Symposium

Evolutionary Conflict between Mobile DNA and Host Genomes*

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abstract: The proportion of eukaryotic genomes composed of active or formerly active mobile elements (MEs) is known to vary widely across lineages, but the explanations for why remain largely unknown. Given that ME activity, like other forms of mutation, is thought to be (on average) slightly deleterious in terms of phenotypic effects, understanding the widespread proliferation of MEs in host genomes requires an evolutionary framework. To better develop such a framework, we review the spectrum of resolutions to the genetic conflict between MEs and their hosts: inactivation of MEs due to mutation accumulation, negative selection (or lack thereof) against hosts with high ME loads, silencing of MEs (by hosts or MEs), ME domestication by their hosts, and the horizontal transfer of MEs to new hosts. We also highlight ecological and evolutionary theory from which ME researchers might borrow in order to explain large-scale patterns of ME dynamics across systems. We hope that a synthesis of the surprisingly significant role played by MEs in the genome, as well as the spectrum of resolutions, applicable theory, and recent discoveries, will have two outcomes for future researchers: better parsing of known variation in ME proliferation patterns across genomes and the development of testable models and predictions regarding the evolutionary trajectory of MEs based on a combination of theory, the comparative method, experimental evolution, and empirical observations.

Keywords: transposable elements, evolutionary conflict, inactivation, silencing, domestication, horizontal transfer.

Introduction

The large proportion of eukaryotic genomes composed of noncoding DNA—and the paucity of genes—was one of the major surprises to emerge from the human genome sequencing project and the whole-genome sequencing revolution (e.g., Lander et al. 2001). Subsequently, it has been shown that a large portion of noncoding DNA comprises currently or formerly active mobile elements (MEs), which include transposons, retrotransposons, and endogenous viruses (~65% of the human genome; de Koning et al. 2011). As new genome sequences are completed, new patterns related to the dynamics of MEs in their host genomes have emerged, fueling the investigation of their role and leading to the discovery of many previously unknown functions and consequences (e.g., the co-option of MEs for gene regulation in mammals; Chuong et al. 2017). In most cases, however, MEs (like other selfish DNA) and their hosts are in conflict (fig. 1) because of the slightly deleterious nature of ME activity (Houle and Nuzhdin 2004; reviewed in Werren et al. 2011). The deleterious phenotypic effects of MEs mirror other forms of mutation, but their elimination can be countered by their ability to replicate and relocate (box 1).

The range of potential resolutions to ME-host conflicts includes a wide array of strategies (see table 1). For example, an ME can be silenced by a host to prevent proliferation or co-opted by the host, resulting in an adaptation. The development of a more complete evolutionary framework for ME-host interactions, as well as a consideration of the potentially applicable evolutionary and ecological theory (table 2), will be useful to pose and answer a variety of emerging questions related to this major component of eukaryotic genomes. For example, how does ME activity vary between unicellular and multicellular organisms, among diploids and polyploids, or in germline versus somatic tissues? In the past, a few specific questions related to ME activity have benefitted from using theory to motivate empirical studies (e.g., patterns of ME proliferation in sexual vs. asexual organisms; Hickey 1982; Arkhipova and Meselson 2000; Wright and Finnegan 2001; Dolgin and Charlesworth 2006). We argue for an expansion of this approach by reviewing the broad spectrum of evolutionary conflicts between MEs and their hosts and highlighting the theory from organismal biology that might be co-opted to explain the variable success of MEs across taxa (see box 1).We hope this synthesis will help propel the field past a purely descriptive phase to an era where hypothesis-

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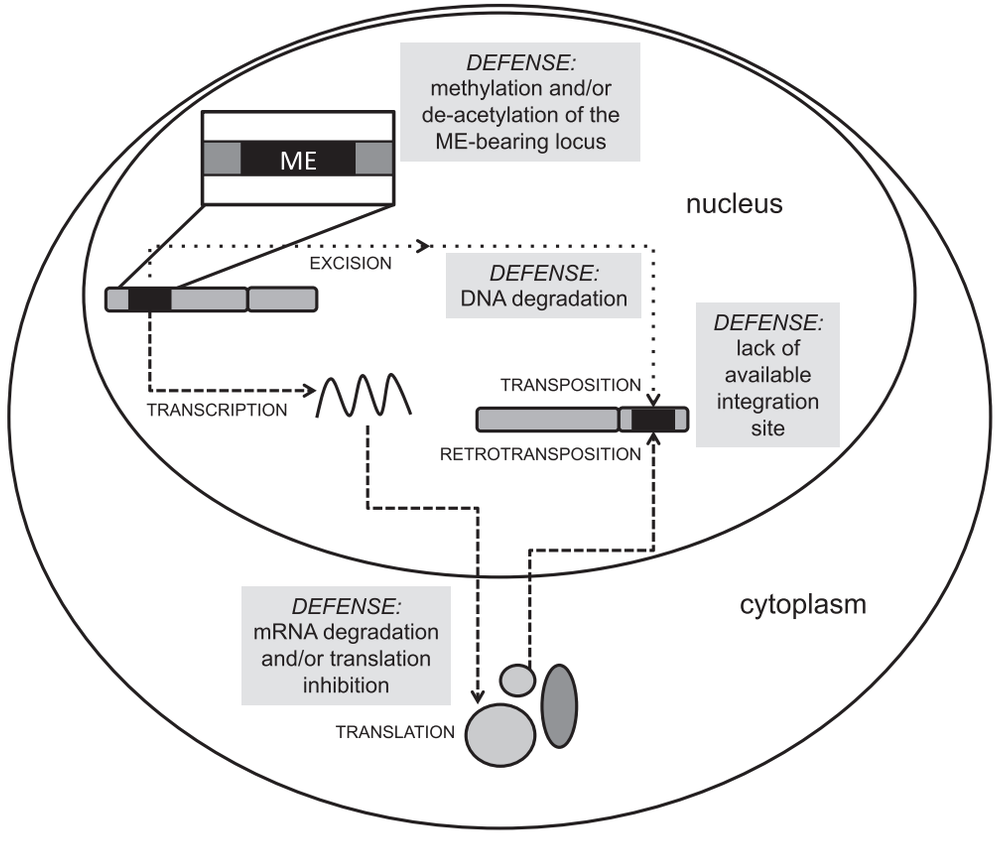

Figure 1: Schematic illustrating points where evolutionary conflicts between mobile DNA and host genomes can occur. The basic mechanisms of proliferation for both copy-and-paste (dashed lines) and cut-and-paste (dotted lines) mobile elements (MEs; black boxes) are shown as an example, with gray boxes highlighting the multiple stages at which hosts can mount a defense.

driven models of genome-wide ME-host dynamics can be tested empirically.

Resolutions to the Evolutionary Conflict between MEs and Their Hosts

Inactivation

Although the majority of many eukaryotic genomes are composed of MEs, frequently these elements are no longer mobile, meaning they are no longer capable of moving within or between genomes as a result of inactivating mutations. In some sense, inactivation of MEs could be thought of as a scenario where the host genome "wins" the evolutionary conflict because the ME "dies" (e.g., Grahn et al. 2005). Although the host still pays the energetic cost of replicating the additional DNA resulting from the existence of the ME, the other translational and mutagenic costs of harboring active elements are eliminated, and the effects of the MEs are likely "nearly neutral" (see table 2). Inactivating mutations do not permanently eliminate the risk of mobility, as back mutations or new mutations could conceivably resurrect an element and reignite the evolutionary conflict. In fact, given the enormous graveyards of MEs in eukaryotic genomes, it is hard to say whether inactivation truly represents a win for hosts, given that MEs appear to be extremely capable of proliferating before hosts manage to inactivate or silence them, and silencing could be a transient, not permanent, solution for the host.

Natural Selection

Like any other mutation, ME activity is thought to be (on average) slightly deleterious; therefore, natural selection should favor individuals with the lowest ME loads, and individuals with new ME insertions should theoretically be less likely to contribute offspring to the next generation (see table 1). In addition to selection against individuals harboring deleterious MEs, loss of individual elements can occur via a variety of mechanisms. Although not all MEs code for the protein machinery required for their own excision, some do, and

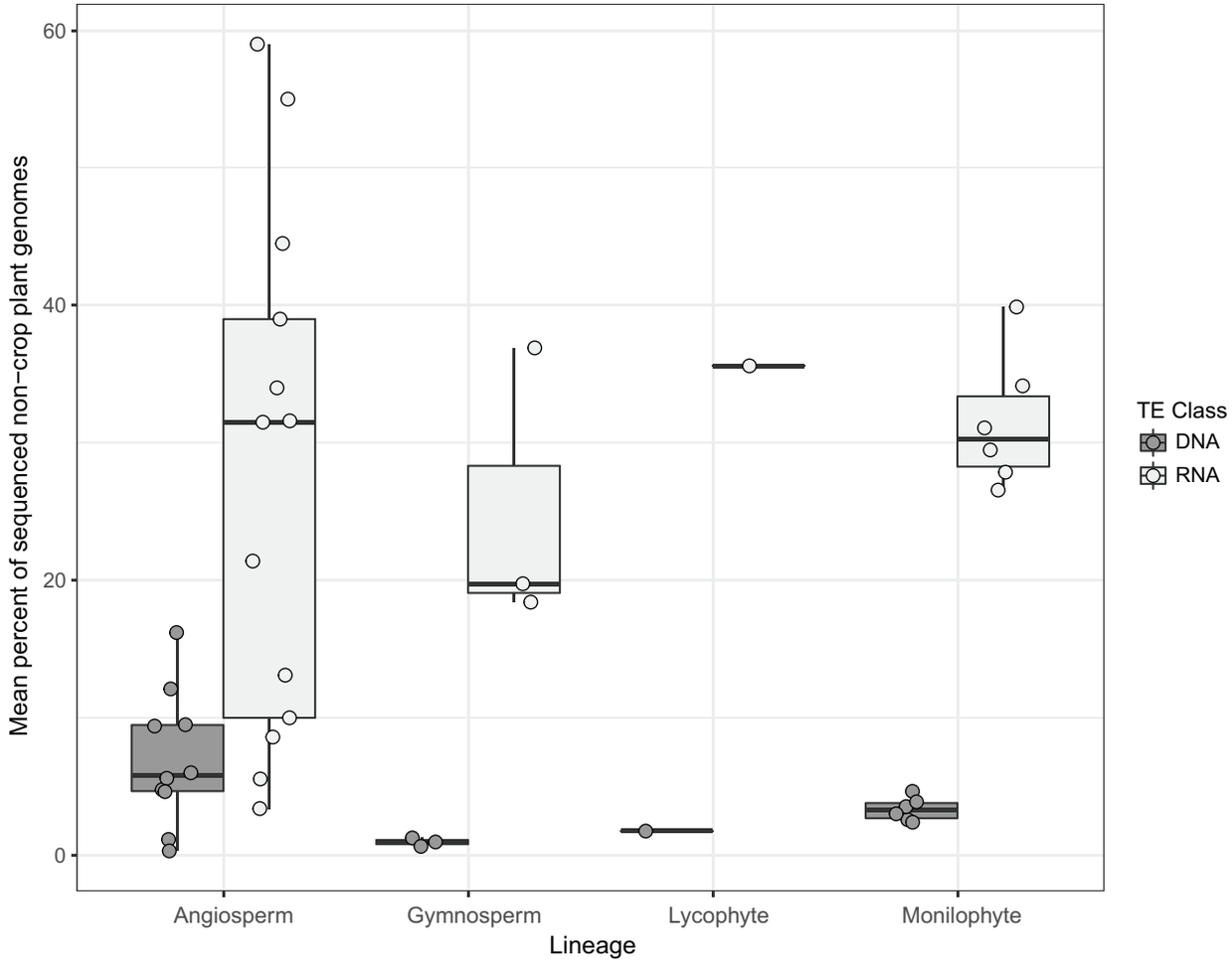

Figure 2: Proportion of mobile element (ME) content in non–crop plant genomes across major lineages from 13 different genome studies. DNA transposons are shown in dark gray, and RNA retrotransposons are shown in light gray. Data and references are in table A1. TE = transposable element.

other MEs can be lost via small- or large-scale deletions. One type of ME, long terminal repeat elements, are famously capable of enhancing the probability of their own loss (or retention) because their terminal repeats provide small islands of highly similar sequence that can increase the risk of ectopic recombination (Frahy et al. 2015). Although the bulk of the selection acting on MEs is presumed to be negative, in cases where MEs are neutral, rampant proliferation may be observed depending on the effective population size (Lynch and Connery 2003) or other features of the host genome (e.g., in plants; see fig. 2 and box 1).

In contrast, another prevalent idea is that ME activity increases in response to environmental stress, putatively in an adaptive manner, to generate the variation on which natural selection can act. McClintock (1984) commented early on the idea that unanticipated "shocks" (e.g., heat shock) to the genome could be disruptive and lead to proliferation of MEs, and this idea has subsequently received support (reviewed in Negi et al. 2016). MEs have been shown to proliferate in a variety of stressful conditions, including hybridization events, heat shock, and whole-genome duplications. Such stressors may trigger important "on-off" switches of regulatory sequences or change kinetics, resulting in mutagenesis or genome reorganization (reviewed in Wessler 1996). Some researchers have gone so far as to suggest that increased ME activity in response to stress gives individuals in a population a genetic variation "lottery ticket" that may equip them to better survive the stressful event (reviewed in Chénais et al. 2012), although this model depends on at least some type of kin or group selectionist argument to offset the potential harm of new ME activity at the level of the individual. This scenario also evokes evolutionary theories of "punctuated equilibrium" put forth to elucidate major morphological leaps in the fossil record (table 2).

Box 1: Variation in mobile element (ME) content across lineages

The widespread success of MEs is clear from the high levels of repeat content in most eukaryotes (Canapa et al. 2015; Wendel et al. 2016). Proliferation of MEs has been offered as a solution to the C-value paradox—the observation that gene content is not correlated with genome size (Oliver et al. 2013; Shiu 2014; Freeling et al. 2015). The large and variable percentages of ME content among taxa show differential success by various elements in different lineages (reviewed in animals in Sotero-Caio et al. 2017), but the causal mechanisms of such patterns are hard to discern. Even among species with relatively recent common ancestors and similar morphologies, the repeat content of the genome can vary tenfold (e.g., among species of *Drosophila*, repeat content ranges from 2.73% to 24.95% in *D. annassae* and *D. simulans*, respectively; *Drosophila* 12 Genomes Consortium 2007), with no known explanation (García Guerreiro 2012).

Another pattern that has been the focus of some inquiry is the apparent difference in activity between MEs that have a DNA versus an RNA intermediate (Chalopin et al. 2015; Wolf et al. 2015; fig. 1). For instance, across plants there are more RNA than DNA intermediate elements in all of the major lineages (Wolf et al. 2015). In some cases, there is activity in a single lineage that contradicts the trends in other members of the order, such as the preponderance of DNA transposons in bats but not in other mammals (Ray et al. 2007, 2008). In some sense, features of different ME families and classes can be thought of as the "life-history" characteristics of the ME, and it may be possible in future studies to predict ME dynamics across lineages based on these features. One of the most provocative hypotheses to emerge from evidence of large bursts of activity of an ME family is the possibility that such mutagenic proliferation fuels adaptive diversification and rapid speciation in the host lineage (Jurka et al. 2011). It is unclear, however, whether speciation events, niche shifts, or other significant macroevolutionary changes are more likely the cause of ME proliferation or a consequence of such proliferation.

Silencing by the Host and by MEs

Active elements can be silenced by the host either before or after transcription via a variety of mechanisms (e.g., DNA methylation, targeted mRNA degradation, or translational inhibition; reviewed in Slotkin and Martienssen 2007). Silencing can be temporary and may not make a given element permanently quiescent (reviewed in Bousios and Gaut 2016). Epigenetic marks, however, can also be inherited, thus impacting evolutionary patterns of ME accumulation across generations (Heard and Martienssen 2014). Work by Hollister and Gaut (2009) in *Arabidopsis* has shown that host-silencing mechanisms that methylate MEs near genes have a deleterious effect on the host, resulting in an evolutionary conflict because they silence nearby functional sequences. Another example of this phenomenon has been observed across *Drosophila* species, where different families of MEs induce different levels of methylation of nearby genes (Lee and Karpen 2017).

MEs can be silenced by the host, but in some cases the mechanism of silencing is derived from MEs themselves (e.g., Piwi-interacting RNAs [piRNAs]). There is now a wealth of empirical support for such silencing (reviewed in Luo and Lu 2017), which was proposed in theory as "self-silencing" (Charlesworth and Langley 1986; Nuzhdin 1999) as a means by which MEs could self-regulate and prevent bringing too much harm to their host as they proliferate. An example of self-silencing is the Piwi-piRNA pathway, an RNA-interference pathway where nested transposable elements (TEs) in piRNA clusters contribute to gene regulation and the silencing of MEs (reviewed in Aravin et al. 2007). Since the fitness cost of transposition for MEs that can form piRNAs would theoretically be lower because they mitigate the negative consequences of transposition, these self-silencing MEs have a higher probability of reaching fixation in natural populations (Lu and Clark 2010). For the host, the machinery for piRNA biogenesis often bears signatures of positive selection, indicating that defense mechanisms evolve in response to evolutionary conflict (Blumenstiel 2016). In *Drosophila*, a piRNA-driven "arms race" in the genus may have even led to reproductive isolation resulting in speciation (Parhad et al. 2017). The reproductive isolation is the result of incompatibilities of the binding and chaperoning genes that complex to localize piRNA clusters, a pattern that evokes ideas from niche theory and the evolution of virulence and cooperation literature (table 2).

MEs have been explicitly implicated in coevolutionary arms races with their host genomes—scenarios where DNA mobilization is suppressed when the host genome mounts a defense, but the defense is later circumvented by genetic changes in the MEs that allow them to remobilize. One of the most well-studied cases of an arms race is between KRAB-containing zinc finger (KZFP) proteins and the TEs they silence in humans (e.g., Thomas and Schneider 2011;

Table 1: Possible outcomes of the evolutionary conflict between mobile elements (MEs) and host genomes

Outcome	Evolutionary framework
Inactivation of MEs	Natural selection will not prevent the accumulation of inactivating, deleterious mutations from occurring within MEs, thus resulting in their inactivation over time and the buildup of "dead" MEs in the host genome
Loss of MEs	Natural selection may favor hosts with the fewest active MEs, since new insertions (like all mutations) are thought to be on average slightly deleterious; MEs can also be lost due to genetic drift
Silencing of MEs	Natural selection may favor hosts with ME suppression mechanisms or may favor hosts bearing MEs capable of self-silencing
Domestication of MEs	Natural selection may favor hosts harboring MEs that have inserted in a specific location or that perform a specific function for the host
Horizontal transfer of MEs	MEs that can escape a given host may colonize a new host genome

Jacobs et al. 2014). However, a recent study in vertebrates has shown that arms race dynamics alone cannot have led to the maintenance of KZFP genes and instead could be an example of co-option of "dead" MEs for gene regulation long after the arms race had been won (Imbeault et al. 2017). Another famous example of genetic conflict where MEs and host genomes exhibit the signature of rapid coevolution is the case of centromeres. Centromeres, while composed of noncoding, highly methylated regions of repeat-rich DNA, perform an essential function for proper chromosome segregation during cell division. Interestingly, centromeric regions are thought to be safe harbors for new TE insertions, centromeric proteins have been shown to often be TE-derived genes, and centromeres play a crucial role in meiotic drive, a form of genetic distortion whereby certain selfish elements can increase the probability of their own transmission (reviewed in Malik and Henikoff 2002).

Domestication

Domestication (or co-option) of MEs is a resolution to evolutionary conflict between an ME and its host whereby the ME performs a function for the host (reviewed in Jangam et al. 2017). Domestication of MEs can take various forms, including the domestication of MEs to silence other MEs (see the previous section). More typically, domestication is thought to be when MEs perform a novel or necessary gene function (including gene regulation) in the host, occasionally using their mobility as part of their functionality (e.g., the maintenance of telomeres by TE insertions in *Drosophila*; Pardue and DeBaryshe 2011). Although domestication (or "exaptation"; table 2) of MEs probably represents one of the most rare resolutions, cases where this occurs are of great interest, and the number and variety of functions performed by MEs for hosts is quite staggering (reviewed in Jangam et al. 2017).

As mentioned previously, domestication events can occur when a single locus is co-opted or, more tantalizingly, when an entire ME family gets co-opted by the host to form, for example, a regulatory network (Chuong et al. 2017). Similarly, a fascinating set of cases of ME domestication is that of syncytins. These placental proteins have been co-opted numerous times in parallel from *env* genes in multiple placental mammalian (and now marsupial; Cornelis et al. 2015) lineages (reviewed in Lavialle et al. 2013). That the same type of ME has been co-opted for a similar function repeatedly in multiple lineages provides compelling evidence that domestication events are not just a collection of spurious "positive" resolutions to the evolutionary conflict between MEs and their hosts. In humans, the most recent case of apparent co-option is the viral-like proteins expressed in neurons to

Table 2: Examples of significant evolutionary and ecological theories and hypotheses that may be usefully applied to studies of mobile element–host conflict and conflict resolution with associated references

Theory	Reference(s)
Ecological models/theories:	
Competition-colonization trade-offs	Hastings 1980
Functional redundancy	Hubbell 2005
Janzen-Connell hypothesis	Janzen 1970; Connell 1970
Keystone predator	Paine 1969
r-/*K*-selection	MacArthur 1962
Lotka-Volterra predator/prey models	Lotka 1920; Volterra 1926
Niche theory	Vandermeer 1972
Island biogeography	MacArthur and Wilson 1967
Evolutionary models/theories:	
Geographic mosaic theory	Thompson 2005
Neutral and nearly neutral theory	Kimura 1968; King and Jukes 1969; Ohta 1973
Red Queen	Van Valen 1973; Lapchin and Guillemaud 2005
Evolution of virulence	Cressler et al. 2016
Evolution of cooperation	Axelrod and Hamilton 1981
Evolutionarily stable strategies	Maynard Smith 1982
Punctuated equilibrium	Eldredge and Gould 1977
Exaptation	Gould and Vbra 1982

encapsulate and deliver materials between synapses (Pastuzyn et al. 2018).

A different type of adaptive resolution to ME-host conflict is that of a beneficial dynamic equilibrium. Oscillating patterns, such as Red Queen dynamics (Van Valen 1973; table 2), have been explored many times in host-parasite evolutionary studies. An example of such stable evolutionary oscillations from the mobile DNA world is that of the effects of the recombination-activating genes (RAGs) on the variable (V), diversity (D), and joining (J) gene segments in jawed vertebrates. These RAG proteins are thought to be TE derived (Huang et al. 2016), and their ability to variably cleave V(D)J sites during lymphocyte production generates a wide variety of cells, which results in a form of adaptive immunity for the host. Although typically thought of as a classic case of ME domestication, given the propensity for errors to occur during the mutation process, the V(D)J example shows the narrow margin of evolutionary conflict between mutualist and mutagen (Roth 2014).

Horizontal Transfer

Another important resolution to, or escalation of, evolutionary conflict is the possibility that an ME can horizontally transfer from one host lineage to another, thereby escaping host-suppression mechanisms in the current genome and entering a potentially naive genome in which the conflict can begin anew (reviewed in Schaack et al. 2010). Whereas horizontal transfer of genes among eukaryotes is thought to be relatively rare, horizontal transfer of MEs might be more prevalent, given that these elements often encode the proteins required for their mobilization and/or transmission. Many hundreds of cases of horizontal transfer of MEs have now been documented in both plants and animals (e.g., El Baidouri et al. 2014; Peccoud et al. 2017*b*).

The likelihood of horizontal transfer as a means of escape may depend on a number of factors, including the mechanism of mobility used by the ME, the availability of vector species or viral go-betweens, the vulnerability of the new host genome to invasion, and the population-genetic environment experienced in the new host (i.e., the strength of natural selection relative to genetic drift). With respect to the likelihood of certain kinds of MEs horizontally transferring more readily than others based on the proteins they encode or their structure (see box 1), it could be useful to borrow from ecological theory and consider the different reproductive strategies used by plants (e.g., *r*- and *K*-selection; table 2). It remains challenging to prove cases of horizontal transfer beyond a shadow of a doubt due to high levels of contamination, template switching during PCR, and spotty taxonomic sampling (reviewed in Peccoud et al. 2017*a*), but the widespread observations of MEs invading new host genome "habitats" and coevolving with host cellular machinery make this an exciting and fertile area of ongoing research that can benefit from an evolutionary conflict framework.

A Case Study: ME Proliferation in the Germline versus Soma

Since the earliest days of ME research, there has been interest in the mechanisms and patterns of transposition in the germline and soma (e.g., Engels 1983). This is an excellent example of a line of inquiry that would benefit from the synthesis of both theory and empirical data (Haig 2016). Empirical studies have long shown that MEs are known to demonstrate tissue- and cell-specific patterns of mobilization between the germline and the soma (Collins et al. 1987). Charlesworth and Langley (1986) pointed out the advantage of germline mobility from the perspective of the TE as a way for TEs to be passed down and the disadvantage of somatic transposition as merely being harmful to their host. From this, they inferred a strong selective advantage for those TEs that do not transpose in the soma. Unfortunately, measuring rates of activity of mobile DNA in somatic cells has been difficult until recent technological advances in single-cell genome sequencing, which make it more feasible now. Despite these advances, measuring rates in the soma and germline of the same organism has rarely been attempted (see table A1; tables A1, A2 are available online). Furthermore, to more accurately determine the interplay of transposition rates and natural selection, it would be essential to have estimates of the phenotypic effects of somatic transpositions. There are not yet enough estimates of somatic rates or phenotypic effects, but when there are more it will be possible to address the long-standing question.

Several interesting questions stem from the possibility of differential mobility between the germline and soma, especially in taxa without a sequestered germline (e.g., many species of plants). In these lineages, somatic mutations can, in a sense, be heritable, thus suggesting that natural selection could favor higher or lower rates of somatic mutation based on life span (as observed in Sarkar et al. 2017). In contrast, several studies have proposed that increased somatic transposition of an ME could be a source of adaptive neuronal plasticity (e.g., LINE1 [L1] elements; Singer et al. 2010; Perrat et al. 2013; Kempen et al. 2017). In humans, high rates of somatic L1 insertion have been observed; however, comparisons to germline L1 activity have been difficult (Richardson et al. 2014; Treiber and Waddell 2017). Genetic mosaicism, while potentially generating diversity, also has negative consequences associated with increased cancer risk and other diseases (Solyom et al. 2012; Bundo et al. 2014; Helman et al. 2014; Tubio et al. 2014). Given the potential costs and benefits of transposition in the germline and soma, untangling the evolutionary conflict between MEs and their host requires both an integrative understanding of the theoretical under-

pinnings and accurate estimates of transposition rates and effects.

Conclusions

While MEs are now known to contribute significantly to most eukaryotic genomes, the explanations for their differential success among host lineages remain elusive. As sequencing technologies and other advances in our ability to collect empirical data have progressed, the need for a robust theoretical framework with which to formulate questions and interpret data is more important than ever. Here, we summarize the five potential resolutions to ME–host genome conflict (table 1), providing examples of recent cases in which these resolutions have been observed in nature. In addition, we argue that different resolutions to evolutionary conflicts between MEs and their hosts may explain the differential success of MEs across eukaryotes, based either on features of the "life history" of the ME or on features of the host genome (box 1). Last, we synthesize some of the major examples of ecological and evolutionary theory that could be borrowed from in future investigations of ME-host interactions in hopes of moving the field from a descriptive phase to an era where comparative studies can test hypotheses more rigorously (table 2).

The technical challenges associated with identifying and characterizing the ME content in the genome remain significant and wide ranging. Sequencing highly repetitive, heavily methylated genomes can be difficult (Grewal and Jia 2007), and assembling such genomes once sequenced can be an elusive goal (e.g., Carvalho et al. 2003). Genomes with high levels of ME content require deeper sequencing coverage and benefit from more recent sequencing technologies that produce longer reads (e.g., McCoy et al. 2014). In fact, much of what was first reported about the ME content of eukaryotic genomes in early sequencing projects was misleading because the genomes that could be sequenced were often those with relatively few repeats (e.g., *C. elegans* Sequencing Consortium 1998; *Arabidopsis* Genome Initiative 2000). Once sequenced, identifying MEs in the genome, especially those that have never before been characterized, can be difficult (Flutre et al. 2011); and once identified, quantifying copy number, relatedness, rates of mobilization, and phenotypic effects are not easy tasks. As these empirical challenges are solved, the need for a robust theoretical framework with which to explain our observations will become more important than ever.

Descriptive investigations of ME patterns have been critically important—they are comparable to exploring the natural history of taxa and the features of the habitat in which they are found (i.e., the community ecology of the genome; Venner et al. 2009). Even in well-studied model organisms like *Drosophila*, previously unidentified ME families can be found long after initial annotation efforts because of improved detection software and/or more affordable short- and long-read sequencing resulting in revised estimates of overall ME content with each release (Gramates et al. 2017). Similarly, in the case of early experimental data sets on transposition rates of well-characterized elements (what can perhaps be thought of as the behavioral ecology of the MEs), it was difficult to accurately estimate ME dynamics because of the limited sensitivity of molecular methods and bioinformatic tools (Maside et al. 2001). More recent efforts to characterize insertion and deletion rates rely not only on whole-genome sequence data but on more sophisticated statistical approaches that help determine the difference in detection bias between different events or test for correlations among mobilization events and genomic context (e.g., Adrion et al. 2017).

While more and more ME studies are motivated by theory (e.g., Serra et al. 2013; McLaughlin and Malik 2017), the gains to be made from unifying conceptual frameworks with data collection and mechanistic studies in the future are still significant. In the same way that naturalists might wish to place their fascinating observations in a larger context by interacting with ecologists and evolutionary biologists who propose, test, and develop theory, molecular biologists and genomicists can benefit from the arsenal of theory- and observation-based models that have been developed in other fields. The evolutionary conflict framework, in particular, can provide a useful context for future investigations of the success and regulation of MEs in their host genomes over long time periods.

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The Red Queen from Lewis Carroll's *Through the Looking-Glass* (1871 illustration by John Tenniel).