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INVASION GENETICS: THE BAKER AND STEBBINS LEGACY

Transposable elements as agents of rapid adaptation may explain the genetic paradox of invasive species

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Abstract

Rapid adaptation of invasive species to novel habitats has puzzled evolutionary biologists for decades, especially as this often occurs in the face of limited genetic variability. Although some ecological traits common to invasive species have been identified, little is known about the possible genomic/genetic mechanisms that may underlie their success. A common scenario in many introductions is that small founder population sizes will often lead to reduced genetic diversity, but that invading populations experience large environmental perturbations, such as changes in habitat and environmental stress. Although sudden and intense stress is usually considered in a negative context, these perturbations may actually facilitate rapid adaptation by affecting genome structure, organization and function via interactions with transposable elements (TEs), especially in populations with low genetic diversity. Stress-induced changes in TE activity can alter gene action and can promote structural variation that may facilitate the rapid adaptation observed in new environments. We focus here on the adaptive potential of TEs in relation to invasive species and highlight their role as powerful mutational forces that can rapidly create genetic diversity. We hypothesize that activity of transposable elements can explain rapid adaptation despite low genetic variation (the genetic paradox of invasive species), and provide a framework under which this hypothesis can be tested using recently developed and emerging genomic technologies.

Keywords: evolutionary genetics, genomics, introduced species, loci of adaptation, mobile elements, naturalized species, nonfunctional DNA

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Rapid adaptation in invasive species

A critical factor in the success of invasive species is their ability to rapidly adapt to new environments following introduction. Identifying key traits that facilitate this rapid adaptation is therefore a major goal in both evolutionary and conservation biology as it can inform us about adaptation and provide opportunity to predict the invasive risk of species. Decades of ecological research have identified many shared ecological features amongst invasive species (e.g. fast growth rates, high fecundity, strong competitive ability, asexual reproduction, human usage), and shared features of invaded communities (e.g. islands, simpler communities and human modified habitats), and some common changes in ecological interactions (e.g. enemy release) following invasion (for review see Baker & Stebbins 1965; Richardson 2011). However, we know little about the genomic or genetic traits that may facilitate invasive success.

The amount and nature of genetic variation that is available to selection during invasions largely determines adaptive potential. Invasive populations often have reduced genetic variation following invasion as a consequence of a genetic bottleneck (Dlugosch & Parker 2008). This loss of genetic variation, and an increase in inbreeding and extinction probability, is thought to limit a population's ability to adapt to novel environments (Baker & Stebbins 1965; Frankham 2010). Indeed,

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the success of an invasive species is strongly dependent on the number of individuals released and the number of release events (Blackburn *et al.* 2015). The surprising ability of invasive species to adapt and colonize new habitats following genetic bottlenecks is referred to as the 'genetic paradox of invasive species' (Frankham 2004). There are several possible explanations that could explain this apparent paradox. Firstly, although lower genetic diversity is a common feature of invasive populations (Dlugosch & Parker 2008), there are cases where invasive populations have greater genetic variation than their native counterparts, presumably as a result of admixture between divergent source populations (e.g. Kolbe *et al.* 2004; Ferrero *et al.* 2015), or intraspecific hybridization in their new habitats (Verhoeven *et al.* 2010; Hohenlohe *et al.* 2013). Secondly, while bottlenecks reduce molecular genetic variation predominantly due to a loss of rare alleles (Wright 1921), their effect on the quantitative trait variation that underlies many fitness-related traits is not clear (Bryant *et al.* 1986; Reed & Frankham 2001; Turelli *et al.* 2006; Willi *et al.* 2006; Taft & Roff 2012). There may be little correspondence between molecular genetic variation and quantitative trait variation (Reed & Frankham 2001), and bottlenecks can increase the additive genetic variation of a trait (Bryant *et al.* 1986; van Heerwaarden *et al.* 2008) or alter the genetic background allowing populations to reach new adaptive peaks (Willi *et al.* 2006; van Heerwaarden *et al.* 2008). Finally, the paradox assumes a constant and low rate of mutation that is independent of processes occurring during introduction. However, mutational processes like transposable elements (TEs) could generate genetic variation in response to environmental and genomic perturbations, which could facilitate adaptation (Casacuberta & González 2013). The rate at which potentially beneficial alleles arise as a result of TE activity is likely to be higher than the background rate of mutation, thus providing opportunity for more rapid adaptation than would be possible from other mutational input. In this perspective, we propose that environmentally induced TE-mediated adaptation can facilitate adaptive potential of invasive populations (Fig. 1). We briefly introduce TEs and highlight their adaptive potential in invasive species, and provide a framework for testing the adaptive role of TEs in invasion, using recently developed and emerging genomic technologies.

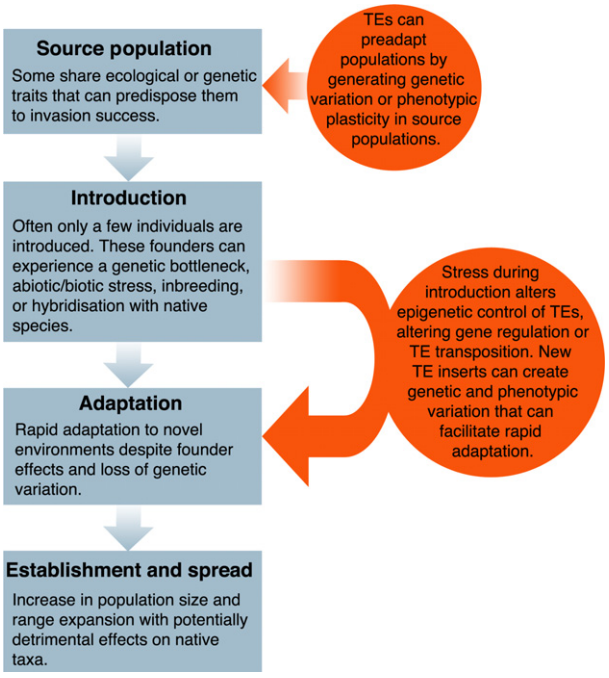

Fig. 1 A schematic of how transposable elements (TEs) could facilitate rapid adaptation in invasive species. TEs, first identified by Barbara McClintock (McClintock 1950), are genetic elements that can replicate and/or move to new positions in the genome. TEs constitute a large fraction of the total genomic DNA and regulate gene expression in many organisms (Schmidt & Anderson 2006; Biémont 2010). TE transcription and replication are controlled by epigenetic mechanisms, which are highly responsive to environmental and genomic stressors. Changes in the environment or hybridization can alter TE regulation of genes and increase TE copy number, generating novel genetic and phenotypic variation that could facilitate adaptation. Increased TE copy number can also promote ectopic (nonhomologous, illegitimate) recombination that can result in large-scale genetic changes (e.g. deletions, insertions) and chromosomal rearrangements (i.e. inversions, translocations). With respect to invasive species, TEs may play an important role in maintaining genetic variation, or contribute to phenotypic plasticity within source populations that could underlie invasive species success. Alternatively, stress experienced by invasive species during introduction could alter TE regulation of genes, increase TE copy number variation and generate small- and large-scale genetic variation.

Transposable elements and adaptation

Although often referred to collectively as a single group, TEs encompass a large diversity of genetic elements that vary in activity, sequence, transposition and replication mechanisms. TEs can be classified based on the presence of an RNA transposition intermediate: Class II or DNA transposons lack an RNA intermediate, while Class II or retrotransposons have an RNA intermediate. In addition, TEs are classified by their ability to transpose; those that are able to transpose on their own are termed autonomous, while those that require another TE to transpose are termed nonautonomous. The types and structures of TEs have been described in detail elsewhere (Slotkin & Martienssen 2007; Böhne *et al.* 2008; Chénais *et al.* 2012; Casacuberta & González 2013). In the process of transposition, TEs can duplicate,

translocate or invert large segments of DNA (DeBolt 2010), generating structural variation (SV), and can alter gene function and expression (Cowley & Oakey 2013). The pervasive view is that TEs insert at random locations with mostly deleterious effects, and their activity is suppressed by the host genome (Mills *et al.* 2007; Arkhipova *et al.* 2012). That a large proportion of TEs are not transcriptionally active and reside in heterochromatin regions supports this view (Lippman *et al.* 2004; Fedoroff 2012; Belyayev 2014). However, data are accumulating that demonstrate that TE activity may not necessarily be deleterious or random, and may have facilitated adaptation in many taxa (for reviews see Casacuberta & González 2013; Oliver *et al.* 2013; Schmidt & Anderson 2006). In rice, the TE *mPing* preferentially inserts into the 5′ flanking regions of genes, avoiding the exons, and it has either no detectable effect or upregulates nearby genes (Naito *et al.* 2009). Across taxa, adaptive TEs include retrotransposons, DNA transposons and both autonomous and nonautonomous elements (see Casacuberta & González 2013). At present, there are no obvious distinctions between random, highly deleterious TEs and targeted, potentially adaptive TEs based on their sequence or replication mechanisms (Class I or II, autonomous or not). However, we may expect that TEs involved in rapid or ongoing adaptation are likely to be those that respond to environmental stressors, are currently or recently transcriptionally active, reside in euchromatic regions and preferentially insert close to genes (i.e. in regulatory regions or introns).

The control of TE activity

The activity of TEs is largely governed by epigenetic mechanisms (Slotkin & Martienssen 2007) that can act pre- or post-transcriptionally and include methylation and RNA interference (Slotkin & Martienssen 2007). While our understanding of how applied stress alters epigenetics and TE activity is incomplete, a strong candidate mechanism is evident, DNA methylation (Rabinowicz *et al.* 2003; Slotkin & Martienssen 2007; Law & Jacobsen 2010). Reduced methylation (hypomethylation) is coincident with increased TE activity in several taxa including diatoms (Maumus *et al.* 2009), *Arabidopsis* (Melamed-Bessudo & Levy 2012), wallabies (O'Neill *et al.* 1998) and mice (Morgan *et al.* 1999). Targeted changes in methylation of specific TEs can also induce phenotypic variation. For example, coat colour variation in mice is induced via changes in methylation state of a transposable element in the *agouti* gene (Waterland & Jirtle 2003). The colour variation can be induced in genetically identical mice by dietary methyl supplementation (Waterland & Jirtle 2003). These alternative epigenetic states, termed epialleles, result in alternative phenotypes in the absence of DNA sequence variation. Phenotypic variation created by changes in methylation of TEs within genetically identical individuals could be an especially important source of phenotypic plasticity for bottlenecked, inbred, asexual and selfing species. The close correspondence between perturbations experienced by invasive species and those that alter methylation, including environmental stress (Verhoeven & van Gurp 2012), hybridization (O'Neill *et al.* 1998; Hegarty *et al.* 2011) and inbreeding (Vergeer *et al.* 2012), further supports the idea that TEs activated by environmental perturbation may facilitate adaptation in invasive species.

The role of TEs in invasion

Evidence supporting a role for TEs in invasion can be direct/causal, or indirect/correlational. The strongest evidence is direct and causal, and demonstrates that a transposable element is the molecular mechanism underlying a trait that aided invasion. Because of the challenges of characterizing, mapping and annotating repetitive elements such as TEs, pinpointing the molecular mechanisms that underlie adaptive phenotypes has mostly been restricted to characterizing, mapping and functional annotation of nonrepetitive DNA. Consequently, the best examples of an adaptive role of TEs during invasion and colonization come from the genetic model species *Drosophila melanogaster* (Barrón *et al.* 2014). Comparisons between populations of *D. melanogaster* revealed several TEs that are likely to have been positively selected during the flies' human-mediated colonization of North America less than 200 years ago (González *et al.* 2008). Most of the 13 putatively adaptive TEs were present in the ancestral African population and thus contributed to the standing genetic variation of these populations (González *et al.* 2008). Most recently, the phenotypic effects of one of these TEs (*pogo*) was characterized in great detail (Mateo *et al.* 2014). The presence of *pogo* results in increased activity of a protein, which protects flies from benzaldehyde, a natural xenobiotic, and from carbofuran, a synthetic insecticide (Mateo *et al.* 2014). The combination of population genetic analysis and functional annotation of this TE provides one of the most complete accounts of how TEs could be involved in adaptation during invasion.

Indirect evidence for a role of TEs in invasive species success comes predominantly from comparative studies comparing TE content or distribution, methylation and ploidy between invasive and noninvasive species or populations. Here, we provide a few examples of these in turn. A recent analysis of an invasive ant

(*Cardiocondyla obscurior*) genome suggests a role of TEs in adaptive evolution (Schrader *et al.* 2014). TEs are concentrated in islands of high TE density, these islands have higher rates of evolution and sequence diversity than regions of low TEs, genes within these islands also have higher rates of evolution, and genes with copy number variation were more commonly within TE islands (Schrader *et al.* 2014). In mosquitoes, the highly invasive and widespread species *Aedes agyptii* has tenfold more TEs than the locally restricted species *Anopheles gambiae* (Zhou *et al.* 2014). In Japanese knotweed, changes in methylation mediate invasion of diverse habitats (Richards *et al.* 2012), and in house sparrows, variation in methylation may compensate for decreased genetic variation facilitating invasion (Schrey *et al.* 2012). Polyploidy is positively associated with invasive species (Ainouche *et al.* 2009; Pandit *et al.* 2011), and bursts of activity of specific TEs following polyploidization have been recorded in plants. These studies identify important patterns, but can only speculate on the underlying processes. More studies that combine population genetics and functional annotation of TEs in invasive species are needed to confirm the role of TEs.

Identifying the processes underlying variation in the number of copies of TEs in the genome, whether due to drift or selection, is an important step in understanding how TEs facilitate invasion. There are numerous examples that TEs are under selection and are adaptive following biotic, abiotic or genomic stress (see Schmidt & Anderson 2006; Casacuberta & González 2013; Barrón *et al.* 2014). For example, in *D. simulans* TE copy number is positively correlated with temperature (Vieira *et al.* 1998) and elements have been positively selected in at least one introduced population (Schlenke & Begun 2004). In wild barley (*Hordeum*), the number of TE copies varies with elevation (Kalendar *et al.* 2000) and there is evidence from *Arabidopsis thaliana* that TEs close to genes are under purifying selection and maintained in the genome under positive selection (Hollister *et al.* 2011). Despite these examples, there is also compelling evidence that variation in TE copy number between populations may simply be driven by demography and drift. In *D. simulans*, activity of the *mariner* element is positively correlated with developmental temperature in the laboratory, but field surveys of populations in native and introduced sites found no consistent relationship between temperature and *mariner* activity: positive and negative correlations were identified across continents (Picot *et al.* 2008). Picot *et al.* (2008) concluded that TE copy number variation in these invasive populations was the result of inefficient selection in bottlenecked populations. Others have also argued that population demography, in particular population subdivision and bottlenecks, is responsible for driving patterns of variation in TE content between populations and species (Lockton *et al.* 2008; Jurka *et al.* 2011; Tollis & Boissinot 2013). Further, the accumulation of TEs in the genome is likely to be associated with increasing genetic load and consequently may constrain adaptation in the invasive population. As we consider the evidence for TEs in invasive species success, it is critically important that we distinguish between the role of selection and drift in driving TE evolution, and the role that TEs play in invasion success.

If TEs do play a role in rapid adaptation, then we must consider whether the abundance of TEs in invasive species genomes is causal, providing standing genetic variation and increases in phenotypic plasticity (i.e. preadapting), or consequentially arising from increased TE activity upon entering a new environment. TEs could be a mechanism that helps maintain standing genetic variation or phenotypic plasticity of a species prior to introduction. If this is the case, then we may expect to see greater TE copy number variation or TE-mediated phenotypic plasticity in invasive species in their native range compared to non-native sister taxa. Phenotypic plasticity has long been positively associated with invasive species success. Although a recent meta-analysis has confirmed this pattern in plants (Davidson *et al.* 2011), contrasting empirical and theoretical data exist that suggest it may not be a general pattern (see Lande 2015). Whether phenotypic plasticity is commonly mediated by TEs is unknown, but investigating how ecological factors that influence phenotypic plasticity, such as population stability, also influence TE activity would be useful. In contrast to TEs preadapting species, their responsiveness to the environment could also facilitate invasive success by releasing phenotypic plasticity (e.g. by altering methylation patterns) and creating novel genetic variation (Oliver & Green 2011). This adaptive change might occur as a consequence of the invasion process through captive breeding and selection for high growth rate, fecundity and competitive ability preceding introduction (Kitajima *et al.* 2006), immediately following introduction but prior to expansion (Colautti & Barrett 2013), or after introduction – during establishment and expansion (Phillips & Shine 2006). Thus, TEs could play a role in 'preadapting' species to invasion, or by generating genetic/phenotypic variation during or after introduction.

Future directions and challenges

We have considered the potential for TE activity to facilitate adaptation of invasive species. How then do we test the hypothesis that TE activity has facilitated rapid adaptation to new environments? We propose a framework to address this question that involves first

identifying a putative role of TEs in an invasive species, to test if the TEs have been under positive selection during invasion, and finally functionally annotated the TEs to confirm their phenotypic effects. This will require the integration of quality genomic and phenotypic data.

Genomics has transformed our ability to identify loci underlying adaptation by enabling the development of genetic resources in model and nonmodel organisms (Stapley *et al.* 2010; Ekblom & Galindo 2011). This explosion of genomic resources has led to the development of genomewide approaches to identify adaptation loci via whole-genome scans including quantitative trait locus (QTL) mapping, genomewide association studies (GWAS), outlier loci scans and tests for selection (for example, see Barrett & Hoekstra 2011; Jones *et al.* 2012; Andrew & Rieseberg 2013; Johnston *et al.* 2013; Linnen *et al.* 2013). These approaches, which we expand upon in the following sections, have different genomic and phenotypic data requirements, but the most useful approach will involve the integration of multiple approaches with high-quality genomic and phenotypic data. Development of quality genome assemblies is likely to present a significant challenge in nonmodel (and some model) species, because mapping structural variation (SV) and repeat regions in the genome requires long contiguous sequences that span the SV or repeat region (Treangen & Salzberg 2012; Hirsch & Buell 2013). Methods to assemble and annotate repeated regions such as TEs and SVs in the genome are developing rapidly (e.g. Kirkpatrick 2010; Alkan *et al.* 2011; Janicki *et al.* 2011; Hamilton & Buell 2012; Hirsch & Buell 2013; McCoy *et al.* 2014; Ritchie *et al.* 2014), and accurate genome assembly will also be enabled by emerging sequencing technologies that generate longer reads (Huddleston *et al.* 2014; McCoy *et al.* 2014).

In addition to quality genomic data, we must emphasize the importance of quality phenotypic data. Mapping adaptation loci is to a large part dependent on accurate ecological characterization of the properties, that is phenotypes, that facilitate invasion (Ungerer *et al.* 2007; Stapley *et al.* 2010; Barrett & Hoekstra 2011). These properties will be highly context- and organism-dependent and so characterization of what makes a species invasive requires careful ecological study (Bossdorf *et al.* 2005; Amundsen *et al.* 2012). Whereas genomic scans of selective sweeps within populations and differentiation between populations can proceed without detailed ecological knowledge (Stapley *et al.* 2010), subsequent analyses such as QTL mapping and GWAS will require well-defined hypotheses of biological pathways and clearly defined phenotypes that are of ecological importance in successful invasions (Barrett & Hoekstra 2011). To effectively resolve the relationship between genome features and invasive success is therefore heavily reliant on the inclusion of appropriate phenotypic information, which for many systems will be often harder to generate/collect than the genomic data.

Identify adaptive loci in invasive populations

Testing for selective sweeps and outlier locus scans

Outlier loci scans look for signatures of selection around selected loci, either by looking at the reduction in genetic variation along a genomic region (selective sweep) or by looking at differences in allele frequencies between populations (outlier loci). Both methods are influenced by the source of genetic variation, that is new mutation or standing genetic variation. Selection on standing genetic variation can produce (often small) shifts in allele frequencies at many positions in the genome (termed a soft selective sweep - Burke 2012; Hermisson & Pennings 2005). As the mutations were present prior to the onset of selection, there is no waiting time for beneficial mutations to arise (Messer & Petrov 2013). In contrast, selection for a new mutation leads to a rapid change in allele frequency at the selected locus, loss of variation at linked loci, but few changes in allele frequency at other genomic regions (a hard selective sweep - Orr 2005). Hard sweeps produce a loss of genetic variation from sites linked to advantageous mutations, generating 'signatures of selection' in the genome that, compared to soft sweeps, are relatively easy to detect (Hermisson & Pennings 2005; Sabeti *et al.* 2006; Burke 2012; Messer & Petrov 2013). To understand the fate of TE-induced adaptive alleles in a population and to map these loci, we need to consider whether adaptive TEs have generated new mutations or have contributed to standing genetic variation, and what sort of signatures of selection they might produce.

A proportion of the adaptive alleles created by TEs will be similar to new adaptive alleles created by other random mutational processes; they will arise at random, on a single genetic background after a relatively long waiting time and then undergo a hard selective sweep. In contrast, TEs that create adaptive alleles in response to perturbations will not have a long waiting time and may not create random mutations. For example, not all TEs respond to the same perturbations, with specific stressors known to activate specific TEs (reviewed in Capy *et al.* 2000; Schmidt & Anderson 2006), and there is evidence that TE insertion sites are not necessarily random (Naito *et al.* 2006). Thus, TE mutations may create the same phenotypic change in many individuals and genomic backgrounds, and as a result, the TE mutation will have attributes similar to standing genetic variation and undergo a soft sweep. TE-induced adap-

tive alleles therefore may share several features of new mutations and standing genetic variation, and they have the potential to produce hard and soft selective sweeps in the genome.

Another important point to consider when identifying loci using selective sweep-type analyses is that it may be difficult to identify the loci under selection if the selective sweep is very recent. Following a soft sweep, heterozygosity will remain high (no alleles fixed), while in the case of hard sweeps, rapid fixation of an allele can reduce variation across large regions in the genome, making it difficult in both cases to identify the causal locus. Further, selection is likely to be less efficient in the small population sizes that are typical of invading populations. Thus, the nature of the genetic variation and the time since invasion will influence our ability to detect adaptation loci using selective sweep-based approaches.

QTL mapping

One of the most promising and powerful approaches to map adaptation loci will be to take advantage of the fact that many invasive species will be amenable to controlled crosses in a laboratory or common garden setting. If invasive and native populations are not reproductively isolated and if large numbers of progeny can be generated, crosses are likely to rapidly fine-map adaptation loci of major contribution to differences in invasive phenotypes (Barrett & Hoekstra 2011). Similarly, in cases where populations display some diversity in a phenotype known to be linked to invasion success (due to, for example, an incomplete selective sweep), mapping loci contributing to within-population diversity can be achieved by crossing extreme phenotypes or generating recombinant inbred lines (RILs) and backcrosses (Barrett & Hoekstra 2011). Importantly, because QTL mapping determines regions of the genome contributing to trait variation, information is integrated across independent variants at the same genomic location (Lynch & Walsh 1998) as may be generated by site-specific TE activity. However, the utility of this approach will be limited if the invasive phenotype is fixed in the invasive population and it is not possible to cross the invasive population with the native population, or if crosses either between or within populations are unable to generate enough offspring to refine the genomic region. In the case where the invasive population is likely to have been founded by multiple source populations or has undergone hybridization in the new range, divergence and admixture mapping are promising approaches to identify adaptive loci (Crawford & Nielsen 2013). Divergence and admixture mapping will be useful to identify adaptive loci as they are minimally effected by bottlenecks; the best choice of approach will be dependent on the divergence between parental populations (Crawford & Nielsen 2013).

Genomewide association studies

GWAS utilize linkage disequilibrium (LD) between markers and causal variants to fine-map loci. In species with a relatively large effective population size, LD decays rapidly (Hill 1981), so population-based GWAS can provide finer precision of the underlying genetic basis of phenotypic differences than QTL mapping, particularly when large test-crosses cannot be established (Hirschhorn & Daly 2005). However, there are some caveats with respect to TEs and invasion events that we should consider when adopting a GWAS approach. Firstly, demographic factors like recent bottlenecks and admixture or hybridization are likely to generate high levels of genomic LD. GWAS may not provide greater mapping precision compared to family-based QTL mapping if there has been insufficient time for recombination to break up LD. Secondly, recombination is often suppressed in TE-rich regions; this is most evident in the heterochromatic regions (Rizzon *et al.* 2002; Dolgin & Charlesworth 2008), but even when TEs occur in the euchromatic regions or close to genes, recombination may be suppressed (e.g. Rizzon *et al.* 2002; Schrader *et al.* 2014). This reduction in recombination could increase regional LD and limit mapping precision.

Comparative genomic approaches

A promising approach to identify candidate adaptive loci, including TEs or SVs, is to use a whole-genome re/sequencing approach (Rubin *et al.* 2010; Hertweck 2013; Schrader *et al.* 2014; Soria-Carrasco *et al.* 2014) to identify loci that differ between native and invasive populations. By sequencing multiple invasive and native populations, and through either *de novo* assembly, or mapping sequences to a high-quality reference genome, we can identify copy number variation in TEs and identify other SV unique to the invaded population (s). For example, specific TEs may have a higher copy number in invasive species (Vieira *et al.* 1999; Schrader *et al.* 2014; Zhou *et al.* 2014), in introduced populations (González *et al.* 2010) or across environmental gradients (e.g. Vieira *et al.* 1998; Kalendar *et al.* 2000; Garcia Guerreiro & Fontdevila 2011). If genome sequence data and genetic map data are available, then estimates of recombination rate across the genome can be obtained. Comparisons of the distribution of recombination events and linkage disequilibrium (LD) between populations that have experienced stress or altered TE activity may provide some interesting insights into the

relationship between TEs and recombination. However, care must be taken when making comparisons between populations that have experienced recent bottlenecks, admixture or hybridization, because these demographic effects will increase the LD between loci. In the absence of whole-genome sequence data, comparison between transcriptome (RNA) sequence could also yield important insights. The RNA sequence data may include TEs close to genes and in regulatory regions, which are the TEs most likely to be involved in rapid adaptation (see above). Although comparisons across transcriptomes is unlikely to provide information about copy number variation, it can provide estimates of the number and diversity of TE families, and be a good starting point in species where little genomic data is available.

The problem with confounding effects of demography

Invasive species may be particularly useful for disentangling demographic effects from selection if multiple populations can be sampled. It is well known that demography and nonrandom sampling can confound the results of outlier scans and selective sweeps. In small populations, alleles may reach very high frequency due to drift rather than selection and selection can be inefficient at removing deleterious mutations (Excoffier *et al.* 2009). Given that many invasive populations are likely to have been founded by a small number of individuals, a major challenge will be to disentangle demographic history, particularly bottlenecks, from selection (White *et al.* 2013; Poh *et al.* 2014). These issues may be ameliorated by comparison of many populations, which is where invasive species provide unique opportunity. Many invasive species are widespread and represent multiple invasion events (Lee 2002), providing opportunity for studies across replicated independent introductions to identify parallel signatures of selection (White *et al.* 2013; Hodgins *et al.* 2015). Outlier loci that are shared amongst the replicates are most likely to reflect selection, and those that are not shared amongst replicates more likely indicate demographic events during invasion (White *et al.* 2013; Hodgins *et al.* 2015). In the case that few or no outlier loci are shared, it may still be possible to demonstrate a role of TEs in enhancing invasion success across populations. For example, taking outliers from all populations and explicitly querying whether TEs are more tightly linked with outlier loci than expected by chance could reveal directly the role of TEs in biological invasions. Correlation between ecological variables and allele frequencies across multiple populations (Kalendar *et al.* 2000; Coop *et al.* 2010; Garcia Guerreiro & Fontdevila 2011) may offer some testable hypotheses of the role of TEs in local adaptation, although again demographic history is likely to complicate such analyses. Further, if across populations certain gene ontologies are overrepresented in the outlier loci, then there is good evidence that the outliers are not the result of drift (Vandepitte *et al.* 2014).

Overall, particularly given the challenges associated with invasive populations including bottlenecks and strong selection, it is likely that complementary approaches that span population and quantitative genomics will be necessary to map and characterize loci of adaptation (Stinchcombe & Hoekstra 2008). In addition, understanding and exploiting differences in the ecology of the source and invasive populations may inform obvious candidate loci in a genomic region with signals of selection or that is linked to an invasive phenotype.

Linking TE activity to adaptive loci and invasions

Having identified a putative adaptation locus in a well-annotated genome, how can we link invasiveness to the presence or activity of a transposable element in that region of the genome? As discussed above, a well-assembled and annotated genome is key to the characterization of the role of TEs in adaptation, and methods to assemble and annotate TEs in the genome are developing rapidly (Huddleston *et al.* 2014; McCoy *et al.* 2014). Although only correlational, the presence of a TE or structural variation within a QTL or GWAS peak, a region with a signature of selection, and copy number variation in TEs in invasive populations, is strong support for the role of the transposable element in the success of the invasive population(s).

In the absence of a strong signal for adaptation in a single region of the genome, or if genomic information is only available for a single invasive population (for example, DNA from the native population and other invasive populations is not available), comparative genomics with other characterized species can offer important insight to relate genome size, TE content, inversions and gene duplications to invasive capacity (e.g. Kalendar *et al.* 2000; Vieira *et al.* 2002). Analysis of whole-genome sequences and genomic properties of a single population can also reveal the relationship between inversions, gene duplications and recombination to provide some indirect evidence of the role of TEs in adaptation (Völker *et al.* 2010; Schrader *et al.* 2014).

Although, functional annotation of noncoding variation is challenging in nonmodel species, several TEs have been charcterized in model species such as *Drosophila* (e.g. González *et al.* 2009; Schmidt *et al.* 2010; Mateo *et al.* 2014), the Silkworm (Sun *et al.* 2014), *Arabidopsis* (DeBolt 2010) and mice (Morgan *et al.* 1999).

These studies and the growing acknowledgement that much of the noncoding genome has important regulatory and functional roles (Cowley & Oakey 2013; Hangauer *et al.* 2013) is likely to encourage increasing research interest in improving our understanding of this part of the genome. The ever-increasing availability of genome sequence will improve our predictions of what is important and may offer important insight into the role of SV and TE in genome evolution.

Conclusive evidence of the role of TE in adaptation will require functional studies to link the presence or absence of TEs to changes in gene expression, using a combination of genomic and transcriptomic approaches. The most promising approach would be to combine RNAseq, to detect gene expression patterns, with, for example, bisulphite sequencing and MeDIP-seq methods to detect epigenetic modifications of the genome in common garden experiments (Frommer *et al.* 1992; Meaburn & Schulz 2012; Guio *et al.* 2014). Congruence between these complimentary approaches will be key to confirming the causal links between epigenetics, TEs and gene expression. Final confirmation of their functional role could be achieved with new methods of creating genetic knockouts in nonmodel species, such as CRISPR (Hwang *et al.* 2013).

Testing if TE activity was induced by environmental or genomic stress

If TE activity is linked to adaptation, it will useful to determine whether this activity was induced by environmental or genomic stressors. This will be best achieved within a laboratory setting, with the purpose of isolating a single putative stressor and recording changes in a variety of endpoints, such as TE copy number (DeBolt 2010; Hertweck 2013), epigenetic mechanisms (methylation patterns), gene expression (Slotte *et al.* 2013) or copy number variation (DeBolt 2010). Similarly, it may be possible to demonstrate that TE activity is indeed induced by a novel environment by transplanting individuals from the native into the invasive range and recording changes in TE activity. Demonstrating that transposons are actively moving in the genome, creating novel genetic variation on which selection can act, is relatively straightforward, especially if the copy number of the elements is increasing and good genomic data are available (Naito *et al.* 2006; Mills *et al.* 2007). Genomic technologies such as targeted enrichment (Teer *et al.* 2010) and long-read sequencing (Huddleston *et al.* 2014; McCoy *et al.* 2014) can, given the sequence of the TE, target sequencing of these elements and capture enough flanking sequence to be able to map them on an assembled genome. Tracking real-time TE activity through generations in a controlled laboratory setting (Naito *et al.* 2006) will enable a greater understanding of the action of TEs including whether they are targeted to particular DNA structures (e.g. heterochromatin vs euchromatin) and how this might impact the adaptive potential of these elements (Naito *et al.* 2009).

Concluding remarks

In this review and perspective, we highlight transposable elements as powerful mutational forces that can rapidly create genetic diversity, and further postulate that TE activity may be pivotal in determining the fate of biological invasions. Finally, we provide a framework in which these hypotheses can be tested that may provide a solution to the genetic paradox of invasive species. Transposable elements have historically been considered to be genomic 'parasites' or 'hitchhikers' and viewed in a negative context, but we suggest that TEs provide an intermediate between rapid adaptation without genomic sequence change that is provided by plasticity, and slow genomic evolution relying on mutational processes. By having near zero effects in benign environments, TEs remain invisible to selection until activated and these secret agents of evolution may provide genomes with rapid response mechanisms to replenish, or even generate *de novo*, genetic variance following genetic bottlenecks and severe environmental changes, providing bursts of allelic and phenotypic diversity upon which selection can act. We are now at the point in 'the genomic revolution' where the superficial questions have been addressed in many taxa and patterns of genomic organization revealed that were not previously imaginable. Using this wealth of data, we can begin to question emergent patterns of genomic elements previously considered nonfunctional and generate hypotheses that may further our understanding of the fundamental processes of adaptation.

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