New Phytologist

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The genomic secrets of invasive plants

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Received: *16 July 2024*
Accepted: *28 November 2024*

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New Phytologist (2025) **245:** 1846–1863
doi: 10.1111/nph.20368

Key words: adaptation, genetic load, hybridisation, invasion genetics, invasion genomics, invasive species, polyploidisation, structural variants.

Summary

Genomics has revolutionised the study of invasive species, allowing evolutionary biologists to dissect mechanisms of invasion in unprecedented detail. Botanical research has played an important role in these advances, driving much of what we currently know about key determinants of invasion success (e.g. hybridisation, whole-genome duplication). Despite this, a comprehensive review of plant invasion genomics has been lacking. Here, we aim to address this gap, highlighting recent discoveries that have helped progress the field. For example, by leveraging genomics in natural and experimental populations, botanical research has confirmed the importance of large-effect standing variation during adaptation in invasive species. Further, genomic investigations of plants are increasingly revealing that large structural variants, as well as genetic changes induced by whole-genome duplication such as genomic redundancy or the breakdown of dosage-sensitive reproductive barriers, can play an important role during adaptive evolution of invaders. However, numerous questions remain, including when chromosomal inversions might help or hinder invasions, whether adaptive gene reuse is common during invasions, and whether epigenetically induced mutations can underpin the adaptive evolution of plasticity in invasive populations. We conclude by highlighting these and other outstanding questions that genomic studies of invasive plants are poised to help answer.

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I. Introduction

Species that are introduced by human activity into novel ranges, and that spread across the landscape disrupting biological communities, economies, and human health, do so under novel genetic and evolutionary circumstances (Prentis *et al.*, 2008). As such, the study of genetic variation and evolutionary processes such as genetic drift, hybridisation, or (mal)adaptation is a bedrock component of invasion science (Lee, 2002; Bock *et al.*, 2015; Hodgins *et al.*, 2018). However, this has not always been the case. In fact, until three decades ago, invasion biologists focussed almost exclusively on ecological questions. Following the lead of prominent British ecologist Charles Elton (Elton, 1958), this research played a critical role in the development of invasion ecology as a highly productive scientific discipline (Ricciardi & MacIsaac, 2008). It revealed, for example, that human activity underpins the global homogenisation of flora and fauna (McKinney & Lockwood, 1999), that processes such as propagule pressure can drive invasions (Lockwood *et al.*, 2005), and that the effects of invasions cascade across trophic levels in recipient communities (Ellis *et al.*, 2011).

The comparatively slow uptake of evolutionary thinking in invasion science may be surprising given that *The Genetics of Colonizing Species* was published in 1965 (Baker & Stebbins, 1965). Edited by leading botanists and evolutionary biologists Herbert G. Baker and G. Ledyard Stebbins, this publication summarised the proceedings of a symposium that occurred in 1964 at Asilomar (California). The meeting was attended by some of the most influential figures in evolutionary biology at the time including Cyril H. Waddington, Ernst Mayr, and Theodosius Dobzhansky (Barrett, 2015). Notably, the Baker & Stebbins (1965) volume was the first to focus squarely on the evolution of adept colonisers. And yet, it was not until the 1980–1990s that invasion biologists began to systematically consider evolutionary processes that occur during invasions (Barrett, 2015). This coincided with technological developments in DNA sequencing that enabled genetic variation to be interrogated in large numbers of populations (Barrett, 2015). Also, it overlapped with a paradigm shift in biological sciences more broadly, which started to more frequently consider the possibility that substantial evolution can occur over the course of just months and years (Reznick *et al.*, 2019) – a timescale sufficiently narrow to influence the course of invasions.

During this time of increased interdisciplinarity, botanical research was at the forefront of intertwining evolution and invasion ecology. Indeed, many of the most prominent evolutionary hypotheses on the origins of invasions can be traced to the study of plants. For example, synthesising earlier evidence of intra- and interspecific genetic exchange that involved introduced plants (e.g. Baker, 1948), botanical workers pointed to the possibility that hybridisation stimulates invasiveness (Ellstrand & Schierenbeck, 2000). Furthermore, drawing on the observation that introduced plants tend to be larger than native counterparts (e.g. Crawley, 1987), botanists proposed that the evolutionary reallocation of resources from defence to growth or reproduction may facilitate invasions in areas where native specialist enemies are scarce (Blossey & Notzold, 1995). Lastly, characteristics of plants such as the frequent occurrence of whole-genome duplication allowed botanists to propose that such large-scale genomic changes enable organisms to negotiate new and more challenging environments (Clausen *et al.*, 1945), and could therefore spur invasions (te Beest *et al.*, 2012).

Over the past decade, improvements in genomic tools have allowed modern invasion biologists to test many of these hypotheses, drawing on data that range from the ecosystem level down to single mutations (Bock *et al.*, 2015; North *et al.*, 2021; McGaughran *et al.*, 2024). This rapidly progressing research programme has supported and, in some cases, challenged hypotheses formulated since 1965. In recognition of the important inflection point that the genomic revolution could represent for invasion science, and to celebrate the 50th anniversary of the Baker and Stebbins symposium, a meeting was held on the same Asilomar conference grounds 10 years ago (Barrett, 2015; Whitney & Gering, 2015). This meeting allowed contributors to consider the ‘known knowns, the known unknowns, and the unknown unknowns’ in the genetics and genomics of invasions, as Loren H. Rieseberg noted in his closing remarks (Whitney & Gering, 2015).

Here, we build on these conclusions, consider lessons that have been learned from invasion genomics over the past decade, and highlight new problems in our understanding of how invasive species evolve. In doing so, we rely predominantly on studies of invasive plants, in keeping with the important role of botanical research for advancing the field. We depart from recent reviews on invasion genomics by covering drivers of invasion that are known to occur in both animals and plants such as hybridisation or adaptation, as well as those that have predominantly been the focus of botanical research such as whole-genome duplication. In addition, we do not cover at length technological and analytical approaches to the genomic study of invasion biology. For in-depth treatments on this topic, we direct the reader to North *et al.* (2021) or McGaughran *et al.* (2024). Rather, we focus specifically on what genomic datasets have taught us about consequences of invasions including bottlenecks and the accumulation of deleterious mutations, as well as causes of invasions including adaptation, polyploidisation, or phenotypic plasticity.

II. Invasion history

Population genomic data, combined with advances in analytical tools, are revolutionising our ability to characterise the demographic processes underlying invasions, including bottlenecks, founder effects, multiple introductions, and hybridisation. Particularly when paired with high-quality reference genomes, these approaches provide unprecedented resolution into these changes over contemporary timescales (Fig. 1; Braasch *et al.*, 2019; Nadachowska-Brzyska *et al.*, 2022). Such genomic resources also enable the assessment of how recent demographic shifts impact both the frequency of deleterious mutations and adaptive genetic variation, offering valuable insights into the evolutionary dynamics of invasion, which we discuss in detail below.

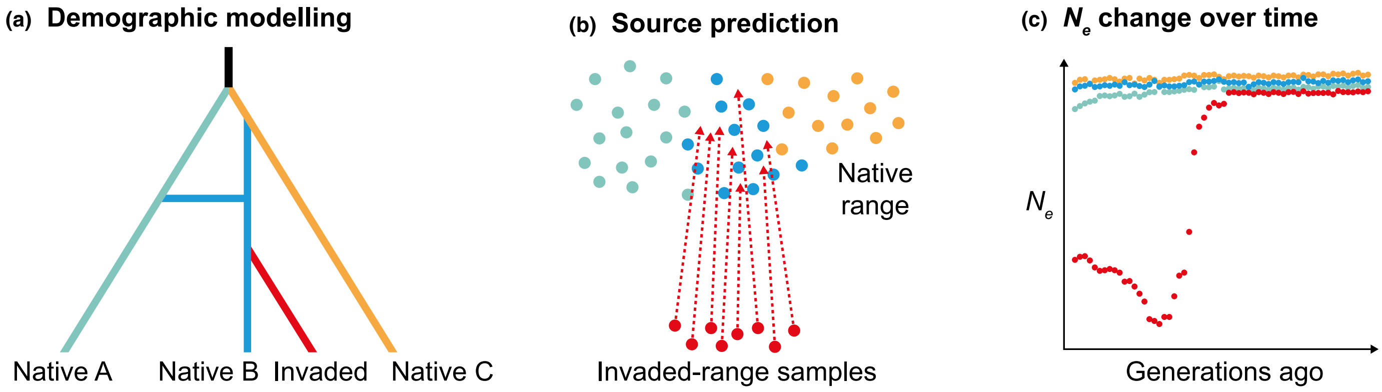


Fig. 1 Genomic strategies for inferring demographic histories of invaders. (a) Demographic modelling (e.g. *DIY-ABC* (Collin *et al*., 2021); *fastsimcoal* (Excoffier *et al*., 2021); *TreeMix* (Pickrell & Pritchard, 2012)) can be used with genomic data to reconstruct the emergence of population structure in the native range, identify admixture events, and trace the ancestral lineage(s) of invaders. (b) Likely source locations of invaders at the individual sample level can be identified using a model of genomic variation across space in the native range (e.g. *locator*; Battey *et al*., 2020). (c) Shifts in effective population size over recent generations can be modelled from genomic patterns of linkage disequilibrium (e.g. *GONE*; Novo *et al*., 2023), providing insights into recent demographic events associated with invasion.

1. Genetic bottlenecks

Bottlenecks associated with colonisation are expected to reduce the effective population size (*Ne*) of introduced populations and deplete genetic variation (Nei *et al*., 1975). In theory, this can limit the capacity of populations to adapt to environmental change, while increasing inbreeding and the frequency of deleterious alleles. These factors can reduce population fitness through maladaptation (Gomulkiewicz & Holt, 1995), inbreeding depression (Charlesworth & Willis, 2009), and drift load (Whitlock & Davis, 2011), which can depress population growth. The success of invaders in the face of these challenges was thought to represent a 'genetic paradox' (Baker & Stebbins, 1965; Allendorf & Lundquist, 2003). As a result, understanding the duration and extent of population bottlenecks and their impact on genetic diversity has been of great interest to invasion biologists.

Genomic analysis now provides detailed insights into changes in *Ne* over timescales relevant to invasions (for review, see Nadachowska-Brzyska *et al*., 2022). However, the application of these methods to invasive species remains relatively rare (Braasch *et al*., 2019). One example is the noxious herbaceous perennial weed *Hypochaeris radicata*, whose invasion history was reconstructed using ddRADseq data and coalescent-based simulations (Lee *et al*., 2024). This study revealed multiple bottlenecked introductions followed by subsequent population expansions, along with a weak relationship between *Ne* and expected heterozygosity across populations, indicating that demographic bottlenecks had limited effects on genome-wide genetic diversity. These findings are consistent with previous research demonstrating that plant invaders typically experience only minor genetic diversity losses, as revealed by both molecular markers (Dlugosch & Parker, 2008; Uller & Leimu, 2011; He *et al*., 2024), and genomic analyses (van Boheemen *et al*., 2017; He *et al*., 2024). This can be partly attributed to the limited impact of genetic drift in rapidly expanding populations following a bottleneck (Nei *et al*., 1975) – a pattern likely to be common among invasive species.

Beyond the tenuous relationship between transient demographic bottlenecks and genetic diversity at neutral loci, questions persist about the effects of invasion bottlenecks on adaptive genetic diversity–particularly given the weak correlation between adaptive and neutral measures of diversity (Reed & Frankham, 2001). In fact, polygenic traits are expected to be buffered from the effects of bottlenecks compared with neutral markers (Barton & Charlesworth, 1984; Dlugosch *et al*., 2015), and additive genetic variation may even increase following a bottleneck through the conversion of epistatic variation (Lee, 2002). In some cases, low genetic variation in the introduced range could be the consequence of adaptation, as selection itself can reduce *Ne* (Estoup *et al*., 2016). Population and quantitative genomic approaches are providing insight into the adaptive component of invader's genomes (see Section III). When combined with estimates of recent *Ne* trajectories (Nadachowska-Brzyska *et al*., 2022), such studies are enhancing our understanding of how the extent and duration of bottlenecks across different invasions may impact adaptive genetic diversity. In the self-incompatible annual weed *Ambrosia artemisiifolia*, reconstructions of *Ne* over recent timescales identified a prolonged 416-fold reduction in *Ne* during the invasion of Australia (Battlay *et al*., 2024a). Despite this substantial demographic bottleneck, signatures of climate adaptation were evident at the phenotypic and genetic level (van Boheemen *et al*., 2019; van Boheemen & Hodgins, 2020; Battlay *et al*., 2024a). Although such studies are presently rare, we anticipate that genomic analysis of both successful and failed invasions will reveal how frequently and under what conditions bottlenecks may constrain invasion through their effects on adaptive genetic variation (Boxes 1 and 2).

Despite questions pertaining to the validity of the genetic paradox of invasions, there are many instances where invasions still appear paradoxical because of the severity of the genetic bottleneck. For example, some well-characterised invasions are formed predominantly from a single clone (Huang *et al*., 2024). Clonality is a trait that is over-represented on the world's worst invader's list, with 81% of invasive plants being capable of clonal reproduction,

Box 1. Failed and stalled invasions

Most population genomic studies of invasive species focus on those that are successful, limiting our understanding of the genetic factors – such as bottlenecks and maladapted source populations–that might hinder invasions. This survivorship bias skews perception of the factors that influence invasion success or failure (Zenni & Nuñez, 2013). Consequently, genomic analyses of introductions in their putative lag phase, which is the time between a species' arrival and its population expansion to invasive status, or those that have failed to spread and are now locally extinct (e.g. using herbarium samples; Box 2) could provide valuable insights (Kim *et al*., 2023). For instance, *Solidago altissima* (late goldenrod) is native to North America and has highly invasive populations in Japan. In Australia, however, populations are noninvasive, despite the climatic and ecological suitability of the region. Genomic data combined with crossing experiments revealed that only two genetically distinct clones are common in Australia. Since this species is self-incompatible, this genetic bottleneck has led to an almost complete absence of sexual reproduction in Australia, resulting in *S. altissima's* inability to spread through long distance seed dispersal. By contrast, the introduction of multiple *S. altissima* genotypes to Japan facilitated invasive spread through seed dispersal (Uesugi *et al*., 2020; Fig. B1). How frequently invasions are constrained by such bottlenecks is not well-understood because of the focus of genomic studies on successful invasions.

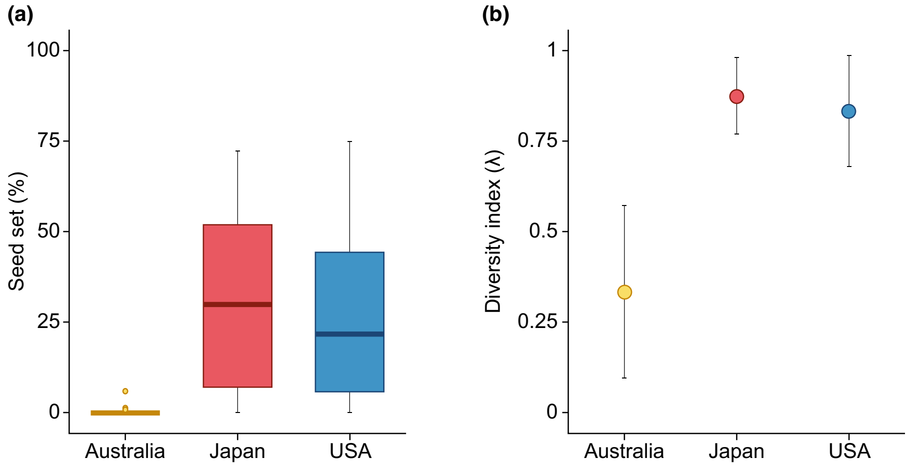


Fig. B1 Low genetic diversity limits seed set of *Solidago altissima* in Australia. (a) Percentage seed set in the field for its native range (USA), and invaded ranges in Australia and Japan. Box plots show the median, upper and lower quartiles, and extremes (within 1.5 × the interquartile range). Dots represent outliers outside the extremes. (b) Simpson's diversity index for multilocus genotypes (confidence intervals calculated using 1000 bootstraps) in each range. After (Uesugi *et al*., 2020).

25% reproducing clonally almost exclusively in their introduced range (Mounger *et al.*, 2021). Uniparental reproduction is advantageous during colonisation due to the reproductive assurance it provides (Baker, 1955). Baker (1965) proposed that such weeds exhibited a 'jack-of-all-trades-master-of-none' strategy arising from 'general purpose' genotypes, where high phenotypic plasticity and broad environmental tolerance were key to their success. Yet even in such species, there can be evidence for adaptation (e.g. *Oduor et al.*, 2016). Fixed heterosis (Ellstrand & Schierenbeck, 2000), epigenetic variation (Mounger *et al.*, 2021), *de novo* mutations including copy number variation (Dlugosch *et al.*, 2015; Exposito-Alonso *et al.*, 2018), and transposable element-derived variation (Stapley *et al.*, 2015) have all been theorised to play an important role in generating beneficial variation following genetically depauperate introductions. Large population sizes common to many invaders could further contribute to adaptation via *de novo* mutations (Colautti & Lau, 2015; Dlugosch *et al.*, 2015). Theoretical studies suggest that mutation and recombination can evolve in populations experiencing range expansion (Cobben *et al.*, 2017; Eriksson & Rafajlović, 2021). Further, even small amounts of sexual reproduction or outcrossing can be advantageous (reviewed in Hartfield, 2016). However, the contributions of these genomic changes in invasions spawned from severe genetic bottlenecks remain poorly understood, as these cases are often excluded from evolutionary studies (but see Exposito-*Alonso et al.*, 2018; Van-Wallendael *et al.*, 2021).

Beyond limiting genetic variation, bottlenecks can also contribute to reduced fitness through deleterious mutations. The genome-wide load of deleterious mutations is sensitive to effective population size as the latter influences the efficacy of selection (Whitlock & Davis, 2011). Bottlenecks can also result in elevated inbreeding, which can reduce fitness through the expression of recessive deleterious alleles (Charlesworth & Willis, 2009). In self-compatible species, higher rates of self-fertilisation that might alleviate Allee effects during colonisation could further increase inbreeding and expressed genetic load (Pannell, 2015). Alternatively, higher rates of inbreeding can also contribute to purging of recessive deleterious alleles (Marchini *et al.*, 2016). Contemporary methods have been developed to estimate genetic load using whole-genome sequence data without relying on direct fitness measurements (e.g. Simons & Sella, 2016). However, these methods have rarely been applied to invasive plants, and the few studies that have done so have discovered no impact of invasion on

Box 2. Invasion museomics

Emboldened by falling sequencing costs and improvements in historical DNA techniques, invasion-genomics research has turned to museums and herbaria where thousands of temporally resolved samples record many of the invasions that have occurred over the last few centuries (Soltis, 2017; Kim *et al*., 2023). Historic samples can now be mined for genomic data, and temporally resolved population genomic datasets spanning native and invaded ranges are now tractable (e.g. Bieker *et al*., 2022; Kreiner *et al*., 2022b).

Invasions are often characterised by complex demographic histories, which can be challenging to infer using modern genomes alone. For example in *A. artemisiifolia*, analysis of hundreds of historic genomes revealed the contribution of admixture in the native range and shifts in population structure in the invaded range to the species' invasion of Europe (Bieker *et al*., 2022). Invasion-genomic studies focus almost exclusively on successful invasions, but herbarium samples allow the study of invasions that have failed (Kim *et al*., 2023). Furthermore, the abundance of deleterious alleles estimated across the time course of an invasion could provide valuable insight into the role of genetic load in invasion success or failure.

Temporally resolved population genomic datasets of invasive species facilitate direct observation of allele frequency shifts over time, which can provide strong evidence for rapid adaptation and furthermore allow estimation of selection coefficients for adaptation candidates (e.g. Kreiner *et al*., 2022b; Battlay *et al*., 2023). The temporal scale of museomic data is far greater than the spans of experimental evolution studies in plants, empowering researchers to quantify responses to weaker selection coefficients. Temporal studies also shed light on the source of adaptive variation, and have emphasised the importance of both *de novo* (Exposito-Alonso *et al*., 2018) and standing (Battlay *et al*., 2023) genetic variation in invasions. In addition to DNA from the specimen itself, herbarium samples contain DNA from associated microbes (Bieker *et al*., 2020), allowing the role of microbes in invasions to be investigated on a temporal scale. Identifying DNA from the natural microbial community, however, is not straightforward, as herbarium samples are prone to microbial contamination (Bieker *et al*., 2020).

The fragmented nature of historic DNA limits herbarium genomics to short-read sequencing, which complicates the identification of structural variants in historic samples. Recent work has, however, demonstrated the utility of short-read approaches in identifying inversions (Battlay *et al*., 2023) and copy number variants (Wilson *et al*., 2024) in historic genomes of invaders. Invasive species pangenomics promises to enhance this area, allowing the imputation of structural variants from the modern pangenome across historic short-read genome sequences. Similarly, genome size is typically measured using flow cytometry, which works best with fresh tissue (Dolezel *et al*., 2007). Thus, flow cytometry estimates of genome size cannot be easily obtained for herbarium specimens, limiting our ability to use temporal contrasts for probing genome size evolution during invasions. However, genomic approaches using historic sequence data may facilitate estimates of genome size across much larger time frames, potentially spanning several centuries. Tools that can be used to estimate genome size based on short-read data are starting to become available even for low coverage datasets (e.g. Guenzi-Tiberi *et al*., 2024), potentially allowing genome size estimates to be obtained for individuals representative of early invasion stages.

estimates of deleterious allele frequencies (Hodgins *et al*., 2015; Gamba *et al*., 2024; Battlay *et al*., 2024a), which is in contrast to other groups such as crops (Makino *et al*., 2018).

During range expansion, genetic load can accumulate at the leading edge of the wave due to the surfing of deleterious variants at expanding range fronts (Edmonds *et al*., 2004). This form of genetic load, termed expansion load (Peischl *et al*., 2013), can be weakened by factors that increase *Ne* at the range edge, such as those that slow the expansion down, including Allele effects or local adaptation (Gilbert *et al*., 2017). Given that many plant invaders have experienced rapid and recent range expansion, and genetic depletion is prevalent at the leading edge of invasions (Mullarkey *et al*., 2013; Braasch *et al*., 2019), signals of expansion load might be expected in some cases. However, most cases of expansion load identified using genomic data have been identified in older range expansions, such as those occurring post glaciation (e.g. Zeitler *et al*., 2023).

2. Introduction source

Identifying the most likely sources of invaders provides valuable information for directing management practices, enabling more specific and targeted strategies (Buckley & Catford, 2016). It can also facilitate meaningful comparisons between native and introduced populations in ecological and evolutionary studies (Hodgins *et al*., 2018). Many invasive plant populations exhibit traits associated with invasiveness, such as vigorous growth and enhanced reproduction, as well as shifts in defence-related traits compared with native populations in common garden studies (Felker-Quinn *et al*., 2013; Gruntman & Segev, 2024). Similarly, the evolution of trait clines is a feature of some widespread invaders (Colautti *et al*., 2009; Colautti & Barrett, 2013; van Boheemen & Hodgins, 2020). A goal of many evolutionary studies is to determine whether such trait differentiation occurred as a result of post-invasion evolution or whether it was caused by sampling pre-existing variation in the native range, therefore determining the source(s) of invasion is critical (Hodgins *et al*., 2018).

Even with high-resolution genomic data and advanced methods of analysis, the source of an invasion can be challenging to identify. Source populations may be extinct or remain undetected, despite extensive sampling efforts (Cristescu, 2015). This is complicated by the fact that many invasions are spawned from other invasions, result from multiple introductions, undergo extensive admixture, or bottlenecks (e.g. van Boheemen *et al*., 2017; Bieker *et al*., 2022). The integration of herbarium samples into population genomic analysis can be key in understanding demographic changes associated with such complex plant invasions (Bieker *et al*., 2022; Kim *et al*., 2023). For instance, in *A. artemisiifolia*, temporal genomic data helped to reconstruct early stages of invasion, which were obscured by later introductions (Bieker *et al*., 2022; Kim *et al*., 2023; Box 2).

The identification of invasion source populations within the native range allows the comparison of native and invaded environments. Species are more likely to invade ranges that share environmental characteristics with their native range (Dlugosch & Parker, 2007; Hamilton *et al*., 2015). Examples of 'pre-adaptation' are prevalent in invasive species (Fig. 2) and may, in some cases, explain the genetic paradox of invasion (Estoup *et al*., 2016).

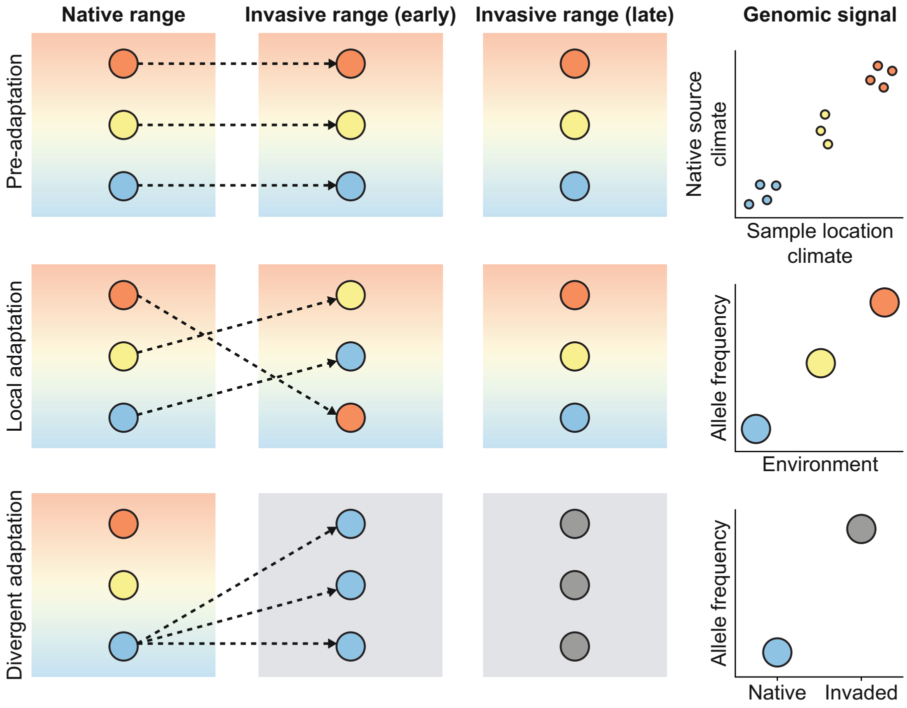


Fig. 2 Genomic footprints of adaptation during range expansion. Circle colours represent populations adapted to a particular environmental optimum. Background colours represent the environmental optimum. Populations inhabiting environments that are similar to those at their native source provides evidence that pre-adaptation has facilitated invasion. When similar environmental variation exists between native and invaded ranges, a haphazardly introduced invader may re-evolve trait clines present in the native range, resulting in parallel patterns of genotype–environmental associations in the native and invaded ranges. Alternatively, when selective pressures in the invaded range are different to those in the native range, adaptation may occur to divergent optima, resulting in loci highly differentiated between ranges.

Correspondingly, invasion success across vast environmental space has been linked with multiple, diverse introductions from the native range (e.g. Bieker *et al.*, 2022), and habitat similarities between native and invaded ranges are becoming increasingly likely through the anthropogenic homogenisation of environments (Hufbauer *et al.*, 2012). Furthermore, recent genomic analyses of invaders have demonstrated the importance of spatially (Battlay *et al.*, 2023; Gamba *et al.*, 2024) and temporally (Stern & Lee, 2020) varying selection across the native range in the maintenance of alleles suited to diverse invaded ranges.

Evidence is growing that the genetic composition of source populations likely has a profound impact on invasion success and subsequent range expansion. Genomically informed species distribution models that leverage differences in habitat preference of genetically distinct source populations are now being used to predict future spread of invaders (e.g. Hudson *et al.*, 2021; Putra *et al.*, 2024). For instance, in *A. artemisiifolia*, genetic clusters from the native range with the greater niche overlap with the current Australian distribution were more prevalent in the Australian invasion, consistent with a pre-adaptation hypothesis. However, there have been few studies that have integrated genomic information, particularly putatively adaptive variation, into predictions of species' spread due to the paucity of genomic data and the corresponding absence of tools. To resolve this gap, more recent approaches have been developed to predict the likelihood of establishment based on matching between the adaptive genetic composition of potential source populations and the climates of the introduced range (i.e. genetic offset; Camus *et al.*, 2024). A recent application of this approach in the selfing annual grass *Bromus tectorum* (cheatgrass) demonstrates geographic variation in genetic offset across the species' introduced range, which is negatively correlated with the invader's abundance (Gamba *et al.*, 2024).

3. Intraspecific admixture and interspecific hybridisation

Hybridisation has long been recognised as a feature of plant invasions (Baker, 1948; Panetsos & Baker, 1967; Ellstrand & Schierenbeck, 2000; Uller & Leimu, 2011; Barker *et al.*, 2019) and is a frequently cited resolution to the genetic paradox of invasion (Estoup *et al.*, 2016). Higher resolution genomic data and improved methods have enhanced our capacity to detect intra- and interspecific gene flow in introduced plants (e.g. Rosinger *et al.*, 2021; Bieker *et al.*, 2022). However a central question in invasion genomics has become how frequently such mixing contributes to invasion and at which points in the invasion process (Bock *et al.*, 2015). While the benefits of admixture and hybridisation for invasive species are numerous – including heterosis, demographic rescue, genetic rescue, and evolutionary rescue (Hodgins *et al.*, 2018) – inter- and intraspecific gene flow might simply appear to be associated with invasion because of increased opportunities caused by the movement of divergent genotypes or species. Further, Allee effects during population establishment may also favour hybridisation–particularly for self-incompatible species–for demographic reasons even without direct benefits derived from lineages mixing (Mesgaran *et al.*, 2016). However, a meta-analysis of studies comparing invasive hybrids to their parents has provided support for the benefits of hybridisation during invasion. Hybrids were found to have greater fecundity relative to parental species that improved with each successive generation, indicating that natural selection significantly influences hybrid performance, and therefore invasiveness, over time (Hovick & Whitney, 2014).

Crossing experiments in various plant invaders have shown that admixture can be particularly advantageous in early-generation hybrids due to heterosis effects (e.g. Mullarkey *et al.*, 2013; Hahn &

Rieseberg, 2017; Li *et al.*, 2018; Barker *et al.*, 2019). Moreover, theory suggests that the accumulation of genetic load during range expansion can lead to heterosis upon secondary contact (MacPherson *et al.*, 2022), potentially overcoming incompatibilities between species. The addition of genomic analysis when combined with other lines of evidence is demonstrating the importance of heterosis for invasion success. In the invasive sunflower *Helianthus tuberosus*, genomic analysis has revealed that heterosis leads to the production of a larger number of asexual propagules, which is an important trait contributing to invasions of this species (Bock *et al.*, 2018).

Evolutionary studies of hybrids have progressed beyond merely documenting hybridisation, focussing instead on genome-wide ancestry patterns and the processes shaping them (Burgarella *et al.*, 2019; Liu *et al.*, 2022; McGaughran *et al.*, 2024). Unfortunately, such studies remain rare for invasive plants despite the link between hybridisation and invasion. Corre *et al.* (2020) examined introgression in introduced annual teosinte (*Zea mays* ssp. *mexicana*) in Europe. This species is an agricultural weed and a close relative of maize. The study demonstrated maladaptive delayed flowering of source populations compared with introduced teosinte in Europe. Local ancestry analysis along the chromosome revealed that introgression from temperate maize at the *ZCN8* gene, a key regulator of flowering and photoperiod sensitivity, was a key driver of the flowering time shift. Additionally, introgression signals from herbicide-resistant maize cultivars were detected. While adaptive introgression in invasive plants remains relatively understudied (but see Whitney *et al.*, 2006), increasing genomic data sets are likely to uncover many more examples.

Although hybridisation can have fitness benefits during the invasion process, inter- and intraspecific gene flow can also come with substantial fitness costs, and it is an open question as to how frequently this might limit invasions by contributing to extinction of colonising populations (Todesco *et al.*, 2016). The mechanisms causing lower fitness in hybrids can be maladaptation (ecological selection), differences in the number of deleterious variants between species (hybridisation load), or negative interactions between genomic regions derived from the two parents (hybrid incompatibilities; Moran *et al.*, 2021). In invasive *Centaurea solstitialis*, interpopulation crosses resulted in heterosis, but fitness benefits declined and were reversed when the source populations were highly genomically divergent, which is consistent with outbreeding depression caused by incompatibilities (Barker *et al.*, 2019). Across species, intermediate levels of divergence within the native range were correlated with a greater likelihood of admixture in the introduced range, suggesting that genetic incompatibilities may constrain admixture as genetic distances increase (Barker *et al.*, 2019). Still, ecological selection against hybrids is likely to be less common in the introduced range compared with the native range due to the disruption of local adaptation (Verhoeven *et al.*, 2011). Extreme traits produced because of hybridisation (i.e. transgressive segregation) might even aid in colonising novel environments of the introduced range (Ellstrand & Schierenbeck, 2000). Consequently, high levels of hybridisation during early invasion stages, where local maladaptation is most prevalent, might be expected.

Genome-wide analysis of hybrids can reveal both adaptive introgression and its counterpart–negative selection against foreign genomic regions. Studies of ancestry patterns across multiple species have identified common genomic principles of hybridisation, including reduced introgression in functionally important regions of the genome (Brandvain *et al.*, 2014; Calfee *et al.*, 2021; Moran *et al.*, 2021). In cases of repeated hybridisation events between the same species, similar genetic interactions and selective pressures are expected to broadly drive repeated patterns of ancestry across the genome (Moran *et al.*, 2021). Invasive species present a compelling opportunity to test this prediction when hybridising taxa encounter one another during multiple independent invasions. However, selection across the genome can be context-dependent. For instance, in the case of hybridisation load, selection may act against ancestry from the parental species with lower *Ne*. Conversely, if deleterious alleles are primarily recessive, selection could favour foreign ancestry, creating patterns that mimic adaptive introgression (MacPherson *et al.*, 2022). Factors such as demographic history, shifts in mating systems, and environmental change during introductions are likely to influence *Ne* and patterns of selection across the genome in complex yet potentially predictable ways. Studies of hybridisation in invasive species offer an opportunity to unravel the dynamics of genome reorganisation following hybridisation, while also shedding light on the mechanisms through which hybridisation can contribute to invasion success.

III. Adaptation

Adaptation over short periods of time was once thought to be unlikely (Reznick *et al.*, 2019), yet experimental evidence in invasive species has demonstrated that adaptation to local environments occurs just as frequently in invasive as native plant species regardless of their reproductive system, life history, mating system, and time since introduction (Oduor *et al.*, 2016). When there are differences in adaptive optima between native and invaded ranges, there is the opportunity for adaptation to occur in introduced populations. Existing theory furnishes us with expectations about the genetic architecture underlying biological invasions. Orr (1998) modelling of adaptive walks demonstrated a role for large-effect mutations early in bouts of adaptation to large environmental shifts. Beneficial large-effect alleles are also more likely to be fixed in small populations compared with small-effect alleles due to drift (Charlesworth, 2009). Further, they are better able to resist the homogenising effects of gene flow and maintain local adaptation between populations connected by migration (Yeaman & Whitlock, 2011), which may aid the spread of invasive species across diverse environmental gradients. Conversely, genetic drift during founder events can reduce variance and shift the means of traits controlled by few large-effect variants (Dlugosch *et al.*, 2015); however, the interplay between forces shaping genetic architecture during invasion has not been directly examined. Invasive species are expected to utilise standing genetic variation over *de novo* mutations during bouts of rapid adaptation, due primarily to the former's immediate availability (Ralph & Coop, 2015). Furthermore, a bias towards standing variation and large-effect mutations is predicted to result in parallel adaptation (Ralph & Coop, 2015; MacPherson & Nuismer, 2017).

Empirical evidence of rapid adaptation in invasive species has classically been inferred from observations of parallel clines in the native and invaded ranges, suggesting rapid local adaptation in the invaded range (Colautti & Barrett, 2013; van Boheemen *et al.*, 2019). Previously restricted to phenotypic variation measured in common gardens or select genetic variants, population genomic data demonstrate that clinal patterns are observable on a genome-wide scale (Fig. 2), with an over-representation of climate adaptation candidates (i.e. loci with genomic signals of climate-mediated selection) shared between native and invaded ranges (van Boheemen & Hodgins, 2020; Battlay *et al.*, 2023, 2024b; Andrade *et al.*, 2024). These genomic signatures of parallel adaptation are not only observed between native and invaded ranges at the local scale but also between distinct invasions at the global scale (Olazcuaga *et al.*, 2020; Battlay *et al.*, 2024b). Furthermore, successful invasions of *A. artemisiifolia* and *Trifolium repens* across the globe in the last two centuries involved rapid shifts in large-effect standing variants from the native range (Battlay *et al.*, 2023, 2024b; Wilson *et al.*, 2024).

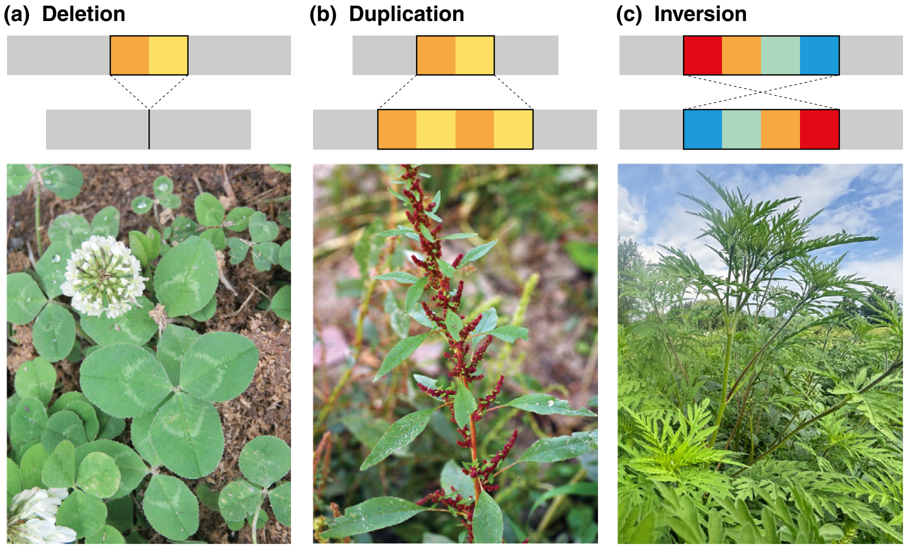
In addition to abiotic factors such as climate, biotic factors also impose selective pressures during invasions. Invasive species, free from pathogens and predators in their native range, may evolve to allocate resources from defence towards increasing competitive ability (The Evolution of Increased Competitive Ability hypothesis; Blossey & Notzold, 1995). Although empirical support for this hypothesis has been mixed (Felker-Quinn *et al.*, 2013; Hodgins *et al.*, 2018), it has been invoked to explain the results of recent population-genetic analyses of invaders. For example, inactivation of a herbivory defence pathway is associated with urbanisation in *T. repens* (Santangelo *et al.*, 2022), although the association is complicated by interactions with other biotic and abiotic factors. Similarly, Sun *et al.* (2022) described an interaction between herbivore resistance and temperature in *A. artemisiifolia* – increased temperatures ameliorate the cost of the weed's herbivory defence. At a genomic resolution, signatures of divergence between native and invaded ranges in *A. artemisiifolia* are enriched for defence genes, which is consistent with shifts in metagenomic signals of pathogens between ranges (Bieker *et al.*, 2022). Invasion genomic studies that include explicit tests for associations with biotic factors (and biotic–abiotic interactions) are important for advancing our understanding of the genetic basis of rapid adaptation, particularly given the conjecture that biotic and abiotic selective pressures may favour distinct genetic architectures (Whiteman, 2022).

As population genomic studies that identify signatures of selection in invasive plants become more common, broad conclusions should be drawn with caution. Genome scans for selection are biassed towards the detection of large-effect loci (Rockman, 2012), while neutral demographic processes such as bottlenecks may result in spurious signatures of selective sweeps (Harris *et al.*, 2018). Genome scans may also be confounded by the genetic structure of populations, particularly if range expansion is correlated with environmental variables (Booker *et al.*, 2023). Furthermore, variation in recombination rate across the genome affects the distributions of a range of selective signatures (Booker *et al.*, 2020). Solutions to these issues include statistical methods that correct for population structure (Gautier, 2015; Olazcuaga *et al.*, 2020), the use of simulations to determine distributions of genome scan results expected under neutrality (e.g. Gautier, 2015; Harris *et al.*, 2018), approaches that partition the roles of large-effect and polygenic background variation (e.g. Koch *et al.*, 2022), and the incorporation of field measurements of fitness (e.g. Battlay *et al.*, 2024b). Results of genome-wide scans for recent selection can also be supplemented by direct observation of allele frequency shifts over time by leveraging herbarium sequences (Box 2).

IV. Structural variation

Genomic structural variants – duplications, deletions, inversions, and translocations of genomic regions – have features that make them theoretically important to invasive species during range expansion. Structural variants have larger genomic footprints than single nucleotide polymorphisms and as such have larger effects on traits (Sudmant *et al.*, 2015). Structural variants that suppress recombination can collect alleles adapted to a particular environment resulting in 'cassettes' of locally adapted variation. For species invading across environmental gradients that are similar to their native range, standing structural variants are predicted to facilitate rapid local adaptation (Kirkpatrick & Barrett, 2015), and by maintaining it by withstanding the swamping effect of gene flow (Yeaman & Whitlock, 2011). However, when range expansion occurs into novel environments, structural variants are predicted to stymie adaptation by constraining optimal combinations of alleles (Roesti *et al.*, 2022). Nevertheless, structural variants have been known to play key roles in species' rapid and local adaptation for almost a century (Fig. 3). The first structural variants described were large chromosomal inversions in *Drosophila* species (Sturtevant, 1921), and variation in their frequencies across geographic space suggested an importance for adaptation (Dobzhansky, 1937). As many of these synanthropic fly species occupied recently colonised ranges, Dobzhansky (1965) also hypothesised that structural variants facilitated the range expansion of these 'animal weeds'. Latitudinal inversion allele frequency clines occur in parallel on multiple continents (Krimbas & Powell, 1992), providing strong evidence for their role in adaptation to local climate, while analysis of the introduction of *Drosophila subobscura* to the Americas demonstrated in real time the rapidity with which these parallel clines could establish in a new range (Prevosti *et al.*, 1988). While, similarly, early evidence of structural variation was available in plants (e.g. Creighton & McClintock, 1931), its role in plant adaptation was not demonstrated until the 21st century (Wellenreuther & Bernatchez, 2018; Wellenreuther *et al.*, 2019).

Historically, structural variants have not been routinely studied beyond model organisms because genetic variants with larger footprints are challenging to identify. As such, empirical evidence of the importance of structural variation to biological invasions is rare. However, discovery of genome-wide structural variants at the population level is becoming tractable in nonmodel systems because of progress in genome sequencing. Low-cost chromosome-level reference assemblies allow the mapping of large variants, while increasing accuracy and decreasing costs of both



Trifolium repens
Defense

Amaranthus tuberculatus
Flowering time

Ambrosia artemisiifolia
Flowering time; height

Fig. 3 Common forms of structural variation and examples of their involvement in adaptive traits in invasive plant species. (a) Deletion of genes involved in the cyanogenesis defence pathway is prevalent in urban populations of *Trifolium repens* (Santangelo *et al*., 2022). (b) In *Amaranthus tuberculatus*, duplications of an ATP synthase gene resulting in as many as 14 copies explain *c*. 20% of the variation in flowering time (Kreiner *et al*., 2023). (c) Inversions in *Ambrosia artemisiifolia* (photo by Nora Hand-Lennon, McGill University and Koffler Scientific Reserve) carry genetic variants affecting key locally adaptive traits such as the timing of flowering (Battlay *et al*., 2023).

short- and long-read technologies have led to an increase in the quality of population resequencing data. In the agricultural weed *Amaranthus tuberculatus*, copy number variation contributes to flowering time, a key locally adaptive trait in the species' native and invasive regions alike (Kreiner *et al*., 2023). Similarly, deletions at two loci underpin variation in a defence phenotype, which is involved in adaptive urban–rural divergence in *T. repens* (Santangelo *et al*., 2022). In *A. artemisiifolia*, large chromosomal inversions and copy number variants are associated with several locally adaptive traits. Sequencing of herbarium specimens of the species from throughout its invasion of Europe demonstrates that structural variant clines in Europe have evolved to parallel those in the native range in < 200 yr (Battlay *et al*., 2023; Wilson *et al*., 2024).

That biological invasions disproportionately draw on structural variants during adaptation is an attractive hypothesis, but empirical evidence supporting it is presently insufficient because few genomic studies of invaders attempt to detect structural variants. Significant insight into this question will be gained from pangenomic studies – which leverage reference genomes comprising *de novo* assemblies of multiple individuals and greatly enhance the detection of structural variants (Danilevicz *et al*., 2020).

V. Polyploidy

Polyploidy, or whole-genome duplication (WGD), occurs in both plants and animals, although it is far more prevalent in plants, where it represents an important mechanism of evolutionary diversification (Otto & Whitton, 2000). As the most dramatic mutation that organisms can sustain, WGD impacts long-term and short-term evolution (Wendel, 2015). Over the long term, processes unfolding over thousands or millions of years such as genome downsising and biassed fractionation (Wendel, 2015) can be a source of reproductive isolation that extends beyond genome doubling per se (Bock *et al*., 2023). In the short term, WGD can mediate morphological and physiological innovation via a range of genome-wide changes such as the remodelling of gene expression and epigenetic profiles, alterations in transposon activity, or by increased genetic variation (te Beest *et al*., 2012; Soltis *et al*., 2014). These rapidly unfolding processes can lead to ecological divergence (Soltis *et al*., 2014), and as such are key for understanding the link between polyploidy and invasion (te Beest *et al*., 2012).

Remarkably, the potential for WGD to mediate ecogeographical transgression was first reported in the 1940s (Soltis *et al*., 2014), thanks to the work of pioneering botanical researchers such as Clausen *et al*. (1945). However, these early studies were largely descriptive and did not receive much attention at the time, leaving no consensus on the potential role of polyploidy as an evolutionary pathway to invasion (Bock *et al*., 2015). While some contributors to the Baker and Stebbins symposium (Baker & Stebbins, 1965) saw the link as weak and indirect (Mulligan, 1965), others highlighted the advantages of polyploid colonisers (Ehrendorfer, 1965). Recent research has facilitated progress in this area via analyses of trait divergence between ploidy levels (e.g. Thébault *et al*., 2011), or by tests of the association between invasive status and ploidy. Pyšek *et al*. (2023), for example, compiled information on genome size and ploidy, as well as naturalisation and invasion success for 11 049 species. Analyses revealed that plants with larger genomes and higher ploidy levels are invasive in more geographical regions (Pyšek *et al*., 2023). In addition, plants that occur as both diploids and polyploids are naturalised more often than plants known only as diploids (Pyšek *et al*., 2023).

Genomic data can complement these analyses by enabling an understanding of the underlying genetic and evolutionary mechanisms. At the most basic level, genomics provides a means to rapidly determine the ploidy of thousands of individuals. Genomic methods can accommodate a range of sequencing technologies including target capture sequencing (Weiß *et al*., 2018), reduced-representation sequencing (Gompert & Mock, 2017), or whole-genome sequencing (Ranallo-Benavidez *et al.*, 2020). Within multiploidy systems, genome-scale data can then be used to establish the parentage of polyploids (Arnold *et al.*, 2015; Glover *et al.*, 2016). When combined with transcriptome sequencing, these analyses can reveal whether gene expression in the polyploid is additive or nonadditive relative to the progenitor species(s) (Yoo *et al.*, 2014). In turn, knowledge of gene expression evolution can help explain why WGD can spur more successful colonisers. For example, in the hybrid tetraploid *Arabidopsis kamchatika*, transcriptome data indicated that additive and nonadditive expression of alleles recruited from parental diploid species that are adapted to contrasting soil types may have enabled the colonisation of a broader range of habitats by the polyploid (Paape *et al.*, 2020).

Polyploidisation can also lead to large-scale mobilisation of transposable elements, either in a burst that occurs shortly after the merger of divergent genomes (e.g. Petit *et al.*, 2010), or more gradually in time, on account of relaxed purifying selection when WGD occurs without hybridisation (e.g. Baduel *et al.*, 2019). Here as well, genomic data have been key for understanding the tempo, scale, and effects of transposable element insertions (Baduel *et al.*, 2019). For example, Yew *et al.* (2023) demonstrated that a transposable element insertion caused a frameshift mutation, which led to the emergence of self-compatibility in *A. kamchatika*. Notably, self-compatibility is key for the establishment of new polyploid species, but it can also facilitate subsequent range expansion and invasions (Hodgins *et al.*, 2018).

Another consequence of WGD is an increase in genomic redundancy. Duplicated genes retained in plant lineages following one or more WGD events may contribute to ecological innovation over contemporary timescales (Soltis *et al.*, 2010). Recently developed analytical tools (e.g. Qi *et al.*, 2021; McKibben & Barker, 2023) enable the identification of WGD-derived gene duplicates and direct tests of this hypothesis. In *C. solstitialis*, two genomic regions that are enriched for ancient gene duplicates are also associated with size-related traits that evolved during the species' invasions. These results are consistent with invasions of *C. solstitialis* being facilitated–at least in part–by genomic redundancy generated via WGD (Reatini *et al.*, 2022).

Lastly, genomic data can illuminate how interactions among species can drive invasions, including via the transfer of adaptive alleles among ploidy levels (e.g. Arnold *et al.*, 2015; Kolář *et al.*, 2017; Baduel *et al.*, 2018; Schmickl & Yant, 2021). The possibility that such ongoing interploidy genetic exchange can have an adaptive value was first proposed by Stebbins (1956), who attributed the ecological success of the tetraploid *Dactylis glomerata* to gene flow from diploid lineages that are locally adapted to contrasting habitats (Schmickl & Yant, 2021). In this case, genetic exchange was hypothesised to be facilitated by sympatry between the widely distributed *D. glomerata* tetraploid and spatially restricted diploids. However, WGD can also lead to the breakdown of strong postzygotic barriers that occur between diploids and that are dosage-sensitive (e.g. Lafon-Placette *et al.*, 2017). In this way, a polyploid species can recruit highly divergent alleles from diploid lineages that are reproductively isolated even in sympatry. Collectively, these processes could provide an explanation for why mixed-ploidy systems are over-represented among naturalised species (Pyšek *et al.*, 2023). Indeed, population genomic analyses are starting to link such interploidy gene flow with the successful colonisation of challenging and highly disturbed habitats such as ruderal sites along railways (e.g. Baduel *et al.*, 2018), which are often dominated by invasive species (Pysek *et al.*, 1998).

Genomic data have been key for improving our understanding of how polyploidisation can drive invasions, highlighting mechanisms that occur shortly after WGD, or more gradually in time during the evolution of polyploids (Fig. 4). We anticipate that genomic studies of polyploid invasive species will continue to accelerate, particularly given the advantages afforded by long-read sequencing for assembling duplicated genomes (Kyriakidou *et al.*, 2018), as well as the rapid uptake of pangenomics in plant biology (Danilevicz *et al.*, 2020). These developments are needed, given that much of what we know about the genomics of invasive polyploids stems from a relatively small number of species. Aside from broadening the taxonomic scope of invasion genomics studies, these advances will also be important for determining how polyploidisation interacts with other genetic mechanisms that can drive invasions. This includes, for example, the extent to which WGD facilitates the more rapid accumulation of structural variants. In the plant genus *Cochlearia*, Hämälä *et al.* (2024) relied on pangenomics and sampling from the ancestral range of this group to demonstrate that polyploid genomes contain a higher proportion of structural variants. While most of these variants appeared to be deleterious and masked from selection due to the additional allelic copies present in polyploids, they were also enriched among climate adaptation candidates. Thus, deleterious

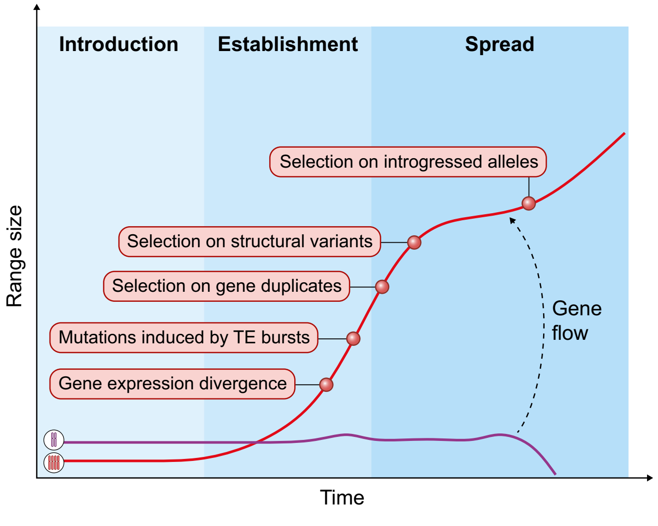


Fig. 4 Mechanisms by which polyploidisation can drive invasions, as identified using genomics. While gene expression divergence and mutations induced by transposable element (TE) accumulation may preadapt a polyploid lineage to become invasive and as such will likely act early on during invasions, variants induced by whole genome duplication or by interploidy gene flow will likely be selected once the polyploid lineage spreads across new environments. The polyploid lineage that becomes invasive is shown in red, while an introduced diploid lineage that eventually becomes locally extinct is shown in purple. For both lineages, range size is tracked over time, as the invasive polyploid lineages progresses through introduction, establishment, and spread.

structural variants that accumulate in polyploid genomes can subsequently be recruited by positive selection in response to environmental change. Whether these processes are even more important in invasive populations, which encounter novel biotic and abiotic selection pressures (Moran & Alexander, 2014), remains to be determined.

VI. Genome size variation

Independent of ploidy, genomes also differ in size due to processes that remove DNA such as segmental deletions or add DNA such as the proliferation of transposons (Petrov, 2001). Since the 1930s, studies have found correlations between genome size and latitude or altitude, which are consistent with an ecological significance of nuclear DNA content (Petrov, 2001). Recently, these results have been expanded to include correlations between genome size and surrogates of growth rate (e.g. cell production rate; Qiu *et al.*, 2019), which is likely under selection at extreme latitudes or altitudes. Overall, this research is consistent with the 'large genome constraint' hypothesis (Knight *et al.*, 2005), whereby large genomes place a lower bound on cell division times, translating to slower growth and reproduction at the organismal level. Thus, after accounting for differences in ploidy, larger genomes should be more permissible when growing seasons are longer, whereas smaller genomes should be favoured when faster growth and reproduction confer a fitness advantage (Qiu *et al.*, 2019).

Given that rapid growth and reproduction frequently translate to greater invasive spread (Hodgins *et al.*, 2018), it follows that invasive species should evolve towards smaller genomes within a ploidy level. Indeed, interspecific comparisons are broadly consistent with this expectation (te Beest *et al.*, 2012; Pyšek *et al.*, 2023). Additional support has recently been provided by in-depth investigations of model invasive plants, which were largely lacking a decade ago (Bock *et al.*, 2015). Cang *et al.* (2024), for example, focussed on *C. solstitialis*, and demonstrated that invasive populations with smaller genome sizes typically reproduce faster. Results further showed that, across the California invaded range of this species, populations evolved either towards larger genomes at the range edge (likely driven by genetic drift in marginal populations) or towards smaller genomes at high elevations (likely driven by selection for faster reproduction (Cang *et al.*, 2024).

Aside from quantifying genome size variation, genomic data can provide in-depth information on the underlying source of such variation, while also enabling contrasts between genome size and other drivers of phenotypic variation. In the agricultural weed *A. tuberculatus*, Kreiner *et al.* (2023) combined genome size estimates based on sequence data with repeat element characterisation and genome-wide associations for flowering time, a trait that has evolved during the colonisation of agricultural environments by this species (Kreiner *et al.*, 2022a). Results confirmed that individuals with smaller genomes and fewer repetitive elements had faster growth and flowered earlier. However, analyses also indicated that genome size explained only 2% of flowering time variation, as compared to copy number variation at a gene or polygenic effects, which explained 11 and 40% of variation in this trait, respectively. While these results indicate genome size may be a minor contributor to traits that can drive invasive spread, we emphasise that more work is needed from other traits and invasive species. These studies will ultimately enable general conclusions to be drawn on the relative importance of genome size variation for plant invasions.

VII. Phenotypic plasticity

Phenotypic plasticity, the property of genotypes to manifest different trait values depending on environment (Richards *et al.*, 2006), has frequently been proposed as a mechanism that can explain invasion success (Richards *et al.*, 2006; Davidson *et al.*, 2011; Hodgins *et al.*, 2018). This research dates back to 1965, when Herbert Baker first considered genotypes that can colonise multiple climatic and edaphic conditions (i.e. 'general purpose' genotypes; Baker, 1965). Since then, meta-analytical support for phenotypic plasticity as a driver of invasions has been mixed (e.g. Davidson *et al.*, 2011; Palacio-López & Gianoli, 2011). Davidson *et al.* (2011), for example, performed a meta-analysis based on 75 invasive/noninvasive species pairs, and demonstrated increased plasticity in invasive taxa in response to high-resource availability. However, higher plasticity did not frequently result in increased fitness in the invasive species, challenging the adaptive value of observed plastic responses.

Several caveats may influence these results, however, preventing an accurate interpretation of the role of plasticity during invasions. First, fitness proxies used in studies of plasticity are usually based on biomass, and only rarely consider reproductive output (Davidson *et al.*, 2011). Thus, at best, fitness is only partially captured. Second, in at least some cases, fitness proxies may be entirely incorrect, because they are measured under controlled conditions rather than under realistic field settings where processes such as competition operate (Davidson *et al.*, 2011). Third, meta-analyses of plasticity do not take into account the possibility that this characteristic can evolve during the invasion process (Hodgins *et al.*, 2018). Specifically, theoretical work supports a gradual transition from highly plastic genotypes early in the invasion, to those with constitutive trait expression at later invasion stages, especially when maintaining a plastic response is costly (Fig. 5; Lande, 2015; Hodgins *et al.*, 2018). Experimental work has started to validate these theoretical predictions, by uncovering genetic variation in plasticity in invasive populations (e.g. Hiatt & Flory, 2020), and by documenting evolution of increased plasticity in lineages representative of early invasion stages (Bock *et al.*, 2018).

Genomic data provide an alternative means for testing whether phenotypic plasticity in invasive populations is adaptive. When used in combination with large experimental populations as part of genome-wide association studies, genomics can detect loci involved in the control of phenotypic plasticity (e.g. Liu *et al.*, 2021; Fournier-Level *et al.*, 2022; Jin *et al.*, 2023). Plants are ideally suited for these experiments, because clonal propagation or selfing can generate replicates which, when grown under different environments, enable the quantification of plasticity and measurements of fitness across environments. Genome scans can then establish whether loci that control phenotypic plasticity are also under selection (e.g. Liu *et al.*, 2021). This approach, however, may be

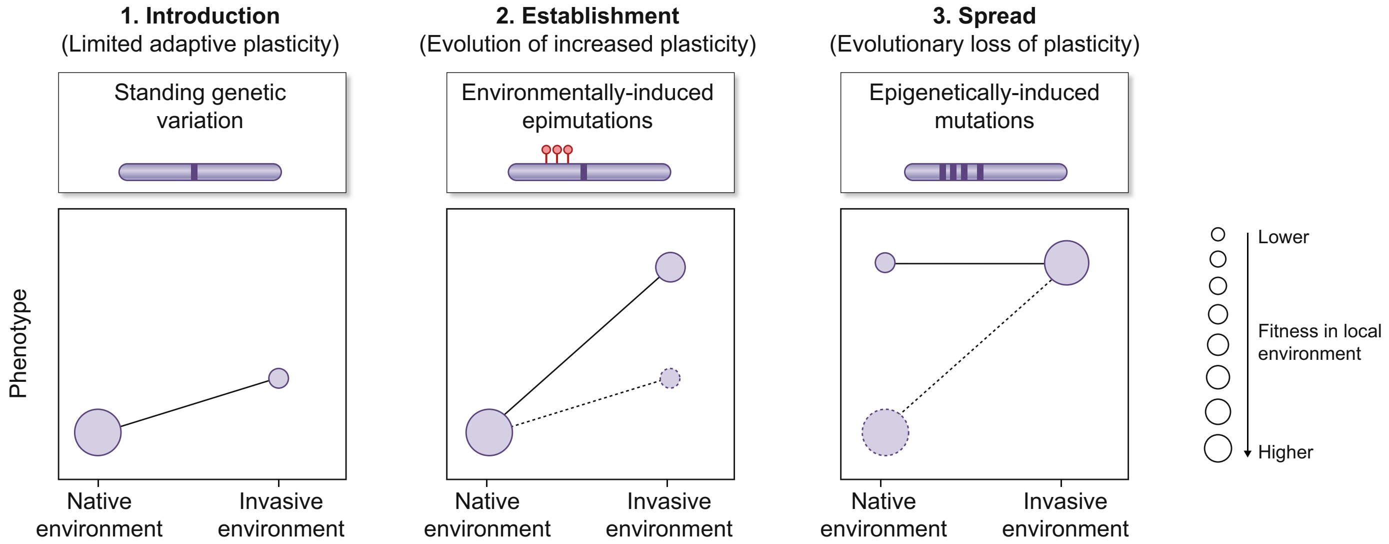


Fig. 5 Evolution of phenotypic plasticity during invasions. After the introduction of genotypes with a moderate level of adaptive plasticity, as conferred by standing genetic variants (Phase 1), selection acts on new heritable environmentally induced epigenetic variants that underpin increased plasticity during establishment (Phase 2). If there are costs to maintaining this plastic response, selection could then act on epigenetically induced mutations to favour loss of plasticity as the invasion progresses (Phase 3; see also Lande, 2015; Hodgins *et al*., 2018; Anastasiadi *et al*., 2021). In all three cases, the upper panels show hypothetical epi(genotypes) at a phenotypic plasticity locus along a chromosome, whereas the lower panels show changes in plasticity (i.e. shifts in the reaction norms of a phenotype under contrasting environments). Reaction norms characteristic of the preceding phase are illustrated with dashed lines.

difficult to implement in some cases, particularly if phenotypic plasticity proves to be highly polygenic (e.g. Liu *et al.*, 2021; Fournier-Level *et al.*, 2022; Jin *et al.*, 2023).

Aside from helping probe the adaptive value of phenotypic plasticity, genomics can also disentangle the mechanisms by which plasticity evolves during invasions. We anticipate that studies interrogating both genomes and epigenomes will be best positioned to make important breakthroughs in this direction. This is because while genome-wide genetic variants have been leveraged to identify molecular mechanisms that control plastic responses (e.g. Liu *et al.*, 2021; Fournier-Level *et al.*, 2022; Jin *et al.*, 2023), less is known about causal epigenetic variants. In *Arabidopsis thaliana*, for example, Zhang *et al.* (2013) surveyed 135 epigenetic recombinant inbred lines (epiRILs), which are genetically homogenous but differ in patterns of DNA methylation. Glasshouse comparisons among epiRILs revealed substantial differences in trait plasticity, as recorded at contrasting levels of water and nutrient availability (Zhang *et al.*, 2013). In this context, the continued development of cost-effective methods for genotyping epigenetic variants across the genome (e.g. Wang *et al.*, 2015) will facilitate rapid progress.

If epigenetic changes that lead to increased plasticity are stable and inherited across generations, they can be subject to selection and may contribute to the evolution of adaptive plasticity. We note, however, that current evidence supporting strictly epigenetic inheritance in adaptation is limited, and more research is needed (Laland *et al.*, 2014; Anastasiadi *et al.*, 2021). Importantly, changes in epigenetic variants such as spontaneous deamination of methylated cytosines can lead to DNA mutations, which may in turn underpin the transition from plastic genotypes to those that express the same trait value irrespective of environment (Fig. 5; Anastasiadi *et al.*, 2021). Thus, studies that combine genomic and epigenomic data can not only help reveal how phenotypic plasticity originates and is maintained but also how it is lost (Anastasiadi *et al.*, 2021).

In addition to information on how plasticity may change with time, we need a better understanding of the conditions under which plasticity is adaptive or maladaptive, and whether such conditions differ between native and invasive populations. In his now classical study on the characteristics of the ‘ideal weed’, Baker speculated that plastic genotypes would be favoured in weedy populations, where reduced competition relaxes the requirement for ‘exquisite’ adaptation to local conditions (Baker, 1965). An alternative nonmutually exclusive possibility is that increased plasticity evolves when invasive populations occupy locales with high environmental predictability – a condition that should favour plastic genotypes (e.g. Tufto, 2015), when plasticity is nonlabile (i.e. a cue experienced early in development determines the adult phenotype; Lande, 2015). Experimental evolution studies of phenotypic plasticity (e.g. Czesak *et al.*, 2006; Leung *et al.*, 2020) can test the effects of competition and environmental predictability. Ideally, such studies would also include the sequencing of genomes and epigenomes, enabling connections between (epi)genotype, phenotype, and fitness (Barrick & Lenski, 2013). While such studies would surely be challenging to complete because of the need to track genetic and epigenetic changes across generations and environments, they have the potential to clarify when, why, and how plastic genotypes are favoured in invasive species.

VIII. Conclusions

Invasive plants have emerged as powerful models for studying evolution in response to environmental shifts and sudden demographic changes. Features such as short generation times and suitability for common garden experiments can make them ideal for evolutionary research. Additionally, invasive species often experience both strong genetic drift and selection, allowing evolutionary changes to be tracked over contemporary timescales. The integration of genomics into invasion biology has enabled the foundational questions posed by Baker & Stebbins (1965) to be addressed, such as the impact of bottlenecks on genetic diversity or the influence of ploidy and plasticity on colonising ability, while also opening new avenues for exploration. Invasion genomics is providing extraordinary insights into the evolutionary dynamics of invasive species, but we are only at the beginning of these discoveries, making it an exciting time to study plant invasions through a genomic lens.

Genomic analysis is providing detailed insights into demographic shifts during invasions, including the extent and duration of colonisation bottlenecks, genetic sources of introductions, and levels of admixture, hybridisation and introgression. Importantly, the effects of these demographic changes on adaptive genetic variation can now be interrogated. To date, invasion bottlenecks appear to have limited impact on genetic diversity, including loci crucial for adaptation as well as deleterious variation (Hodgins *et al.*, 2015; van Boheemen *et al.*, 2019; van Boheemen & Hodgins, 2020; Gamba *et al.*, 2024; Battlay *et al.*, 2024a). However, current genomic studies that address these questions are limited to a few plant species and focus mainly on successful invaders. Studies of putative invasion lags indicate that the lack of critical genetic variation can hinder invasion (Box 1; Uesugi *et al.*, 2020). Furthermore, new research reveals that adaptive genomic composition matters for invasion success (Gamba *et al.*, 2024). Hybridisation is a catalyst for invasion (Baker, 1948; Panetsos & Baker, 1967; Ellstrand & Schierenbeck, 2000; Uller & Leimu, 2011; Barker *et al.*, 2019), yet its occurrence often remains hidden. Genomics has been pivotal in revealing these hidden events (e.g. Rosinger *et al.*, 2021; Bieker *et al.*, 2022) and assessing their adaptive value (Bock *et al.*, 2018; Corre *et al.*, 2020). These initial advances are beginning to uncover the genetic mechanisms by which hybridisation drives invasion success, providing a foundation for much needed future research.

Genomics has opened the door to understanding the genetic changes underlying adaptation during invasion. Despite short time spans since introduction in many cases, adaptation is common (Oduor *et al.*, 2016) and can even occur in invasions involving asexual species and those experiencing genetic bottlenecks (e.g. Oduor *et al.*, 2016; van Boheemen *et al.*, 2019). We now have a greater understanding of the genetic basis of adaptation during plant invasion where defence response genes, flowering time, and stress response genes are often over-represented among candidate genes (e.g. Bieker *et al.*, 2022; Battlay *et al.*, 2023). Further, we have made important progress in dissecting the genetic basis of traits involved in range expansion (e.g. Bock *et al.*, 2018; Corre *et al.*, 2020). However, despite some commonalities in the types of genes involved in adaptation, we lack a systematic analysis of the reuse of genes (i.e. parallelism and convergence) involved in adaptation during invasion within and across species (e.g. Nocchi *et al.*, 2024; Whiting *et al.*, 2024).

Genomic advances have renewed our ability to resolve large-scale structural changes associated with invasion. Structural variants appear to play an important role for adaptation during invasion in some species (Dobzhansky, 1937; Santangelo *et al.*, 2022; Battlay

Box 3. Outstanding questions in invasion genomics

We summarise key outstanding questions raised in this review that genomic data can help resolve.

Bottlenecks and founder effects

- Do bottlenecks frequently constrain invasion? Is variation at adaptive loci a better predictor of invasion than neutral markers?
- How does genetic load evolve during different phases of the invasion and for different types of invaders (e.g. different reproductive systems, admixed vs nonadmixed populations)? Can it constrain invasion?
- What are the genomic secrets of invasion success in species experiencing severe genetic bottlenecks?

Hybridisation and admixture

- How frequently does intraspecific admixture or interspecific hybridisation help or hinder invasion, and what are the primary mechanisms?
- How does the genome restructure post-hybridisation, and does invasion influence this process?

Adaptation

- Is adaptation during invasions predictable, and if so, at what levels of biological organisation (genetic variant; gene; pathway; trait)?
- What is the relative importance of pre-existing vs *de novo* mutation for adaptation for different types of plant invaders (e.g. different introduction histories, mating systems)?
- How do demographic changes experienced during range expansion impact the genetic architecture of adaptation (e.g. effect size) and our ability to detect loci involved in adaptation?

Structural variation

- Do large structural variants such as inversions favour or constrain invasions (and under what conditions)?

Polyploidy and genome size

- What are the mechanisms that drive the association of polyploidy and invasion (e.g. hybridisation vs whole-genome duplication in allopolyploids)?
- How does WGD interact with other genetic mechanisms influencing invasions such as hybridisation or structural variation?
- What is the relative importance of genome size in mediating invasiveness trait divergence, relative to other genetic drivers of trait change?

Plasticity

- What is the role of genetic vs epigenetic variants during the evolution of adaptive plasticity and what are the environmental drivers of these changes (e.g. high environmental predictability of relaxed competition)?

et al., 2023, 2024b; Wilson *et al.*, 2024), but we still do not know how frequently structural variants facilitate invasion and under what circumstances. We have evidence that standing variation and large-effect mutations contribute to rapid adaptation in several invasive plants and observe substantial parallelism (e.g. Battlay *et al.*, 2023). However, such studies are biassed towards identifying large-effect loci (Rockman, 2012). Empirical studies in animals have shown that loci under balancing selection due to fluctuating environments in the native range can contribute to range expansion (Stern & Lee, 2020) but this remains an open question in plants.

Polyploidy plays a crucial role in plant invasion (Pyšek *et al.*, 2023), with genomic studies revealing its extensive genome-wide effects, including the restructuring of gene expression and epigenetic patterns, altered transposon activity, and increased genetic variation (te Beest *et al.*, 2012; Soltis *et al.*, 2014). These effects may enhance the adaptive potential of polyploids, facilitating their colonisation and invasion. Moreover, WGD can lead to interploidy gene flow, allowing polyploid invaders to recruit adaptive alleles from diploid relatives (e.g. Arnold *et al.*, 2015; Kolář *et al.*, 2017; Baduel *et al.*, 2018; Schmickl & Yant, 2021), further enhancing their ecological success. Expanding the taxonomic diversity of these studies and employing advanced genomic tools will be essential to fully understand the role of polyploidy in plant invasions. Within ploidy levels, genome size also shapes invasion dynamics, as smaller genomes are associated with faster growth and reproduction, traits advantageous for colonisation (te Beest *et al.*, 2012; Pyšek *et al.*, 2023). However, early research suggests that genome size alone accounts for only a small proportion of variation in adaptive traits (Kreiner *et al.*, 2023), highlighting the need for future studies to disentangle its role.

Phenotypic plasticity has long been considered a potential driver of invasion success (Baker, 1965; Richards *et al.*, 2006; Davidson *et al.*, 2011; Hodgins *et al.*, 2018). But research on plasticity in invasive species has yielded mixed results (Davidson *et al.*, 2011; Palacio-López & Gianoli, 2011), with some studies showing increased plasticity but unclear adaptive benefits. Genomic approaches now provide a means to investigate whether plasticity in invasive populations is indeed adaptive by identifying loci associated with plastic responses and interrogating these regions for signals of selection. Additionally, emerging research on epigenetics highlights its role in controlling plastic responses (Anastasiadi *et al.*, 2021). However, further research is needed to understand whether epigenetic changes contribute to adaptive plasticity across generations.

The quest to define the traits of the 'ideal weed' through comparative approaches has been a cornerstone of invasion biology since Baker's seminal idea (Baker, 1965) and was a major theme of Baker & Stebbins (1965). Can we now extend this framework to identify genomic predictors of invasion success? By studying invasion lags, failed invasions, and comparing invasive species with their noninvasive relatives, we can gain a deeper understanding of the genomic factors that constrain or drive invasions. With the growing availability of chromosome-level genome assemblies and population genomic data sets for invasive plants, comparisons across diverse taxa are poised to answer the pressing questions outlined in Box 3, offering new insights into the genomic causes and consequences of plant invasions.

Acknowledgements

We apologise to all authors whose work we could not include in this review owing to space limitations. We would like to thank Daniel Ortiz-Barrientos, Holly Slater and Liam Dolan for suggesting that we write this review and three anonymous reviewers for their helpful comments. Support for this research was provided by Australia Research Council grants to KAH (DP220102362 and DP240102637).

Competing interests

None declared.

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References

Allendorf FW, Lundquist LL. 2003. Introduction: population biology, evolution, and control of invasive species. *Conservation Biology: The Journal of the Society for Conservation Biology* **17**: 24–30.

Anastasiadi D, Venney CJ, Bernatchez L, Wellenreuther M. 2021. Epigenetic inheritance and reproductive mode in plants and animals. *Trends in Ecology & Evolution* **36**: 1124–1140.

Andrade P, Alves JM, Pereira P, Rubin C-J, Silva E, Sprehn CG, Enbody E, Afonso S, Faria R, Zhang Y *et al.* 2024. Selection against domestication alleles in introduced rabbit populations. *Nature Ecology & Evolution* **8**: 1543–1555.

Arnold B, Kim S-T, Bomblies K. 2015. Single geographic origin of a widespread autotetraploid *Arabidopsis arenosa* lineage followed by interploidy admixture. *Molecular Biology and Evolution* **32**: 1382–1395.

Baduel P, Hunter B, Yeola S, Bomblies K. 2018. Genetic basis and evolution of rapid cycling in railway populations of tetraploid *Arabidopsis arenosa*. *PLoS Genetics* **14**: e1007510.

Baduel P, Quadrana L, Hunter B, Bomblies K, Colot V. 2019. Relaxed purifying selection in autopolyploids drives transposable element over-accumulation which provides variants for local adaptation. *Nature Communications* **10**: 5818.

Baker HG. 1948. Stages in invasion and replacement demonstrated by species of melandrium. *The Journal of Ecology* **36**: 96–119.

Baker HG. 1955. Self-compatibility and establishment after 'long-distance' dispersal. *Evolution; International Journal of Organic Evolution* **9**: 347–349.

Baker HG. 1965. Characteristics and modes of origin of weeds. In: Baker HG, Stebbins GL, eds. *The genetics of colonizing species*. New York, NY, USA: Academic Press, 147–168.

Baker HG, Stebbins GL. 1965. *Genetics of colonizing species*. New York, NY, USA: Academic Press.

Barker BS, Cocio JE, Anderson SR, Braasch JE, Cang FA, Gillette HD, Dlugosch KM. 2019. Potential limits to the benefits of admixture during biological invasion. *Molecular Ecology* **28**: 100–113.

Barrett SCH. 2015. Foundations of invasion genetics: the Baker and Stebbins legacy. *Molecular Ecology* **24**: 1927–1941.

Barrick JE, Lenski RE. 2013. Genome dynamics during experimental evolution. *Nature Reviews. Genetics* **14**: 827–839.

Barton NH, Charlesworth B. 1984. Genetic revolutions, founder effects, and speciation. *Annual Review of Ecology, Evolution, and Systematics* **15**: 133–164.

Battey CJ, Ralph PL, Kern AD. 2020. Predicting geographic location from genetic variation with deep neural networks. *eLife* **9**: e54507.

Battlay P, Craig S, Putra AR, Monro K, De Silva NP, Wilson J, Bieker VC, Kabir S, Shamaya N, van Boheemen L *et al.* 2024a. Rapid parallel adaptation in distinct invasions of *Ambrosia artemisiifolia* is driven by large-effect structural variants. *bioRxiv*. doi: 10.1101/2024.09.18.613801.

Battlay P, Hendrickson BT, Mendez-Reneau JI, Santangelo JS, Albano LJ, Wilson J, Caizergues AE, King N, Puentes A, Tudoran A *et al.* 2024b. Structural variants underlie parallel adaptation following global invasion. *bioRxiv*. doi: 10.1101/2024.07.09.602765.

Battlay P, Wilson J, Bieker VC, Lee C, Prapas D, Petersen B, Craig S, van Boheemen L, Scalone R, de Silva NP *et al.* 2023. Large haploblocks underlie rapid adaptation in the invasive weed *Ambrosia artemisiifolia*. *Nature Communications* 14: 1–15.

te Beest M, Le Roux JJ, Richardson DM, Brysting AK, Suda J, Kubesová M, Pysek P. 2012. The more the better? The role of polyploidy in facilitating plant invasions. *Annals of Botany* **109**: 19–45.

Bieker VC, Battlay P, Petersen B, Sun X, Wilson J, Brealey JC, Bretagnolle F, Nurkowski K, Lee C, Barreiro FS *et al.* 2022. Uncovering the genomic basis of an extraordinary plant invasion. *Science Advances* **8**: eabo5115.

Bieker VC, Sánchez Barreiro F, Rasmussen JA, Brunier M, Wales N, Martin MD. 2020. Metagenomic analysis of historical herbarium specimens reveals a postmortem microbial community. *Molecular Ecology Resources* **20**: 1206–1219.

Blossey B, Notzold R. 1995. Evolution of increased competitive ability in invasive nonindigenous plants: a hypothesis. *The Journal of ecology* **83**: 887–889.

Bock DG, Cai Z, Elphinstone C, González-Segovia E, Hirabayashi K, Huang K, Keais GL, Kim A, Owens GL, Rieseberg LH. 2023. Genomics of plant speciation. *Plant Communications* **4**: 100599.

Bock DG, Caseys C, Cousens RD, Hahn MA, Heredia SM, Hübner S, Turner KG, Whitney KD, Rieseberg LH. 2015. What we still don't know about invasion genetics. *Molecular Ecology* **24**: 2277–2297.

Bock DG, Kantar MB, Caseys C, Matthey-Doret R, Rieseberg LH. 2018. Evolution of invasiveness by genetic accommodation. *Nature Ecology & Evolution* **2**: 991–999.

van Boheemen LA, Atwater DZ, Hodgins KA. 2019. Rapid and repeated local adaptation to climate in an invasive plant. *New Phytologist* **222**: 614–627.

van Boheemen LA, Hodgins KA. 2020. Rapid repeatable phenotypic and genomic adaptation following multiple introductions. *Molecular Ecology* **29**: 4102–4117.

van Boheemen LA, Lombaert E, Nurkowski KA, Gauffre B, Rieseberg LH, Hodgins KA. 2017. Multiple introductions, admixture and bridgehead invasion characterize the introduction history of *Ambrosia artemisiifolia* in Europe and Australia. *Molecular Ecology* **26**: 5421–5434.

Booker TR, Yeaman S, Whiting JR, Whitlock MC. 2023. The WZA: a window-based method for characterizing genotype-environment associations. *Molecular Ecology Resources* **24**: e13768.

Booker TR, Yeaman S, Whitlock MC. 2020. Variation in recombination rate affects detection of outliers in genome scans under neutrality. *Molecular Ecology* **29**: 4274–4279.

Braasch J, Barker BS, Dlugosch KM. 2019. Expansion history and environmental suitability shape effective population size in a plant invasion. *Molecular Ecology* **28**: 2546–2558.

Brandvain Y, Kenney AM, Flagel L, Coop G, Sweigart AL. 2014. Speciation and introgression between *Mimulus nasutus* and *Mimulus guttatus*. *PLoS Genetics* **10**: e1004410.

Buckley YM, Catford J. 2016. Does the biogeographic origin of species matter? Ecological effects of native and non-native species and the use of origin to guide management. *The Journal of ecology* **104**: 4–17.

Burgarella C, Barnaud A, Kane NA, Jankowski F, Scarcelli N, Billot C, Vigouroux Y, Berthouly-Salazar C. 2019. Adaptive introgression: an untapped evolutionary mechanism for crop adaptation. *Frontiers in Plant Science* **10**: 4.

Calfee E, Gates D, Lorant A, Perkins MT, Coop G, Ross-Ibarra J. 2021. Selective sorting of ancestral introgression in maize and teosinte along an elevational cline. *PLoS Genetics* **17**: e1009810.

Camus L, Gautier M, Boitard S. 2024. Predicting species invasiveness with genomic data: is genomic offset related to establishment probability? *Evolutionary Applications* **17**: e13709.

Cang FA, Welles SR, Wong J, Ziaee M, Dlugosch KM. 2024. Genome size variation and evolution during invasive range expansion in an introduced plant. *Evolutionary Applications* **17**: e13624.

Charlesworth B. 2009. Effective population size and patterns of molecular evolution and variation. *Nature Reviews. Genetics* **10**: 195–205.

Charlesworth D, Willis JH. 2009. The genetics of inbreeding depression. *Nature Reviews Genetics* **10**: 783–796.

Clausen J, Keck D, Hiesey WM. 1945. *Experimental studies on the nature of species. II. Plant Evolution through amphidiploidy and autoploidy, with examples from the Madiinae.* Washington, DC, USA: Carnegie Institute of Washington Publications.

Cobben MMP, Mitesser O, Kubisch A. 2017. Evolving mutation rate advances the invasion speed of a sexual species. *BMC Evolutionary Biology* **17**: 150.

Colautti RI, Barrett SCH. 2013. Rapid adaptation to climate facilitates range expansion of an invasive plant. *Science* **342**: 364–366.

Colautti RI, Lau JA. 2015. Contemporary evolution during invasion: evidence for differentiation, natural selection, and local adaptation. *Molecular Ecology* **24**: 1999–2017.

Colautti RI, Maron JL, Barrett SCH. 2009. Common garden comparisons of native and introduced plant populations: latitudinal clines can obscure evolutionary inferences. *Evolutionary Applications* **2**: 187–199.

Collin F-D, Durif G, Raynal L, Lombaert E, Gautier M, Vitalis R, Marin J-M, Estoup A. 2021. Extending approximate Bayesian computation with supervised machine learning to infer demographic history from genetic polymorphisms using DIYABC Random Forest. *Molecular Ecology Resources* **21**: 2598–2613.

Corre VL, Siol M, Vigouroux Y, Tenaillon MI, Délye C. 2020. Adaptive introgression from maize has facilitated the establishment of teosinte as a noxious weed in Europe. *Proceedings of the National Academy of Sciences, USA* **117**: 25618–25627.

Crawley MJ. 1987. What makes a community invasible? In: Gray Mjcapje AJ, ed. *Colonization, succession and stability.* London, UK: Blackwell, 429–453.

Creighton HB, McClintock B. 1931. A correlation of cytological and genetical crossing-over in Zea Mays. *Proceedings of the National Academy of Sciences, USA* **17**: 492–497.

Cristescu ME. 2015. Genetic reconstructions of invasion history. *Molecular Ecology* **24**: 2212–2225.

Czesak ME, Fox CW, Wolf JB. 2006. Experimental evolution of phenotypic plasticity: how predictive are cross-environment genetic correlations? *The American Naturalist* **168**: 323–335.

Danilevicz MF, Tay Fernandez CG, Marsh JI, Bayer PE, Edwards D. 2020. Plant pangenomics: approaches, applications and advancements. *Current Opinion in Plant Biology* **54**: 18–25.

Davidson AM, Jennions M, Nicotra AB. 2011. Do invasive species show higher phenotypic plasticity than native species and, if so, is it adaptive? A meta-analysis: invasive species have higher phenotypic plasticity. *Ecology Letters* **14**: 419–431.

Dlugosch KM, Anderson SR, Braasch J, Cang FA, Gillette HD. 2015. The devil is in the details: genetic variation in introduced populations and its contributions to invasion. *Molecular Ecology* **24**: 2095–2111.

Dlugosch KM, Parker IM. 2007. Molecular and quantitative trait variation across the native range of the invasive species Hypericum canariense: evidence for ancient patterns of colonization via pre-adaptation? *Molecular Ecology* **16**: 4269–4283.

Dlugosch KM, Parker IM. 2008. Founding events in species invasions: genetic variation, adaptive evolution, and the role of multiple introductions. *Molecular Ecology* **17**: 431–449.

Dobzhansky T. 1937. *Genetics and the origin of species.* New York, NY, USA: Columbia University Press.

Dobzhansky T. 1965. 'Wild' and 'domestic' species of *Drosophila*. In: Baker HG, Stebbins GL, eds. *The genetics of colonizing species.* New York, NY, USA: Academic Press, 533–546.

Dolezel J, Greilhuber J, Suda J. 2007. Estimation of nuclear DNA content in plants using flow cytometry. *Nature Protocols* **2**: 2233–2244.

Edmonds CA, Lillie AS, Cavalli-Sforza LL. 2004. Mutations arising in the wave front of an expanding population. *Proceedings of the National Academy of Sciences, USA* **101**: 975–979.

Ehrendorfer F. 1965. Dispersal mechanisms, genetic systems, and colonizing abilities in some flowering plant families. In: Baker HGSG, ed. *The genetics of colonizing species.* New York, NY, USA: Academic Press, 331–351.

Ellis BK, Stanford JA, Goodman D, Stafford CP, Gustafson DL, Beauchamp DA, Chess DW, Craft JA, Deleray MA, Hansen BS. 2011. Long-term effects of a trophic cascade in a large lake ecosystem. *Proceedings of the National Academy of Sciences, USA* 108: 1070–1075.

Ellstrand NC, Schierenbeck KA. 2000. Hybridization as a stimulus for the evolution of invasiveness in plants? *Proceedings of the National Academy of Sciences, USA* 97: 7043–7050.

Elton CS. 1958. *The ecology of invasions by animals and plants*. London, UK: Methuen.

Eriksson M, Rafajlović M. 2021. The effect of the recombination rate between adaptive loci on the capacity of a population to expand its range. *The American Naturalist* 197: 526–542.

Estoup A, Ravigné V, Hufbauer R, Vitalis R, Gautier M, Facon B. 2016. Is there a genetic paradox of biological invasion? *Annual Review of Ecology, Evolution, and Systematics* 47: 51–72.

Excoffier L, Marchi N, Marques DA, Matthey-Doret R, Gouy A, Sousa VC. 2021. FASTSIMCOAL2: demographic inference under complex evolutionary scenarios. *Bioinformatics* 37: 4882–4885.

Exposito-Alonso M, Becker C, Schuenemann VJ, Reiter E, Setzer C, Slovak R, Brachi B, Hagmann J, Grimm DG, Chen J *et al.* 2018. The rate and potential relevance of new mutations in a colonizing plant lineage. *PLoS Genetics* 14: e1007155.

Felker-Quinn E, Schweitzer JA, Bailey JK. 2013. Meta-analysis reveals evolution in invasive plant species but little support for Evolution of Increased Competitive Ability (EICA). *Ecology and Evolution* 3: 739–751.

Fournier-Level A, Taylor MA, Paril JF, Martínez-Berdeja A, Stitzer MC, Cooper MD, Roe JL, Wilczek AM, Schmitt J. 2022. Adaptive significance of flowering time variation across natural seasonal environments in *Arabidopsis thaliana*. *New Phytologist* 234: 719–734.

Gamba D, Vahsen ML, Maxwell TM, Pirtel N, Romero S, Van Ee JJ, Penn A, Das A, Ben-Zeev R, Baughman O *et al.* 2024. Local adaptation to climate facilitates a global invasion. *bioRxiv*. doi: 10.1101/2024.09.12.612725.

Gautier M. 2015. Genome-wide scan for adaptive divergence and association with population-specific covariates. *Genetics* 201: 1555–1579.

Gilbert KJ, Sharp NP, Angert AL, Conte GL, Draghi JA, Guillaume F, Hargreaves AL, Matthey-Doret R, Whitlock MC. 2017. Local adaptation interacts with expansion load during range expansion: maladaptation reduces expansion load. *The American Naturalist* 189: 368–380.

Glover NM, Redestig H, Dessimoz C. 2016. Homoeologs: what are they and how do we infer them? *Trends in Plant Science* 21: 609–621.

Gompert Z, Mock KE. 2017. Detection of individual ploidy levels with genotyping-by-sequencing (GBS) analysis. *Molecular Ecology Resources* 17: 1156–1167.

Gomulkiewicz R, Holt RD. 1995. When does evolution by natural selection prevent extinction? *Evolution; International Journal of Organic Evolution* 49: 201–207.

Gruntman M, Segev U. 2024. Effect of residence time on trait evolution in invasive plants: review and meta-analysis. *NeoBiota* 91: 99–124.

Guenzi-Tiberi P, Istace B, Alsos IG, Coissac E, Lavergne S, Aury J-M, Denoeud F. 2024. LocoGSE, a sequence-based genome size estimator for plants. *Frontiers in Plant Science* 15: 1328966.

Hahn MA, Rieseberg LH. 2017. Genetic admixture and heterosis may enhance the invasiveness of common ragweed. *Evolutionary Applications* 10: 241–250.

Hämälä T, Moore C, Cowan L, Carlile M, Gopaulchan D, Brandrud MK, Birkeland S, Loose M, Kolář F, Koch MA *et al.* 2024. Impact of whole-genome duplications on structural variant evolution in Cochlearia. *Nature Communications* 15: 5377.

Hamilton JA, Okada M, Korves T, Schmitt J. 2015. The role of climate adaptation in colonization success in *Arabidopsis thaliana*. *Molecular Ecology* 24: 2253–2263.

Harris RB, Sackman A, Jensen JD. 2018. On the unfounded enthusiasm for soft selective sweeps II: Examining recent evidence from humans, flies, and viruses. *PLoS Genetics* 14: e1007859.

Hartfield M. 2016. Evolutionary genetic consequences of facultative sex and outcrossing. *Journal of Evolutionary Biology* 29: 5–22.

He Z-Z, Stotz GC, Liu X, Liu J-J, Wang Y-G, Yang J, Li L-F, Zhang W-J, Nan P, Song Z-P. 2024. A global synthesis of the patterns of genetic diversity in endangered and invasive plants. *Biological Conservation* 291: 110473.

Hiatt D, Flory SL. 2020. Populations of a widespread invader and co-occurring native species vary in phenotypic plasticity. *New Phytologist* 225: 584–594.

Hodgins KA, Bock DG, Hahn MA, Heredia SM, Turner KG, Rieseberg LH. 2015. Comparative genomics in the Asteraceae reveals little evidence for parallel evolutionary change in invasive taxa. *Molecular Ecology* 24: 2226–2240.

Hodgins KA, Bock DG, Rieseberg LH. 2018. Trait evolution in invasive species. *Annual Plant Reviews Online* 20: 459–496.

Hovick SM, Whitney KD. 2014. Hybridisation is associated with increased fecundity and size in invasive taxa: meta-analytic support for the hybridisation-invasion hypothesis. *Ecology Letters* 17: 1464–1477.

Huang Y, Guo L, Xie L, Shang N, Wu D, Ye C, Rudell EC, Okada K, Zhu Q-H, Song B-K *et al.* 2024. A reference genome of Commelinales provides insights into the commelinids evolution and global spread of water hyacinth (*Pontederia crassipes*). *GigaScience* 13: 1121.

Hudson J, Castilla JC, Teske PR, Beheregaray LB, Haigh ID, McQuaid CD, Rius M. 2021. Genomics-informed models reveal extensive stretches of coastline under threat by an ecologically dominant invasive species. *Proceedings of the National Academy of Sciences, USA* 118: 3210.

Hufbauer RA, Facon B, Ravigné V, Turgeon J, Foucaud J, Lee CE, Rey O, Estoup A. 2012. Anthropogenically induced adaptation to invade (AIAI): contemporary adaptation to human-altered habitats within the native range can promote invasions. *Evolutionary Applications* 5: 89–101.

Jin M, Liu H, Liu X, Guo T, Guo J, Yin Y, Ji Y, Li Z, Zhang J, Wang X *et al.* 2023. Complex genetic architecture underlying the plasticity of maize agronomic traits. *Plant Communications* 4: 100473.

Kim AS, Kreiner JM, Hernández F, Bock DG, Hodgins KA, Rieseberg LH. 2023. Temporal collections to study invasion biology. *Molecular Ecology* 32: 6729–6742.

Kirkpatrick M, Barrett B. 2015. Chromosome inversions, adaptive cassettes and the evolution of species' ranges. *Molecular Ecology* 24: 2046–2055.

Knight CA, Molinari NA, Petrov DA. 2005. The large genome constraint hypothesis: evolution, ecology and phenotype. *Annals of Botany* 95: 177–190.

Koch EL, Ravinet M, Westram AM, Johannesson K, Butlin RK. 2022. Genetic architecture of repeated phenotypic divergence in *Littorina saxatilis* ecotype evolution. *Evolution; International Journal of Organic Evolution* 76: 2332–2346.

Kolář F, Čertner M, Suda J, Schönswetter P, Husband BC. 2017. Mixed-ploidy species: progress and opportunities in polyploid research. *Trends in Plant Science* 22: 1041–1055.

Kreiner JM, Caballero A, Wright SI, Stinchcombe JR. 2022a. Selective ancestral sorting and *de novo* evolution in the agricultural invasion of *Amaranthus tuberculatus*. *Evolution; International Journal of Organic Evolution* 76: 70–85.

Kreiner JM, Hnatovska S, Stinchcombe JR, Wright SI. 2023. Quantifying the role of genome size and repeat content in adaptive variation and the architecture of flowering time in *Amaranthus tuberculatus*. *PLoS Genetics* 19: e1010865.

Kreiner JM, Latorre SM, Burbano HA, Stinchcombe JR, Otto SP, Weigel D, Wright SI. 2022b. Rapid weed adaptation and range expansion in response to agriculture over the past two centuries. *Science* 378: 1079–1085.

Krimbas CB, Powell JR. 1992. *Drosophila inversion polymorphism*. Boca Raton, FL, USA: CRC Press.

Kyriakidou M, Tai HH, Anglin NL, Ellis D, Strömvik MV. 2018. Current strategies of polyploid plant genome sequence assembly. *Frontiers in Plant Science* 9: 1660.

Lafon-Placette C, Johannessen IM, Hornslien KS, Ali MF, Bjerkan KN, Bramsiepe J, Glöckle BM, Rebernig CA, Brysting AK, Grini PE *et al.* 2017. Endosperm-based hybridization barriers explain the pattern of gene flow between *Arabidopsis lyrata* and *Arabidopsis arenosa* in Central Europe. *Proceedings of the National Academy of Sciences, USA* 114: E1027–E1035.

Laland K, Uller T, Feldman M, Sterelny K, Müller GB, Moczek A, Jablonka E, Odling-Smee J, Wray GA, Hoekstra HE *et al.* 2014. Does evolutionary theory need a rethink? *Nature* 514: 161–164.

Lande R. 2015. Evolution of phenotypic plasticity in colonizing species. *Molecular Ecology* 24: 2038–2045.

Lee CE. 2002. Evolutionary genetics of invasive species. *Trends in Ecology & Evolution* 17: 386–391.

Lee S-R, Choi T-Y, Son D-C. 2024. Multiple introductions of divergent lineages and admixture conferred the high invasiveness in a widespread weed (*Hypochaeris radicata*). *Evolutionary Applications* 17: e13740.

Leung C, Rescan M, Grulois D, Chevin L-M. 2020. Reduced phenotypic plasticity evolves in less predictable environments. *Ecology Letters* 23: 1664–1672.

Li Y, Stift M, van Kleunen M. 2018. Admixture increases performance of an invasive plant beyond first-generation heterosis. *The Journal of Ecology* **106**: 1595–1606.

Liu N, Du Y, Warburton ML, Xiao Y, Yan J. 2021. Phenotypic plasticity contributes to maize adaptation and heterosis. *Molecular Biology and Evolution* **38**: 1262–1275.

Liu W, Zhang Y, He H, He G, Deng XW. 2022. From hybrid genomes to heterotic trait output: challenges and opportunities. *Current Opinion in Plant Biology* **66**: 102193.

Lockwood JL, Cassey P, Blackburn T. 2005. The role of propagule pressure in explaining species invasions. *Trends in Ecology & Evolution* **20**: 223–228.

MacPherson A, Nuismer SL. 2017. The probability of parallel genetic evolution from standing genetic variation. *Journal of Evolutionary Biology* **30**: 326–337.

MacPherson A, Wang S, Yamaguchi R, Rieseberg LH, Otto SP. 2022. Parental population range expansion before secondary contact promotes heterosis. *The American Naturalist* **200**: E1–E15.

Makino T, Rubin C-J, Carneiro M, Axelsson E, Andersson L, Webster MT. 2018. Elevated proportions of deleterious genetic variation in domestic animals and plants. *Genome Biology and Evolution* **10**: 276–290.

Marchini GL, Sherlock NC, Ramakrishnan AP, Rosenthal DM, Cruzan MB. 2016. Rapid purging of genetic load in a metapopulation and consequences for range expansion in an invasive plant. *Biological Invasions* **18**: 183–196.

McGaughran A, Dhami MK, Parvizi E, Vaughan AL, Gleeson DM, Hodgins KA, Rollins LA, Tepolt CK, Turner KG, Atsawawaranunt K *et al.* 2024. Genomic tools in biological invasions: current state and future frontiers. *Genome Biology and Evolution* **16**: evad230.

McKibben MTW, Barker MS. 2023. Applying machine learning to classify the origins of gene duplications. *Methods in Molecular Biology* **2545**: 91–119.

McKinney ML, Lockwood JL. 1999. Biotic homogenization: a few winners replacing many losers in the next mass extinction. *Trends in Ecology & Evolution* **14**: 450–453.

Mesgaran MB, Lewis MA, Ades PK, Donohue K, Ohadi S, Li C, Cousens RD. 2016. Hybridization can facilitate species invasions, even without enhancing local adaptation. *Proceedings of the National Academy of Sciences, USA* **113**: 10210–10214.

Moran BM, Payne C, Langdon Q, Powell DL, Brandvain Y, Schumer M. 2021. The genomic consequences of hybridization. *eLife* **10**: e69016.

Moran EV, Alexander JM. 2014. Evolutionary responses to global change: lessons from invasive species. *Ecology Letters* **17**: 637–649.

Mounger J, Ainouche ML, Bossdorf O, Cavé-Radet A, Li B, Parepa M, Salmon A, Yang J, Richards CL. 2021. Epigenetics and the success of invasive plants. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences* **376**: 20200117.

Mullarkey AA, Byers DL, Anderson RC. 2013. Inbreeding depression and partitioning of genetic load in the invasive biennial *Alliaria petiolata* (Brassicaceae). *American Journal of Botany* **100**: 509–518.

Mulligan GA. 1965. Recent colonization by herbaceous plants in Canada. The genetics of colonizating plants. In: Baker HG, Stebbins GL, eds. *The genetics of colonizing species.* New York, NY, USA: Academic Press, 127–143.

Nadachowska-Brzyska K, Konczal M, Babik W. 2022. Navigating the temporal continuum of effective population size. *Methods in Ecology and Evolution* **13**: 22–41.

Nei M, Maruyama T, Chakraborty R. 1975. The bottleneck effect and genetic variability in populations. *Evolution; International Journal of Organic Evolution* **29**: 1–10.

Nocchi G, Whiting JR, Yeaman S. 2024. Repeated global adaptation across plant species. *bioRxiv*. doi: 10.1101/2024.04.02.587814.

North HL, McGaughran A, Jiggins CD. 2021. Insights into invasive species from whole-genome resequencing. *Molecular Ecology* **30**: 6289–6308.

Novo I, Ordás P, Moraga N, Santiago E, Quesada H, Caballero A. 2023. Impact of population structure in the estimation of recent historical effective population size by the software GONE. *Genetics, Selection, Evolution: GSE* **55**: 86.

Oduor AMO, Leimu R, van Kleunen M. 2016. Invasive plant species are locally adapted just as frequently and at least as strongly as native plant species. *The Journal of Ecology* **104**: 957–968.

Olazcuaga L, Loiseau A, Parrinello H, Paris M, Fraimout A, Guedot C, Diepenbrock LM, Kenis M, Zhang J, Chen X *et al.* 2020. A whole-genome scan for association with invasion success in the fruit fly Drosophila suzukii using contrasts of allele frequencies corrected for population structure. *Molecular Biology and Evolution* **37**: 2369–2385.

Orr HA. 1998. The population genetics of adaptation: the distribution of factors fixed during adaptive evolution. *Evolution; International Journal of Organic Evolution* **52**: 935–949.

Otto SP, Whitton J. 2000. Polyploid incidence and evolution. *Annual Review of Genetics* **34**: 401–437.

Paape T, Akiyama R, Cereghetti T, Onda Y, Hirao AS, Kenta T, Shimizu KK. 2020. Experimental and field data support range expansion in an allopolyploid arabidopsis owing to parental legacy of heavy metal hyperaccumulation. *Frontiers in Genetics* **11**: 565854.

Palacio-López K, Gianoli E. 2011. Invasive plants do not display greater phenotypic plasticity than their native or non-invasive counterparts: a meta-analysis. *Oikos (Copenhagen, Denmark)* **120**: 1393–1401.

Panetsos CA, Baker HG. 1967. The origin of variation in 'wild' *Raphanus sativus* (Cruciferae) in California. *Genetica* **38**: 243–274.

Pannell JR. 2015. Evolution of the mating system in colonizing plants. *Molecular Ecology* **24**: 2018–2037.

Peischl S, Dupanloup I, Kirkpatrick M, Excoffier L. 2013. On the accumulation of deleterious mutations during range expansions. *Molecular Ecology* **22**: 5972–5982.

Petit M, Guidat C, Daniel J, Denis E, Montoriol E, Bui QT, Lim KY, Kovarik A, Leitch AR, Grandbastien M-A *et al.* 2010. Mobilization of retrotransposons in synthetic allotetraploid tobacco. *New Phytologist* **186**: 135–147.

Petrov DA. 2001. Evolution of genome size: new approaches to an old problem. *Trends in Genetics* **17**: 23–28.

Pickrell J, Pritchard J. 2012. Inference of population splits and mixtures from genome-wide allele frequency data. *Nature Precedings* **2**: 10.

Prentis PJ, Wilson JRU, Dormontt EE, Richardson DM, Lowe AJ. 2008. Adaptive evolution in invasive species. *Trends in Plant Science* **13**: 288–294.

Prevosti A, Ribo G, Serra L, Aguade M, Balaña J, Monclus M, Mestres F. 1988. Colonization of America by Drosophila subobscura: experiment in natural populations that supports the adaptive role of chromosomal-inversion polymorphism. *Proceedings of the National Academy of Sciences, USA* **85**: 5597–5600.

Putra AR, Hodgins KA, Fournier-Level A. 2024. Assessing the invasive potential of different source populations of ragweed (*Ambrosia artemisiifolia* L.) through genomically informed species distribution modelling. *Evolutionary Applications* **17**: e13632.

Pyšek P, Lučanová M, Dawson W, Essl F, Kreft H, Leitch IJ, Lenzner B, Meyerson LA, Pergl J, van Kleunen M *et al.* 2023. Small genome size and variation in ploidy levels support the naturalization of vascular plants but constrain their invasive spread. *New Phytologist* **239**: 2389–2403.

Pysek P, Prach K, Mandák B. 1998. Invasions of alien plants into habitats of Central European landscape: an historical pattern. In: Starfinger U, Edwards K, Kowarik I, Williamson M, eds. *Plant invasions: ecological mechanisms and human responses.* Leiden: Backhuys Publishers, 23–32.

Qi X, An H, Hall TE, Di C, Blischak PD, McKibben MTW, Hao Y, Conant GC, Pires JC, Barker MS. 2021. Genes derived from ancient polyploidy have higher genetic diversity and are associated with domestication in *Brassica rapa*. *New Phytologist* **230**: 372–386.

Qiu F, Baack EJ, Whitney KD, Bock DG, Tetreault HM, Rieseberg LH, Ungerer MC. 2019. Phylogenetic trends and environmental correlates of nuclear genome size variation in Helianthus sunflowers. *New Phytologist* **221**: 1609–1618.

Ralph PL, Coop G. 2015. The role of standing variation in geographic convergent adaptation. *The American Naturalist* **186**: S5–S23.

Ranallo-Benavidez TR, Jaron KS, Schatz MC. 2020. GENOMESCOPE 2.0 and smudgeplot for reference-free profiling of polyploid genomes. *Nature Communications* **11**: 1432.

Reatini B, Pelosi JA, Cang FA, Jiang Q, McKibben MTW, Barker MS, Rieseberg LH, Dlugosch KM. 2022. Chromosome-scale reference genome and RAD-based genetic map of yellow starthistle (*Centaurea solstitialis*) reveal putative structural variation and QTL associated with invader traits. *bioRxiv*. doi: 10.1101/2022.09.28.509992.

Reed DH, Frankham R. 2001. How closely correlated are molecular and quantitative measures of genetic variation? A meta-analysis. *Evolution; International Journal of Organic Evolution* 55: 1095–1103.

Reznick DN, Losos J, Travis J. 2019. From low to high gear: there has been a paradigm shift in our understanding of evolution. *Ecology Letters* 22: 233–244.

Ricciardi A, MacIsaac HJ. 2008. The book that began invasion ecology. *Nature* 452: 34.

Richards CL, Bossdorf O, Muth NZ, Gurevitch J, Pigliucci M. 2006. Jack of all trades, master of some? On the role of phenotypic plasticity in plant invasions. *Ecology Letters* 9: 981–993.

Rockman MV. 2012. The QTN program and the alleles that matter for evolution: all that's gold does not glitter. *Evolution; International Journal of Organic Evolution* 66: 1–17.

Roesti M, Gilbert KJ, Samuk K. 2022. Chromosomal inversions can limit adaptation to new environments. *Molecular Ecology* 31: 4435–4439.

Rosinger HS, Geraldes A, Nurkowski KA, Battlay P, Cousens RD, Rieseberg LH, Hodgins KA. 2021. The tip of the iceberg: Genome wide marker analysis reveals hidden hybridization during invasion. *Molecular Ecology* 30: 810–825.

Santangelo JS, Ness RW, Cohan B, Fitzpatrick CR, Innes SG, Koch S, Miles LS, Munim S, Peres-Neto PR, Prashad C *et al.* 2022. Global urban environmental change drives adaptation in white clover. *Science* 375: 1275–1281.

Schmickl R, Yant L. 2021. Adaptive introgression: how polyploidy reshapes gene flow landscapes. *New Phytologist* 230: 457–461.

Simons YB, Sella G. 2016. The impact of recent population history on the deleterious mutation load in humans and close evolutionary relatives. *Current Opinion in Genetics & Development* 41: 150–158.

Soltis DE, Buggs RJA, Doyle JJ, Soltis PS. 2010. What we still don't know about polyploidy. *Taxon* 59: 1387–1403.

Soltis PS. 2017. Digitization of herbaria enables novel research. *American Journal of Botany* 104: 1281–1284.

Soltis PS, Liu X, Marchant DB, Visger CJ, Soltis DE. 2014. Polyploidy and novelty: Gottlieb's legacy. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences* 369: 20130351.

Stapley J, Santure AW, Dennis SR. 2015. Transposable elements as agents of rapid adaptation may explain the genetic paradox of invasive species. *Molecular Ecology* 24: 2241–2252.

Stebbins GL. 1956. Cytogenetics and evolution of the grass family. *American Journal of Botany* 43(890): 905.

Stern DB, Lee CE. 2020. Evolutionary origins of genomic adaptations in an invasive copepod. *Nature Ecology & Evolution* 4: 1084–1094.

Sturtevant AH. 1921. A case of rearrangement of genes in Drosophila. *Proceedings of the National Academy of Sciences, USA* 7: 235–237.

Sudmant PH, Rausch T, Gardner EJ, Handsaker RE, Abyzov A, Huddleston J, Zhang Y, Ye K, Jun G, Fritz MH-Y *et al.* 2015. An integrated map of structural variation in 2504 human genomes. *Nature* 526: 75–81.

Sun Y, Züst T, Silvestro D, Erb M, Bossdorf O, Mateo P, Robert C, Müller-Schärer H. 2022. Climate warming can reduce biocontrol efficacy and promote plant invasion due to both genetic and transient metabolomic changes. *Ecology Letters* 25: 1387–1400.

Thébault A, Gillet F, Müller-Schärer H, Buttler A. 2011. Polyploidy and invasion success: trait trade-offs in native and introduced cytotypes of two Asteraceae species. *Plant Ecology* 212: 315–325.

Todesco M, Pascual MA, Owens GL, Ostevik KL, Moyers BT, Hübner S, Heredia SM, Hahn MA, Caseys C, Bock DG *et al.* 2016. Hybridization and extinction. *Evolutionary Applications* 9: 892–908.

Tufto J. 2015. Genetic evolution, plasticity, and bet-hedging as adaptive responses to temporally autocorrelated fluctuating selection: a quantitative genetic model. *Evolution; International Journal of Organic Evolution* 69: 2034–2049.

Uesugi A, Baker DJ, de Silva N, Nurkowski K, Hodgins KA. 2020. A lack of genetically compatible mates constrains the spread of an invasive weed. *New Phytologist* 226: 1864–1872.

Uller T, Leimu R. 2011. Founder events predict changes in genetic diversity during human-mediated range expansions. *Global Change Biology* 17: 3478–3485.

VanWallendael A, Alvarez M, Franks SJ. 2021. Patterns of population genomic diversity in the invasive Japanese knotweed species complex. *American Journal of Botany* 108: 857–868.

Verhoeven KJF, Macel M, Wolfe LM, Biere A. 2011. Population admixture, biological invasions and the balance between local adaptation and inbreeding depression. *Proceedings of the Royal Society B: Biological Sciences* 278: 2–8.

Wang S, Lv J, Zhang L, Dou J, Sun Y, Li X, Fu X, Dou H, Mao J, Hu X *et al.* 2015. MethylRAD: a simple and scalable method for genome-wide DNA methylation profiling using methylation-dependent restriction enzymes. *Open Biology* 5: 150130.

Weiß CL, Pais M, Cano LM, Kamoun S, Burbano HA. 2018. nQuire: a statistical framework for ploidy estimation using next generation sequencing. *BMC Bioinformatics* 19: 122.

Wellenreuther M, Bernatchez L. 2018. Eco-evolutionary genomics of chromosomal inversions. *Trends in Ecology & Evolution* 33: 427–440.

Wellenreuther M, Mérot C, Berdan E, Bernatchez L. 2019. Going beyond SNPs: the role of structural genomic variants in adaptive evolution and species diversification. *Molecular Ecology* 28: 1203–1209.

Wendel JF. 2015. The wondrous cycles of polyploidy in plants. *American Journal of Botany* 102: 1753–1756.

Whiteman NK. 2022. Evolution in small steps and giant leaps. *Evolution; International Journal of Organic Evolution* 76: 67–77.

Whiting JR, Booker TR, Rougeux C, Lind BM, Singh P, Lu M, Huang K, Whitlock MC, Aitken SN, Andrew RL *et al.* 2024. The genetic architecture of repeated local adaptation to climate in distantly related plants. *Nature Ecology & Evolution* 8: 1933–1947.

Whitlock MC, Davis B. 2011. Genetic load. *Current Biology* 34: R1216–R1220.

Whitney KD, Gering E. 2015. Five decades of invasion genetics. *New Phytologist* 205: 472–475.

Whitney KD, Randell RA, Rieseberg LH. 2006. Adaptive introgression of herbivore resistance traits in the weedy sunflower *Helianthus annuus*. *The American Naturalist* 167: 794–807.

Wilson J, Battlay P, Bieker VC, van Boheemen L, Connallon T, Martin MD, Hodgins KA. 2024. Copy number variation contributes to parallel local adaptation in an invasive plant. *bioRxiv*. doi: 10.1101/2024.07.03.601998.

Yeaman S, Whitlock MC. 2011. The genetic architecture of adaptation under migration-selection balance. *Evolution; International Journal of Organic Evolution* 65: 1897–1911.

Yew C-L, Tsuchimatsu T, Shimizu-Inatsugi R, Yasuda S, Hatakeyama M, Kakui H, Ohta T, Suwabe K, Watanabe M, Takayama S *et al.* 2023. Dominance in self-compatibility between subgenomes of allopolyploid Arabidopsis kamchatica shown by transgenic restoration of self-incompatibility. *Nature Communications* 14: 7618.

Yoo M-J, Liu X, Pires JC, Soltis PS, Soltis DE. 2014. Nonadditive gene expression in polyploids. *Annual Review of Genetics* 48: 485–517.

Zeitler L, Parisod C, Gilbert KJ. 2023. Purging due to self-fertilization does not prevent accumulation of expansion load. *PLoS Genetics* 19: e1010883.

Zenni RD, Nuñez MA. 2013. The elephant in the room: the role of failed invasions in understanding invasion biology. *Oikos* 122: 801–815.

Zhang Y-Y, Fischer M, Colot V, Bossdorf O. 2013. Epigenetic variation creates potential for evolution of plant phenotypic plasticity. *New Phytologist* 197: 314–322.