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INVASION GENETICS: THE BAKER AND STEBBINS LEGACY

CONCLUSIONS

What we still don't know about invasion genetics

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Abstract

Publication of *The Genetics of Colonizing Species* in 1965 launched the field of invasion genetics and highlighted the value of biological invasions as natural ecological and evolutionary experiments. Here, we review the past 50 years of invasion genetics to assess what we have learned and what we still don't know, focusing on the genetic changes associated with invasive lineages and the evolutionary processes driving these changes. We also suggest potential studies to address still-unanswered questions. We now know, for example, that rapid adaptation of invaders is common and generally not limited by genetic variation. On the other hand, and contrary to prevailing opinion 50 years ago, the balance of evidence indicates that population bottlenecks and genetic drift typically have negative effects on invasion success, despite their potential to increase additive genetic variation and the frequency of peak shifts. Numerous unknowns remain, such as the sources of genetic variation, the role of so-called expansion load and the relative importance of propagule pressure vs. genetic diversity for successful establishment. While many such unknowns can be resolved by genomic studies, other questions may require manipulative experiments in model organisms. Such studies complement classical reciprocal transplant and field-based selection experiments, which are needed to link trait variation with components of fitness and population growth rates. We conclude by discussing the potential for studies of invasion genetics to reveal the limits to evolution and to stimulate the development of practical strategies to either minimize or maximize evolutionary responses to environmental change.

Keywords: adaptation, colonization, hybridization, invasion, rapid evolution, weeds

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Introduction

Ecologists and evolutionary biologists have a love–hate relationship with invasive species, defined here as widespread nonindigenous species. Although we dislike the harm they cause to the economy and environment, we appreciate their attributes as study organisms. They are easy to propagate and often have short generation times and small genomes (at least in plants). In addition, they typically produce very large numbers of offspring and frequently have the capacity for selfing or asexual reproduction, which can facilitate experimentation. Most importantly, at least from a scientific perspective, they represent natural ecological and evolutionary experiments unfolding in a recent historical time frame, thereby providing a window on ecological and evolutionary processes. This aspect is especially valuable to evolutionary biologists, who often are limited to making indirect inferences about evolutionary processes from DNA sequences, museum samples or from brief snapshots of evolution in contemporary populations.

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Evolutionary studies of invasive lineages have generated two main kinds of information. First, as alluded to above, they have yielded valuable insights into evolutionary processes, especially with respect to the speed of adaptation and to the role of population bottlenecks in evolution. Second, these studies have informed us regarding the features of the invading organisms themselves and the evolutionary processes and the genetic changes that underlie these features (Handley *et al.* 2011; Lee 2002). Here, we focus on this second kind of information, as our review is about the evolutionary genetics of invasive lineages. However, the inferences we make about the genetics of invaders are broadly relevant to understanding how organisms successfully colonize new environments, regardless of whether they conform to any particular definition of an invasive species.

We first discuss genetic and genomic variation in invasive lineages. We ask about the sources of genetic variation, the roles of intra- and interspecific hybridization in invasions and whether certain kinds of genomic changes might serve as stimuli for invasiveness (Glossary). We then examine key evolutionary processes, exploring the roles of genetic drift and pre- and post-introduction adaptation in successful invasions. We consider phenomena associated with the adaptive evolution of invasive lineages, including invasion 'lag phases', evolutionary trade-offs and phenotypic plasticity. Lastly, we describe what is known about the architecture of genetic changes associated with successful invasions and evaluate different approaches for identifying these changes. In keeping with the theme of this volume, we consider these topics in the context of what was known by the contributors to the Baker and Stebbins (1965) volume while identifying what we still don't know about each issue. Where possible, we suggest experiments or other kinds of studies that have the potential to address still-unanswered questions.

Genetic variation

Sources of variation

What is the primary source of genetic variation employed by natural selection during the evolution of invasive lineages? This question, which relates mainly to post-introduction adaptation, was a topic of discussion by the Baker and Stebbins (1965) contributors and remains unsolved. However, analyses of new genomic data sets are beginning to yield answers.

While some successful invaders arrive well-suited to their new environments, the success of others appears to depend on rapid local adaptation. Adaptation relies on two main sources of variation: pre-existing standing genetic variation and new beneficial mutations. Adaptation from standing genetic variation is generally faster and more predictable because standing variants typically have higher initial frequencies, which increases both the probability and speed of their fixation (Barrett & Schluter 2008; Prentis *et al.* 2008). In contrast, adaptation from new mutations is slowed by the waiting period for them to occur and reach fixation, which could be critical to the fate of the invasion. Despite the greater efficiency of adaptation from standing genetic variation compared with that from new mutations, Baker and Stebbins (1965) contributors gave greater credence to the latter (Dobzhansky 1965; Mayr 1965), especially as an explanation for the lag phase (Box 1). A third source of variation, which represents a distinct kind of standing variation, is the introgression of alleles from other species (Hedrick 2013). This process was deemed likely by the Baker and Stebbins (1965) contributors (see Interspecific hybridization, below).

The relative contributions of these sources of variation to the adaptation of invasive lineages are not obvious. Due to intra- and interspecific admixture, invasive lineages often harbour significant levels of standing variation (see below). On the other hand, Fisher's geometric model of adaptation (Fisher 1930) implies that new mutations are more likely to be beneficial in a population that is far from its adaptive optimum, which is likely for a new invader. Moreover, mutation accumulation experiments frequently find evidence of beneficial mutations (Heilbron *et al.* 2014). In practice, the rapid evolution of invasive lineages may involve more than one type of variation, and analytical approaches may fail to distinguish between them, especially when multiple colonizations have occurred or when selection is weak (Hermisson & Pennings 2005).

However, there are features of invasions, as well as new techniques, that may permit the different sources of variation to be determined, at least under some circumstances and for some loci. Most importantly, many invasions are recent, and source regions can often be identified, so one can ask whether variants under selection in the invaded range are present in relevant native populations. Also, by examining herbarium or museum specimens, it may be possible to pinpoint the source of variation and assess how allele frequencies have changed over time. Such an approach was recently employed by Vandepitte *et al.* (2014) to show that the genetic changes underlying flowering time adaptation in colonizing populations of the Pyrenean rocket arose from standing variation. This study and others also illustrate how genome scans for footprints of selection, differentiation and hybridization permit detection of candidate genes and genomic regions that are associ-

Box 1 Lag phases

Biologists have long noted that, on occasion, the rate of spread of an invasive species accelerates after a long period of quiescence, more rapidly than would be expected on the basis of a standard population model (Fig. 1). The lag phase, the period from introduction to acceleration, can in some instances be over a century in length. Anecdotes of lags abound, although it is rarely possible to estimate their length with any accuracy. The data are simply too poor: while the species is in its lag phase, recording intensity is inevitably extremely low and occurrences will be overlooked, while search effort may be increased as the species becomes of concern, creating bias (Cousens & Mortimer 1995). Even so, many invasion ecologists now appear to regard lags as the norm, rather than the exception.

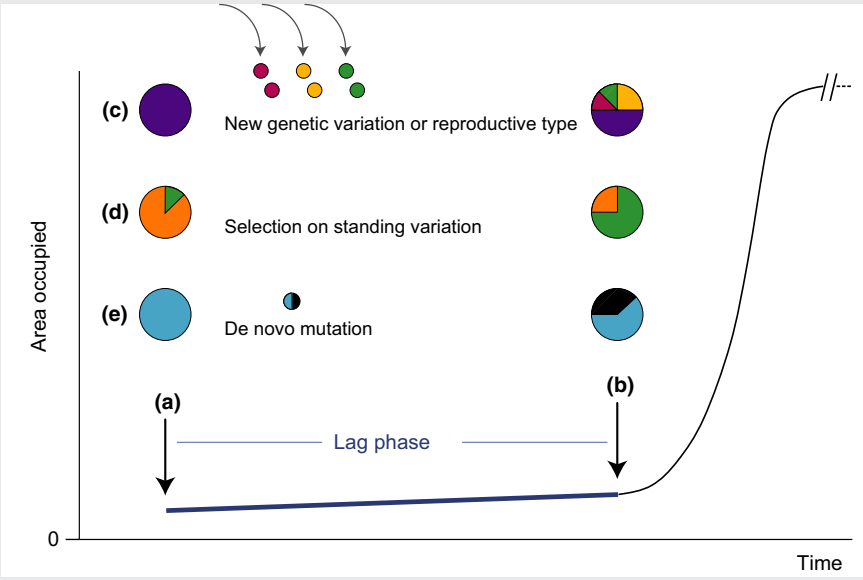

Fig. I Example of the lag phase and potential genetic causes, showing hypothetical patterns of genetic variation at a single locus at (a) initial establishment and before (b) the onset of accelerated expansion. (c) shows an increase in genetic variation following immigration of new genotypes or new sexes. (d) illustrates selection on standing variation. (e) represents the origin of a *de novo* adaptive mutation.

There have been many plausible explanations given for lag phases (e.g. Crooks & Soulé 1999), although it is only rarely possible to ascribe a cause to a particular instance. Potential genetic causes include the following: evolution from standing genetic variation or new mutations resulting in increased local adaptation or dispersal capability; the introduction of genotypes that are either more fit or that allow novel, fitter gene combinations to be generated (Kolbe *et al.* 2004); and the introduction of the opposite sex where previously only one sex was present, thus allowing sexual reproduction to occur. Such genetic mechanisms were in the forefront of the thinking of the Baker and Stebbins (1965) contributors, in particular the need for sufficient time to elapse after introduction before a species became adapted to the novel conditions. This thinking persists, even without specific reference to a lag, with many researchers commenting routinely on the requirement for invaders to adapt to novel conditions. In some instances, however, the environment may not be so different from the native range, and plasticity may be sufficient for the invader to spread rapidly. Adaptation will no doubt occur in these situations, but more as a means of fine-tuning than overcoming a major fitness hurdle, and the change in rate of spread may be hard to discern.

There are also several possible nongenetic causes for lags. These include the following: sudden/rapid change in an environmental factor (e.g. land management, introduction of a symbiotic species, loss of a predator); overcoming an Allee effect (Aikio *et al.* 2010); eventual dispersal into another region of more suitable habitat (e.g. across a barrier around the initial introduction site); reaching an area where a more effective dispersal vector is available (Ridley 1930); and a threshold age for physiological maturity (Wangen & Webster 2006). An apparent lag may occur where early occurrence records in fact represent repeated failed establishments, but these are later followed by an event in which the species successfully establishes and then spreads.

It is tempting to argue that we need to better establish the mechanism behind particular instances of lag phase. Do genetic or nongenetic processes predominate and, if so, which ones? However, we face the challenge that we cannot monitor all invasions in fine detail in their early stages—there are simply too many and the areas too extensive—and those having extensive lag phases are only identified well after the lag has ended. Although herbarium or museum specimens may allow us to retrospectively search for genetic changes (e.g. Vandepitte *et al.* 2014), demographic and detailed population data typically will not be available through which to rule out the alternative mechanisms.

ated with invasiveness, and when compared with native populations, reveal the likely source(s) of adaptive genetic changes (Prentis *et al.* 2008; Scascitelli *et al.* 2010; Tollenaere *et al.* 2013; Brown *et al.* 2014). Given wider implementation of these approaches over the coming decade, this major question is likely to be resolved soon.

Multiple introductions, genetic diversity and intraspecific admixture

Modern molecular techniques permit reconstruction of the phylogeographic histories of invaders, revealing invasion routes and putative source populations (e.g. Muirhead *et al.* 2008; Gray *et al.* 2014; Martin *et al.* 2014; Zhang *et al.* 2014)—information that typically was not available to the Baker and Stebbins (1965) contributors. Such studies indicate that many successful invasions are associated with multiple introductions and subsequent mixing (Bossdorf *et al.* 2005; Dlugosch & Parker 2008; Simberloff 2009). In part, this association may be driven by a correlation between number of introductions and propagule pressure (Simberloff 2009), because increased propagule pressure is known to aid founding populations in overcoming stochastic processes that would otherwise lead to extinction. However, increased propagule pressure is also likely to be associated with increased genetic diversity as a larger fraction of the native range alleles are likely to be sampled. Genetic diversity can have independent effects on colonization/invasion success through two sets of mechanisms, distinguished by timescale.

On shorter timescales, increased genetic diversity may increase colonization success through predominantly ecological mechanisms analogous to the species diversity effects seen in biodiversity-ecosystem functioning studies. These include selection effects, in which high-diversity founder populations have increased probabilities of containing (and becoming dominated by) a genotype with high invasive potential, and complementarity effects, in which either facilitation between genotypes or trait differences among genotypes lead to improved performance of mixtures over monocultures (Crawford & Whitney 2010; Forsman 2014). Forsman's recent (2014) meta-analysis found a significant positive effect of genetic diversity on measures of colonization success in plants and animals. This result should perhaps be interpreted with caution, as the meta-analysis did not correct for phylogenetic nonindependence and further, included several studies suffering from pseudo-replicated genetic diversity treatments. Further progress in this area will involve experimentally decoupling the purely numeric component of propagule pressure effects from the genetic diversity component and determining why their relative importance can vary in the field (e.g. Erfmeier *et al.* 2013).

On longer timescales, intraspecific genetic admixture may benefit invaders via the same set of evolutionary mechanisms proposed to benefit interspecific hybrids (Rius & Darling 2014). These include (i) an increase in genetic variation, providing a larger pool of raw material for adaptive evolution (Anderson 1949; Anderson & Stebbins 1954); (ii) the creation of novel or transgressive phenotypes through previously unexplored allele and gene combinations (Stebbins 1969; Lavergne & Molofsky 2007); (iii) heterosis, particularly when stabilized by nonsexual forms of reproduction (Baker 1965); and (iv) the masking or purging of deleterious mutations, which may reduce potentially negative effects of genetic bottlenecks and inbreeding (Ellstrand & Schierenbeck 2000; Keller & Waller 2002). Many observational and a few experimental studies indicate that admixture can contribute to invasion success (e.g. Kolbe *et al.* 2004; Wolfe *et al.* 2007; Keller & Taylor 2010; Verhoeven *et al.* 2011). For example, Keller & Taylor (2010) found that the level of genetic admixture in the invasive plant *Silene vulgaris* was associated with increased fecundity and thus may contribute to its success in the invaded range. However, in all cases, it is not possible to determine whether the invasion would have been successful without admixture. More experimental manipulations that directly test the effect of admixture on colonization success are needed (Rius & Darling 2014).

While genetic admixture and invasion appear to be linked, the specific roles of the four above-mentioned evolutionary mechanisms are less clear. In general, increased genetic diversity and the creation of novel genotypes should have long-lasting effects by enhancing

the adaptive potential of a population. This may be most beneficial to invaders experiencing novel environmental conditions, whereas in native environments, genetic admixture may result in the loss of local adaptation (Verhoeven *et al.* 2011). In contrast, the beneficial effects of heterosis are thought to be transitory in sexual populations and thus mainly important to establishment (Rius & Darling 2014). However, this depends on the genetic basis of heterosis (Hochholdinger & Hoecker 2007; Lippman & Zamir 2007). Genetic models for the evolution of heterosis include the following: dominance (enhanced performance due to the masking of deleterious recessive alleles from one parent by dominant alleles from the other parent), overdominance (enhanced performance due to beneficial interactions of alleles from different lineages at a single locus) and epistasis (enhanced performance due to beneficial interactions between loci from different lineages). Heterosis due to dominance (considered most common) and epistasis can be fixed by selection, resulting in the long-term preservation of heterotic effects and purging of genetic load.

Interspecific hybridization

As alluded to previously, the Baker and Stebbins (1965) contributors were well aware of the hypothesis that interspecific hybridization could act as an evolutionary stimulus, perhaps triggering colonizing and invasive behaviour. Several contributors had or were about to publish seminal papers on the issue (Heiser 1951; Anderson & Stebbins 1954; Stebbins 1959; Lewontin & Birch 1966; Panetsos & Baker 1968), and reference to the idea permeated the presentations and discussions. However, it was not obvious at the time how prevalent the process was, with E. O. Wilson asking whether 'introgression commonly results in a considerable increase in the fitness of a species' (Baker & Stebbins (1965) p. 213).

We have a much better handle on the issue today. Ellstrand & Schierenbeck (2000) published lists of species that were both hybrid derived and invasive, describing potential cases among a broad taxonomic array of plant invaders. Importantly, investigators also began to experimentally test performance of hybrids vs. parents in many systems, allowing cases of neutral or incidental hybridization in already-invasive taxa to be distinguished from cases where hybridization is a causal driver of increased invasiveness. Recently, these studies have been compiled in a systematic review and meta-analysis (Hovick & Whitney 2014) focusing on studies in which hybridization has been putatively associated with colonizing behaviour, and in which the performance of hybrids vs. their parental species has been experimentally tested. Meta-analyses of fecundity, survival and size (as proxies for population growth rate, λ) determined that wild hybrids are typically larger and more fecund than their parental species, while not differing in survival. Further, hybrid fecundity generally increases with generation, suggesting that natural selection can play an important role in shaping hybrid performance (and thus invasiveness) over time. However, these results are driven by tests in plants and further work is needed to understand patterns in animals and fungi. Also, hybridization and polyploidy are confounded in this and earlier studies, so more work is needed to understand both their independent and their synergistic effects.

Substantial progress has also been made in the identification of the genomic regions/alleles potentially involved in introgression events in colonizing or invasive species. Specific genes or quantitative trait loci (QTL) have been identified that control introgressing traits such as inflorescence morphology affecting pollination in *Senecio vulgaris* (Kim *et al.* 2008; Chapman & Abbott 2010), resistance to anticoagulant poisons in the Western European house mouse (Song *et al.* 2011), several fitness, ecophysiological, architectural and phenological traits in *Helianthus annuus texanus* (Whitney *et al.* 2015) and fitness traits in crop–wild *Lactuca* hybrids (Hartman *et al.* 2013). Other studies have used molecular signatures of selection or geographical clines in marker frequencies to identify introgressing genomic regions, without identification of the phenotypic traits affected (e.g. *Ambystoma*, Fitzpatrick *et al.* 2009; *Tamarix*, Gaskin & Kazmer 2009). In all of these examples, introgression is associated with increased adaptation, although in most cases, the link between the introgression of specific alleles and increased invasiveness has not been made. Nevertheless, the fact that specific alleles are frequently found that increased adaptation in the recipient species could be interpreted as support for the 'novel phenotypes' mechanism discussed in the previous section, while not ruling out contributions from other mechanisms.

It remains unclear, however, *why* hybridization sometimes results in increased colonization success and sometimes does not. One approach is to view the problem as a genotype × genotype × environment (G×G×E) interaction, where the interactions between the alleles provided by the donor species, the genome of the recipient species and the environment in which the hybrids are located determine whether the overall outcome is an increase, decrease or no change in λ. This approach emphasizes the extremely contingent nature of the process. However, it may be that even if each case is not individually predictable, the process across many cases is predictable at a statistical level. To our knowledge, there have not yet been attempts to evaluate such predictability. We suggest three hypotheses, one previously articulated and two perhaps new: (i) parental species separated by

intermediate genetic distances might give rise to more successful hybrids than will less or more divergent parents (Ellstrand & Schierenbeck 2000; see also Stelkens & Seehausen 2009); (ii) the greater the adaptive fit of the donor parent to the environment to be colonized, the higher the chance that beneficial alleles are available via hybridization, and the more successful the hybrid; and (iii) the higher the frequency of hybridization between two parents, the greater the chance of 'hitting the G×G×E jackpot' and thus the greater the probability that a successful hybrid will arise.

Genomic variation

Punctuated changes in the structure and organization of the genome may also contribute to the evolution of invasiveness. Three types of genomic variation—namely polyploidy, genome size variation and chromosomal rearrangements—have been considered in this context.

Polyploidy. The first attempts to address the role of polyploidy in the evolution of invasiveness date from the early part of the twentieth century. These efforts relied on assessments of the frequency of polyploids among invasive species (e.g. Muntzing 1935; Gustafsson 1948) and were generally idiosyncratic, providing inconclusive answers. The views of the Baker and Stebbins (1965) contributors were mixed as well, with Mulligan (1965) writing that 'there is no evidence that polyploid weeds are particularly favoured for the colonization of newly available areas.' In contrast, Ehrendorfer (1965) listed polyploidy as one of the characteristics of good colonizers and provided examples of polyploid species that are considerably more widespread than their diploid progenitors.

Estimates of the frequency of polyploids among invasive species have expanded in both taxonomic scope and accuracy during the past two decades, with the implementation of methods such as flow cytometry, which allow ploidal levels to be identified *en masse*, and with the establishment of electronic databases of genome size and chromosome numbers. With these advances came more frequent reports that polyploids are over-represented among invasive species in regional floras (e.g. Verlaque *et al.* 2002; Pandit *et al.* 2006). In broad taxonomic surveys as well, polyploidy was found to be associated with invasiveness. Pandit *et al.* (2011), for example, compiled data from 81 invasive species and 2356 of their congeners and showed that being invasive is 20% more likely for polyploid species than for closely related diploid species.

However, the factors driving these patterns remain unclear. While studies comparing closely related diploid and polyploid species in their native and introduced ranges (e.g. Hahn *et al.* 2012a) can generate strong hypotheses, experimental work is needed to link the effects of polyploidization to invasiveness. These effects include genetic and epigenetic changes such as the masking of deleterious alleles, fixed heterozygosity and epigenetic remodelling, as well as morphological/physiological changes such as increased body size, altered drought tolerance and altered phenology (Soltis & Soltis 2000; te Beest *et al.* 2012). For allopolyploid invaders, some of these effects may be due to hybridization (e.g. Hegarty *et al.* 2011). Therefore, the most valuable studies will be those that experimentally decouple the effects of hybridization from polyploidy through comparisons of the invasiveness of both diploid parental species with both diploid and polyploid hybrids.

Even if differences in invasive potential are detected between ploidal levels, studies using natural polyploids are likely to overestimate the contribution of genome doubling, because of the confounding effects of genetic differences that accumulate after polyploid formation. To address this issue, experiments should be performed that use not only field-collected samples of varying ploidal levels, but also artificially obtained neo-polyploids. This approach has been used recently by Ramsey (2011) to show that genome duplication as well as post-polyploidization evolution facilitated adaptation of hexaploid cytotypes of the noninvasive wild yarrow (*Achillea borealis*) to Mediterranean habitats on the Pacific coast of North America.

Genome size. Inherently linked to ploidy, genome size has also been proposed to contribute to the evolution of invasiveness, albeit in the opposing direction (te Beest *et al.* 2012). Small genomes have been associated with traits such as short generation time, which may facilitate reproductive success under ephemeral conditions, or small seeds, which may enhance reproductive output and dispersal ability (Knight *et al.* 2005; te Beest *et al.* 2012). Support for this prediction has so far mainly come from broad surveys of the distribution of genome size values among invasive species (e.g. Kubesova *et al.* 2010; Pandit *et al.* 2014).

Evidence from specific systems, which may provide clues to the underlying traits and mechanisms, has, by comparison, been much more difficult to find. One possible exception is the study by Lavergne *et al.* (2010). The authors estimated genome sizes as well as rates of vegetative growth under glasshouse conditions for native (European) and invasive (North American) diploid genotypes of reed canary grass (*Phalaris arundinacea*). Patterns were in the expected direction: invasive genotypes had lower average genome sizes and displayed higher early growth rates than native

genotypes. However, many previous reports of intraspecific variation in plant genome size have been discounted due to methodological issues (Greilhuber 1988; Price *et al.* 2000), and it is not clear whether the *Phalaris* study avoided these issues. Future work is required to confirm the genome size variation, elucidate the mechanisms linking genome size and growth rate and to make the connection between early growth rate and propensity to invade in the field.

Chromosomal rearrangements. In *The Genetics of Colonizing Species*, Carson (1965) and Dobzhansky (1965) make a distinction between 'flexible' and 'rigid' chromosomal inversions and discuss their dynamics in populations of cosmopolitan *Drosophila* species. 'Flexible' polymorphisms are shown to vary in frequency along environmental gradients and are hypothesized to contribute to local adaptation. 'Rigid' polymorphisms are shown to maintain unchanged frequencies in drosophilid populations and are proposed to result from heterozygote advantage. Under currently accepted models for the spread of chromosomal inversions (Hoffmann & Rieseberg 2008), 'flexible' polymorphisms can occur if inversions bring together alleles that are locally adapted, with or without epistasis. Similarly, 'rigid' polymorphisms can arise via overdominance, when inverted and noninverted arrangements carry different deleterious alleles (Hoffmann & Rieseberg 2008).

More recent work has provided some experimental support that inversion polymorphisms contribute to adaptation during biological invasions. For example, Prevosti *et al.* (1988) calculated correlations between chromosomal rearrangement frequencies and latitude for populations of *Drosophila subobscura* established along the Pacific coasts of North and South America. Striking similarities were observed along the two latitudinal clines, providing strong indication that chromosomal inversions are adaptive.

Few other studies have established a link between invasion success and inversion polymorphisms (although see Kirkpatrick & Barrett 2015). Even less is known about the potential contributions of other kinds of rearrangements to invasions. Future research should therefore aim to identify the role of chromosomal rearrangements in invasion potential in other systems. Moreover, the genes responsible for the associations between invasion success and inversion polymorphisms are unknown. One approach to address this is to use genome scans to identify targets of spatially variable selection within inversions (e.g. Fabian *et al.* 2012). The success of this approach will depend, however, on whether some recombination has occurred within the inverted region, breaking-up linkage disequilibrium away from the inversion breakpoints.

Epigenetic variation and invasion

The Baker and Stebbins (1965) contributors were aware of epigenetic variation: Waddington had previously coined the term, defining it as 'the branch of biology that studies the causal interactions between genes and their products, which bring the phenotype into being' (Waddington 1942). However, epigenetics did not feature in the symposium discussions, in part because it was largely a theoretical concept. We now know that epigenetic phenomena provide an information layer above the DNA sequence level and can contribute to variation in gene expression and phenotype via multiple molecular mechanisms including DNA methylation, histone modifications, small RNAs and noncoding RNA (Kinoshita & Jacobsen 2012). Moreover, some epigenetic modifications are elicited by environmental factors and can be transmitted across generations (Verhoeven *et al.* 2010; Dowen *et al.* 2012).

Because invaders often exhibit reduced genetic variation in their new range (Dlugosch & Parker 2008), there has been interest in whether epigenetic variation could ameliorate this apparent handicap. For example, epigenetic diversity appears to compensate for the loss of genetic diversity and inbreeding in recently introduced Kenyan house sparrows (Liebl *et al.* 2013). In Japanese knotweed, successful invasion of diverse habitats was correlated with epigenetic differentiation in response to new and dynamic microclimate conditions (Richards *et al.* 2012). Experimental studies have shown that epigenetic modifications can be induced by specific abiotic and biotic stresses (Verhoeven *et al.* 2010; Dowen *et al.* 2012) and contribute to increased population biomass (Latzel *et al.* 2013). These results are consistent with a possible role for epigenetic variation in invasive species via adaptive phenotypic plasticity and by compensating for losses in genetic variability. However, the adaptive significance of epigenetic variation remains largely unknown, especially in the context of plant invasions. Field and common garden experiments are needed to differentiate between plastic and heritable epigenetic variation and to link this variation to specific phenotypes and to fitness (Richards *et al.* 2010).

Evolutionary processes

Genetic drift and invasion

Newly introduced populations often experience a genetic bottleneck, which can have potentially important consequences for their evolution and ultimate fate. The relationship between bottlenecks and variation in Mendelian traits such as molecular markers is well understood theoretically (Wright 1931; Dlugosch & Par-

ker 2008), leading to the following predictions: (i) the loss of Mendelian variation via drift should correlate with both the severity and length of the bottleneck (Wright 1931); (ii) bottlenecks should cause greater reductions in allelic richness than in expected heterozygosity (Nei *et al.* 1975); and (iii) large shifts in allele frequencies are likely, especially for rare alleles that survive the bottleneck (Peischl *et al.* 2013). All three predictions have been validated by empirical studies of species invasions (Dlugosch & Parker 2008; Uller & Leimu 2011; Tsuchida *et al.* 2014). Such losses of diversity and/or drift-induced changes in allele frequencies have the potential to impede adaptive evolution, at least to the extent that Mendelian traits affect fitness. As discussed earlier, however, multiple introductions often restore lost diversity, not infrequently resulting in higher levels of diversity than in the native range because of admixture from genetically different source populations (Dlugosch & Parker 2008; Uller & Leimu 2011).

The effects of genetic bottlenecks on quantitative genetic variation, which is generally believed to underlie the majority of fitness related traits (Falconer & Mackay 1996), are less severe. As pointed out by Lewontin (1965, p. 481):

> If there is colonization by a single fertilized female... one-half of the additive, three-quarters of all the dominance variation, and a large amount of the epistatic variation are present in the offspring.

Moreover, theoretical and empirical studies indicate that population bottlenecks can convert dominance (Robertson 1952; Cockerham & Tachida 1988) and epistatic variance (Goodnight 1988; Whitlock *et al.* 1993; Cheverud & Routman 1995) to additive variance (Bryant *et al.* 1986). While conversion of the former is restricted to cases where the initial frequency of the recessive allele is low in the source population, conversion of epistatic variance is less restricted (Whitlock *et al.* 1993). Thus, it is perhaps unsurprising that comparisons of phenotypic and/or quantitative genetic variation in source and introduced populations have generally failed to find differences in variance (reviewed in Dlugosch & Parker (2008), although see Simberloff *et al.* (2000) and Van Heerwaarden *et al.* (2008) for examples of reduced and increased quantitative genetic variation, respectively). Ample evidence of rapid post-introduction adaptive differentiation (see below) further implies that genetic variation is generally not limiting in invaders.

The Baker and Stebbins (1965) contributors were enthusiastic about the possibility that bottlenecks associated with colonization might enable invaders to reach a new adaptive peak through a process put forward by Wright (1931). These ideas have gained some support from studies demonstrating gains in additive genetic variation following bottlenecks (e.g. Bryant *et al.* 1986), and from recent theory indicating that range expansions increase the frequency of peak shifts (Burton & Travis 2008). Nonetheless, we are unaware of examples where such gains have contributed to Wrightian peak shifts in introduced populations (Van Heerwaarden *et al.* 2008).

Another possible consequence of population bottlenecks and subsequent population expansion is the accumulation of deleterious mutations, which could limit invasion success (Peischl *et al.* 2013). Simulations indicate that extreme drift is created at the wave front of expanding populations because population density is low and growth rate is high (Edmonds *et al.* 2004). New and standing mutations at the wave front can 'surf' to high frequency whether they are neutral or deleterious (Klopfstein *et al.* 2006), creating what has been termed 'expansion load' (Peischl *et al.* 2013). Expansion load can reduce fitness over much of the newly expanded range and persist for thousands of generations.

The extent of expansion load in invading lineages is largely unknown, although an excess of deleterious mutations has been reported in non-African human populations (Peischl *et al.* 2013). Likewise, we are unaware of examples of colonization failure due to expansion load or of the operation of compensating mechanisms such as admixture (see above) or Allee effects (Glossary), which might limit its severity. Methods now exist for detection of deleterious mutations from genomic scan data (e.g. Adzhubei *et al.* 2010), so the extent of expansion load in invading lineages can be estimated (see Hodgins *et al.* 2015 and Peischl & Excoffier 2015). Linking load to failed invasions will be more challenging because of the confounding effects of other demographic and genetic factors associated with invasion success.

Pre-introduction adaptation

For a species to establish in a new location, its intrinsic rate of increase must be positive. This condition will be more likely if there is a close match between native and recipient environments. In other words, species should be pre-adapted to at least some novel geographical locations. Even in cases of a partial environmental match, adaptive phenotypic plasticity may be sufficient for the colonizer to survive and reproduce. Some indication of invasive potential may come from the breadth of the native range: a wide realized niche will increase the possibility that (if introduced) at least one genotype will be suited to a set of novel conditions. Researchers attempting to understand the degree of matching of

environments in native and recipient regions, however, face a number of pitfalls associated with extrapolation (Mesgaran *et al.* 2014).

Perhaps because predicting the environmental match between organism and location is both case specific and data intensive, there has been a long-standing tradition of instead searching for universal, pre-adapted traits that are associated with invasive behaviour. For plants, Baker's list of traits (Baker 1965) that together would result in the 'ideal weed' is a touchstone, emphasizing a capacity for asexual reproduction, high fecundity, rapid growth to maturity, phenotypic plasticity and broad environmental tolerance. Similar lists have since been suggested for animals (e.g. Kolar & Lodge 2001). Many of the traits correspond to the concept of 'r-selected' species and are likely to result in high rates of increase.

Tests of the idea that invasive species come pre-equipped with particular traits have had mixed success. Many authors have observed that 'Baker traits' were both present in some noninvasive species and absent in some invasive species (Perrins *et al.* 1992; Mack 1996). More recently, there has been some success in distinguishing traits of invasive vs. noninvasive species (Rejmánek & Richardson 1996; Pyšek & Richardson 2007; van Kleunen *et al.* 2010). For example, Rejmánek & Richardson (1996) used multivariate techniques to identify short juvenile periods, a short interval between large seed crops, and small seed mass as good predictors of increased invasiveness in *Pinus*. A meta-analysis (van Kleunen *et al.* 2010) found evidence for higher values of performance traits in invasive vs. noninvasive plants, although we point out that samples of the former often derive from the invaded range, so trait values for invasives (and thus effect sizes) do not necessarily reflect pre-adaptation and could instead reflect post-introduction adaptation. However, these successes have been balanced with other cases in which invasiveness was not correlated with biological traits (Caley & Kuhnert 2006), and have been followed by scepticism as to whether the pursuit of predictive traits is worthwhile (Thompson & Davis 2011; Moles *et al.* 2012).

The future of the pre-adaptation paradigm is unclear. Certainly, it appears that further comparative studies of traits in highly invasive vs. noninvasive taxa will be carried out. A new approach involves breaking the invasion process into stages to isolate the traits that matter at each stage, with perhaps a higher chance of identifying consistent trait differences between invasive and noninvasive species (van Kleunen *et al.* 2015). It is also likely that others will continue to argue that other factors, for example post-introduction adaptation and the environmental context in which a colonizing species finds itself (e.g. when predators and pathogens have been left behind, aka 'enemy release'), are more explanatory than pre-existing traits in determining invasiveness.

Post-introduction adaptation

The success of many biological invasions may depend on the capacity of invasive species to adapt to novel environmental conditions. Such post-introduction adaptation was considered by a number of the Baker and Stebbins (1965) contributors: although the evidence provided was often indirect, rapid evolution of life history, reproductive and dispersal traits in plants (Ehrendorfer 1965; Harper 1965) and *Drosophila* (Dobzhansky 1965) was considered important to the colonization process (Lewontin 1965). Since then, observational and experimental studies have documented adaptive changes in invasive relative to native populations (reviewed in Dlugosch & Parker 2008; Prentis *et al.* 2008; Whitney & Gabler 2008; Felker-Quinn *et al.* 2013). Rates of evolution can be quite rapid, with many examples occurring in <50 years (Whitney & Gabler 2008). This makes sense, as environmental differences between native and invaded ranges should generate strong selective pressures. Indeed, rates of adaptive phenotypic change may be higher in human disturbed environments than in undisturbed contexts (Hendry *et al.* 2008), and a survey of herbarium specimens across 150 years in Australia showed significantly more morphological changes in introduced species than in Australian natives (Buswell *et al.* 2011).

Increased growth rate or reproductive capacity is frequently reported from field observations in the invaded range (Elton 1958; Crawley 1987; Thebaud & Simberloff 2001; Parker *et al.* 2013; Pandit *et al.* 2014; see also discussion of the EICA hypothesis in Box 2) and increasingly from common garden experiments (reviewed in Felker-Quinn *et al.* 2013). This improved fecundity could contribute to rapid spread and population growth in the invaded range. However, many experimental studies are limited in scope, thus limiting the generality of their conclusions. Observing the phenotypes of a single generation of native and invasive populations in a common environment may be insufficient to demonstrate adaptation to a novel habitat. Differences between native and invasive populations caused by maternal environmental effects need to be taken into account, as do the effects of latitudinal or environmental clines (Colautti *et al.* 2009).

Some of the best evidence supporting post-introduction adaptation and contemporary evolution in invasive species comes from recapitulation of clinal variation of species in their native ranges. Latitudinal clines in morphological wing traits, chromosome inversion frequencies, genetic variation and physiological resistance in invasive *Drosophila* populations have evolved to parallel

Box 2 Evolutionary trade-offs and invasion

Increased performance in competitive ability, size and fecundity of invasive populations relative to their native conspecifics has been addressed by multiple hypotheses, several of which invoke evolutionary trade-offs between self-defence, growth and reproduction (Fig. 2). Underlying all trade-off hypotheses is the assumption that organisms are unable to be both highly competitive or have high reproductive output and be highly tolerant of stressful conditions (Grime 1977). Many of the trade-offs described in the context of invasive species are based on modifications of interspecific interactions in the introduced range, such as release from natural enemies.

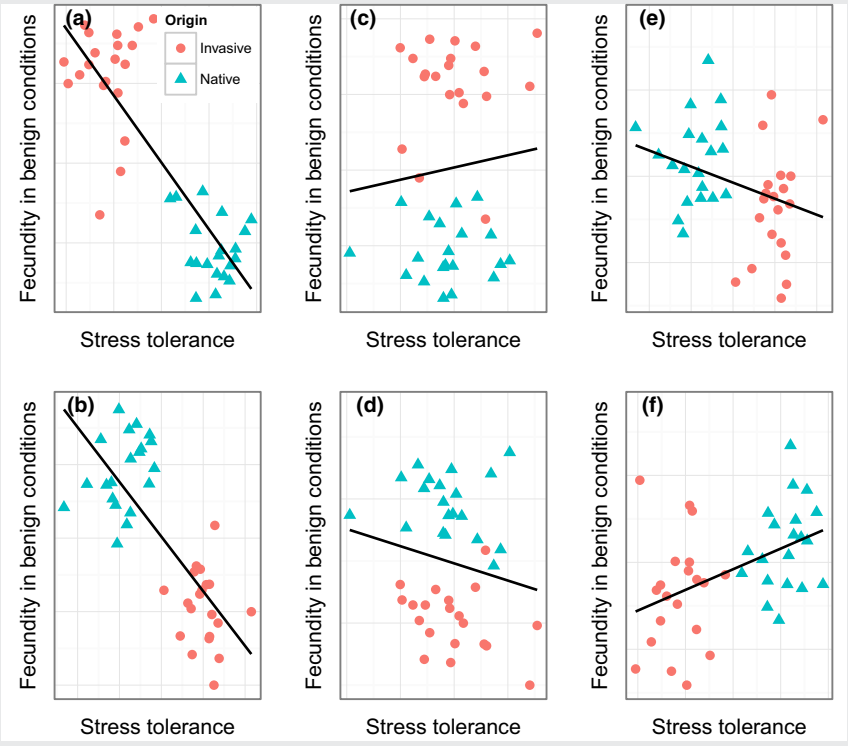

Fig. 2 Simulated population means suggestive of evolution in the invaded range in a two-trait comparison. (a) and (b) represent trade-offs in resource allocation for increased fecundity (a) or increased stress tolerance (b) in the invaded range. (c–f) represent the result of introduction bottlenecks for either increased (d) or decreased (e) fecundity, or increased (e) or decreased (f) tolerance, but without correlated change in the other trait.

The most studied hypothesis in invasive plant species is the evolution of increased competitive ability (EICA) hypothesis, which posits that selection will favour genotypes with reduced allocation to herbivore defence and increased allocation to growth, reproductive output or competitive ability in the absence of herbivores characteristic of the native range (Blossey & Notzold 1995). Although increased performance in invasive individuals relative to natives is often observed in common garden experiments, shifts in defences are less common (Kumschick *et al.* 2013). As EICA is only supported if increases in plant growth are linked to decreases in defence, evidence for EICA is equivocal (Felker-Quinn *et al.* 2013).

Trade-offs in invasive plant species may not be limited in response to herbivore defence only, but may include tolerance to stressful abiotic conditions (Bossdorf *et al.* 2005; He *et al.* 2010; Turner *et al.* 2014). Such trade-offs have been demonstrated in both natural and invasion contexts. For example, tolerance to serpentine soils in serpentine sunflower or drought in common ragweed comes at the expense of competitive ability and growth rate (Sambatti & Rice 2007; Hodgins & Rieseberg 2011). These studies and others suggest that invasive individuals may evolve a lower tolerance to biotic or abiotic stress to increase competitive ability, vigour and/or fecundity and therefore will perform relatively poorly under stressful conditions (Hodgins & Rieseberg 2011; Lachmuth *et al.* 2011; Kumschick *et al.* 2013;

Turner *et al.* 2014). However, detecting a trade-off that has occurred can be complex and can depend on testing performance under the correct stressor. Trade-offs can occur in multiple directions (Fig. 2) and may include dispersal or competitive ability rather than a simple two-way relationship between performance and defence or tolerance (e.g. Burton *et al.* 2010). To thoroughly investigate potential trade-offs in invasive plant species, future studies should consider defence responses to specialist and generalist herbivores separately (Joshi & Vrieling 2005), differentiate constitutive and induced resistance (Kempel *et al.* 2011), assess the level of resistance in different plant tissue types (young and old, above and below ground; Alba *et al.* 2012) and account for confounding abiotic factors, latitudinal origins and climate effects (Colautti *et al.* 2009; Felker-Quinn *et al.* 2013; Rypel 2014). Furthermore, strategies favoured by selection may change over time, between different phases of an invasion (Dietz & Edwards 2006) or depending on the habitats invaded (Lachmuth *et al.* 2011).

native-range clines (Gilchrist *et al.* 2001, 2004, 2008; Hoffmann *et al.* 2002). Likewise, thermal adaptations in body size between a specialist and a generalist invasive *Drosophila* species showed positive corresponding variation between altitudinal and latitudinal gradients (Folguera *et al.* 2008). Finally, similar altitudinal clines in growth and reproductive traits were found for native and invasive populations of Asteraceae plants (Alexander *et al.* 2009).

Although the prevalence of post-introduction adaptation is well established, at least two critical areas remain to be investigated. First, post-introduction evolutionary change complicates risk-assessment schemes which aim to quantify the invasion potential of individual taxa (Whitney & Gabler 2008; Box 3). Such schemes would benefit from the development of better metrics of genetic variation, hybridization propensity and other features associated with adaptive potential. Second, and most importantly, it remains unclear how important post-arrival adaptation is to invasion outcomes. In other words, does evolutionary change ever tip the scales from a failed to a successful invasion? Or does such change simply accelerate the rate (or impact) of an invasion that would have been successful anyway? Answering these questions could close a major chapter in our understanding of the relative importance of evolutionary vs. ecological factors in invasions.

Phenotypic and developmental plasticity

As an alternative to coping with novel environmental conditions through local adaptation (above), a successful invader might employ generalist strategies that produce high performance under a wide range of conditions. In his classic study, Baker (1965) introduced the term 'general purpose genotype' to describe a genotype that possesses broad environmental tolerance and should be frequently found in weeds. Here, we consider the general purpose genotype in the broader framework of phenotypic and developmental plasticity.

In the context of invasions, we are most interested in plastic responses that confer fitness advantages to invaders. Richards *et al.* (2006) outlined three possible scenarios by which an invader may benefit from phenotypic plasticity: (i) plastic responses in morphological and physiological traits that permit fitness to be maintained across different stressful or unfavourable environments (i.e. fitness homeostasis), a 'Jack-of-all-trades' strategy; (ii) an invader may increase its fitness under favourable conditions (i.e. opportunism), a 'Master-of-some' strategy; or (iii) a combination of the first two strategies (i.e. 'Jack-and-Master'), which permits both fitness homeostasis and opportunism.

Many studies have compared phenotypic plasticity of invasive vs. noninvasive species and populations (Rice & Mack 1991; Sexton *et al.* 2002), and in some weedy plants, plasticity has also been shown to be adaptive (Funk 2008; Hahn *et al.* 2012b). However, recent meta-analyses (Davidson *et al.* 2011; Palacio-Lopez & Gianoli 2011) came to conflicting conclusions concerning whether plasticity is generally important in invasions, a result that might be due to the transient evolution of plasticity itself (Lande 2009; Sultan *et al.* 2013). Thus, future meta-analyses should consider time since introduction in the interpretation of such data sets.

Theory predicts that frequent fluctuations of the environment will select for phenotypic and developmental plasticity (Meyers *et al.* 2005), while infrequent fluctuations favour local adaptation. Possibly, anthropogenic disturbance in the ancestral range may select for plastic genotypes that are then pre-adapted for invasion elsewhere (Hufbauer *et al.* 2012). Adaptation to extreme environmental changes, such as at the start of a biological invasion, can also favour rapid evolutionary increase of plasticity (Lande 2009, 2015). After an initial benefit from plasticity, however, an invader may lose its ability to express different phenotypes in favour of the expression of a locally adapted fixed phenotype through genetic assimilation (Pigliucci & Murren 2003; Lande 2015). The latter prediction assumes that there are costs and constraints associated with the maintenance of the genetic and physiological machinery required for plasticity (van Kleunen & Fischer 2005). The transient nature of plasticity was recognized by the

Box 3 Adaptation in biological control agents

Post-introduction adaptation is of particular interest in introduced biological control agents (Roderick *et al.* 2012) because it can lead to unwanted host shifts or other nontarget interactions in the introduced range. Even without evolutionary change, predicting the ecological impact of biological controls is difficult (Louda *et al.* 2005). The Baker and Stebbins (1965) contributors shared these concerns and discussed the likelihood of host shifts and post-introduction adaptation in biological control species (DeBach 1965; Wilson 1965). Collaborative work between geneticists and biological control workers was encouraged, with the goal of identifying adaptive genetic changes in introduced biological control populations.

Recent data confirm that biological control agents can evolve post-introduction. A well-known example is the cane toad, which was introduced into Australia 70 years ago to control populations of the cane beetle. Unfortunately, cane toads eat essentially anything that moves, spread disease, are toxic to naïve predators and have evolved longer legs—factors that have contributed to the species' rapid spread and devastating ecological impact (Fig. 3; Roderick *et al.* 2012; Rollins & Shine 2015). Phylogenetic studies of host shifts, for example interkingdom host jumps and changes in habitat preference, nutrition mode and ecological role in the fungal genus *Trichoderma* (Chaverri & Samuels 2013), provide an additional cautionary note. Such results should provide pause to the biological control community, as host shifts and other nontarget impacts may become more likely with evolutionary change.

While host shifts remain challenging to predict, modelling techniques using food networks successfully predicted host shifts from native herbivores to non-native plant species in central Europe (Pearse & Altermatt 2013). Also, genetic improvement of biological control agents through artificial selection (Roderick *et al.* 2012) has the potential to sharpen target specificity and impact, reduce nontarget effects and possibly slow evolutionary responses to new environments.

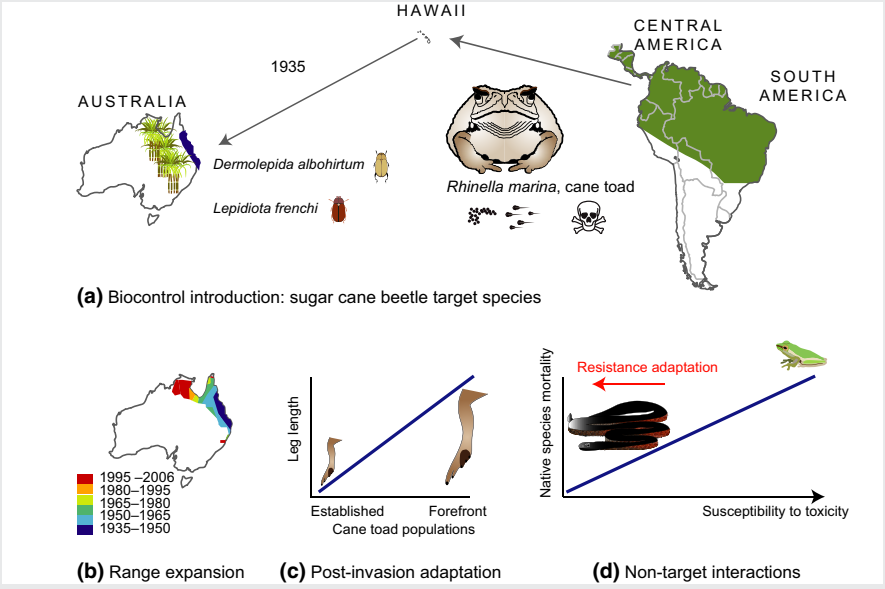

Fig. 3 The cane toad (*Rhinella marina*) was introduced (a) to control cane beetles in sugar cane fields in northeastern Queensland in 1935 after successful use in Hawaii. Since then, cane toads have (b) expanded across tropical and subtropical Australia (Urban *et al.* 2008), increasing their rate of spread through, among others, (c) evolution of longer legs (Phillips *et al.* 2006). Cane toads eat a wide variety of nontarget invertebrates reducing their population sizes. (d) Most native predators have declined as well due to lethal toxic ingestion of the toads, tadpoles and/or eggs with one known exception: the Australian black snake that has evolved physiological resistance to cane toad toxins (Phillips & Shine 2006; Shine 2010).

Baker and Stebbins (1965) contributors, who considered the future of general purpose genotypes to be 'rather dim' (Mayr 1965, p. 171). There were also suggestions that plasticity was associated with autogamy, apomixis, vegetative reproduction, hybridization and polyploidy (Baker 1965). While heterosis (whether expressed in diploid or polyploid hybrids) is known to stabilize fitness across environments (Schlichting 1986; Lippman &

Zamir 2007), evidence for the other proposed associations remains sparse (te Beest *et al.* 2012; Hahn *et al.* 2012b).

Conceptual and theoretical work during the latter half of the 20th century predicted that two main kinds of genetic mechanisms would be responsible for phenotypic plasticity: (i) loci with environmentally sensitive alleles and (ii) regulatory loci that modify gene expression levels across different environments (Via *et al.* 1995). Since then, numerous 'plasticity genes' have been cloned and characterized and the molecular genetic mechanisms underlying plasticity are more diverse than previously surmised (reviewed in Des Marais *et al.* 2013; Pierik *et al.* 2013). While the majority of loci are environmentally sensitive loci such as photoreceptors or regulatory loci, other kinds of genes are involved as well. There is also evidence that epigenetic modifications such as DNA methylation and chromatin modification play a role in adaptive plasticity, in some cases by providing a type of epigenetic memory that enables accurate prediction and response to future conditions (Bastow *et al.* 2004). A future focus should be to link such mechanisms to evolutionary changes in plasticity that have accompanied biological invasions.

Genetic architecture and invasion

Understanding the genetic and molecular mechanisms that underlie the formation of invasive genotypes has been a central goal of invasion genetics, yet knowledge on the topic remains limited. The few currently available examples indicate—in agreement with theoretical expectations—that invasiveness is often underpinned by a small number of genes. Moreover, rapid evolution in invasive taxa does not appear to be mutation limited (above). Below, we discuss the genetic architecture of invasiveness in the framework of two general approaches, top-down (or forward) genetics and bottom-up (or reverse) genetics.

The top-down approach. The top-down approach starts with knowledge on the phenotypic traits that vary between invasive and noninvasive genotypes, or that have been targets of selection during the evolution of invasiveness. The task then becomes to identify loci that underlie those traits. This can be achieved through candidate gene analyses and through genomewide association or quantitative trait locus (QTL) mapping.

In some cases, dissecting the genetic basis of invasiveness can be relatively straightforward, if a list of candidate genes known to affect the phenotypes under investigation is available. Some of the best-known invasiveness genes come from studies in this category. One example comes from studies of the fire ant (*Solenopsis invicta*), in which multi-queened introduced populations are more ecologically destructive and show less aggression to conspecifics than single-queened native populations (Porter & Savignano 1990). Krieger & Ross (2002) were able to identify *Gp-9*, a gene that encodes an odorant-binding protein, as the locus underlying polymorphism in this social behaviour in *S. invicta*. Another example is the dopamine receptor D4 gene, which is associated with novelty seeking and activity behaviour in introduced populations of yellow-crowned bishops (Mueller *et al.* 2014).

More often than not, no information is available on the likely genetic underpinnings of invasiveness. In this case, efforts have been directed towards finding associations between genetic markers and phenotypes of interest in pools of unrelated individuals, or in experimental populations derived from crosses between parents that show extreme trait values.

This latter approach, known as QTL-mapping, has been used with some success in weed genomics (Basu *et al.* 2004). In allopolyploid invasive Johnson grass (*Sorghum halepense*), Paterson *et al.* (1995) used crosses between the two species progenitors to understand the genetic basis of rhizomatousness, a weediness trait in this system. A small number of QTLs, most of which show additive or dominant gene action, were identified. More recently, Whitney *et al.* (2015) investigated loci involved in adaptive introgression associated with range expansion in the natural hybrid sunflower *H. annuus texanus*. Three donated QTLs were found that increased components of male and female fitness in the recipient species, likely as pleiotropic effects of phenological and architectural trait QTLs that colocalized with the fitness QTLs.

The bottom-up approach. The bottom-up approach does not require prior knowledge on traits that contribute to the propensity to invade. Instead, this strategy involves searching for changes in gene expression or allele frequency between pools of native and invasive genotypes, and making inferences about the traits involved based on knowledge of gene function.

Transcriptome analyses use microarrays or direct sequencing of RNA to identify genes that are differentially expressed in native and invasive genotypes. Lockwood & Somero (2011), for example, investigated the transcriptional response to low-salinity stress in two species of blue mussels (genus *Mytilus*). One of these, *M. galloprovincialis*, is invasive and has spread along the Pacific coast of California except areas North of Bodega Bay. This area is characterized by lower salinity and is still dominated by the native species *M. trossulus*. The authors performed a microarray analysis of *M. galloprovincialis* and *M. trossulus* individ-

uals grown under benign conditions as well as those simulating abrupt decreases of salinity. Results revealed that most differentially expressed genes in response to salt stress are shared between the two species. Thus, either a small number of genes limit the spread of the invader, or most species-specific differences in tolerance to osmotic stress are mediated downstream of transcription (Lockwood & Somero 2011).

Similar studies have been performed for invasive plants. Hodgins *et al.* (2013), for example, examined differential gene expression between native and invasive genotypes of common ragweed (*Ambrosia artemisiifolia*) across 45 062 unigenes. In this case as well, a small fraction of the genes were differentially expressed between native and invasive samples. The functional categories over-represented among the differentially expressed genes were also in agreement with results from a common garden experiment in this system (Hodgins & Rieseberg 2011) and highlighted genes involved in oxidoreductase activity, response to blue light, as well as abiotic and biotic stress response, as strong candidates for invasiveness genes in this system.

At the genome level, bottom-up approaches rely on finding the signature of positive selection, which can include regions that show high levels of genetic differentiation or shifts in the site frequency spectrum of mutations. Puzey & Vallejo-Marín (2014), for example, performed one such genome scan analysis to detect the signature of positive selection during the invasion of monkeyflowers (*Mimulus guttatus*) in the UK. While a specific target of selection was not identified, genes located in swept regions were shown to be associated with flowering time, as well as biotic and abiotic stress (Puzey & Vallejo-Marín 2014). Moreover, two of these regions were positioned near or at a chromosomal inversion polymorphism associated with a number of morphological and life history differences in monkeyflowers (Puzey & Vallejo-Marín 2014).

In another recent example, Vandepitte *et al.* (2014) investigated the genetic basis of adaptation following the 1824 introduction of the Pyrenean rocket (*Sisymbrium austriaum* subsp. *chrysanthum*) in Belgium using native, contemporary invasive samples and herbarium specimens collected in the introduced area. Six genes involved in flowering were identified as outliers of genetic differentiation and experienced allele frequency changes over the course of the invasion process.

A concern with the bottom-up approach is false positives, which can arise due to nonequilibrium demographic histories (Lotterhos & Whitlock 2014), as well to genomic heterogeneity in mutation and recombination rates (Renaut *et al.* 2014). These issues can be especially problematic in invaders, as generally little is known about their genomes. Also, as previously discussed, populations at the invasion front undergo extreme drift, allowing neutral and deleterious alleles to surf to high frequency, mimicking the signature of selection. Further, the loci identified as 'invasion loci' remain hypotheses until further work confirms that they control actual invasiveness in the field.

The small number of studies investigating the genetic architecture of invasiveness currently precludes the making of many generalizations. It is unclear, for example, whether and how often the genetic architecture of invasiveness traits differs from that of other traits differentiating natural populations or species. For example, are recessive QTLs more frequently established in invasive populations? Theory predicts that the probability of fixation for advantageous mutations is higher if they are dominant (Haldane's sieve; Turner 1977). Because of frequent bottlenecks, this process might be less effective in invasive populations. Also, the extent to which evolution re-uses the same genes or genomic regions during the evolution of invasiveness remains unclear.

Conclusions

We have learned a great deal about invasion genetics since the *Genetics of Colonizing Species* was published 50 years ago. Thanks in part to the widespread application of molecular marker techniques, we have elucidated the geographical origin(s) of many invaders, as well as their invasion routes. We have discovered that invaders are surprisingly variable genetically and that their variability depends in large part on whether they result from single or multiple introductions. Strong evidence has accumulated in favour of a positive role for intraspecific admixture, hybridization and polyploidy in invasion success. On the other hand, the balance of evidence indicates that population bottlenecks and genetic drift likely have negative or no effects on invasion success, despite the potential for gains in additive genetic variation or increases in the frequency of peak shifts. We understand the environmental conditions favouring the evolution of phenotypic and developmental plasticity and have cloned and functionally characterized genes underlying plasticity and invasiveness. Most importantly, we now know that rapid adaptation of invaders is common and generally does not appear to be limited by genetic variation.

In addition to the things we think we know, certain hypotheses appear to be gaining support, while others are falling out of favour. For example, both theoretical and empirical evidence suggests that natural selection in invaders relies mainly on standing genetic variation. Likewise, there is increasing support for evolutionary trade-offs between abiotic stress tolerance and growth

and reproduction, but support for similar trade-offs involving resistance to biotic stress appears to be declining (Box 2).

There also are numerous things that we don't know, which we have highlighted throughout this review and below, including:

- the relative roles of the numeric and genetic diversity components of propagule pressure in successful invasions;
- why hybridization sometimes results in increased colonization success and sometimes does not;
- whether chromosomal rearrangements, epigenetic modifications and shifts in genome size are important contributors to invasion success;
- whether the accumulation of deleterious mutations limits invasions and/or if compensatory mechanisms reduce the severity of expansion load;
- what traits or trait combinations, if any, best predict invasion success;
- why some invaders exhibit strong local adaptation and others do not;
- the generality and main cause of the lag phase;
- whether phenotypic plasticity evolves in a predictable way during the course of an invasion;
- which of the different strategies by which an invader may benefit from adaptive plasticity are most frequent;
- whether the genetic architecture of invasiveness traits differs from that of other traits differentiating natural populations or species;
- the extent of gene re-use during the evolution of invaders.

We also have suggested experiments or approaches to answer these questions. For example, many unknowns relating to sources of genetic variation, chromosomal rearrangements, genetic load, genetic architecture and gene re-use will fall to the power of evolutionary genomic approaches, perhaps within the next decade. Other questions will be more challenging, but manipulative experiments in model organisms have potential for decoupling the numeric and genetic diversity components of propagule pressure, assessing the potential role of epigenetic modification in the colonization of new habitats, and testing theoretical predictions regarding the temporal evolution of phenotypic plasticity. Of course, such experiments complement rather than replace classical reciprocal transplant and field-based selection experiments, which are required to connect trait variation with components of fitness and population growth rates. There also will continue to be a place for comparative studies, especially those that consider context, such as phylogenetic relationships, latitude, environment and the stage of the invasion, when making such inferences.

The Baker and Stebbins (1965) contributors were keenly aware that findings on the genetics of colonizing species, while fascinating in their own right, were important because they ramified throughout evolutionary biology. Evolutionary genetic studies of invasions tell us how species are likely to respond evolutionarily to changes in their environments, whether these changes come about through range expansions or occur *in situ*. We wish to know: What strength of selection (and over how long a period) can populations withstand? How much can they change their phenotype? How is their future evolutionary potential affected by past evolutionary change? More generally, what are the limits to evolution? These questions have become increasingly important as organisms must adapt to a changing world or face extirpation. Evolutionary genetic studies of invasive species have given us hope for the future by demonstrating multiple strategies by which organisms successfully respond to new environments, including rapid evolutionary change. Over the next 50 years, we expect studies of invasion genetics to reveal the limits to evolution (Blows & McGuigan 2015; Day 2015), as well as practical strategies to either minimize evolutionary change (such as in biological control agents) or maximize evolutionary potential (such as in native species facing environmental challenges), depending on the desired outcome.

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Glossary

Adaptive introgression	The movement of fitness-increasing alleles from one species to another via hybridization and backcrossing
Additive genetic variance	The proportion of genetic variance in a phenotypic trait that is due to the additive or main effect of alleles
Adaptive peak	A high point on an adaptive landscape (the surface of a three-dimensional graph that is used to visualize the relationship between genotypes or phenotypes and fitness)
Allee effect	The phenomenon by which the per capita rate of increase decreases, or reaches 0 or negative values, in populations for which conspecifics are not numerous enough
Dominance genetic variance	The proportion of genetic variance in a phenotypic trait due to dominant gene action, which is an interaction between alleles at the same gene locus
Epigenetic variation	Functionally relevant variation in the genome that does not involve modifications in the underlying DNA sequence, such as DNA methylation, histone modifications or noncoding RNA
Epistasis	The condition by which two or more independently inherited genes interact to control a phenotype
Epistatic (interaction) genetic variance	The proportion of genetic variance in a phenotypic trait that is due to interactions between alleles at two or more gene loci
Expansion load	Gradual accumulation of deleterious mutations during range expansion, which occurs because of increased genetic drift at the leading edge of the expansion front
Heterosis	Phenotypic superiority of a hybrid over its parents, due to increased levels of heterozygosity
Invasiveness	The ability of a species to become widespread when introduced to locations outside its natural geographical range
Lag phase	The time between initial introduction and subsequent rapid population growth
Linkage disequilibrium	Nonrandom association of alleles at two or more loci. Note that while such nonrandom associations most commonly result from genetic linkage, they can also arise due to selection or extreme drift
Local adaptation	Enhanced fitness of local populations compared to nonlocal populations, driven by spatial variation in selection pressures
Noninvasive species	A nonindigenous species that, contrary to an invasive species, does not achieve widespread distribution in its new environment
Outbreeding depression	The reduction in fitness for offspring resulting from crosses between individuals of different populations
Overdominance	The condition by which the heterozygote produces a phenotype more extreme than that of either homozygote
Phenotypic plasticity	The ability of a genotype to produce different phenotypes in response to environmental variation. This plasticity may be adaptive, maladaptive or neutral
Pleiotropy	The condition by which a gene affects more than one phenotypic character
Pre-introduction adaptation	A situation in which an invader is already well adapted to the conditions in its introduced range, typically because of a close match between the native and introduced environments
Propagule pressure	The total number of individuals introduced at a given location, which is the product of the number of introduction events (propagule number) and mean the number of individuals introduced per event (propagule size) (Lockwood *et al.* 2009)
R selection	Selection for increased rates of reproduction, associated with a reduced investment per capita offspring.
Realized niche	The niche an organism occupies in an environment as a result of factors such as competition for resources, which constrain the acquisition of the fundamental (or potential) niche
Selective sweep	Rapid increase in the frequency of an allele and nearby linked neutral variants under strong positive selection for the allele
Transgressive segregation	The formation of extreme phenotypes (relative to those of the parental lines) in segregating hybrid populations