plants, an increase in photorespiratory metabolites has been observed in engineered chloroplast-chloroplast adhesions, demonstrating the influence of organellar positioning has on metabolic pathways [65]. The modulation of these and more concentration landscapes may well be a contributor to mitochondrial positioning control in the plant cell.

As sessile organisms, plants are particularly sensitive to environmental changes, and have sophisticated response mechanisms to factors such as light levels, oxygen concentration and temperature. Mitochondrial dynamics contribute to these responses. Mitochondria have been shown to alter their position in the cell in response to different strengths of treatment light in a reversible manner, slowing down their dynamic movement when they have reached their destination [67]. These responses are mediated by photoreceptors, but also by the influence of photosynthetic processes [66,67]. High light also induces peroxule-mitochondria interactions, mediated by ROS signalling [70]. It has also been shown that nuclear loci, specifically the CAB locus

encoding chlorophyll a/b binding proteins, has been shown to relocate and alter its radial distribution in response to light [38]. This physical relocation and related transcriptional activation of an organelle-related gene demonstrates a link between organellar positioning and cellular signaling, in response to an environmental change. The dynamic population of mitochondria has also been shown to respond to changes in temperature. In *Arabidopsis*, mitochondria demonstrated an increased fragmentation in response to cold stress (4ºC) [8]. In algae *Micrasterias denticulata*, aquatic plant *Lemna sp.*, and the cold-adapted plant *Ranunculus glacialis,* mitochondria demonstrate an increase in fusion and networked organelles in response to freezing stress (−2°C), in order to increase or at least maintain respiration rates [155]. In both cases, it is predicted that mitochondrial morphology alterations allow the cell to respond to the environmental stress, and modulate their surface area and respiration rates accordingly [161].

In dividing cells, an essential priority is controlling the inheritance of mitochondrial content between daughter cells. Both the physical and the genetic content of mitochondria must be partitioned appropriately, which in different developmental circumstances may involve the faithful inheritance of content, or the generation of variability, between daughters [52,71,78,83]. In the plant germline, mtDNA variability is generated remarkably quickly [19,20]. The mechanisms behind this rapid segregation, called the "mtDNA bottleneck" in the animal world [156,174,79], likely involve a combination of stochastic partitioning at cell divisions and gene conversion supported by mtDNA encounters [33]. Recent theory has shown that the physical structure of the mitochondrial population can control both the physical and genetic inheritance of mitochondria at cell divisions, providing another cellular and developmental priority addressable with mitochondrial dynamics [52,71].

One less-studied topic is how the physical behaviour of mitochondria influences their capacity for cellular signalling. Individual mitochondria are involved in a wide range of signal transduction processes – with a recent review highlighting their status as "information processing organelles" [125], paralleling the picture of plant mitochondria as a signal integration hub [158]. All cellular signals are fundamentally conveyed through physical means. In the case of fragmented mitochondria, the question of individual identification arises: if one of the hundreds of mitochondria in the cell require new nuclear-encoded machinery, how does that mitochondrion specifically receive it [103,2,3,51,56,77]? In the absence of a signal that labels an individual mitochondrion's identity, perhaps (transient) colocalisation with the nucleus can allow individual response. Anchoring of mitochondria to other organelles or to polar ends of cells may facilitate signalling, particularly through calcium or lipid transfer [85]. Retrograde signalling between mitochondria, chloroplasts and the nucleus is vital for regulation of gene expression in response to functional change in these organelles [30,179]. Therefore, cross-organelle signalling capacity and fidelity will be shaped by the physical arrangement and proximity of the compartments involved.

6. Translational aspects of plant mitochondrial behaviour

The dynamics of mitochondria in plants have received much less attention than those in the animal kingdom. Part of this disparity is no doubt due to their perceived lack of importance compared to the role of mitochondrial dynamics in human disease. But we, predictably, will (gently) argue that plant mitochondria should not be ignored from the translational perspective – not primarily for human disease, but for their essential role in human health via global food production, a target of the One Health initiative and UN Sustainable Development Goal 2.[3]

Of course, as in the vast majority of free-living eukaryotes, plant mitochondria are central to bioenergetics and metabolism and play a multitude of essential roles in signalling, stress responses, and more. Some of these are shared across kingdoms [112]; some, including photorespiration, alternative oxidase activity, and signalling and metabolic responses to pathogens, are more plant-specific [111]. However, in part due to the limited mitochondrial dynamics mutants available in plants, direct connections between mitochondrial dynamics (our focus here) and these roles remain to be explored. The pronounced and cross-system phenotype of plants where mitochondrial dynamics are compromised highlight their importance [36]; and *any instance where a resolution to a tradeoff has evolved (as above) raises the possibility of rational re-engineering of that resolution to suit more human priorities*.

The importance of mitochondrial behaviour – not just their individual and collective motion – in plant (and therefore crop) physiology has been reviewed elsewhere [111,46], and the translational importance of plant mitochondria highlighted by a recent large-scale survey identifying considerably more routes to yield improvement via modulation of respiration than photosynthesis [46]. With our focus on plant mitochondrial dynamics, some particular cases are worth emphasising here, including links between the plant, and crop, domain with several well-known roles of mitochondria in human pathophysiology. One example is human ischemia-reperfusion injury: tissue damage to the heart or brain caused by the rapid reoxygenation of cells after the vascular blockage leading to heart attack or stroke is cleared [24]. Reoxygenation injury in plants is also considerable, following drainage after flooding or perturbations to soil [90], and as it is directly shaped by mitochondrial activity [75], is coupled to the position of mitochondria in the cell. It is also worth noting that mitochondrial positioning in the cell is an early and essential step in the evolution of efficient C_4 photosynthesis (helping scavenge photorespiratory CO_2) [136,135]. A study inferring the evolutionary pathways of C_4 by studying the properties of C_3-C_4 intermediates identified it as an early precursor step to further metabolic and genetic changes [182]. Inter-organelle signalling is essential in co-ordinating stress and environmental responses within the cell, and as discussed above is modulated by the spatial arrangement of organelles [30].

This review has emphasised the capacity of physical mitochondrial dynamics to shape the genetic population of mtDNA in the cell [10,33]. As in humans, pathological mtDNA variants exist in plants and have strong phenotypic consequences. Although detrimental to the plant, these consequences can be tremendously beneficial to human agriculture [104]. Many plants experience male sterility as a result of mtDNA variants (called cytoplasmic male sterility or CMS) [23,63,165]. But male sterility is an essential feature in the production of high-yielding F1 hybrid crops, as it prevents self-fertilisation of the parental inbred lines [22,58]. Exploiting CMS thus allows plant breeders to create hybrids without manually or chemically sterilising plants.

A wide-reaching (but somewhat dated) review [58] has attempted to estimate the prevalence of CMS-produced hybrid crops in a global setting. Not all crops are hybrids and not all hybrids are produced with CMS, but for several crops CMS dominates production. The following paragraph summarises findings from [58] as of 2004. Onion markets were dominated by hybrids, the vast majority of which are produced using CMS from a single cytoplasmic line. Rapeseed hybrids made up 10–50% of seeds in North America and Europe and more in Asia; 40% of these in Europe and Canada were produced using CMS. Carrot hybrids using CMS made up around half the world market. Maize was one of the first crops to be hybridised using CMS, although early lines' sensitivity to pathogen *Bipolaris maydis* led to an epidemic in the 1970 s and a decrease in the reputation and uptake of CMS approaches [127]. Havey [58] notes that statistics on CMS maize use was hard to find but it was (and is) certainly widespread. Hybrid millet made up nearly 100% of the US market and 50% in India; most pearl millet was produced using CMS with a particular cytoplasm line. Rye hybrids reflected 10–60% of production in central Europe, using CMS. Sunflower hybrids produced using CMS dominated many global production areas.

Clearly, pluralities and often majorities of many real-world crops are produced by harnessing what is effectively mtDNA disease in plants.

[3] https://www.fao.org/one-health/en; https://sdgs.un.org/goals

However, CMS lines are not flawless; the aforementioned maize epidemic involving maternally inherited susceptibility [127] provides a cautionary tale, and [58] references several instances where CMS lines have negative traits including cold susceptibility and decreased attraction to pollinators. Research on mechanisms generating new CMS variants may have translational promise in circumventing such issues [104, 73]. One intriguing avenue is the MSH1 protein responsible for (among other things) mtDNA maintenance [1,185]; recent work has generated new *Arabidopsis* mtDNA variants with an *msh1* mutant that were then rapidly segregated to homoplasmy after backcrossing to recover MSH1 functionality [19], suggesting a route to generate mtDNA diversity. The recent establishment of targeted base editing in plant mtDNA [116] represents another exciting research avenue on this topic.

7. From plants to general principles: resolving cellular tensions with mitochondrial dynamics

The structure and dynamics of mitochondria differ across kingdoms, species, organisms, and cells. But many of the cellular priorities discussed above have at least some universal presence across eukaryotes [112]. The behaviour of mitochondria in many organisms can influence these aspects of the "cellular society", so it is tempting to speculate that some universal principles may underlie the structure and dynamics of mitochondria across life. Perhaps the different behaviours observed in animals, plants, fungi, and protists – and across different tissue types and disease states – may correspond to resolutions for different weightings of the tensions in Table 1.

Our recent work would suggest a (highly hypothetical) picture coupling plant mitochondrial dynamics, genetics, and evolution. Here, plant mitochondria retain relatively high gene complements to enable rapid and organelle-specific responses to their pronounced diurnal fluctuations in environmental energetic and metabolic demands [47, 50]. This retention differs across plants: self-pollination and clonal plants transfer more mitochondrial genes to the nucleus [17,16]; herbaceous plants seem to retain fewer mtDNA genes (perhaps because they experience comparatively limited environmental variability over their lifetimes) [49], but generally plants retain comparatively many mtDNA genes. As plants do not sequester a germline in the same way as animals, they mitigate the consequent genetic susceptibility through an alternative genetic bottleneck, via gene conversion [19,20,33]. The necessity for recombination requires mitochondrial fusion in the SAM [144]. But recombination can also have unwanted side-effects like genomic fragmentation and the appearance of selfish elements (for example, fragments of mtDNA containing an origin of replication but limited genetic content) [184]. Given that most plant mitochondria contain less than a full copy of the genome [126,149], restricting mitochondrial fusion physically limits the amount of recombination that can occur. This "control lever" could be employed to support variance generation in the SAM (where cells that do end up dominate by selfish elements can perhaps be sacrificed) while limiting recombination by enforcing fragmented mitochondria containing less than a full genome in other tissues [126,149]. This fragmentation also allows rapid reconfiguration of mitochondria to fulfil other roles from Table 1, including modulating chemical concentrations and controlling signalling between organelles.

This picture raises several future questions that could test and refine the hypotheses involved. In plants that exploit clonal proliferation (for example, seagrasses [188]), do the same physical-genetic principles shape mitochondrial behaviour in the somatic line? In other organisms that do not sequester a mammal-like germline and support mtDNA recombination, do mitochondria stay largely fragmented outside the replicative line? Some observations suggest that this is the case in, for example, freshwater sponges [171] and filamentous algae [39].

A review [61] suggested several potential reasons for mitochondrial fusion (while [183] provide arguments for remaining as a collection of individuals). Why are these potentially advantageous points apparently less important in plants? Complementation and shaping the genetic population are two of these hypotheses, which have been discussed in the plant context above. Here, we speculate that the "social network" arising from transient interactions between dynamic plant mitochondria replaces the physical network found in other eukaryotic contexts – *in plants, mitochondria are connected through time rather than through space*. Increased ATP production, through various potential mechanisms including membrane shape, proton leak, modulating mitophagy, and nonlinear kinetics has also been suggested. If this is an important determinant of fusion then it would suggest that increasing ATP production is not a high priority in the majority of plant cell situations. The large-scale fusion observed upon imbibing seeds [121] is a possible counterexample; clearly the ignition of multiple developmental processes will constitute a highly energy-demanding time. The rapidly replicating shoot apical meristem (SAM) may also reflect strong energy demands, corresponding to the aforementioned hypothesis about ATP delivery to the nucleus [144]. Calcium buffering, signalling, and physical buffering against thermodynamic and biochemical perturbations have also been proposed as fusion advantages; further theoretical work harnessing plant-specific observations will help compare and select between these mechanisms [125,173].

The key hypothesis unifying the content of this review is that *mitochondrial dynamics and arrangements provide context-specific resolutions to tradeoffs between physical and "social" priorities in plants (and across eukaryotes)*. The adaptability of these resolutions to different challenges suggests that the physical arrangement of mitochondria provides the cell with a rapid, non-genetic control mechanism for shaping exchange and metabolism in the face of changing demands.

We began with a perspective drawing on the systems biology pillars of "system dynamics" and "design method". Through this lens, the urban planning metaphor above, for the design of dynamic cellular "societies", bears a further extension. In urban planning, tools from operations research, control theory, maths, and simulation are used to design optimal and robust structures. We suggest that *such quantitative approaches provide a promising way of harnessing the unprecedented volume and quality of data describing cellular societies to identify and test potential governing principles*. It has been argued that "optimization is the only approach biology has for making predictions from first principles" [157]. Our work has suggested that some of the resolutions that plant mitochondria provide to the tension between competing priorities do approach optimality [27,26,48]. Going forward, we believe the fields of set- and multi-objective optimisation [29,82] may provide strong theoretical tools to explore optimality in these stochastic, multi-objective systems. Identifying the principles by which evolution has shaped these resolutions may help the rational re-engineering of cell behaviour to suit human priorities – both in plants and across kingdoms.

Declaration of Competing Interest

The authors declare that no competing interests exist.

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