Biological Invasion: The Influence of the Hidden Side of the (Epi)Genome

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Functional Ecology

DR DOMINIQUE BARLOY (Orcid ID : 0000-0001-5810-4871)
PROFESSOR CRISTINA VIEIRA (Orcid ID : 0000-0003-3414-3993)

Article type : Review

Section: Evolutionary Ecology
Editor: Dr Anthony Herrel

Biological Invasion: The Influence of the Hidden Side of the (Epi)Genome

Pierre Marin[1*], Julien Genitoni[2,3*], Dominique Barloy[2], Stéphane Maury[3], Patricia Gibert[1], Cameron K. Ghalambor[4], Cristina Vieira[1]

[1] Université de Lyon, Université Lyon 1, CNRS, Laboratoire de Biométrie et Biologie Evolutive UMR 5558, F-69622 Villeurbanne, France
[2] ESE, Ecology and Ecosystem Health, Agrocampus Ouest, INRA, Rennes, France
[3] LBLGC EA 1207, INRA, Université d'Orléans, USC 1328, 45067, Orléans, France
[4] Department of Biology and Graduate Degree Program in Ecology, Colorado State University, Fort Collins, CO, USA

* Equally contributed

Correspondence: cristina.vieira@univ-lyon1.fr

This article has been accepted for publication and undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/1365-2435.13317

Abstract

1. Understanding the mechanisms underlying biological invasions and rapid adaptation to global change remains a fundamental challenge, particularly in small populations lacking in genetic variation. Two under-studied mechanisms that could facilitate adaptive evolution and adaptive plasticity are the increased genetic variation due to transposable elements, and associated or independent modification of gene expression through epigenetic changes.

2. Here we focus on the potential role of these genetic and non-genetic mechanisms for facilitating invasion success. Because novel or stressful environments are known to induce both epigenetic changes and transposable element activity, these mechanisms may play an underappreciated role in generating phenotypic and genetic variation for selection to act on. We review how these mechanisms operate, the evidence for how they respond to novel or stressful environments, and how these mechanisms can contribute to the success of biological invasions by facilitating adaptive evolution and phenotypic plasticity.

3. Because genetic and phenotypic variations due to transposable elements and epigenetic changes are often well regulated or "hidden" in the native environment, the independent and combined contribution of these mechanisms may only become important when populations colonize novel environments. A focus on the mechanisms that generate and control the expression of this variation in new environments may provide insights into biological invasions that would otherwise not be obvious.

Global changes and human activities impact on ecosystems and allow new opportunities for biological invasions. Invasive species succeed by adapting rapidly to new environments. The degree to which rapid responses to environmental change could be mediated by the epigenome – the regulatory system that integrates how environmental

and genomic variation jointly shape phenotypic variation - requires greater attention if we want to understand the mechanisms by which populations successfully colonize and adapt to new environments

Keywords

Adaptation, Biological Invasion, Epigenetics, Phenotypic Plasticity, Transposable Elements

Introduction

In the face of current and future environmental changes, there are two possible outcomes for surviving organisms: to shift their geographical range or to adapt (Aitken *et al.*, 2008). Dispersal to more suitable geographical areas is possible for some organisms but can be challenging for others depending on the mode of dispersal. Adapting to new environments can occur either through adaptive phenotypic plasticity or adaptive evolution. Phenotypic plasticity is defined as the ability for a genotype to express several phenotypes according to different environmental cues and is adaptive when the environment shift the distribution of phenotypes towards the local optimum (Ghalambor *et al.* 2007). Such plastic responses represent the initial morphological, physiological, or behavioral response to environmental change (Pigliucci *et al.* 2006). Evolutionary responses occur across generations and the rate at which populations can evolve is predicted to be a function of the strength of selection and the amount of standing genetic variation (*e.g.* Lande & Arnold, 1983). Adaptive plasticity has been proposed as one mechanism allowing for populations to colonize and persist in new environments (Davidson, Jennions, & Nicotra, 2011; C. K. Ghalambor, McKAY, Carroll, & Reznick, 2007; C. L. Richards, Bossdorf, Muth, Gurevitch, & Pigliucci, 2006). Yet, invasive populations also pose an interesting dilemma in that they often display evidence of rapid

adaptive evolution despite lacking genetic variation, thus invasive populations constitute good models to tackle the questions related to the mechanisms that contribute to adaptive plasticity and evolution (Simberloff & Rejmanek, 2011). A biological invasion can be defined as the success of a species to establish, develop and maintain populations outside its geographical area of origin ((Theoharides & Dukes, 2007); Fig. 1). The development of international trade and intercontinental transportation has accelerated the movement of non-native species to new habitats (Early *et al.*, 2016). These non-native species can cause damage to ecological systems (*e.g.* Lodge, 1993), human health and economy (*e.g.* Pimentel *et al.*, 2001). Biological invasions also represent 'natural experiments' for evolutionary biologists, allowing investigation of evolutionary processes in real time (Huey, Gilchrist, & Hendry, 2005).

The process of introduction into a new location and of spatial expansion from this point of introduction often imposes a transitory reduction in population size (*e.g.* Dlugosch & Parker, 2008; Peischl & Excoffier, 2015); Fig. 1). Such population bottlenecks are predicted to increase inbreeding depression, increase genetic drift and decrease genetic diversity. A variety of compensatory mechanisms may act to counter the loss of genetic variation and facilitate adaptive evolution for population expansions, such as hybridization, multiple introduction events, and propagule pressure, such that genetic diversity in the invasive and native area could be comparable or even greater in invaded regions (Baltazar-Soares, Paiva, Chen, Zhan, & Briski, 2017; Bock *et al.*, 2015; Estoup *et al.*, 2016; Facon *et al.*, 2011; Prentis, Wilson, Dormontt, Richardson, & Lowe, 2008; Wellband, Pettitt-Wade, Fisk, & Heath, 2017). However, studies of genetic diversity and adaptive change in invasive populations rarely take into account the potential role played by transposable elements (TEs) and epigenetic changes (Oliver & Greene, 2009; Oliver, McComb, & Greene, 2013; Rey, Danchin, Mirouze, Loot, & Blanchet, 2016; Schrader *et al.*, 2014; Stapley, Santure, &

Dennis, 2015). These mechanisms can act together to rapidly increase genetic and phenotypic diversity in a population. TEs are repeated sequences present in virtually all genomes. They behave "parasitically" within the genome in that they are able to replicate and insert themselves across chromosomes. The amount of the genome made of TEs can vary from a few percent, such as in yeast (3%; Kim *et al.*, 1998) up to 80% in maize (Schnable *et al.*, 2009; Vitte, Fustier, Alix, & Tenaillon, 2014). Despite variation in their activity and impact across species, TEs have been proposed to be a relevant source of genetic variation. For example, in plants, TEs are considered as a source of genetic and epigenetic variability and thus drivers of evolution (Belyayev, 2014; Lisch, 2013; Vitte, Fustier, Alix, & Tenaillon, 2014). Similarly, diversification and rapid evolution in angiosperms have been attributed to TE sequences (Oliver *et al.*, 2013). In animal model systems like *Drosophila*, TEs have been described as potentially playing a role in speciation since they can be responsible for hybrid incompatibility (Kidwell & Lisch, 2001). More recently, TEs have been hypothesized to facilitate adaptation in invasive species to new environments (Schrader *et al.*, 2014; Stapley *et al.*, 2015). A fundamental challenge in testing this hypothesis in natural populations is linking the genetic variation generated by TEs to changes in fitness. However, the relationship between TEs and fitness is mediated by a variety of mechanisms that have evolved to find and silence deleterious TE-induced changes in gene expression. These silencing systems are largely epigenetic mechanisms, which play a role in influencing gene expression and have been proposed to independently and jointly with TEs facilitate adaptive plasticity and evolution (Lanciano & Mirouze, 2018; Rey *et al.*, 2016; Stapley *et al.*, 2015).

Here we use a broad definition of epigenetics as any non-genetic molecular modification of the genome that alters gene expression. Epigenetic modifications are non-genetic changes in the sense that there is no DNA sequence change that is passed on in the germline, although there is evidence that some epigenetic modifications can sometimes be transmitted across

generations (Meyer, 2018). Epigenetic mechanisms encompass three levels of well characterized modifications: 1) DNA methylation, 2) histone modifications and 3) non-coding RNA (Allis & Jenuwein, 2016; Duncan, Gluckman, & Dearden, 2014), which can rapidly change gene expression and affect mobility of TEs. Epigenetic changes or epimutations refer to the stable modification of epigenetic marks (such as DNA methylation) on a locus that does not affect the DNA sequence. Epimutation on a locus generates an epiallele corresponding to one of two or more alleles of a given gene differing in their epigenetic properties and encoding different phenotypic characteristics. The epigenetic mechanism silencing TE insertions can be very effective, although the exact mechanism varies across species. For example, in *Drosophila*, silencing of TEs is mainly operated by histone modifications and small RNAs (Aravin, Hannon, & Brennecke, 2007) while in mammals and plants, DNA methylation is the major mechanism.

Both epigenetic mechanisms and TE activity can be sensitive to the environment (Fablet & Vieira, 2011; Lanciano & Mirouze, 2018). For example, histone modifications and DNA methylation have been shown to be modified by abiotic and biotic changes to the environment (Blake & Watson, 2016; Nätt & Thorsell, 2016; Alonso, *et al.* 2018), and the alteration of these epigenetic mechanisms can in turn alter TE induced changes that would otherwise not occur. Thus, the linkage between environmental change and epigenetic-TE behavior leads to the expectation that TEs and epigenetics can contribute to an increase of the genetic and phenotypic diversity following the colonization of a new environment. The environmental induction of these mechanisms can be placed into a larger conceptual framework that considers how phenotypic plasticity can facilitate evolutionary change.

Phenotypic plasticity provides a unifying conceptual framework for incorporating the molecular mechanisms that contribute to invasion success within and across generations. Plastic responses to the environment may be reversible or non-reversible depending on the trait (Piersma & Drent, 2003; Pigliucci, 2005; West-Eberhard, 2005), and they can be adaptive, non-adaptive, or neutral with respect to fitness (C. K. Ghalambor *et al.*, 2007). We can thus view the predictable environmental induction of TEs and epigenetic changes from the same perspective used to study other environmentally induced plastic traits (Horváth, Merenciano, & González, 2017). Indeed, the epigenetic changes associated with different environmental cues provide a mechanistic understanding for the observed patterns of phenotypic plasticity, and have been hypothesized to facilitate invasions by allowing organisms to express advantageous phenotypes across a broader range of environments (Baker & Stebbins, (1965); Bradshaw, 1965; Richards, 2006; Sultan, 2001). The hypothesis that greater plasticity contributes to the success of an invasion has been supported by some studies (*e.g.* Daehler, 2003; Davidson, Jennions, & Nicotra, 2011; Nyamukondiwa, Kleynhans, & Terblanche, 2010; Sexton, Mckay, & Sala, 2002; Trussell & Smith, 2000), but not by others (*e.g.* Chown, Slabber, McGeoch, Janion, & Leinaas, 2007; Godoy, Valladares, & Castro-Díez, 2011; Matzek, 2012; Palacio-López & Gianoli, 2011). Thus, while we have a compelling argument for how phenotypic plasticity and genetic variation can contribute to adaptive evolution and facilitate the invasion processes, we know far less about the contribution of TEs and epigenetic variation (Stapley *et al.*, 2015) and specific tests of these mechanisms in natural populations remain largely unexplored (Schlichting & Wund, 2014).

In this review, we first cover the mechanistic underpinnings of how mobile TEs and epigenetic systems alter genetic variation and gene expression. We use the conceptual framework of how phenotypic plasticity or how environmentally induced changes can facilitate invasions through these mechanisms. We first review the role of transposable elements as drivers of genetic diversity and the epigenetic system that regulates these genomic rearrangements. We discuss the evidence that TEs generate genetic variation, and environmental conditions can release this variation and make it available to selection. We then review the epigenetic mechanisms that control gene expression, the evidence for transmission across generations, the patterns of environmental induction, and how all these processes may contribute to invasion success. We conclude that by focusing on the epigenome (*i.e.* the regulatory system that integrates how environmental and genomic variations jointly shape phenotypic variation) we will gain key insights into the mechanisms that contribute to invasion success.

I. Transposable elements (TEs) in invasive populations

1. TEs and their regulation in the genome

Altered environmental conditions are thought to act as a source of selection that shifts genotype and phenotype frequencies towards new optima. The classical perspective is that selection acts either on standing genetic variation or on spontaneous mutations underlying the phenotypes under selection, leading to adaptive evolutionary change (Orr, 2005). However, there is an increasing appreciation that other types of genetic modifications could contribute to the genetic variation selection acts on. One such mechanism is the genetic variation generated by transposable elements (TEs). First discovered by Barbara McClintock in maize (McClintock, 1950), TEs are defined as mobile repeated DNA sequences that can move in the

genome by generating new copies of themselves and induce new mutations (Casacuberta & González, 2013; Chuong, Elde, & Feschotte, 2017; Slotkin & Martienssen, 2007). Because TEs can induce mutations in genes, alter gene regulation, and disrupt recombination they can have deleterious consequences (Slotkin & Martienssen, 2007), however they can also generate new variation on which selection can act (Kidwell & Lisch, 2001; Stapley *et al.*, 2015). TEs are formally classified into two families according to the transposition process. Type I, or retrotransposons, use an RNA intermediate to produce new copies of themselves. Type II DNA transposons code a transposase enzyme and transpose by a cut and paste mechanism (Fig. 2). The majority of TE insertions in a genome are neutral or slightly deleterious, such that only small effects are expected on individual fitness. However, when they insert in locations that disrupt gene function or change gene regulation TE can have negative or (rarely) positive fitness consequences (Lanciano & Mirouze, 2018; Slotkin & Martienssen, 2007). Negative impacts of TE insertion should be under natural selection and potentially purged but could also be a source of disease as reported in human health population studies (Wang & Jordan, 2018). Positive fitness effects of TE insertions are now also reported. For example, a role for TEs in the adaptive evolution was proposed in stress response of *Drosophila melanogaster* (González, Karasov, Messer, & Petrov, 2010) and *Arabidopsis thaliana* (McCue, Nuthikattu, Reeder, & Slotkin, 2012), as well as in plant defense against fungi (Hayashi & Yoshida, 2009) or osmotic tolerance (Ito *et al.*, 2016). Yet, to fully understand how TEs spread and are regulated throughout the genome it is important to understand the various mechanisms that have evolved to manage the consequences of these insertions.

Genomes and TEs are locked in an on-going evolutionary arms race, where TEs act like selfish “parasites” attempting to increase their numbers, and genomes respond with a variety of mechanisms to reduce the mobility of TEs and regulate the impacts of these

insertions on gene expression (Aravin, Hannon, & Brennecke, 2007; Slotkin & Martienssen, 2007). The regulation of TEs can occur both by transcriptional gene silencing (TGS) or post-transcriptional gene silencing (PTGS) (Castel & Martienssen, 2013; Pumplin & Voinnet, 2013; Slotkin & Martienssen, 2007) and different types of epigenetic regulation have been described in this process. These mechanisms can either use small RNAs, histone modification, or DNA methylation. Small non-coding RNA from the piwi RNA (piRNA) family are implicated in TE regulation either by TGS or PTGS. The piRNA can directly cleave transcripts of TE, avoiding the protein production and transposition, but they can also act on TE insertion and drive histone modifications which will silence the TE copies (Pedersen & Zisoulis, 2016; Sienski, Dönertas, & Brennecke, 2012; Slotkin & Martienssen, 2007; Song & Cao, 2017; Zhang *et al.*, 2016). These piRNA pathways are well conserved among eukaryotes (Fablet, Akkouche, Braman, & Vieira, 2014; Fablet, Salces-Ortiz, Menezes, Roy, & Vieira, 2017) and are considered a reliable immunity system contributing to the stability of the genomes (Sienski *et al.*, 2012; Slotkin & Martienssen, 2007; Zhang *et al.*, 2016). At the histone level, methylation of the histone 3 lysine 9 (H3K9me3) is known to limit TEs expression in *Drosophila* and mammals (Lee, 2015; Matsui *et al.*, 2010). Some data also indicates that the histone modifications that target a specific TE insertion can spread on the region and affect the expression of nearby genes (Elgin & Reuter, 2013; Lanciano & Mirouze, 2018; Lee, 2015; Rebollo *et al.*, 2011; Slotkin & Martienssen, 2007). DNA methylation also affects TE expression. For example, mouse embryo mutants for the DNA methyl transferase 1 (*Dnmt-1)* have higher levels of intracisternal A-particle (IAP) retrotransposon expression (Walsh, Chaillet, & Bestor, 1998). In rice, mutation of DNMT *OsMet1b* gene, reduced CG methylation and induced necrotic death in the seedlings (Yamauchi, Johzuka-Hisatomi, Terada, Nakamura, & Iida, 2014). DNA methylation can be induced by environmental conditions (see below) as shown with classic–examples of TE

generated epialleles. For example, in mice, the *agouti* gene inducing coat color modification is due to IAP retrotransposon presence, while in morning glory flowers (*Convolvulaceae*), a methylated MuLE transposon induces petal color streaks (Slotkin & Martienssen, 2007). Thus, despite the potentially negative consequences of TEs, there are a suite of mechanisms that allow organisms to not only mitigate these impacts, but to also coopt them in the process of adaptation. Yet, these mechanisms are also potentially sensitive to the environmental context organisms occur in, leading to the expectation that novel or stressful environments may induce changes that fuel evolution.

2. TEs and environmental stress

Environments that fall outside the historic or normal range experienced by a population may impose stress on organisms by challenging their physiological and cellular systems and reducing fitness. How stressful environments impact the mechanisms by which organisms cope with them is thus of great interest to those interested in invasion biology because invasive populations often encounter novel environmental conditions (*e.g.* Stapley *et al.*, 2015). Barbara McClintock was the first to hypothesize that the variation generated by TEs can over evolutionary time help populations survive of under stressful conditions, but limited empirical data were available to test these ideas at that time (Casacuberta & González, 2013). Specifically, novel or stressful environments have been implicated in increasing TE activity (Capy, Gasperi, Biémont, & Bazin, 2000; Lanciano & Mirouze, 2018) and disrupting the epigenetic regulation of TEs, such that previously silenced TEs become reactivated (Guerreiro, 2012; Horváth *et al.*, 2017; Stapley *et al.*, 2015; Vieira, Aubry, Lepetit, & Bie mont, 1998). The expectation is that such loss of control over TEs will promote a rapid increase in genetic and phenotypic variation available for selection to act on, and that in some of these cases TEs will evolve to become part of the adaptive stress response

(Guerreiro, 2012; Horváth *et al.*, 2017; Stapley *et al.*, 2015; Vieira *et al.*, 1998). However, in a recent review of published studies, Horvath *et al.* (2017) argue the links between stress and TE response are far from being a generality.

For TEs to become involved in the stress response there needs to be a functional link between them. In rice, Naito *et al.* (2009) found that *mping* TE insertions had no effect on the transcriptome under control conditions, but clearly affected the expression of nearby genes under stressful cold and saline conditions. There was no indication any of these changes were adaptive or whether the TE driven changes in gene expression were due to stress-induced disruption of the epigenetic regulatory mechanisms or some other mechanism. Using a comparative approach, González *et al.* (2010) identified several TE insertions that could potentially be implicated in the adaptation of *D. melanogaster* to the most southern and northern populations exhibiting clinal variation in Australia and North America. They examined both putatively neutral and adaptive insertions that had increased in these derived populations and found strong evidence for selection on a subset of the putatively adaptive insertions. When they examined the neighboring genes to these insertions, they found they were genes previously known to be involved in adaptation to a variety of environmental factors (González *et al.*, 2010). Other studies have also found direct links between the presence of the insertion and adaptive phenotypes related to insecticide resistance (Magwire *et al* ., 2011; Merenciano *et al* ., 2016), cold stress response, oxidative stress (Guio, Barrón, & González, 2014; Guio, Vieira, & González, 2018), xenobiotic stress (Mateo, Ullastres, & González, 2014), and resistance to sigma virus (Magwire *et al.*, 2011). Thus, there is growing evidence that TEs can be under selection to alter patterns of expression on neighboring genes; leading to adaptive changes in phenotypes.

3. TEs in the context of phenotypic plasticity and invasion

The potential for TEs to resolve the invasion paradox of rapid evolution in response to new environments despite small population sizes and reduced genetic variation, is in part, dependent on how TEs and their epigenetic control systems respond on ecological time scales (Stapley *et al.*,, 2017). In other words, there must be a predictable increase in genetic and phenotypic variation when populations colonize new environmental conditions, and this should be caused by either the increased production of TEs or the reduced control of TEs. If the expression of TEs predictably changes in response to environmental cues, then we can study the context dependent nature of TE activity as we would any other phenotypically plastic trait (Rey *et al.*, 2016; Stapley *et al.*, 2017). Thus, we can view how a change in environmental cues (*e.g.* a stressor) alters TE activity as a form of phenotypic plasticity, but we can also view how TEs change gene expression; providing a mechanistic explanation for patterns of phenotypic plasticity observed in fitness related traits. In either case, the expectation is that populations likely harbor hidden genetic variation caused by TEs that is rarely revealed in their native range, because selection shapes the epigenetic control mechanisms that tightly regulate TE activity (Slotkin & Martienssen, 2007). However, in novel environments where selection has not had an opportunity to act on the epigenetic control of TEs, we expect increased misregulation of the epigenetic control system which can lead to a "burst" of TE related variation (Stapley *et al.*, 2015). This burst of genetic and related phenotypic variation should fuel evolutionary responses to selection. Yet, to date no study has documented that these various mechanisms have directly contributed to the success of an invasive population, but various indirect lines of evidence suggest it is highly plausible.

The role of TEs in the success of invasive populations was reviewed by Stapley *et al.* (2015) where they outlined the potential ways in which TEs contribute to population expansion and adaptation, and then review the largely indirect evidence supporting this view. Here we briefly highlight two studies where TEs have been implicated in facilitating adaptation in invasive populations. Goubert *et al.* (2017) studied populations of the tiger mosquito *A. albopictus* in their native range of Vietnam and in their invasive range throughout Europe. They used TEs as neutral markers to identify genomic regions under selection (Goubert *et al.*, 2017). They found the majority of outlier loci had a higher frequency of insertions in the European populations, suggesting that TEs could be linked to genes that have a role in adaptation to temperate environments. Dennenmoser *et al.* (2017) studied copy number and TEs richness in the invasive hybrid sculpin fish, *Cottus spp*., and found an increase in TE copy numbers in invasive *Cottus* populations potentially caused by hybridization. In combination with the studies described above, these studies suggest we can no longer ignore the role of TEs in the evolutionary process, although we still lack a basic understanding of what role TE-related variation plays in the early stages of population divergence when invasive populations are colonizing new environments. Future studies will need to take advantage of recently established populations or experimentally generate new populations to explicitly test if TEs and their epigenetic control play a role in biological invasions.

II. Epigenetic mechanisms, a component of the rapid response to environmental changes in the invasion process

1. Epigenetic mechanisms play a role in response to environmental change

So far we have considered epigenetic mechanisms in the context of how they control TE activity, but far more research has investigated the independent effects of epigenetic changes for their role in altering patterns of gene expression in response to both biotic and abiotic environmental variation (Amarasinghe, Clayton & Mallon, 2014; Crisp, Ganguly, Eichten, Borevitz, & Pogson, 2016; Gómez-Díaz, Jordà, Peinado, & Rivero, 2012; Y. Song *et al.*, 2015; Spannhoff *et al.*, 2011). In a diversity of organisms, ranging from invertebrate to vertebrate animals and from annual to perennial plants, environmentally induced epigenetic changes can result in adaptive responses to new and stressful environments (Becker *et al.*, 2011; Bräutigam *et al.*, 2013; Conde *et al.*, 2017; Gibert, Peronnet, & Schlötterer, 2007; Kawakatsu *et al.*, 2016; Lafon-Placette *et al.*, 2018; Meyer, 2015; Schmitz *et al.*, 2011; Seymour & Becker, 2017). Some of the best examples of adaptive epigenetic effects are observed in plants. For example, following an abiotic or biotic stress plants will exhibit epigenetic changes that prime the defenses against future repeated stress, resulting in a "epigenetic memory" that and allows an individual to respond more quickly and effectively when the stress appears again (Lämke & Bäurle, 2017). The duration of the primed state within an individual is a key factor to survival and adaptation, and several studies have shown that the epigenetic modifications involved can be transgenerational (Mauch-Mani, Baccelli, Luna, & Flors, 2017).

The transgenerational nature of environmentally induced epigenetic variants (*i.e.* epimutations or epialleles) provides the critical link between plastic changes in one generation influencing the next generation. However, unlike sequence-based changes that are relatively stable and predictably transmitted regardless of environmental conditions, epigenetic variants occur at higher frequency, and exhibit rapid flexibility and reversibility (Heard & Martienssen, 2014; Law & Jacobsen, 2010). For example, with respect to frequency, the rate of epimutations has been estimated at $3x10^{-4}$ or five orders of magnitude higher than the DNA substitution rate in *A. thaliana* (10^{-9}; see van der Graaf *et al.*, 2015). At the same time, we also know that patterns of methylation and demethylation can rapidly shift in response to different environmental cues within and between generations, making epigenetic patterns also very flexible (Heard & Martienssen, 2014; Law & Jacobsen, 2010; Meyer, 2015). How well this epigenetic information is stored and transmitted from one generation to the next, and how effective this information is in preparing subsequent generations for responding to environmental challenges ultimately determines the importance of epigenetic variation for adaptive plasticity and evolution in response to new environments. The Arabidopsis 1001 Epigenomes project provides evidence that DNA methylation is correlated with geography and climate of origin and could be involve in local adaptation (Kawakatsu *et al.*, 2016). Schmid *et al.* (2018) reported in *Arabidopsis* that epigenetic variation is subject to selection and can play a role in fast adaptive responses. However, the relative extent to which genetic and epigenetic variation contribute to plant adaptation remains to be elucidated and likely depends on the reproductive mode of the investigated species. If environments are predictable across generations, heritable epigenetic changes could provide an adaptive anticipatory response. However, if environments change between generations, a mismatch can arise between the transmitted epigenetic information and the

environment of the descendants. Thus, a critical link that remains to be established is the degree to which heritable epigenetic variation underlies adaptive phenotypes.

The fundamental challenge facing researchers is to disentangle genetic and epigenetic effects on specific phenotypic traits. Establishing the genetic basis of any complex traits is already challenging given that many loci of variable effect all contribute to explain some of the phenotypic variation observed. Then, epigenetic control is an additive layer of complexity in direct interaction with genetic variation (*e.g.* control of TE insertion and spontaneous deamination of 5-methylcytosine into thymine) leading to a complex situation. One powerful approach that has been used to separate genetic from epigenetic effects is to create epigenetic recombinant inbred lines (epiRILs) (Johannes *et al.*, 2009; Reinders & Paszkowski, 2009) where epigenetic variants are placed on a homogeneous genetic background. This approach allows for the quantification of phenotypic variation between different lines and assumes they must be due to the epigenetic variants given the rest of the genomic background is held constant. Zhang, Fischer, Colot, & Bossdorf, (2013) used this approach in *A. thaliana* and demonstrated that epigenetic marks were responsible for phenotypic variance and for plasticity. Similar approaches reveal that for a diversity of traits epiRILs not only explain phenotypic variation, but also that transgenerational inheritance and patterns of plasticity may be purely caused by epigenetic effects (Bossdorf, Arcuri, Richards, & Pigliucci, 2010; Kooke *et al.*, 2015; Zhang *et al.*, 2013). For example, (Cortijo *et al.*, 2014) demonstrated that "Differentially Methylated Regions" (DMR) of epiRIL lines act as epigenetic quantitative trait loci for several traits and could be stably inherited and that DNA methylation could contribute to the phenotypic plasticity of a trait, especially in stressful conditions. The same result was found in the fungus (*Neurospora crassa)* using mutants for the different epigenetic pathways (DNA methylation, histone methylation and RNA interference; (Kronholm, Johannesson, & Ketola, 2016). Another similar strategy to isolate the epigenetic basis of traits

is to use clones as proposed by Conde *et al.* (2017), Lafon-Placette *et al.* (2018) and Le Gac *et al.* (2018) who have analyzed poplar (*Populus spp.*) clones subjected to distinct environmental conditions (cold, water availability). These studies show that developmental plasticity and memory are associated with DEGs (Differential Expressed Genes) overlapped by DMRs. These genes were related with abiotic stress response and phytohormone pathways involved in complex traits such as developmental transitions during the annual cycle or drought tolerance. While such methods are not feasible in most plant and animal systems, they demonstrate that environmentally induced epigenetic modification does explain phenotypic variation and examining patterns of epigenetic changes in invasive populations is a worthwhile endeavor.

2. Epigenetic landscape in invasive species

In the case of invasive species, the critical question is: What is the evidence that epigenetic mechanisms play a role in the successful colonization of new environments? To date, relatively few studies have differentiated between the role of genetic versus epigenetic variation on successful invasions (Prentis *et al.*, 2008; Vogt, 2017). We suspect that in time evidence will accumulate that epigenetic mechanisms act as a complementary mechanism in conjunction with standing genetic variation to shape the phenotypic variation exposed to selection. Current attempts to quantify the epigenetic contribution to population divergence is to measure genome-wide patterns of DNA cytosine methylation polymorphism using Methylation-Sensitive Amplified Fragment Length Polymorphism (MS-AFLP). This technique uses a number of restriction enzymes that are varyingly sensitive to cytosine DNA methylation, and cut DNA depending on the methylation status of the cytosine. Using such an approach it is possible to compare the patterns obtained after digestion between individuals or

populations to estimate genetic and epigenetic diversity (Richards, Schrey, & Pigliucci, 2012). In many studies, the experimental design consists of comparisons of epigenetic patterns between populations of the same species from several geographical areas for which the colonization history is known. While such an approach does not explicitly test the relationship between specific epigenetic changes and trait variation, it provides insight into how different environmental conditions induce genome wide patterns of methylation. Below we briefly discuss some of the relevant plant and animal studies that have used this design and their major findings.

Invasive Plant Examples

Given their sedentary nature, plants rely extensively on phenotypic plasticity and epigenetic mechanisms to cope with changing environments (Seymour & Becker, 2017), and there is growing evidence the patterns of methylation vary across populations occupying different environments (Foust *et al.*, 2016; Guarino *et al.*, 2015; Herrera & Bazaga, 2016; Preite *et al.*, 2015). Thus, it is reasonable to expect that epigenetic variation may underlie invasive plant populations colonizing new environments (Richards *et al.*, 2012). We highlight some of these examples here.

Gao *et al.*, (2010) investigated how epigenetic profiles change within and among natural populations of alligator weed (*Alternanthera philoxeroides*), an invasive plant in China that can grow in aquatic and terrestrial environments. This species exhibits significant changes in morphology depending on the environment it inhabits, and Gao *et al.*, (2010) induced these differences by simulating aquatic and terrestrial environments in a common garden experiment using clones derived from natural populations occupying both environments. They found more than half of the epigenetic markers were shared under the

same common garden environment independent of the population's origin, this similarity indicates that environmentally induced epigenetic reprogramming is a predictable response to environmental cues (Gao *et al.*, 2010). Because the number of polymorphic loci were close to zero among these populations, but the epigenetic diversity was much higher (*e.g.* approximately a quarter of markers being polymorphic), the conclusion is that the epigenetic changes directly contribute to phenotypic changes underlying invasion success (Gao *et al*.,2010)

Richards *et al.* (2012) examined the correlation between epigenetic variation and phenotypic response for two invasive *Fallopia* species (Japanese Knotweed) in the USA. They compared individuals coming from 16 different sites in the USA and found that epigenetic differentiation was higher between populations than between both species. Moreover, they observed greater epigenetic differentiation than genetic differentiation across haplotypes. Thus, because the genetic diversity in the introduced range is less than the epigenetic diversity, it likely originated from the environmental conditions the plants experienced (Richards *et al.*, 2012). More recently, Zhang, Parepa, Fischer, & Bossdorf (2016) have shown that epigenetic variants in Japanese knotweed are correlated with patterns of phenotypic variability of different clones, providing a link between the environment, epigenetic variation, and plant phenotypes.

The specific link between methylation, gene expression and phenotypic response to the environments has recently been demonstrated by (Xie *et al.*, 2015) in A*geratina adenophora* (crofton weed). This plant originates from Mexico and was introduced to tropical regions of China before invading the north of China with differentiated cold tolerant populations. Xie *et al.* (2015) studied methylation state in the C-repeat/dehydration-

responsive element Binding Factor (CBF) pathways which are responsible for the plant cold response via activation of cold responsive genes. They sampled several distinct geographical populations and tested their cold tolerance. All populations were screened for expression of seven inducers of CBF pathways (RT-qPCR) and methylation state (bisulfite-PCR). In parallel, they analyzed CBF inducer sequences to confirm no genetic difference in these genes or no impact at expression level. They observed a negative correlation between the methylation level of one CBF inducer, ICE1, and cold tolerance among populations. Most cold-sensitive populations were more methylated than cold-tolerant plants. The methylated state seems to be stable due to the same level in the four populations after several cold time treatments (Xie *et al.*, 2015). These results provide some of the strongest evidence demonstrating a strong link between methylation state and rapid response to an environmental stress.

Yet, the relationship between epigenetic variation, population divergence, and transgenerational inheritance across environments is not universal. Recently, Liu *et al.* (2018) examined genetic and epigenetic variation of native and invasive lineages of the common reed (*Phragmites australis*) in North America. They grew populations from different environments under controlled common garden conditions to test the stability of epigenetic inheritance and to partition genetic from epigenetic variation. While they found epigenetic variation to be greater than genetic variation across populations, they did not find convergence in epigenetic markings between populations occupying similar environments, suggesting the relationship between environmentally heritable induced epigenetic variations may not be stable and also dependent on the genetic background (Liu *et al.*, 2018). In comparison of two salt marsh species, Foust *et al.* (2016) found while one species showed a significant correlation between epigenetic variation and habitat, the other species did not.

Both Foust *et al.* (2016) and Liu *et al.* (2018) found a correlation between the amount of genetic and epigenetic variation within a population, suggesting that separating the contribution from these sources of variation will present challenges for interpretation of these patterns.

Invasive Animal Examples

Unlike plants that are more sedentary, many animals are mobile and experience a diversity of environments within and between generations. Epigenetic mechanisms may therefore underlie plastic responses of animals expanding their geographic ranges into new environments. Schrey *et al.* (2012) analyzed genetic and epigenetic variation in the invasive house sparrow (*Passer domesticus*). This bird has spread all over the world and exhibits phenotypic variability in different geographical areas, despite a recent invasive period (less than 150 years). Schrey *et al.* (2012) studied epigenetic variability of populations in Nairobi (Kenya) which was colonized 50 years ago, and Tampa (Florida, USA) which was colonized over 150 years ago (Schrey *et al.*, 2012). Comparisons of these populations reveal that the amount of within-population epigenetic variation is greater than the between-population variation, and that it was associated with a poor genetic diversity. Schrey *et al.* (2012) suggest that epigenetic variation may have compensated for the low genetic variability and contributed to phenotypic differentiation. In another study of the same species, Liebl *et al.* (2013) sampled seven geographically separated Kenyan populations and found a negative correlation between genetic and epigenetic diversity; higher epigenetic diversity was correlated with a decrease in heterozygosity and an increase in inbreeding (Liebl, Schrey, Richards, & Martin, 2013). The authors suggested that epigenetic variation could be a compensatory mechanism for low genetic diversity in an invading population, allowing an increase in phenotypic variability by expression of cryptic genotypes or by phenotypic

plasticity. However, in a more recent comparison of Australian house sparrows, (Sheldon, Schrey, Andrew, Ragsdale, & Griffith, 2018) failed to detect such compensatory effects. Despite considerable phenotypic differences, Liebl, Schrey, Richards and Martin (2013) also fail to find a relationship between epigenetic variation and populations occupying different environments.

Ardura, Zaiko, Morán, Planes, & Garcia-Vazquez (2017) studied epigenetic variation in populations of the invasive pygmy mussel (*Xenostrobus secures*). Specifically, they compared a newly established population to existing older populations to test the hypothesis that epigenetic patterns change over time as the population undergoes sequential invasive steps. They observed a significant hypomethylation in recent invasive populations compared to older ones and suggested that hypomethylation could increase gene expression or genetic recombination thus impacting the phenotype. The same hypomethylation was detected in one other species, *Ficopomatus enigmaticus* (Ardura *et al.*, 2017). However, the experimental design of these studies presents potential biases due to confounding effects of different environments and the invasive status of the populations.

Huang *et al.* (2017) used the MSAP technique to reveal changes in DNA methylation frequency, intragroup methylation divergence and methylation differentiation after rapid environmental stress in the marine species *Ciona savignyi*. They induced variation of DNA methylation frequency and DNA methylation divergence after 1hr of treatment, but these differences disappeared after 48hrs. These results demonstrate that DNA methylation can also be short-lived in response to environmental changes and may not persist.

Lastly, Oppold *et al.* (2015) and Kreß *et al.*, (2017) studied epigenetic modifications in the Asian tiger mosquito (*Aedes albopictus)*, which has spread from South East Asia to every continent except Antarctica and Australia. Invasive populations of this vector for many diseases have low genetic diversity (Kreß *et al.*, 2017; Oppold *et al.*, 2015), yet exhibit rapid adaptive responses to different environments, such as chemical compounds or resistance to cold, and showed alteration of DNA methylation levels. These results should be taken with caution since the levels of DNA methylation in insects are in general very low and some recent papers suggest an absence of significant DNA methylation in Diptera (Bewick, Vogel, Moore, & Schmitz, 2016; *Provataris et al.*,, 2018).

Collectively these publications highlight the potential role of epigenetics in the invasion process by correlating epigenetic patterns of variation with natural environmental variation, but definitive evidence that such correlations underlie invasion success remains elusive. We see several fundamental challenges that need to be overcome before we can move beyond correlation and establish causal links between environmentally induced epigenetic variation and the phenotypic changes that allow populations to expand in new environments. First, when examining genome wide patterns of genetic and epigenetic diversity it is extremely difficult to partition out how much each of these sources of variation contribute to observed phenotypes without using experimental designs that control for genetic background. Such experiments are simply not possible for many organisms. Second, while comparing methylation patterns of invasive populations across different environments may reveal higher epigenetic diversity relative to background genetic variation, rarely is there a clear link with the phenotypic response. This challenge is not unique to epigenetic studies, as establishing the genetic basis of any complex trait is exceedingly difficult in non-model organisms. Lastly, many of the current methods such as the MSAP approach sample only a

small subset of the genome, which underestimates the epi/genetic diversity and makes it even more difficult to find clear relationships with phenotypes. New genome wide approaches are emerging to better evaluate genetic and epigenetic diversity (Pu & Zhan, 2017; van Gurp *et al.*, 2016). Thus, while there are compelling reasons to suspect epigenetic changes to be important in the process of adapting to new environments, we still lack the necessary evidence to draw any general conclusions.

Conclusions and future directions

In this review, we highlight significant literature that shows potential links between TE, epigenetics and phenotypic plasticity and their role in rapid adaptation of invasive species to new environments. No broad studies have deal with these three notions concomitantly. It is clear that limited empirical data is available to test this idea, particularly because it is difficult to disentangle genetic and non-genetic variability.

Species introductions can be considered evolutionary experiments and constitute good models for investigating adaptive processes operating at short time scales (Gibert *et al.*, 2016). The two most commonly used approaches to investigate how invasive populations respond to new environments are: 1) the synchronic approach, where comparisons of native and invasive populations are made, and 2) the diachronic approach, where the invasive population's genetic variation is monitored over time. Ideally, both approaches should be combined, but more importantly if we are to better understand the mechanisms that facilitate and constrain population expansion in new environments, we need to study populations during the initial stages of the invasion. This is the time period when population size, genetic variation, and patterns of plasticity will dictate the evolutionary trajectory of the population.

While detection and study of such populations is needed, the use of large-scale experiments using mesocosms and experimental introductions to simulate the invasion may ultimately be needed if we are to draw any general conclusions about what mechanisms are most important.

It has been hypothesized that invasive species or populations exhibit greater adaptive phenotypic plasticity or rapidly evolve to new environments (Ghalambor *et al.*, 2007; Prentis *et al.*, 2008; E. J. Richards, 2006; Fig. 1). The molecular mechanisms underlying these responses have yet to be totally elucidated, but a growing body of literature shows that TEs contribute to genetic variation and epigenetic changes underlie phenotypic plasticity in gene expression. The epigenome provides a mechanistic explanation on how environmental and genomic variations are transformed into phenotypic differences. Thus, while we have reason to suspect TEs and epigenetic changes contribute to adaptive phenotypic plasticity and adaptive evolution, establishing mechanistic links between genes, the environment, gene expression, and phenotypes is an exceedingly difficult task. These challenges are part of the larger debate and problem of mapping genotypes to phenotypes (Debat & David, 2001; Wagner & Altenberg, 1996). We know that the environmental context is critical to shaping how the genome is transformed into different phenotypes by altering patterns of gene expression throughout development, but by considering how the epigenome responds to different environments we can potentially gain a better understanding of this mapping problem (Duncan *et al.*, 2014). Our perspective here is that the potential of the epigenome to facilitate biological invasions may be hidden within the native range, where natural selection has had the opportunity to shape epigenetic control mechanisms (see also Rey *et al.*, 2016; Stapley *et al.*, 2017). The general hypothesis is that if new environments disrupt the epigenetic control of genetic variation, then we will observe the release of this hidden

variation which in turn will facilitate biological invasions through adaptive plasticity and or increased genetic variation. Robust tests of this hypothesis will need to capture the processes occurring during the early stages of colonization and will require comparative studies of the source and descendent populations. Such studies will also need to resolve some fundamental challenges that all epigenomic approaches face. We briefly discuss these below.

1. Phenotypic Plasticity vs. Stochastic Environmental Effects

Phenotypic plasticity is defined as the capacity of a genotype to produce different phenotypes in response to different environmental cues (*e.g.* Ghalambor *et al.*, 2007). A defining feature of plasticity is that it represents a predictable response to the environment, such that phenotype of an individual genotype can be predicted based on the environment (Pigliucci, 2005). In contrast, environmentally-induced variation that is stochastic, such as developmental noise, represents unpredictable variation (Raser & O'Shea, 2005). This distinction matters when we consider the role the epigenome plays in biological invasions. If new environments induce predictable changes in the epigenetic control of gene expression, then natural selection can shape the pattern of epigenetic change throughout the genome. The concept of predictability is central to determining whether environmentally induced variation is adaptive or non-adaptive, and how selection will act on the distribution of phenotypes (Ghalambor *et al.*, 2015). For example, if plasticity is adaptive then it can allow populations to persist under new conditions and allow time for beneficial mutations to arise (Corl *et al.*, 2018; Ghalambor *et al.*, 2007; Pigliucci, 2005).

However, if new or stressful environments induce stochastic changes in patterns of methylation or histone modification, then the contribution to adaptive evolution is largely based on the probability that by chance some of the variation is beneficial. Under such a

scenario, the stability of these patterns across generations is critical to the process, otherwise the patterns of variation generated in one generation will be uncorrelated with the patterns in subsequent generations. It is therefore imperative that future studies examining the role of the epigenome in biological invasions quantify how predictable environmentally induced epigenetic change is, and how heritable these changes are across generations in the presence and absence of the environmental cues.

2. Linking Epigenetic Marks to Traits and Separating Genetic from Epigenetic Variation

Identifying the genetic basis of complex traits is a fundamental and on-going challenge in evolutionary biology, and this is particularly the case for polygenic traits subject to environmental influences. Under controlled environmental conditions, breeding experiments can be designed to partition phenotypic variation into the contributions made by genetic, epigenetic, and environmental sources. However, when studying natural populations establishing links between the epigenome and specific traits is often an exceedingly difficult task. To date, most studies investigating the contribution of epigenetic mechanisms to biological invasions have simply quantified the patterns of epigenetic marks for populations occupying different environments. The assumption is that variation in epigenetic marking underlies differences in phenotypes, but such correlations are rarely tested in outbred natural populations.

Establishing the relationship between epigenetic changes and phenotypes is closely related to the general problem of separating genetic from epigenetic effects on phenotypic variation. In other words, when we observe phenotypic differences between invasive populations occupying different environments, how much of the observed variation can be directly attributed to the underlying epigenetic vs. genetic diversity? In model organisms,

clonal or isogenic lines can be generated that place epigenetic variation against a common genetic background, thus allowing for observed phenotypic differences to be attributed to the epigenetic effects (but see Menezes *et al.* 2018 for the difficulty to disentangle the two sources of variation). However, in natural populations not only does the genetic background vary across individuals and populations, epigenetic and genetic diversity can be positively correlated with each other making results even more difficult to interpret. We anticipate that future sequencing technology and advances in bioinformatic tools will continue to improve and help overcome some of these challenges, especially for non-model species (Billet *et al.*, 2017). Thus, epigenome-wide association study (EWAS) is an interesting approach to associate single methylation polymorphisms (used as marker) with a phenotype of interest (Lin, Barton, & Holbrook, 2016).

3. Separating Genetic and Environmentally Induced Changes in TE Activity

It will be important to quantify how much the role TEs play in adaptive changes in response to new environments requires a joint understanding of the epigenetic control of genetic variation. TE regulation is mediated by epigenetic marks and the modification of their expression in new environments could reflect increased mobility or misregulation of the epigenetic control mechanisms. Distinguishing between these alternatives is important if we are interested in knowing whether new environments increase genetic variation through TE activity or simply expose existing variation that was previously hidden. Disentangling epigenetic from genetic variability is becoming easier but remains a very challenging problem, in particular if TEs are accounted for. We envision studies that do a better job of quantifying genome size, the percentage of the genome made up of TEs, and the total number of small TE variations (Goubert *et al.*, 2015; Lerat *et al.*, 2017). This could be done by simulating genomes with different TE amounts and using software's like

DNApipe TE (Goubert *et al.*, 2015) for which no reference genome is needed. Still, the identification of insertion polymorphism in natural population which will give us indications of increased genetic variance is not an easy task, despite incredible bioinformatics developments (Villanueva□Cañas, Rech, Cara, & González, 2017). The availability of third-generation sequencing technologies will be decisive to facilitate both the incorporation of TEs in the genetic variability analyses and also the identification of specific epigenetic changes associated with TE.

ACKNOWLEDGEMENTS

This work was supported by the ANR (grant SWING ANR to PG and CV), by the "INRA Département EFPA" (grant PI EFPA-2016 to DB and SM), the RTP3E-CNRS (http://rtp-3e.wixsite.com/rt3e), the Fondation Rovaltain (France), the "Région Bretagne and INRA Département EFPA" (PhD grant to JG). CKG was supported by a National Science Foundation grant (IOS-1457383) and the University Lyon 1.

AUTHORS' CONTRIBUTIONS

C.V. and P.G. conceived the idea and initial structure of the manuscript, P.M. and J.G. wrote the first draft of the manuscript, S. M. and D. B. contributed to the epigenetics section, C. K. G. significantly reorganized and correct the manuscript. All the co-authors significantly contributed to the writing and revisions.

DATA ACCESSIBILITY

No data were collected for this manuscript.

Figure 1: Process of invasion and its epi/genomics, life mode, environmental, and human activities determinants

A species in its native area (*white frame*) is transported in a new environment (*grey frame*), often by human activities, voluntary or not. Genetic diversity (*blue shape*) is recognized as lower in introduced area. Installation of this new population can be facilitated by repeated introductions (propagule pressure). A bottleneck can take place and lead to the disappearance of the introduced species. Organisms who survive can have a different genetic (*shape*) and epigenetic (*color*) diversity that from native area. Population becomes established and continues in time, some species can be naturalized while others are going to continue their expansion until they become invasive. There is an uncontrolled expansion with a selection of the most performing genetic and epigenetic variants in a given environment. These invasive species can then colonize new environments and cause economic and ecological issues. During this process, a multitude of factors comes into play:

*Epigenetic and genetic mechanisms can modify gene expression and thus the capacity for local adaptation and phenotypic plasticity.

*Characteristics of organisms are also important factors to consider for the success of a biological invasion: life cycle, type of multiplication, especially clonal, possibility of hybridization (*combined shape*) and the capacities of dispersal, can favor the installation of the population in a new environment.

*Environmental factors, biotic and abiotic, represent the sensibility of the ecosystems. Absence of predators and competitors in the introduction area, as noted in Enemy release hypothesis (ERH) and its corollary Evolution of increased competitive ability (EICA) as well as the presence of empty ecological niche can partially explain the success of an invasion. Besides, global climate change has been identified as a source of opportunity for new invasions.

*Finally, human activities are a key factor of either success or failure of an invasion. The human being acts at the origin of the process by the action of transport, voluntary or not. On the other hand, he can, with legislation and prevention, decrease or control this mechanism. He can either promote invasions by altering the ecosystems or limit their impact by controlled actions and management.

Figure 2: Transposable element structures

Transposable elements can be classified in type I and II according to RNA intermediate production and insertion mechanisms.

Type I, called retrotransposon use RNA intermediate via a reverse transcription step, and can be divided into 2 groups. LTR (long terminal repeats) elements with direct repeats both at beginning and end of the element. It presents POL and GAG sequences, related to retroviral protein genes. Non-LTR element also use an RNA intermediate and are characterized by a polyA tail. SINEs (short interspersed nuclear elements) are non-autonomous TE that will use LINE enzymatic machinery to transpose.

Type II elements, named DNA transposons present TIR (terminal inverted repeats) at each extremity, which are recognized by transposon's transposase and allow the integration in other genome. MITEs (miniature inverted repeats) have no ORFs (open reading frames) and are non-autonomous TEs. Then helitron transposons, use DNA helicase mechanisms to be transposed and don't present TIR pattern. Hence, the recently discovered Maverick sub-group, seems to use a self-encoded DNA polymerase and have TIRs.

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