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SPECIAL ISSUE: THE ROLE OF GENOMIC STRUCTURAL VARIANTS IN ADAPTATION AND DIVERSIFICATION

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The impact of transposable elements in adaptive evolution

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Abstract

The growing knowledge about the influence of transposable elements (TEs) on (a) long-term genome and transcriptome evolution; (b) genomic, transcriptomic and epigenetic variation within populations; and (c) patterns of somatic genetic differences in individuals continues to spur the interest of evolutionary biologists in the role of TEs in adaptive evolution. As TEs can trigger a broad range of molecular variation in a population with potentially severe fitness and phenotypic consequences for individuals, different mechanisms evolved to keep TE activity in check, allowing for a dynamic interplay between the host, its TEs and the environment in evolution. Here, we review evidence for adaptive phenotypic changes associated with TEs and the basic molecular mechanisms by which the underlying genetic changes arise: (a) domestication, (b) exaptation, (c) host gene regulation, (d) TE-mediated formation of intronless gene copies—so-called retrogenes and (e) overall increased genome plasticity. Furthermore, we review and discuss how the stress-dependent incapacitation of defence mechanisms against the activity of TEs might facilitate adaptive responses to environmental challenges and how such mechanisms might be particularly relevant in species frequently facing novel environments, such as invasive, pathogenic or parasitic species.

KEYWORDS
exaptation, genomic plasticity, HSP90, molecular domestication, retrogene formation, stress-induced TE activity

1 | THE PERSPICACITY OF BARBARA MCCLINTOCK

Transposable elements (TEs)—also frequently called "jumping genes"—are mobile genetic units possibly evolutionarily related to viral components and present in virtually every genome. The presence and particularly the transposition of TEs have profound consequences for a genome's structure, stability and likely also its evolvability. However, over the elapsed 70 years of TE research, the potential of these still enigmatic genetic elements in adaptive evolution has remained contentious (Biémont, 2010). Barbara McClintock was the first to recognize a mobile genetic element that "may change its position in the chromosome" (McClintock, 1948) and whose "presence […] at or near the locus of a known gene may affect the action of this gene" (McClintock, 1956), when the established scientific consensus at the time was that genes were neatly aligned along chromosomes. Today, due to increasing research efforts combined with technical refinements, TEs are commonly recognized as ubiquitous and influential genetic elements populating the genomes of almost all organisms (Fedoroff, 2012). We only now begin to fully appreciate the involvement of TEs in adaptive evolution, where they provide an extraordinarily versatile source of genetic and epigenetic variation. At last, we review recent progress in understanding the adaptive potential unfolding from TEs and TE-induced structural variation, focusing on how adaptive novelty can evolve from TE-associated genetic changes, in particular under challenging environmental conditions.

2 | THE COMPLEX DIVERSITY OF TRANSPOSABLE ELEMENTS

While initially considered as rare specialized genetic elements, the prevalence and diversity of TEs became fully apparent following the advent of whole genome studies (Hurst & Werren, 2001), which revealed that several thousand copies of different TEs typically populate a genome. For example, they make up ~85% of the maize genome and ~69% of the human genome (de Koning, Gu, Castoe, Batzer, & Pollock, 2011; Schnable et al., 2009). Intact TEs vary in length, from a few hundred to several thousand base pairs (Feschotte & Pritham, 2007), depending on their coding facility to achieve replication.

A unifying classification system was developed in 2007, dividing mobile genetic elements into two classes with nine orders and 29 superfamilies based on mechanistic and enzymatic criteria (Wicker et al., 2007); see also (Kapitonov & Jurka, 2008). Note, however, that alternative classification systems are still being proposed (e.g., based on functional and structural features of TEs [Arkhipova, 2017]), emphasizing that many challenges revolving around in TE identification and classification remain unresolved to date (Arensburger, Piégu, & Bigot, 2016; Piégu, Bire, Arensburger, & Bigot, 2015; Seberg & Petersen, 2009).

Class I comprises retrotransposons that propagate via RNA intermediates and rely on the activity of reverse transcriptases and integrases. This "copy-and-paste" mechanism leads to the duplication of elements with each cycle of transposition, inserting a novel, reverse-transcribed copy at a new locus while retaining the template element at its original locus. Class I elements are further divided into five orders representing two subclasses LTR (long terminal repeats) and non-LTR retrotransposons. Class II elements are DNA transposons and depend on transposase enzymes to catalyse excision and insertion. DNA transposons are further divided into two subclasses based on the number of DNA strings that are cut during transposition. Most elements in Subclass I move via a "cut-and-paste" mechanism and contain terminal inverted repeats, which are recognized by transposase enzymes cutting both DNA strands during transposition (Fedoroff, 2013). Subclass II, with its two orders Helitron-like elements and Maverick-like elements, comprises DNA transposons that replicate by a "copy-and-paste" mechanism in which only a single DNA strand is cut (Wicker et al., 2007). It is interesting that even Class II "cut-and-paste" DNA transposons can achieve duplication by exploiting the host's DNA replication. One of the newly synthesized DNA strands containing the replicated DNA transposon located behind the replication fork might be exposed to transposase-mediated excision and reinsertion anywhere else in the genome. The gap at the origin locus is filled by homology-dependent gap repair using the opposite strand DNA transposon as a template (Skipper, Andersen, Sharma, & Mikkelsen, 2013).

In general, TE propagation depends on the activity of proteins that recognize, mobilize and finally reintegrate the element in the genome. Full-length autonomous TEs by definition contain the necessary genes to achieve this transposition. For example, a Class I autonomous retrotransposon of the LTR subclass contains an open reading frame coding for the full molecular toolset necessary to catalyse its own transposition: a reverse transcriptase, a proteinase, an RNase and an integrase (Wicker et al., 2007). Mutational events can however impair the ability of TEs to independently produce their transpositional machinery, giving rise to nonautonomous elements that exploit the enzymes produced by other active, autonomous TEs. Intriguingly, nonautonomous TEs can also evolve directly from non-protein-coding (npc) genetic elements, without ever being autonomous transposons or even containing a single protein-coding gene. For example, short interspersed elements (SINEs) are the most abundant nonautonomous Class I retrotransposons present in many vertebrates and are derived from host cell-specific small RNAs (tRNA, 7SL RNA and 5S RNA). According to their origin, they are free of protein-coding components. More than one million copies of 7SL RNA-derived *Alu* SINEs successfully dispersed throughout our own genome and now occupy approximately 10% of genomic space (Lander et al., 2001).

3 | TAMING TES

Historically, TEs were long considered to be purely parasitic elements that, without any benefit, inflated genomes with "junk" sequences (Ohno, 1972)—a reputation that was bolstered by the discovery of substantial and deleterious mutations generated by TEs (Kazazian, 1998). TEs indeed are highly mutagenic, either directly (e.g., by harmful insertions in coding or regulatory regions) or indirectly based on their high genomic abundance and transposon-mediated chromosomal rearrangements (also known as nonhomologous ectopic recombination or nonallelic homologous recombination). Furthermore, insertions of TEs can have drastic effects on surrounding genes by changing the structure and the regulatory and/or epigenetic environment.

Given the strong mutagenic potential of TEs, it is not surprising that molecular countermeasures evolved to suppress their activity, thus keeping their disruptive potential in check. Chromatin modifications, which are the genome's first line of defence, suppress transcription of TEs through methylation, histone modification or chromatin packing. Posttranscriptionally, TE-derived transcripts are targeted for cleavage directed by npc silencing (siRNAs) and *piwi*-interacting RNAs (piRNAs; Slotkin & Martienssen, 2007). It is interesting that there is evidence that the earliest siRNAs as well as several other classes of npcRNAs in fact originally suppressed TEs and that a more general involvement in transcriptional regulation emerged only secondarily (Levin & Moran, 2011; Shabalina & Koonin, 2008; Slotkin & Martienssen, 2007). At the posttranslational level, regulation of transposition has been studied less extensively, despite some remarkable evidence for "multimer poisoning" and autoregulatory suppression in P and mariner transposases through processes called "overproduction inhibition" and "dominant-negative complementation" (Lohe & Hartl, 1996). Both these processes involve the formation of enzymatically inactive transposases or

transposase complexes, either through oligomerization or through competition for transposon binding. While dominant-negative complementation requires the expression of a mutated, nonfunctional transposase, overexpression inhibition can occur when a critical threshold of active transposons is reached in a cell (Claeys Bouuaert, Lipkow, Andrews, Liu, & Chalmers, 2013; González & Petrov, 2012; Lohe & Hartl, 1996; Skipper et al., 2013). Likewise, immobilized copies of TEs can hinder transposition of other elements in a process called transposon titration, if transposon binding sites are retained in the immobile elements (Hartl, Lohe, & Lozovskaya, 1997; Simmons & Bucholz, 1985).

Whether such posttranslational mechanisms in fact constitute defence mechanisms or are adaptive for TEs themselves as autoregulatory mechanisms remains elusive and calls for further research into the underlying evolutionary dynamics.

While epigenetic modification and posttranscriptional/translational suppression can act against activation of TEs, there is no known general molecular process for a targeted removal of individual transposable elements. However, unequal homologous recombination between invariant, TE-flanking target site duplications is a viable mechanism to purge exclusively young TEs that are usually flanked by such perfect short duplications (van de Lagemaat, Gagnier, Medstrand, & Mager, 2005). The efficiency of such homology-dependent illegitimate recombination is also known for the formation of solo-LTRs (Shirasu, Schulman, Lahaye, & Schulze-Lefert, 2000). Unequal homologous recombination imposes strong selection against TEs, either by eliminating TEs from a genome or by producing inviable chromosomal aberrations. Nevertheless, TEs have accumulated in most eukaryotic genomes, raising the question whether evolution of epigenetic silencing mechanisms controlling recombination might have been key in allowing the invasion of eukaryotic genomes by TEs (Fedoroff, 2012).

In accordance with and as a general consequence of recombination and recombination-controlling mechanisms, genomic regions of high recombination frequency are more likely to eliminate TEs and intermediate sequences. Hence, the distribution of TEs in a genome is often negatively correlated with recombination rate, with accumulations of TEs in low-recombining, gene-rich regions (Kent, Uzunović, & Wright, 2017; Montgomery, Charlesworth, & Langley, 1987)—likely due to Hill–Robertson effects (Dolgin & Charlesworth, 2008).

While nonhomologous allelic recombination can generate larger mutations removing entire TEs, single-base mutations and small indels in functionally integral regions can render TEs immobilized, while leaving most of their functional elements intact. Following this incapacitation, disrupted TEs in most cases continue to evolve largely neutrally so that genomes of most organisms are densely populated by TEs in various stages of degeneration and fragmentation. Despite being inactive, such TE fossils often still contain coding or noncoding elements able to interact with the molecular and genomic machinery (Elbarbary, Lucas, & Maquat, 2016), thus providing a rich substrate for evolutionary novelty and genetic innovation (Chuong, Elde, & Feschotte, 2017).

4 | TES IN ADAPTIVE EVOLUTION: A RICH AND DIVERSE COLLECTION

Despite their apparent menace to genome integrity and host fitness (Hedges & Deininger, 2007), the reputation of TEs changed when evidence for TE-conveyed beneficial genetic innovation surfaced (reviewed in Volff, 2006; Oliver & Greene, 2009; Fedoroff, 2013). Today, there is relatively broad consensus that TEs have been key contributors to various fundamental innovations in adaptive evolution, such as gene regulatory networks (Chuong et al., 2017), for example, by serving as seeds for small RNAs (Berezikov, 2011; Slotkin & Martienssen, 2007) and long npcRNAs (lncRNA; Kapusta et al., 2013; Johnson & Guigo, 2014).

The molecular mechanisms by which TEs can generate novel and potentially adaptive genetic variants are diverse and can broadly be divided into five different categories: (a) Genomic TE protein-coding genes or parts thereof can be domesticated and co-opted by providing the original codon structure to express a novel and adaptive host trait (Jangam, Feschotte, & Betrán, 2017; Miller, McDonald, & Pinsker, 1997); (b) after insertion of a TE into an intron of a host gene, parts of the TE can be co-opted by including a novel, emerging TE exon to an existing protein-coding gene in a process called exonization. Such "features that now enhance fitness but were not built by natural selection for their current role" are called exaptations (Gould & Vrba, 1982); (c) TE transposition into the proximity of genes can affect their regulatory environment and thus transcription (Feschotte, 2008); (d) by the activity of the transposition machinery of Class I long interspersed elements (LINE1), therian gene transcripts can be reverse-transcribed and inserted into the genome as intronless retrocopies (Casola & Betrán, 2017; Kaessmann, Vinckenbosch, & Long, 2009); and (e) the presence of paralogous copies of TEs in a genome can provide the substrate for aberrant transposition (Gray, 2000; Weil, 2009) and ectopic recombination (Carvalho & Lupski, 2016; Robberecht, Voet, Zamani Esteki, Nowakowska, & Vermeesch, 2013; Startek et al., 2015) leading to novel structural rearrangements and genomic plasticity. In the following, we review each of the categories in more detail and provide recent examples that illustrate the evolutionary and adaptive significance of different kinds of TE-induced variations.

4.1 | Domestication

TE-derived proteins have been co-opted so abundantly and recurrently in evolution that TE domestication is now considered a generally important adaptive mechanism for evolutionary innovation (Jangam et al., 2017). It is in fact agreed upon today that the evolution of various fundamental cellular mechanisms in many species originally arose through TE domestication. A classic example for the evolutionary significance of TE domestication is found in *Drosophila*, where telomeres are maintained not by telomerases but by two domesticated, non-LTR Jockey LINE-like retrotransposons, HeT-A and TART (Pardue & DeBaryshe, 2003). HeT-A and TART actively and in tandem add their long repeats at terminal regions of

chromosomes to compensate for the loss of terminal nucleotides during DNA replication. Another remarkable example of TE domestication is found in *Paramecium*. Developmentally programmed genome rearrangements that occur during sexual reproduction involve a domesticated *piggyBac* transposase called PiggyMac, which catalyses the precise excision of several thousand loci during the formation of the somatic macronucleus (Baudry et al., 2009; reviewed in Catania & Schmitz, 2015). Likewise, the RAG1 gene essential for somatic V(D)J recombination in humans and other jawed vertebrates evolved through domestication of the transposase of *Transib*, an ancient DNA transposon, producing a chimeric gene combining host- and TE-coding sequences (Huang et al., 2016; Kapitonov & Jurka, 2005).

While the above-mentioned examples involve domestication of functional (retro)transposases, other TE-derived genes and proteins have been co-opted to harbour specific traits as well, and it is likely that the diversity of genes contained in endogenous TEs greatly facilitates the evolution of alternative, domesticated functions. One prominent example is syncitine that is co-opted from the envelope gene of an endogenous retrovirus (ERV) to mediate cell–cell fusion during the host's placental development (Figure 1). It is interesting that, in viviparous mammals, syncitine was domesticated multiple times independently within the last 150 million years (reviewed in Kaneko-Ishino, 2012).

4.2 | Exaptation

In contrast to domestication, exaptation is the evolution of a trait so as to have a different function and different usage of nucleotides than in its original form, such as the exonization of non-protein-coding nucleotides into novel, protein-coding exons. Such exonized sequences are usually short (a few tens of functional de novo codons), as interruptive stop codons or open reading frame shifts will occur by chance in longer stretches. Exapted sequences are mostly shaped over long evolutionary periods commencing as minor alternative splice variants (Schmitz & Brosius, 2011). Intriguingly, it is not necessarily the additional protein-coding feature that leads to the novel adaptive value. For example, in the vertebrate ZNF639 gene, orthologous exaptations in intron 5 of completely different elements led to a characteristic exonization in mammals and birds (Figure 2). In this exceptional case, the optimized topology of the ZNF639 protein structure is suggested to be the adaptive value (Krull, Petrusma, Makalowski, Brosius, & Schmitz, 2007).

4.3 | Host gene regulation

Like the recruitment of protein-coding sequences to the expression of novel traits, the transposition of TEs in proximity to protein-coding genes can trigger novel adaptive phenotypes. Newly inserted (or excised) TEs can affect these genes by changing the regulatory or coding environment at a given locus. As such, insertion in promoter or regulatory intronic regions can modify expression patterns of downstream genes. A particularly striking case of such adaptive transposition-conferred change is the colour polymorphism of the peppered moth: The adaptive, "industrial melanism" phenotype in this species evolved through the intronic insertion of a DNA transposon, resulting in an increase in transcript abundance of the affected cortex gene (Van't Hof et al., 2016; Figure 3). Likewise, several cases have been reported over the years, in which TE insertions conferred pesticide resistance by affecting expression profiles of metabolic genes (Guio, Barrón, & González, 2014; Le Goff & Hilliou, 2016; Mateo, Ullastres, & González, 2014; Rostant, Wedell, & Hosken, 2012). TE insertions can also lead to modifications at the epigenetic level (methylation and chromatin packing), which can again affect the expression of neighbouring genes (Horvath & Slotte, 2017). In *Escherichia coli*, insertion of a single TE was shown to drive the adaptive transition from a commensal to a pathogenic lifestyle (Proença, Barral, & Gordo, 2017).

4.4 | Retrogene formation

RNA-mediated retroposition of transcribed genes is a well-known source for gene duplications and genetic novelty in eukaryotic and prokaryotic genomes (Chen, Krinsky, & Long, 2013). New retrocopies can arise when particularly highly transcribed and polyadenylated mRNAs are randomly recognized by the transposition machinery of LINE1 transposons, leading to the reverse transcription and reintegration of an intronless copy of the original gene back into the genome at a randomly selected locus. The fate of such initially

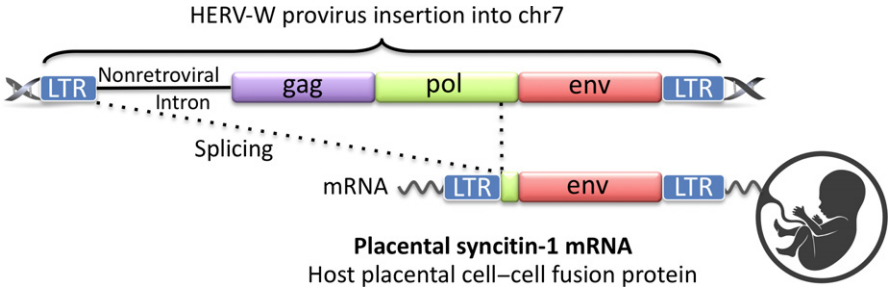

FIGURE 1 Domestication: Acquisition of a TE-derived functional genetic element. An HERV-W provirus inserted about 35 mya into germ line chromosome 7 of the ancestral Catarrhini lineage (Old World monkeys and apes). The original retroviral ORF of the spliced envelope gene was domesticated as a single-exon gene. In apes, including humans, the expressed mRNA mediates the physiological trophoblast cell–cell fusion essential for normal placental development (symbolized by the human embryo)

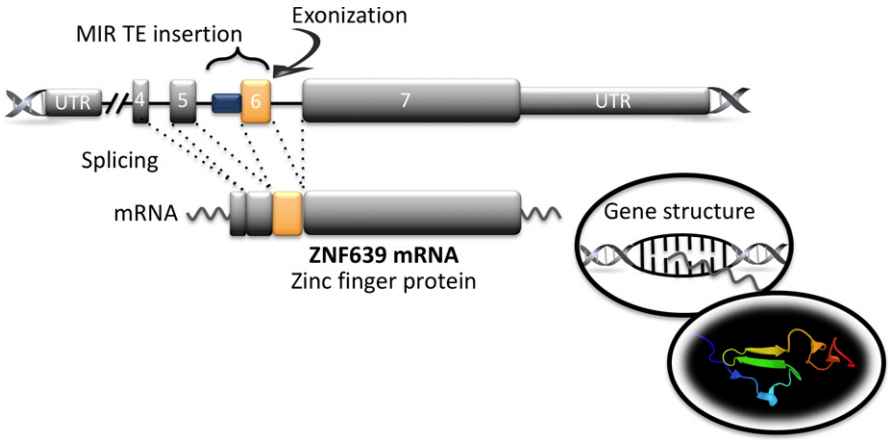

FIGURE 2 Exaptation: Adaptive incorporation of TE-derived sequence as a functional genetic element. The constitutive inclusion of the MIR cassette (exon 6) into the mRNA transcribing the ZNF639 zinc finger protein is the result of an exonization of a new protein-coding unit from a part of a non-protein-coding mammalian-wide interspersed element (MIR). This mutation resulted in an altered ZNF639 gene architecture in all mammals and a convergently evolved anonymous insertion into the orthologous intron in birds (see text)

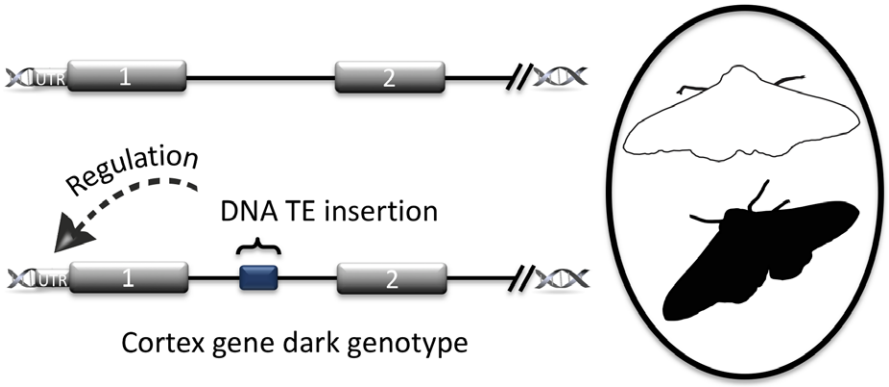

FIGURE 3 Regulation: Regulatory modification of gene expression after integration of TE cassettes. The industrial melanism phenotype in peppered moths results from a tandemly repeated DNA transposon (*carbonaria*) in the first intron of the gene, *Cortex*. The TE insertion positively affects expression during early wing development, generating an adaptive wing colour phenotype in a period of heavy pollution during the industrial revolution

functionless retrocopies ("retropseudogenes") that are separated from their original flanking regulatory units depends on how subsequently arising mutations, selection and drift change the structure and regulation of the retrocopy. Potentially, these events can lead to the evolution of an adaptive, novel trait conferred by a young and functional retrogene (e.g., neofunctionalization or subfunctionalization). Examples of retrocopies that evolved adaptive functions are abundant and many cases have been studied in detail in domestic dogs (Parker et al., 2009; Figure 4), fruit flies (Zhang, Dean, Brunet, & Long, 2004), apes and human (Baertsch, Diekhans, Kent, Haussler, & Brosius, 2008; Rosso et al., 2008), *Arabidopsis* (Matsuno et al., 2009) and many other organisms (Carelli et al., 2016; Chen et al., 2013; Kubiak & Makałowska, 2017).

4.5 | Genomic plasticity

While it has long been recognized that aberrant transposition (Busseau, Pelisson, & Bucheton, 1989; Weil & Wessler, 1993) and ectopic recombination (Kupiec & Petes, 1988) between paralogous TEs can generate substantial genetic mutations in a genome, there is only limited insight into the potential for adaptive evolutionary change in these mechanisms. However, comparative and functional genomic studies begin to shed more light on the role of TE-conferred genome plasticity in the adaptive evolution of both prokaryotes (Vandecraen, Chandler, Aertsen, & Van Houdt, 2017) and eukaryotes (Albertin et al., 2015; Daron et al., 2014; Grabundzija et al., 2016; Vicient & Casacuberta, 2017). Most prominently, genomic studies on filamentous plant pathogens have repeatedly revealed a remarkable pattern described as "two-speed genomes." In these genomes, certain fast-evolving regions are enriched in TEs, have increased frequencies of single-nucleotide and large-scale mutations and often harbour genes related to pathogenicity and host–pathogen interactions (Croll & McDonald, 2012; Dong, Raffaele, & Kamoun, 2015; Raffaele & Kamoun, 2012; Seidl & Thomma, 2017). The significant contribution of TEs to the variability of these regions led to the conclusion that transposons can be considered the major driving

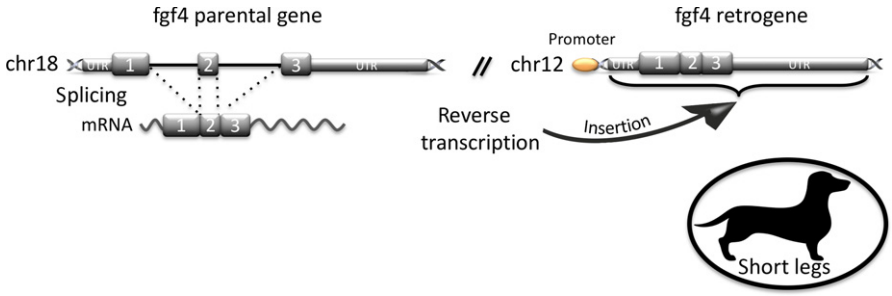

FIGURE 4 Retrogene formation: Random TE-driven reverse transcription and insertion of a spliced mRNA. Expression of a functional retrocopy of *fgf4* causes the short leg phenotype, known as chondrodystrophy, in dogs. This phenotypic variation is a human-domesticated trait expressed for example in dachshund breeds. One of two known functional *fgf4* retrogenes is located on chromosome 12 and is regulated by an adjacent preexisting promoter (orange oval). The retrogenes were most likely generated and inserted by a LINE1-mediated reverse transcription from the spliced parental/ancestral *fgf4*mRNA

force for adaptive genome evolution in such pathogens (Bao et al., 2017; Faino et al., 2016). Likewise, TE-conferred genomic plasticity as a major determinant of their adaptive potential has been proposed for some prokaryotic pathogens (Siguier, Gourbeyre, & Chandler, 2014; Vandecraen et al., 2017), such as *Coxiella*, in which gene loss through transposon-mediated chromosomal rearrangements is the main source of genomic diversity in populations (Beare et al., 2009). In general, the TE-conferred genome plasticity in different pathogens has been argued to essentially be an adaptation for adaptability (Möller & Stukenbrock, 2017), enabling the pathogens to rapidly adapt to evolutionary challenges stemming from their host's defensive mechanisms—a concept that has also been discussed in more general evolutionary contexts (Crombach & Hogeweg, 2007; Wolf & Linden, 2011).

Probably, the strongest argument in favour of an adaptionist view on the structure and composition of these genomes is that they have evolved convergently in distinct lineages of eu- and prokaryotes (Dong et al., 2015; Raffaele & Kamoun, 2012). Furthermore, the observed increased evolutionary rates, higher rates of positive selection, more lineage-specific elements and overabundance of effector genes involved in host–parasite interactions in the repeat-rich parts of these genomes strongly suggest that adaptive processes have driven the evolution of such two-speed genomes. The genetic diversification in the quickly evolving regions is driven by structural variation through aberrant recombination and/or transposition, but also repeat-induced point mutations (Dong et al., 2015; Faino et al., 2016; Fudal et al., 2009; Raffaele & Kamoun, 2012).

Two-speed genomes appear to be an elegant evolutionary solution for frequent change in some parts and conservation in other parts of the genome. However, the question remains, how such genome architecture can evolve by natural selection. The current model borrows from the evolutionary concept of clade selection (Williams, 1992) and argues that pathogen lineages with more flexible genomes outcompete other lineages by faster coevolution with the host but also more frequent host shifts (Dong et al., 2015; Raffaele & Kamoun, 2012). Furthermore, it has been proposed that the heavy invasion of TEs in these genomes may have occurred during periods where effective population sizes were small, for example, due to population bottlenecks or periods of extended asexuality and that intragenomic differences in drift and selection (e.g., caused by recombination rate variation) produced the bimodal distribution of TEs we see in extant genomes (Möller & Stukenbrock, 2017).

There are only limited examples of similar mechanisms promoting adaptability in nonpathogenic, free-living species. One such example could be the invasive ant *Cardiocondyla obscurior*, whose genome displays a similar peculiar bimodal distribution of TEs. The genome of this species is populated by distinct and quickly evolving accumulations of TEs ("TE islands;" Figure 5), spanning approximately 7% of the genome and harbouring genes suspected to be particularly important during the founder populations' adaptations to novel environments (Schrader et al., 2014). In invasive species, each founding event drastically reduces the effective population size, thereby decreasing genetic diversity ("founder effect;" Mayr, 1942). At the same time, introduced populations usually face novel environmental conditions that require an adaptive response despite the reduced adaptive capability of small, genetically homogeneous populations. In the case of *Cardiocondyla*, it is suspected that environmental stress following founding events induces transposition bursts that relatively rapidly generate inheritable genetic variation over a few generations, thus facilitating the evolution of locally adapted phenotypes. Intriguingly, there is further empirical evidence implicating TEs in the adaptive radiation of primates (Pace & Feschotte, 2007), bats (Platt et al., 2014) and *Anolis* lizards (Feiner, 2016). In *Anolis*, for example, following a burst of activity, TEs have populated the vicinity of *Hox* gene clusters, that is, the genomic regions that are associated with morphological adaptations to different habitats in these species. More recently, a more general correlation between TEs and speciation/diversification has been discussed for several mammalian lineages (Ricci, 2018). Together, these findings raise the question whether the stress-induced transposition of TEs is a more general driver of diversifying evolution beyond certain fungal and prokaryotic pathogens (Shapiro, 2017).

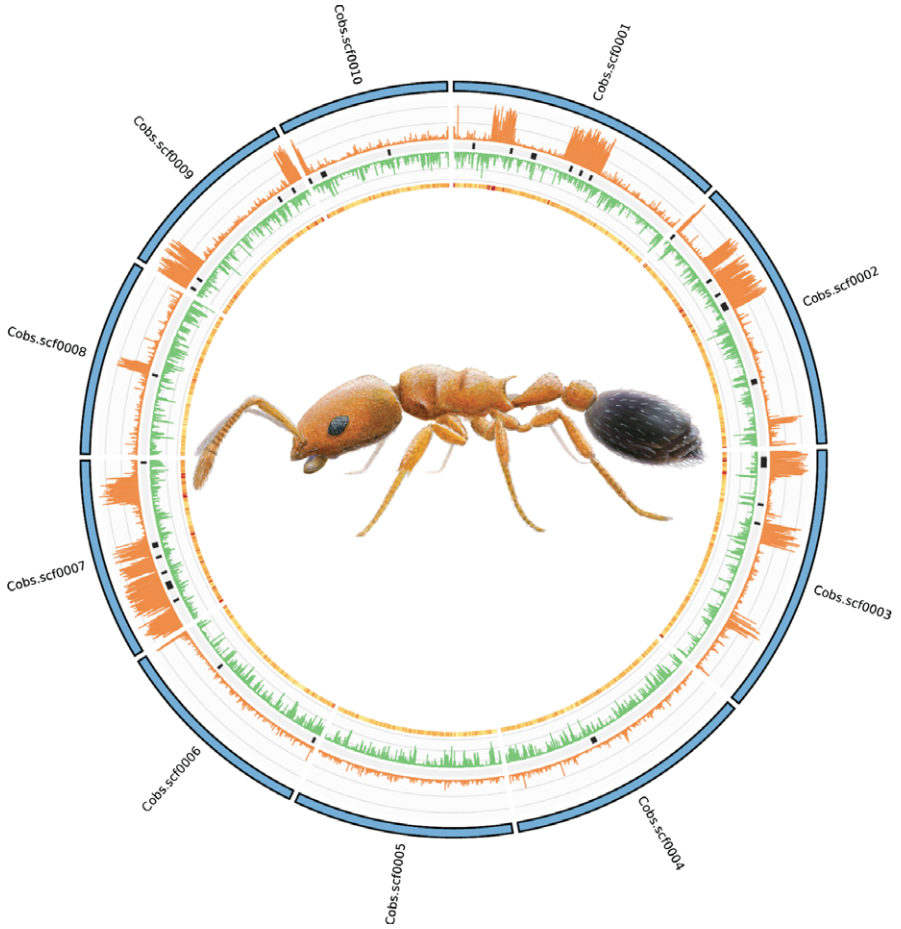

FIGURE 5 Genomic plasticity: Increased frequency of genomic rearrangements and mutations, directly or indirectly caused by TEs. To establish stable populations from genetically homogeneous founding populations, ants of the species *Cardiocondyla obscurior* are required to adapt to novel environmental challenges. Well-defined regions in the genome are enriched in TEs and genes likely involved in environmental adaptation (e.g., olfactory receptors). These "TE islands" are suspected to generate genetic novelty under environmental stress, thus facilitating the genetic diversification of incipient populations and ultimately enabling adaptation to novel environments. The figure shows the genomic architecture for the ten largest scaffolds of the *C. obscurior* draft assembly. The bar plots show TE content (orange) and gene content (green) in 10 kb windows. Black bars indicate the location of OR gene arrays, and GC content is shown in the heatmap

5 | STRESS, TES AND ADAPTIVE EVOLUTION IN NATURAL POPULATIONS

Classic evolutionary theory states that constantly and randomly emerging genetic mutations generate mild phenotypic differences in a population, thus providing the substrate for gradual evolutionary progress through selection and adaptation. However, the narrative of "evolution through gradual change" fails to explain episodes of rapid adaptation and organismal diversification (Gould, 1980). The discovery that TEs spurred and affected fundamental evolutionary innovations evoked a new perspective in evolutionary genetics and that genetic change caused by transposing TEs can in fact generate novel and adaptive phenotypes.

The ratio of beneficial to deleterious mutations is similar for single-nucleotide and TE-induced mutations (Akagi, Li, & Symer, 2013; Barrón, Fiston-Lavier, Petrov, & González, 2014), with most changes being deleterious or nearly neutral (Nellåker et al., 2012). It is an ongoing topic of debate, whether TE-induced mutations are more likely than single-nucleotide mutations to produce stronger phenotypic effects. Given TE-associated mutations often involve shuffling and rewiring of entire functional genetic elements, it is tempting to speculate that major evolutionary changes are in fact more likely to emerge from a single TE-associated mutational event than from a single-nucleotide substitution (Chuong et al., 2017; Ellison & Bachtrog, 2015; Feschotte & Pritham, 2007; Trizzino et al., 2017; Wicker et al., 2016).

Transposable elements are also a major source for intrapopulational genetic variation (e.g., Lockton, Ross-Ibarra, & Gaut, 2008; Stewart et al., 2011). As other genetic mutations, the population-wide fixation of TEs depends not only on fitness effects and generation time, but also significantly on demographic parameters and in particular the effective size of the population (N_e). At low N_e, it is more likely that TEs are fixed by genetic drift; thus, for example, facilitating the invasive fixation of TEs in the genomes of a population after

genetic bottlenecks (Matzke et al., 2012). In addition, the site of insertion can also affect the likelihood of fixation; for example, if genetic linkage to proximate genes leads to genetic hitchhiking effects. Depending on the size of a population and the generation time, fixation of neutral changes in a species can take several million years (Kimura, 1962).

Apart from the regular vertical transmission of TEs from parent to offspring, horizontal transmission across species boundaries also occurs at a relatively low, but evolutionarily relevant frequency (Peccoud, Cordaux, & Gilbert, 2017; Wallau, Vieira, & Loreto, 2018). Evidence from plants, mammals and particularly insects suggests that the introduction of a TE to a new genomic environment by horizontal transmission constitutes the basis for burst-like propagation of the newly acquired TE (Walsh, Kortschak, Gardner, Bertozzi, & Adelson, 2013) and may enable subsequent coactivation of nonautonomous SINEs (Gogolevsky, Vassetzky, & Kramerov, 2008). An unusual alliance between a horizontally transferred, transcriptionally inactive, non-LTR transposable element (RTE), and a small RNA gene was found in the genome of platypus. Interaction between both partners allows the truncated transposon to be cotranscribed with the RNA gene and the RNA gene to be cotransposed with the RTE into more than 40,000 partially active, functional copies, so-called snoRTEs, that are spread over the entire platypus genome (Schmitz et al., 2008).

Transposition occurs primarily during the breakdown of the nuclear envelope in dividing cells (Abyzov et al., 2013), paving the way for the cytoplasmic, transpositionally active enzymes to access the genome. While transposition is most abundant in the hypomethylated germ line (Blumenstiel, 2010), more and more studies have shown that transposition can also propagate in somatic cells; for example, deregulated TEs in cancer (Anwar, Wulaningsih, & Lehmann, 2017), the well-known DNA-transposon-induced mosaic phenotype in maize kernels (McClintock, 1948), and the mosaic patterns of LINE1 activity in the embryonic mouse brain (summarized in (Faulkner & Garcia-Perez, 2017)). In the last case, the question arises of what the phenotypic consequences of such brain LINE1 mosaic activity might be. Does it promote behavioural plasticity or memory formation or does it constitute a neurodegenerative disease? While this question remains ultimately unanswered at this point, new studies suggest that L1 mosaicism in the hippocampus might indeed enable memory formation (Bachiller, del-Pozo-Martín, & Carrión, 2017). It should be mentioned that any of the few active, inheritable LINE1 elements (ten LINE1 elements, for example, are expected to be active in the human genome; Brouha et al., 2003) can become incapable of transposing autonomously, as was recently described for LINE1 activities in megabats (Cantrell, Scott, Brown, Martinez, & Wichman, 2008) and a South America rodent lineage (Casavant et al., 2000). However, as somatic mosaicism cannot be directly inherited, selection can only act on the original, inheritable, active elements in the germ line that drive the somatic mosaicism.

The fact that TE transposition activity fluctuates over evolutionary time and appears to peak during periods of stress (Capy, 2012) provided grounds for extended scenarios for the role of TEs in adaptive evolution. Finding that TEs are both mutagenic and periodically active led to the recent rise in interest in TEs with regard to adaptive evolution, culminating in such compelling models as the "epi-transposon hypothesis" and the "TE thrust hypothesis" (Oliver & Greene, 2010, 2012; Zeh, Zeh, & Ishida, 2009). These and similar models advocate an important role for TEs in adaptive evolution and speciation by driving leaps of rapid genetic and phenotypic diversification. The basis for these hypotheses was the observation that, either by directly activating transposition or by inhibiting genomic silencing mechanisms (Slotkin & Martienssen, 2007), environmentally induced physiological or genomic stress can lead to the liberation and activation of TEs, thus enabling the restructuring and rapid diversification of genomes. In response to changes in the environment, the physiological and genomic stress response can hence trigger random genetic and ultimately phenotypic variation that provides the necessary diversity in a population for subsequent adaptation through natural selection (Oliver & Greene, 2010; Piacentini et al., 2014; Piskurek & Jackson, 2012; Rey, Danchin, Mirouze, Loot, & Blanchet, 2016; Zeh et al., 2009). Further research is required to unravel how such mechanisms could evolve by natural selection and how, for example, changes in effective population size and recombination rate or neutral processes can be accounted for into these models.

The relationship between TEs and environmental stress is complex, and both activation and repression of transposition under stressful conditions have been reported (Horváth, Merenciano, & González, 2017). Unravelling the proximate molecular mechanisms linking TEs and stress is challenging, due to the abundant cellular pathways involved in their epigenetic and transcriptional regulation. In recent years, however, efforts to decipher the potential molecular connections between environmental or physiological stress and the activation of TEs have advanced, uncovering a remarkable interplay between the stress-sensitive heat-shock protein 90 (HSP90) chaperone machinery and the *piwi*-interacting RNA (piRNA) pathway, a molecular suppressor of TE activity (Fanti, Piacentini, Cappucci, Casale, & Pimpinelli, 2017; Gangaraju et al., 2010; Hull, Cruz, Jack, & Houseley, 2017; Hummel et al., 2017; Ichiyanagi et al., 2014; Karam, Parikh, Nayak, Rosenkranz, & Gangaraju, 2017; Sato & Siomi, 2010; Specchia et al., 2010). HSP90 is a broadly conserved, ubiquitously expressed, ATP-dependent chaperone involved in such fundamental processes as the cell cycle, signal transduction and cellular transport (e.g., McClellan et al., 2007). In addition, HSP90 received significant interest from evolutionary biologists over the last decades, due to its role in conferring phenotypic robustness. Initially described as a heat-shock protein (Lindquist, 1986), expression and function of HSP90 are highly sensitive to various forms of environmental stress (Schopf, Biebl, & Buchner, 2017). Stress-induced changes in HSP90 function have repeatedly been shown to lead to the generation of phenotypic variation through disruption of canalized processes and the release of cryptic genetic variation, in particular during development (Chen & Wagner, 2011; Jarosz & Lindquist, 2010; Rohner et al., 2013; Rutherford & Lindquist, 1998; Sangster et al., 2008; Wong & Houry, 2006). More recently, HSP90 has gained interest from

evolutionary biologists due to its role in the regulation of transposable element activity (Kaplan & Li, 2012; Piacentini et al., 2014; Sato & Siomi, 2010; Siegal & Masel, 2012). Similar to its role in canalization, HSP90 appears to be integral to the proximate cellular mechanism leading to the stress-induced activation of TEs observed in many different organisms (Horváth et al., 2017). By interacting with the piRNA pathway (Gangaraju et al., 2010; Ichiyanagi et al., 2014) and the cochaperone Hop (Hsp70/Hsp90-organizing protein; Karam et al., 2017), HSP90 is assumed to be important in the transcriptional suppression of TE transposition—a system that is sensitive to environmental stress. The precise nature of the interplay between HSP90 and piRNAs is still not fully resolved, but it is suspected that HSP90 contributes to the maturation of piRNAs by interacting with Argonaute proteins (Ichiyanagi et al., 2014). Hence, the current model of how environmental stress can lead to HSP90-dependent phenotypic variation has been extended to accommodate the emergence of de novo TE-induced genetic mutations, in addition to the more classic contention of the release of cryptic genetic variation.

6 | CONCLUSIONS

In general, stress and altered selection pressures tend to increase mutation rate, which can either be considered an adaptive organismal response or a simple by-product of physiological, cellular and genomic stresses (reviewed in Lee & Gelembiuk, 2008). Among the mutagenic factors responsive to stressful conditions, as described above, TEs are particularly powerful players in the generation of genetic variation. Thus, increased TE activity may provide the means for genetic diversification within natural populations, enabling the emergence and subsequent selection and fixation of novel adaptive variants through natural selection (Barrón et al., 2014; Prentis, Wilson, Dormontt, Richardson, & Lowe, 2008; Stapley, Santure, & Dennis, 2015).

Thus, TEs are no longer only recognized as the negligible fraction of genomes, but they are considered as potential contributors to evolutionary adaptation. Following liberation from the host's silencing mechanisms, they might cause significant spontaneous changes as demonstrated in some of the above-mentioned case studies. With the ongoing advances in sequencing technology, high-quality, long-read-based genome assemblies now begin to provide the foundation for studying the role of TEs in genome evolution and adaptations in an unprecedented matter.

In the face of climate change and ever-increasing anthropogenic disturbance of natural habitats, research on rapid adaptation is becoming an increasingly important field in ecology, conservation biology and evolutionary biology (Lee, 2002; Lee & Gelembiuk, 2008; Prentis et al., 2008; Shimada, Ishii, & Shibao, 2010). Empirically demonstrating the evolutionary significance of de novo TE-induced mutations, however, remains challenging, as the emergence and subsequent spread and fixation of an adaptive TE-induced variant in a natural population remain the exception rather than the rule.

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