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Review

Jumping genes and epigenetics: Towards new species

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ABSTRACT

Transposable elements (TEs) are responsible for rapid genome remodelling by the creation of new regulatory gene networks and chromosome restructuring. TEs are often regulated by the host through epigenetic systems, but environmental changes can lead to physiological and, therefore, epigenetic stress, which disrupt the tight control of TEs. The resulting TE mobilization drives genome restructuring that may sometimes provide the host with an innovative genetic escape route. We suggest that macroevolution and speciation might therefore originate when the host relaxes its epigenetic control of TEs. To understand the impact of TEs and their importance in host genome evolution, it is essential to study TE epigenetic variation in natural populations. We propose to focus on recent data that demonstrate the correlation between changes in the epigenetic control of TEs in species/populations and genome evolution.

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1. Introduction

Speciation can be a slow process in which genetic differences between individuals become fixed, either as a result of changes in their fitness, ecological specialization, or simply the induction of genetic incompatibility, followed in both cases by micro-population isolation. Speciation is also thought to happen quickly in the context of "non-genic" speciation, i.e., when important karyotypic differences between individuals of the same species lead to sexual isolation. Our exploration of the dynamics of transposable elements (TEs) in natural populations led us to ask how TE mobilization is involved in speciation (Vieira et al., 1999; Rebollo et al., 2008; Fablet et al., 2009). In this short-review we argue that TEs are able to induce speciation through chromosomal rearrangements since, 1) chromosomal rearrangements are able to induce speciation (Noor et al., 2001; Baird et al., 2009; Greig, 2009), 2) bursts of TE transposition can cause chromosomal rearrangements (Geurts et al., 2006; Weil, 2009; Zhang et al., 2009), and 3) bursts of TE transposition may be driven by the selective release of active elements as the result of an epigenetic response to the environment (Lisch, 2009). We also discuss the importance of TE-induced speciation compared to that of other speciation mechanisms.

2. Transposable elements and the genome: a partnership on the move

Genome-sequencing programs have provided new clues explaining the lack of correlation between phenotypic complexity and genome size—which is known as the "C value" paradox—by revealing that most of the differences in genome size between species reside in the non-coding parts (reviewed in Biemont and Vieira, 2006). For instance, the human genome is composed of ~98% of non-coding DNA (International Human Genome Sequencing Consortium, 2004), whereas the fruit fly, *Drosophila melanogaster*, has a very compact genome with far fewer sequences of this type (Dowsett and Young, 1982; Hoskins et al., 2002; Kaminker et al., 2002; Clark et al., 2007).

This variable part of the genome consists mostly of repetitive sequences, such as satellite DNAs and TEs. We will focus mainly on the latter in this short review (see Fig. 1 for a classification of eukaryotic TEs). The evolutionary importance of TEs is no longer open to question. In general, all families of DNA repeats could potentially have an impact on genome organization, either by generating genome instability, since multicopy elements are known to be powerful recombinogenic substrates (Hedges and Deininger, 2007), or as being components of essential chromosomal domains, such as centromeres and telomeres in many species (Wong and Choo, 2004; Lamb et al., 2007). It is clear that TE replication might induce genetic mutations via transposition, as reported for some maize lineages, where *Ac*/*Ds* alternative transposition (from the ends of two different elements) is directly responsible for major chromosomal rearrangements (translocations, duplications, inversions…) (Zhang et al., 2009). The immediate effects

Abbreviation: TE, Transposable element.

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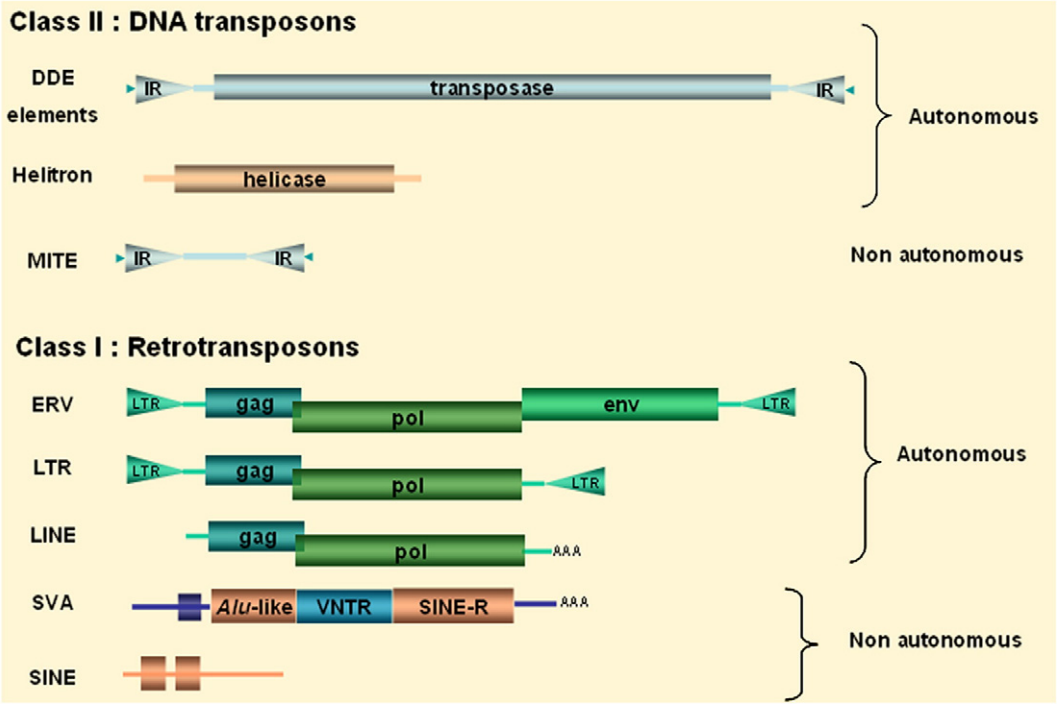

Fig. 1. General classification of eukaryote transposable elements. TEs are abundant and ubiquitous mobile sequences capable of jumping inside the genome. TEs are divided into two major classes on the basis of differences in their transposition mechanisms: Class I Retrotransposons "copy and paste" through an RNA intermediate, whereas Class II DNA transposons just "cut and paste" their own molecule. Autonomous retrotransposons harbor long terminal repeats in their ends (LTR) or not (LINE-like), and can be infectious agents (endogenous retroviruses). Non-autonomous retrotransposons, such as SINEs, are dependent on autonomous elements to be "copied and pasted" *in trans*. The same dependency is observed among DNA transposons, where MITEs need a full-length transposase coded by autonomous DNA transposons to be "cut and pasted" *in trans*. Full-length helitrons, recently identified Class II DNA transposons, play an important role in exon shuffling thanks to their "rolling circle" replication mechanism. For a recent classification of eukaryote TEs, please refer to Wicker et al., 2007. Boxes represent open reading frames, triangles are either inverted repeats (IR) in blue, or long terminal repeats (LTR) in green, and small blue arrows correspond to duplicated insertion site representations. DDE elements: transposases carrying the aspartate (D), aspartate (D), glutamate and (E) motif. MITE: miniature inverted repeated elements; ERV: endogenous retrovirus; LINE: long interspersed nuclear element; SVA: composite element composed of parts of SINE (short interspersed nuclear element), VNTR (variable number of tandem repeats) and *Alu* repeats—the first box represents CCCTCT hexamer repeats; SINE red boxes indicate a diagnostic feature; Gag, Pol, Env: retroviral-like proteins coded by TE open reading frames.

of transposition can be detrimental, as illustrated by several human diseases (reviewed in Callinan and Batzer, 2006). Despite these damaging effects, TEs have been maintained in almost all genomes either as full-length or truncated copies. Full-length copies have kept their ability to mutate the genome as a result of transposition, while the truncated copies have often lost their capacity to transpose. However, the truncated versions might also be recruited by the genome. Indeed, recent reports have proposed a scenario of co-evolution of TEs and hosts, in which TEs (often as truncated copies) are involved in complex genomic processes such as post transcriptional gene regulation, gene protein translation enhancement, etc. (Muotri et al., 2007; Sinzelle et al., 2009). Truncated copies may also act as recombinogenic substrates for other truncated or full-length copies, inducing genome rearrangements. TE copies have been shown to give rise to new regulatory sequences, alternative splice sites, polyadenylation signals (Marino-Ramirez et al., 2005), and new transcription-factor binding sites (Polavarapu et al., 2008). TEs also enhance genome regulation as, for example, when they give rise to microRNAs, which are able to regulate gene expression (Hasler et al., 2007; Piriyapongsa et al., 2007). Since TEs are widespread in the genome and have so many different influences on gene regulation, several authors have suggested that TEs may play a vital role in creating, remodelling and regulating gene networks (McClintock, 1984; Feschotte, 2008). We therefore hypothesize that bursts of TE activity may have profound impacts on genome structure and gene regulation.

3. Bursts of TE transposition drive speciation

TEs have been observed in all sequenced genomes analyzed to date, and comparative genomics gives us a broad insight into the variability of TE content between genomes of different species. For instance, TEs represent 85% of the maize genome (Schnable, 2009), but only 14% of *Arabidopsis thaliana*'s (The Arabidopsis Genome Initiative, 2000). Variations in genome size between closely related species are often related to differences in the amount of TEs. For instance, in the *D. melanogaster* species subgroup, larger genomes are partly attributable to high TE-like sequence content, as estimated from the amount of reverse transcriptase-related sequences determined by dot blot (Boulesteix et al., 2006). In cotton, differences in genome size between species, ranging from 40 to 65%, have been associated with a particular TE subfamily, the *gypsy*-like *Gorge3* element (Hawkins et al., 2006). Differences in the relative proportions of TEs can also occur within a species, as observed for copies of several TE families when counted in the euchromatic arms on polytene chromosomes in natural populations of *D. simulans* (Vieira et al., 1999).

TE abundance, TE-derived genomic features and chromosomal rearrangements involving TE sequences are frequently lineage specific and, therefore, suggest that TEs have contributed to the process of speciation, either as a cause, or an effect (Marino-Ramirez et al., 2005; Bohne et al., 2008). One should note that correlating TE transposition consequences and speciation is rather tricky, since it is difficult to determine the exact timing of TE bursts and natural species diversification. We hypothesize that TE burst of transposition might induce rapid speciation, but the debate is still open. A variety of factors such as gene transfers and losses, mutations affecting "speciation genes", endosymbiotic interactions, antirecombination mechanisms during meiosis, and introgression could also account for reproductive isolation (Presgraves et al., 2003; Scannell et al., 2006; Lexer and Widmer, 2008; Lowry et al., 2008; Greig, 2009). In addition, an influence of TEs on speciation apparently without transposition bursts was reported in

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some species. For instance, the formation of the recent invasive species *Spartina anglica* through natural allopolyploidization was accompanied by major structural and epigenetic remodelling (CpG methylation changes) in the vicinity of TEs (Parisod et al., 2009), but bursts of transposition have not been detected (Baumel et al., 2002). Heterochromatin, which is highly enriched in TEs and other repeats, also seems to play a role in speciation, as observed by Ferree and Barbash in *Drosophila* hybrids (Ferree and Barbash, 2009; Hughes and Hawley, 2009). Furthermore, bursts of transposition are not always associated with rapid speciation. It should be kept in mind that *P* and *I* elements have recently invaded natural populations of *D. melanogaster*, but no speciation has been observed. Also, in a few rice strains, nearly 40 new copies of the DNA transposon *mPing* are observed per plant generation. For the present, the only consequence of this burst of transposition has been slight transcriptional deregulation of a few genes that have *mPing* copies inserted into their 5′ flanking region. However, it is interesting to note that a few of the genes containing *mPing* in their flanking regions are salt- and cold-inducible as a result of the presence of the DNA transposon, suggesting that a few insertions may play an adaptive role (Naito et al., 2009). No massive restructuration of the genome has been observed in rice despite the high rate of transposition, and no speciation seems to be happening in this species. We hypothesize that in order for bursts of transposition to induce rapid speciation, massive genome restructuring or mutations in "speciation genes" have to occur. Several researchers have found examples of concordant timing between bursts of transposition or massive TE extinction and speciation (Table 1). While this short-review was in revision, two bibliographic analyses of the punctuated equilibrium theory, and the general impact of TEs in genomes were published, and both reinforce the hypothesis that TEs may drive macroevolution via bursts of transposition (Oliver and Greene, 2009; Zeh et al., 2009). Significant TE activity is observed in several species, often during periods of radiation, suggesting that massive speciation and massive TE activity may be associated. The genetic distance between two organisms is calculated as a function of their genetic divergence, so every episode that creates divergence, such as lineage-specific transposition events, could contribute to the reproductive isolation of those organisms. TE patterns that differ between individuals of the same species, whether as a cause or a consequence of genetic differentiation, may not only provide genetic markers for researchers, but also constitute evidence of a speciation process occurring within the species concerned (Esnault et al., 2008). For instance, significant TE insertion site polymorphism can be observed in the Japonica and Indica cultivars of rice, and accounts for 14% of their genetic differences (Huang et al., 2008).

Table 1
TE transposition bursts concomitant with radiation periods.

TE events/Species history	References
Decreased L1 and SINE accumulation during emergence of African apes (14–15 Mya).	Consortium (2002)
Generation of L1 subfamilies in less than 0.3 Mya concomitant with intense speciation in *Rattus sensu stricto*.	Verneau et al. (1998)
The timing of Lx family (L1 ancestral family) amplification is close in time to the murine radiation.	Pascale et al. (1990)
Rapid speciation in the genus *Taterillus* (gerbil) occurred, and massive transposition of TEs in new lineages was observed.	Dobigny et al. (2004)
DNA elements were extremely active during the *Myotis* radiation.	Ray et al. (2008)
DNA transposon transposition bursts are concomitant with speciation events in pseudotetraploid salmonids and occurred after genome duplication.	de Boer et al. (2007)
Acquisition and consequent transposition of an endogenous retrovirus element in *Entamoeba histolytica*, and lineage specific enrichment in TEs might affect speciation and pathogenicity.	Lorenzi et al. (2008)

Since the exact evolutionary history of a species is difficult to determine, interspecies crosses provide useful macroevolution study models. Indeed, interspecies hybrids are classic examples of bursts of transposition that have caused severe dysfunctions, and could potentially induce rapid speciation (reviewed in Fontdevila, 2005; Michalak, 2009) (Table 2). Three independent hybrids of the sunflower species have a genome that is 50% larger than the parental lines as the result of a massive TE transposition, and they are thought to have undergone rapid speciation (in fewer than 60 generations in one case) (Ungerer et al., 1998, 2006). Also, in dosage-dependent crosses between *A. thaliana* and *A. arenosa* (crosses where the amount of the maternal and paternal genomes are variable, and so may be different), high expression of the paternal *A. arenosa Athila* element in the hybrid is correlated with seed lethality (Josefsson et al., 2006). Such observations are essential for understanding hybrid "compatibility", since *A. arenosa* and *A. thaliana* hybridization has been successful at least once in nature (Jakobsson et al., 2006). In insects, *D. buzzatii* and *D. koepferae* are still able to interbreed, and share common TE families that are maintained in both genomes. Crosses between these two species considerably induce the transposition of *Osvaldo* in the resulting hybrids, even though it is repressed in both parental genomes (Labrador et al., 1999). In wallabies, interspecies hybrids contain variable centromeres, composed of satellite repeats and newly replicated TE copies (Metcalfe et al., 2007). McFadden and Knowles have designed an algorithm in order to model evolution in asexual digital organisms with or without TEs. They conclude that transposon-mediated mutations were associated with punctuated bursts of rapid evolution and appearance of new adaptive peaks, in contrast to the stasis trap observed in organisms without transposons (McFadden and Knowles, 1997). All these examples suggest that bursts of TE transposition occurring during hybrid speciation may induce important karyotypic changes because of the ability of TEs to induce major chromosomal rearrangements and ectopic recombination (Hedges and

Table 2
Hybrid analysis: epigenetic remodelling and TE activation.

Experimental conclusions	References
Mus musculus and *M. caroli* crosses induce retroelement hypomethylation on chromosome 10, the substrate of double minute chromosome formation in interspecies hybrids.	Brown et al. (2008)
Intraspecific crosses of *D. melanogaster* can result in hybrid dysgenesis, associated with mobilization of *P* or *I* elements, dependent on rasiRNA production in the germinal cell line and causing several abnormalities (such as female sterility).	Brennecke et al. (2008)
Crosses between *D. buzzatti* and *D. koepferae* activate *Osvaldo* copies in the hybrid.	Labrador et al. (1999)
Interspecific macropodid hybrids (*Macropus rufogriseus* and *M. agilis*) present centromeric instability due to TEs and satellite replication, probably inducing karyotypic isolation from the parental species.	Metcalfe et al., 2007
Genome-wide hypomethylation and centromeric expansion, due to TE activation, are observed in *M. eugenii* or *Wallabia bicolour* hybrids.	O'Neill et al., 1998
In *A. thaliana* and *A. arenosa* dosage-dependent crosses, the usually silenced paternal *Athila* elements are activated concomitantly with the deregulation of polycomb complex-dependent gene regulation.	Josefsson et al. (2006)
Wheat allotetraploid formation is accompanied by TE activation, DNA methylation and gene expression alterations.	Kashkush et al., 2002
Helianthus annuus and *H. petiolari* hybrids have a genome 50% larger than parental individuals due to TE amplification.	Ungerer et al. (1998), Ungerer et al. (2006)
DNA introgression in *Zizania latifolia* causes TE activation through modifications in DNA methylation and morphological deviations from the primordial line. Note that *de novo* stable silencing of TEs is observed in the introgressed lines.	Liu and Wendel (2003)

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Deininger, 2007; Weil, 2009). In this way, TE mobilization can result in novel phenotypes followed by the ecological isolation of micro-populations, which are the components required for rapid and divergent evolution.

Both transcription and transposition activities of TEs are controlled via a variety of mechanisms. The expression of TEs is dependent on the presence of transcription factors, as illustrated by the evolution of the L1 lineage in humans. Indeed, the recruitment of regulatory regions in new L1 subfamilies harboring new transcription-factor binding sites is essential for L1 expression (Khan et al., 2006). Furthermore, cellular inhibitors may influence TE transposition post-transcriptionally, as has been observed for some members of the APOBEC family, which are capable of reducing HERV-K infectivity (50 fold) (Lee and Bieniasz, 2007), and block *Alu* transposition in a manner independent of ORF1p L1 (Hulme et al., 2007). Moreover, transposition of mobile elements induces DNA breaks, suggesting that an interaction occurs between the host DNA repair machinery and TEs. ERCC1-XPF heterodimers are implicated in DNA repair processes and limit L1 insertion (Gasior et al., 2008). Apart from cellular inhibitors and transcription factor dependency, TEs are also transcriptionally and post-transcriptionally regulated through epigenetic pathways (Lisch, 2009). However, we know that epigenetic mechanisms are labile in response to environmental changes, and so TEs may occasionally escape silencing, and in some cases could cause genome rearrangements. In order to understand how speciation occurs as a result of transposition bursts, it is therefore essential to understand epigenetic reprogramming.

4. Epigenetic reprogramming of TEs

Epigenetic regulation of TEs involves interdependent pathways, such as chromatin remodelling factors, DNA methylation and non-coding small RNAs (Lisch, 2009; Obbard et al., 2009) (Table 3 for an overview of TE epigenetic regulation). In rice, for instance, specific mutants of histone H3K9 methyltransferase induce DNA demethylation of *Tos17* (*copia*-like retrotransposon) and, consequently, lead to transposition (Ding et al., 2007). In plants, RNA-dependent DNA methylation (RDDM) of TEs and genes is often observed. This is reversible, since it is dependent on the presence of small interfering RNAs (Matzke et al., 2007). Recent investigations have highlighted the central role of RNA in controlling TE activity: such a system was probably present in a common eukaryote ancestor as it is well conserved between species; and it may act as an immunological system against non-self RNAs (Obbard et al., 2009). Also, small RNAs allow for target specificity of DNA methylation or histone modification in a given sequence. For instance, epigenetic instability in long-term cultured cells of *A. thaliana* evolves into the hypomethylation of specific TEs and subsequent activation (Tanurdzic et al., 2008). Indeed, *Athila* or *copia* elements are hypomethylated, regardless of their location, whereas no change is observed for *gypsy* class elements (Tanurdzic et al., 2008). Such specificity is possibly due to the fact that siRNAs are produced differently in TE families subjected to stress of this type, varying from 21 nt and 24 nt for hypomethylated activated TEs, but with only 24 nt for silenced *gypsy* class elements (Tanurdzic et al., 2008).

The TE epigenetic regulation system is in fact rather efficient. It is general in nature, because the TE families capable of invasion are multiple and divergent but, at the same time, it also appears to be specific, and targets single TE families through sequence-specific small RNAs. Each pathway in the epigenetic regulation of TEs seems, therefore, to be both essential and extremely rigorous. Naturally the question arises as to how TEs can possibly invade a genome if they are trapped in an inviolable prison. In fact, we know that TEs often do transpose at a very low rate, suggesting that the prison is after all, somewhat permeable. Indeed, it has recently been suggested that small RNAs can be linked with the total or partial silence of elements, as observed in *Drosophila* hybrid dysgenesis. Indeed, intraspecies *Drosophila* crosses may cause hybrid dysgenesis of *P* and *I* elements, resulting in very seriously deleterious effects, such as female sterility or chromosomal abnormalities (Bucheton et al., 1984; Castro and Carareto, 2004). In these crosses, individuals of the same species have different amounts of TEs since one of the parents has an "empty" genome. A deficit in a small interfering RNA (piRNA) in the maternal gamete allows originally silenced TEs to transpose in the hybrids (Brennecke et al., 2008; Chambeyron et al., 2008).

The study of natural populations and the observation of the natural variability that exists in epigenetic host control can explain TE-induced macroevolution. Epigenetic variation in hybrids, in allopolyploid species, and in single individuals could arouse the TEs, induce a burst of transposition and, as described above, increase karyotypic changes followed by ecological isolation. TE epigenetic regulation has been reported both in somatic tissues (Barbot et al., 2002; Malone et al., 2009) and in germline tissues (Malone et al., 2009). Both types of regulation can influence population behavior by creating potentially heritable phenotypic variations. Variation in TE epigenetic regulation has been observed, for instance, in the LINE-like element *Sadhu*, that displays epigenetic variation (DNA methylation and different silencing states) in three different *A. thaliana* ecotypes (Rangwala et al., 2006). Other epialleles, or differences in the epigenetic regulation of a given sequence in different tissues and/or individuals belonging to the same population, have been reported, mostly in plants and mice. However, further progress in population epigenetics is still necessary, along with ecological epigenetic studies, if we are fully to understand natural population variation in epigenetic regulation (Bossdorf et al., 2008; Johannes et al., 2008; Richards, 2008). TE

Table 3
General view of TE epigenetic regulation.

Histone modifications	Position effect variegation (PEV) is the mechanism behind variation in the transcription of a given gene, and is correlated to its chromatin localization. Mutations in Su(var) genes responsible for such variegation are often accompanied by TE amplification. The major function of this gene family is to post-translationally modify histone N terminal ends. Usually histone methylation in lysine residues (H3K9me, H3K27me, and H4K20me) typically occurs in a closed chromatin conformation, in contrast to the acetylation of histones and methylation in H3K4, which are often observed in open chromatin structures. TEs are closely associated with repressive marks, like H3K9me3 in humans, H4K20me3 in *Drosophila* and H3K9me2 in plants.
DNA methylation	In plants and mammals, DNA methylation plays an important role in silencing TEs. In insects, DNA methylation is observed as a silencing process in genes and TEs.
Non-coding RNAs	Post translational gene silencing (PTGS) via small interfering RNAs (siRNA) processed by the AGO/DICER/RISC complex is another mechanism that can be used to silence TEs. Indeed siRNAs derived from TE copy transcripts can target full-length and putatively active TE transcripts, thus preventing TE transposition. Piwi related RNAs (piRNAs or rasiRNAs, standing for repeated associated small interfering RNAs) in *Drosophila* are processed via the Piwi/Aub/AGO3 pathway, are 24–30 nt and are known to silence TEs in the germline, whereas endo-siRNA (endogenous small interfering RNAs) processed by DICER2/AGO2 are 21 nt, and are capable of somatic silencing TEs. Germinal and somatic silencing are therefore possible thanks to non-coding RNAs. However, the presence and the transcription of a TE copy in the genome are essential to engage PTTES (post translational transposable element silencing). The idea that this constitutes an immune system is therefore appropriate, since having non-coding RNAs of a given TE family will protect the genome from further invasions.

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epigenetic regulation is, therefore, a variable and flexible mechanism that can induce massive TE transposition in the germline, and consequent chromosomal rearrangements.

The gibbon species has rapidly accumulated chromosomal rearrangements and, hence, offers an interesting model for karyotypic evolution and speciation. Carbone et al. recently reported an example of differences in the epigenetic regulation of *Alu* elements in humans and gibbons that is associated with breakpoints between the species (Carbone et al., 2009). They observed that CpG content was higher in the gibbon *Alu* elements near the breakpoints (typical of active elements), and that these elements were undermethylated relative to human *Alu*. *Alu* elements present in the breakpoints are probably active and responsible, in part, for the rapid chromosomal remodelling in the gibbon. The authors propose that "the association between undermethylation and chromosomal rearrangement in gibbons suggests a correlation between epigenetic state and structural genome variation in evolution".

Conjugating two different genomes in the same organism, as in hybrids or in allopolyploids, may require significant adaptations of all the regulatory mechanisms, including TE epigenetic regulation (reviewed in Michalak, 2009) (Table 2). In wallabies, interspecies crosses cause a burst of transposition of a retrotransposon, together with genome-wide hypomethylation (O'Neill et al., 1998). Such a burst of transposition targets a single parental genome, and results in extended centromeres, suggesting rapid karyotype differentiation from the parents (O'Neill et al., 1998). The authors also analyzed some other natural crosses, and found that hypomethylation of the hybrids was always observed as *de novo* chromosomal changes. In allopolyploidization, TE transposition may also be concomitant with genome-wide epigenetic changes (Liu and Wendel, 2003). These examples show how genome remodelling could occur after epigenetic variation in TE copies. However, we need to identify the causes of genome-wide epigenetic modifications and subsequent TE activation. Interspecies crosses induce genomic stress, i.e. changes in genomic stability (chromatin changes, density of repeats…) and organization (DNA recombination, TE replication, retroposed or duplicated genes…), that could indeed have an impact on epialleles and provoke TE activation. Genome-wide epigenetic changes might play a role in genome adaptation to environmental changes. One can readily imagine that TE arousal occurs due to epigenetic changes, and that these changes originate in one individual in response to specific environmental changes. The ecological outcomes of TE mobilization due to environmental changes may be numerous, including things such as survival of the host, increase of host fitness, micro-population isolation etc. The subsequent spread of these factors within populations could lead to sexual isolation and speciation.

5. The environment induces epigenetic reprogramming

Several studies have demonstrated that modifications in the environment can induce epigenetic modifications and, therefore, transcription state changes (Jaenisch and Bird, 2003). Such transcriptional changes are a source of phenotypic variability that may be exploited by organisms to increase the "adaptative potential" of the host. Indeed, diet changes, temperature variation, stress etc. all have an impact on gene regulation (Waterland and Jirtle, 2004; Cropley et al., 2006; Gibert et al., 2007; Chinnusamy and Zhu, 2009). In addition, diet changes, temperature variations, stress etc. could all affect TE transposition (El-Sawy et al., 2005; Hashida et al., 2006; Ebina and Levin, 2007; Cho et al., 2008). Consequently, activation of TEs could result from the relaxation of epigenetic control induced by environmental changes.

There is a huge amount of literature relating the activation of TEs to environmental stress, but only a few examples suggest a link between environmental epigenetic instability and the activation of TEs. Early nutrition has an impact on the epigenetic regulation of TEs, especially via DNA methylation, as reviewed by Waterland and Jirtle, 2004. The *agouti* gene controls hair color in mice (brown in wild type), and the insertion of an IAP retrotransposon in the first exon induces ectopic and variable expression of *agouti*. LTR from IAP elements are regulated by DNA methylation, which varies between individuals. Dietary supplementation (with methyl donors) shifts the phenotype to the wild type brown colour, which is indicative of higher DNA methylation in the IAP element (Waterland and Jirtle, 2003).

In *D. melanogaster*, both heat treatment and aging induce the transcription of older heterochromatic I copies and, hence, the production of small interfering RNAs (rasiRNA) that repress active I elements in the germline (Dramard et al., 2007). DNA methylation of L1 and *Alu1* elements is decreased in individuals exposed to the pollutant benzene (Bollati et al., 2007) and, similarly, benzo(*a*)pyrene increases retrotransposition of L1 elements in HeLa cells (Stribinskis and Ramos, 2006). In mice, a long-term peroxisome proliferating diet induces hypomethylation of satellites, IAP and L1/L2 elements (Pogribny et al., 2007).

6. Conclusion

The fact that TE copies are subject to epigenetic regulation has two main consequences: 1) the environment can have a direct influence on TE activity through epigenetic instability and 2) TE sequences are present in the host genome in a "harmless" state. Since bursts of transposition have been observed in several species it is tempting to suggest that their defense systems have, at least temporarily, broken down. However, this failure is transient, and the host may rapidly silence any *de novo* TE copies produced. Although the benefit is not immediate, transposition might have a long term advantage. Indeed, transposition bursts have numerous consequences, resulting in a renewal of genetic diversity, which is the major prerequisite for genome evolution and selection to occur. Genetic diversity is fundamental for gene networks to be renewed, allowing new species to emerge. Each environmental change indirectly creates an increase in host genetic variability, which means that selection can act over a larger repertoire of genetic information. Epigenetic instability of TEs would lead to significant genetic variability, and the subsequent selection of the best adapted organism.

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