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Genomic parasites or symbionts? Modeling the effects of environmental pressure on transposition activity in asexual populations

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ABSTRACT

Transposable elements are DNA segments capable of persisting in host genomes by self-replication in spite of deleterious mutagenic effects. The theoretical dynamics of these elements within genomes has been studied extensively, and population genetic models predict that they can invade and maintain as a result of both intra-genomic and inter-individual selection in sexual species. In asexuals, the success of selfish DNA is more difficult to explain. However, most theoretical work assumes constant environment. Here, we analyze the impact of environmental change on the dynamics of transposition activity when horizontal DNA exchange is absent, based on a stochastic computational model of transposable element proliferation. We argue that repeated changes in the phenotypic optimum in a multidimensional fitness landscape may induce explosive bursts of transposition activity associated with faster adaptation. However, long-term maintenance of transposition activity is unlikely. This could contribute to the significant variation in the transposable element copy number among closely related species.

The evolution of species depends on both the strength of selection and the species' capacity to evolve. Small environmental changes tend to generate moderate stress on populations, which are likely to reach the new phenotypic optimum from standing genetic variation. On the contrary, large and fast shifts in the environment may generate substantial selection pressure, endangering the survival of the species, and adaptation may require the accumulation of several mutational changes (Barrett and Schluter, 2008; Durand et al., 2010). In any case, the ability for the population to generate new variants through mutation remains a crucial feature that conditions its capacity to cope with environmental challenge. or evolvability, are still not fully understood (Hansen, 2006; Partridge and Barton, 2000; Pigliucci, 2008). Both theory and empirical observations suggest that, in some conditions, adaptive evolution of mutation enhancers is realistic (Taddei et al., 1997). In this context, mobile and mutagenic sequences such as Transposable Elements (TEs) appear as natural candidates for evolvability helpers (Blot, 1994; Chao et al., 1983; Schneider and Lenski, 2004).

Transposable elements are self-duplicating DNA sequences that are present in virtually all living species (Biémont, 2010). Yet, understanding their presence, distribution, copy number, insertion patterns, and their propensity to be maintained in constant or changing environments is still under theoretical investigation (Charlesworth et al., 1994; Le Rouzic and Deceliere, 2005). Generally considered as genomic parasites in sexual organisms (Charlesworth and Charlesworth, 1983; Doolittle and Sapienza, 1980; Hickey, 1982; Orgel and Crick, 1980), their mobility promotes both deleterious mutations and genetic innovation. However, the spread of such selfish DNA requires sexual reproduction, and this mechanism cannot explain the persistence of TEs in selfing, parthenogenetic, and clonal organisms (Wright and

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Finnegan, 2001). Indeed, theoretical developments generally predict that active deleterious TEs should either be eliminated from asexual lineages, or drive them to extinction (Charlesworth and Charlesworth, 1983; Wright and Schoen, 1999; Dolgin and Charlesworth, 2006; Boutin et al., 2012), which has often been supported empirically (Zeyl et al., 1996; Arkhipova and Meselson, 2005). The presence of TE sequences in asexuals is thus generally attributed to rare but recurrent intra- or inter-specific horizontal transfers, compensating the extinction of TE-carrying lineages (Moody, 1988; Basten and Moody, 1991; Bichsel et al., 2010).

Understanding the impact of TEs on evolution and their role in the response to environmental pressure remains particularly challenging, as these sequences can be both beneficial and detrimental for their host (Capy et al., 2000). Indeed, being mutagenic by nature, they are, on average, deleterious. Most insertions that are not neutral tend to disrupt useful genes, and only a small fraction of TE-driven mutations has the potential to be favored by natural selection, a process often referred to as 'molecular domestication' (Miller et al., 1992, 1997). TE-promoted evolutionary innovations include insertions, deletions, and recombinations, but may also involve TE sequences themselves as new genes or part of chimeric transcripts (Sinzelle et al., 2009). Consequently, TEs are generally considered as major contributors to genomic plasticity (Capy, 1998).

In clonal organisms, the rare occurrence of advantageous mutations may balance the fitness cost of carrying TEs, allowing the persistence of active copies in genomes. Interestingly, the dynamical properties of TEs in asexuals have led to little theoretical investigation compared to sexual populations. The possibility that prokaryotic TEs might act as evolvability enhancers was confirmed theoretically (Sawyer and Hartl, 1986; Martiel and Blot, 2002), but simulations were stopped after a single adaptive walk, leaving unexplored the dynamics of TEs once the fitness peak was reached. In the model proposed in McFadden and Knowles (1997), they are maintained for a long time because TE-promoted mutations allow TE-carrying lineages to cross adaptive valleys and thus explore more efficiently the adaptive landscape. Although exciting, this model strongly relies on the hypothesis that TE-mediated mutations have significantly larger phenotypic effects than 'regular' background mutations, which does not appear to be supported empirically (Stoebel and Dorman, 2010). The idea that TEs could be maintained on a long-term due to recurrent environmental changes was developed more recently in Edwards and Brookfield (2003) and McGraw and Brookfield (2006), where the authors identified the timing of environmental shifts as the major factor conditioning the survival of TEs in clonal organisms. However, such models were explored only in simple cases (e.g. shifts between only two environments, unconditionally neutral insertions, no or limited evolution of TE sequences).

In particular, intra-genomic competition between TE copies may prevent TE-host systems from reaching an equilibrium. It is well-known that super-parasitic, non-autonomous elements are often successful and can seriously impact the evolutionary dynamics of autonomous copies (Brookfield, 1996; Hartl et al., 1992; Le Rouzic and Capy, 2006). Such intra-genomic competition between TE copies may lead to complex evolutionary dynamics, including TE loss or successive bursts of re-invasion, closely matching empirical observation (Le Rouzic et al., 2007).

In this paper, we develop a general model of TE evolution in clonal organisms accounting for TE polymorphism (including autonomous and non-autonomous copies). Several environmental scenarios were considered (two being shown here), determining the size and the frequency at which TE-related mutations can be favored by natural selection, and the long-term dynamics of the TE-host system were explored for thousands of generations.

2. Method and results

Here we present a stochastic computational model of TE proliferation that enables exploration of the interplay between environmental changes and TE activity. We considered populations of 10,000 clonally propagating individuals carrying both autonomous and non-autonomous TEs. Each organism is defined by its phenotype together with its TE genomic content. Simulations are initialized by introducing a single autonomous element in every individual of a population well-adapted to the current environment (all individuals are at the phenotypic optimum). See Fig. 1 for the general outline of the model.

2.1. Phenotype and natural selection

The phenotype-fitness map is adapted from Fisher's geometric model (Fisher, 1930; Martin and Lenormand, 2006) with a moving optimum (Kopp and Hermisson, 2009; Orr, 2005). The phenotype of an individual is represented as a vector of n real numbers, each coordinate representing an independent trait involved in the adaptation of the organism to the environment.

The carrying capacity of the environment is m, i.e. the actual number of organisms fluctuates slightly around m. Associated with the environment is an 'optimal phenotype', i.e. a combination of phenotypes for which fitness is maximal.

Organisms whose phenotypes are close to the optimum are considered more 'fit' than organisms with phenotypes distant from the optimal phenotype. The fitness function is calculated from the standard n-dimensional Euclidean distance between the phenotype of an individual o (denoted by $\boldsymbol{\pi}(o) = [\pi_i(o)]_{i=1\ldots n}$) and the optimal phenotype $\hat{\boldsymbol{\pi}} = [\hat{\pi}_i]_{i=1\ldots n}$, as follows:

$$F(o) = \exp(-\mathrm{dist}(\boldsymbol{\pi}(o), \hat{\boldsymbol{\pi}})^2) = \exp\left(-\sum_{i=1}^{n}(\pi_i(o) - \hat{\pi}_i)^2\right).$$

The fitness function does not depend on the TE count of an organism, and as such, does not enforce an artificial transposition–selection equilibrium.

Environmental change is modeled by shifting the optimal phenotype. We assume that among the n traits, $n/2$ have invariant optima and the other $n/2$ traits change every T generations by a deterministic factor s so that the change is directional. The fixed traits are introduced in order to model more realistically a natural environment (which might be changing in some aspects, while remaining stationary in other). Additional simulations (not shown) confirm that the model behaves in a similar fashion for a wide range of 'fixed' traits (between 0 and about $0.8n$). In the scenario called 'Global Warming', the optimal phenotype changes by a small amount ($s_{\mathrm{GW}} = 0.0002$) every generation ($T = 1$). In the 'Meteor Impact' scenario, the change is larger ($s_{\mathrm{MI}} = 0.075$) and occurs every $T = 500$ generations.

Generations are non-overlapping. The number of offspring produced by an organism is drawn from Poisson distribution with the mean proportional to the organism's fitness. The relative fitness is multiplied by a scaling factor, chosen in each generation in such a way that the expected number of offspring equals the carrying capacity of the environment.

2.2. Transposition

Our model considers two kinds of transposable elements: autonomous and non-autonomous copies. Autonomous copies transpose with a constant rate τ per copy and per generation. Non-autonomous copies, which can "parasitize" the transposition enzymes produced by autonomous copies, transpose at a rate of

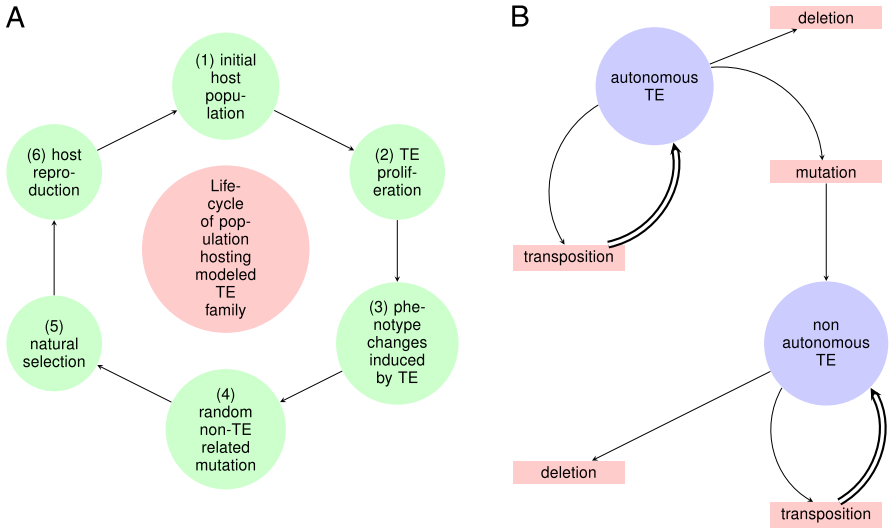

Fig. 1. General outline of the model. (A) Life cycle of the population: (1) simulation starts with a population of 10,000 individuals; (2) both autonomous and non-autonomous transposable elements are mobilized by the transposition machinery produced by autonomous TEs; (3) each TE inserted causes a mutation of the host phenotype; (4) non transposition-related mutations also modify the phenotype; (5) better adapted individuals (i.e. closer to the *optimal phenotype*) have greater probability of survival; (6) surviving individuals reproduce to fill the environment back to its capacity. (B) Evolution of transposable elements: an autonomous element can duplicate during the process of transposition or become non-autonomous; a non-autonomous element can still proliferate using the transposition machinery produced by autonomous elements.

$\tau \cdot [A]$ per copy and per generation, where $[A]$, the concentration of transposition enzymes, is proportional to the number of autonomous copies in the cell. Therefore, non-autonomous copies cannot transpose in the absence of autonomous copies, and their transposition rate increases with the autonomous copy number. In our stochastic simulations, the actual number of transpositions for each copy was sampled in a Poisson distribution. In addition to proliferating, autonomous TEs can spontaneously turn into non-autonomous copies with probability Δ_α, and both autonomous and non-autonomous TEs can disappear (by deletion or by being mutated beyond recognition) with probability Δ_β.

2.3. Mutations

Insertion events create *de novo* genetic variation, which in vivo may result in a range of functional alterations, ranging from gene knockouts to subtle regulatory shifts. In addition to transposition-related mutations, transposition-unrelated mutations (e.g. nucleotide substitutions) occur with a constant rate of $\rho = 0.003$. Both types of mutations have the same effect on the phenotype, shifting a single random phenotypic trait by a random number drawn from normal distribution, $Norm(0, \mu)$ where $\mu = 0.1$, the mutational standard deviation, is a parameter of our model. Note that the phenotypic change inflicted by transposition stays with the phenotype regardless of further fates (such as deletion) of the transposon which caused it. Mutations are not pleiotropic, i.e. they do not affect several traits at once (in other words, the set of traits can be understood as independent phenotypic directions). Unlike the situation in most models, a positive number being randomly drawn does not necessarily result in a helpful mutation (just like a negative number need not result in a detrimental mutation). The effect of a mutation depends on the relative position of the host organism's phenotype and the optimal phenotype: for well-adapted organisms, most mutations are detrimental, as they push them away from the optimum. With mutational effects being drawn from a normal distribution, some mutations will be almost silent, while others will have noticeable impact on the phenotype, and the mutations coming from the tails of normal distribution are likely to have an immediately lethal effect. Table 1 presents parameter settings fixed in simulations.

2.4. Constant environmental pressure ('Global Warming' (GW) scenario)

The pressure exerted on the host population by slow, gradual environmental changes was modeled by a cumulative, directed shift of the 'optimal phenotype' in each consecutive generation. Both transposition activity and TE copy number increase with the intensity of environmental change (Fig. 2). If the level of environmental change is very low to nonexistent, TEs are only deleterious and disappear from the population.

A transposition–selection–drift equilibrium can be frequently observed under a moderate environmental change (Fig. 3(B)). Active transposition maintains a stable number of TE copies, as well as a moderately high mutation rate (accounting for both transposition-related and transposition-unrelated mutations). Although most mutations are deleterious, some are beneficial and become fixed in the host population. If the environment is constant (not shown), transposition activity is only deleterious, and clones carrying TE copies are lost.

When autonomous TEs can mutate into non-autonomous TEs with frequency $\Delta_\alpha = 0.0003$ per generation, after an initial stage similar to the previous case (autonomous elements are active and stimulate the mutation rate), non-autonomous copies amplify, and the number of autonomous copies decreases (Fig. 3(C)). The transposition rate (and the induced mutation rate) are maintained, since only a few autonomous copies are enough to stimulate the transposition of many non-autonomous copies. However, this stage is followed by the loss of all autonomous copies, which eventually leads to the loss of transposition activity. At the end of the simulations, all TEs disappear, and the evolvability of populations (its capacity to track environmental changes) is reduced. Fig. 3(A) presents simulation without TEs for reference. In this case transposition-unrelated mutations manage to track the optimal phenotype, but the average fitness is lower, than with the presence of TEs.

2.5. Rapid environmental change ('Meteor Impact' (MI) scenario)

When the environmental change is large and instantaneous, populations do not have the possibility to track the optimum. The optimum shift is thus followed by a stage of directional selection, during which the average fitness in the population remains below

Table 1
Parameter values used in simulations presented in: Fig. 3(B) (Global-warming without non-autonomous TEs), Fig. 3(C) (Global-warming with non-autonomous TEs), Fig. 3(A) (Global warming without TEs), Fig. 4(A) (Meteor impact without TEs), Fig. 4(B) (Meteor impact without non-autonomous TEs), and Fig. 4(C) (Meteor impact with non-autonomous TEs).

Parameter	Symbol	GW without non-autoTEs (Fig. 3(B))	GW with non-autoTEs (Fig. 3(C))	GW without TEs (Fig. 3(A))	MI without TEs (Fig. 4(A))	MI without non-autoTEs (Fig. 4(B))	MI with non-autoTEs (Fig. 4(C))
Dimension of phenotypic space	n	10	10	10	10	10	10
Mutation stdev.	μ	0.1	0.1	0.1	0.1	0.1	0.1
Non-TE-related mutation rate	ρ	0.003	0.003	0.003	0.003	0.003	0.003
Niche size	m	10 000	10 000	10 000	10 000	10 000	10 000
Autonomy loss probability	Δ_α	0.0	0.0003	–	–	0.0	0.0003
Deletion probability	Δ_β	0.003	0.003	–	–	0.003	0.003
Transposition rate	τ	0.003	0.003	–	–	0.003	0.003
Environmental change[a]	–	0.0002	0.0002	0.0002	0.075	0.075	0.075

[a] Measured in phenotypic units per generation for GW and phenotypic units per impact (every 500 generations) for the MI model.

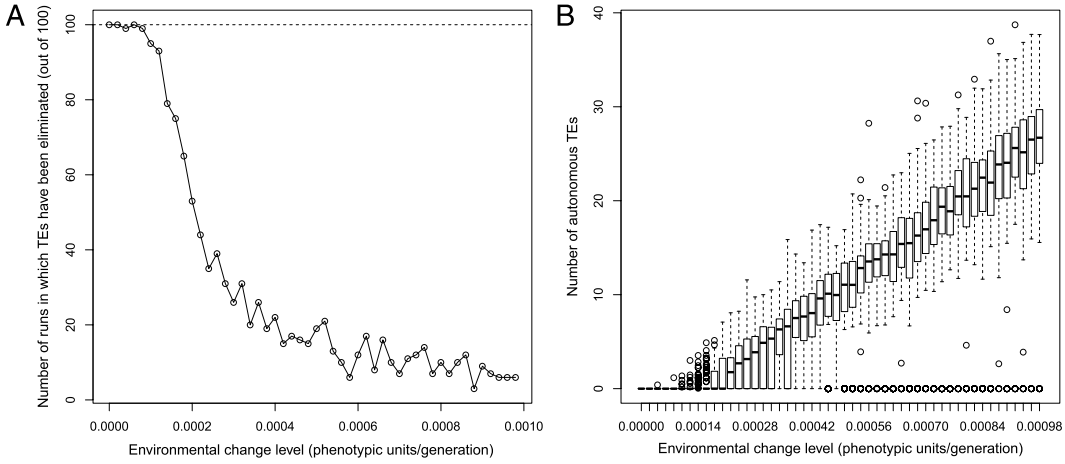

Fig. 2. (A) Number of runs (out of 100) in which autonomous TEs are eliminated by generation 5000 under the "Global Warming" scenario (no non-autonomous copies). In low stress levels all TEs are lost, and the phenotypic optimum is tracked purely through TE-unrelated mutations. With higher levels of environmental change, TEs are maintained more often and assist in tracking the phenotypic optimum by providing extra mutations. (B) Distribution of the average number of autonomous TE copies at generation 5000, from 100 simulation runs at each change level. The TE copy number at generation 5000 increases linearly with the rate of environmental change above the minimal threshold allowing for TE persistence.

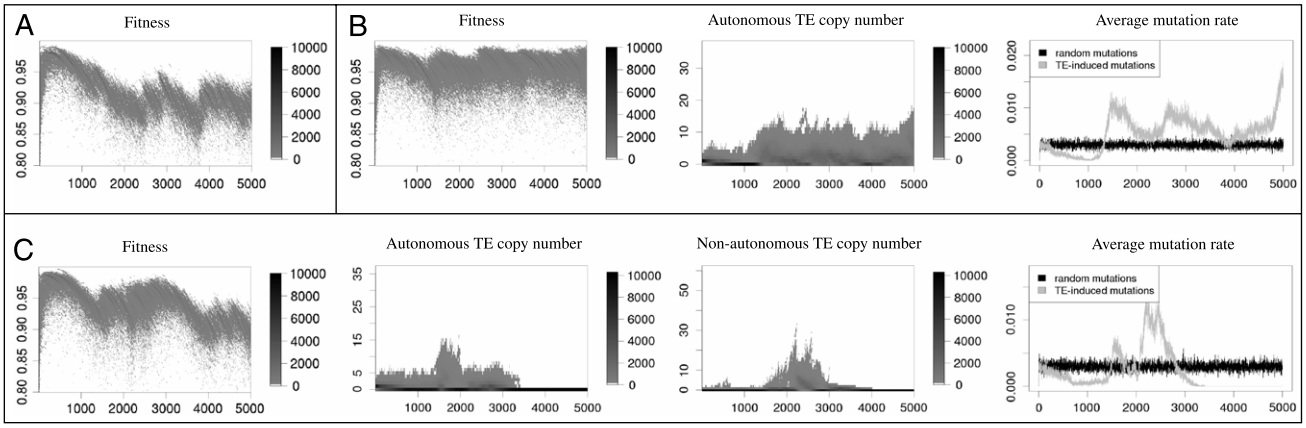

Fig. 3. Effect of smooth environmental change ('Global Warming' scenario). (A) Reference simulation without TEs: transposition-unrelated mutations are enough to track the optimum in the long term. The distance of the population from the optimum is larger than with TEs (the average fitness is lower). The color scale is proportional to the density distribution of fitnesses in the population. (B) Non-autonomous copies disabled. TEs assist in tracking of the phenotypic optimum. (C) Non-autonomous copies enabled.

the original fitness. If the population is evolvable enough, it can reach the new optimum between two "impacts"; otherwise, stages of directional selection follow each other.

Fig. 4 shows a situation in which the changes are too large and too frequent to be tracked efficiently by transposition-unrelated mutations only: if there are no TEs, the population never reaches the phenotypic optimum (the absence of individuals having the optimal fitness); see Fig. 4(A). When autonomous TEs are present, the mutation rate increases, and the population can reach the optimum. Once at the optimum, TE activity is only deleterious, and the copy number tends to drop. If a new environmental change happens before the loss of all copies, active TEs proliferate again, which can lead to the long-term maintenance of active TE copies (Fig. 4(B)).

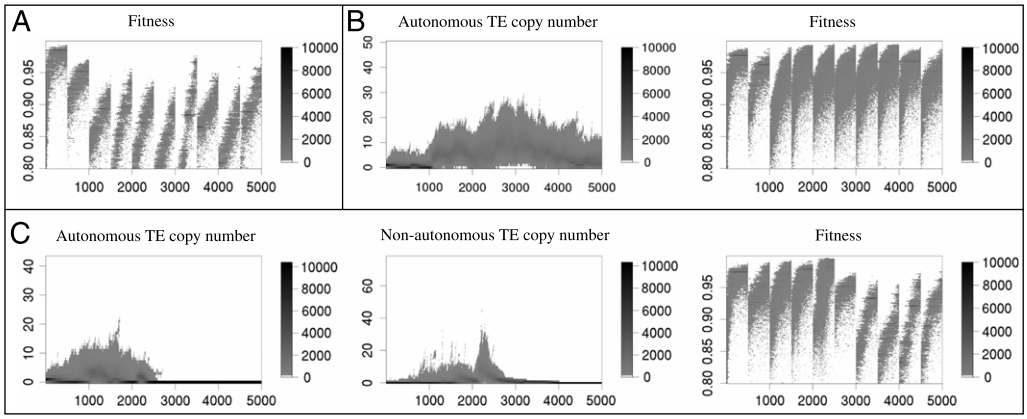

Fig. 4. Effect of periodic dramatic environmental shifts ('Meteor Impact' scenario). (A) Without TEs the population diverges from the phenotypic optimum. (B) With autonomous copies the population is able to track more closely the phenotypic optimum. (C) With both autonomous and non-autonomous copies, the population follows the phenotypic optimum, until TEs are eliminated.

Introducing non-autonomous copies has a similar effect as in the GW scenario. Non-autonomous mutants take over the autonomous copies, up to the point where all active copies are eliminated. The major consequence of the loss of transposition activity is a decrease in evolvability, leading to a fitness drop, and the inability to cope efficiently with environmental challenges (Fig. 4(C)).

2.6. General properties of the model

We tested our model using a range of parameter settings, including different population sizes, transposition and mutation rates, etc. In addition to the simulation engine, we have developed a GUI interface for browsing the results accessible from http://bioputer.mimuw.edu.pl/transp.

The model generally behaves in a stable fashion. As a rule, the increase of the environmental change level results in a raise in the TE activity. TEs proliferate most intensely under long-lasting directional changes. In contrast, realistic levels of non-cumulative random environmental fluctuations did not result in any significant TE activation: even if the level of stress imposed by random fluctuations is close to being lethal to the host population, the number of TEs rises, but remains an order of magnitude lower than that in the GW scenario. The figures (especially Fig. 3) show that the main mechanism of evolution relies on selective sweeps by clones carrying beneficial mutations (almost instantaneous fitness increase), followed by stages of slow fitness decay, during which the different lineages tend to accumulate deleterious mutations independently. This mode of evolution is reminiscent of the patterns observed in "mutator" models (see Section 3).

3. Discussion

3.1. Model

The model described in this paper aims at understanding and predicting the long-term evolution of transposable elements in asexual species living in changing environments. Compared to the relevant literature, this model brings several significant improvements: (i) the diversity of TEs is represented through autonomous and non-autonomous copies, (ii) the mutational effect of TE mobility on phenotype is modeled explicitly, (iii) fitness is determined according to the distance to a phenotypic optimum, and (iv) this setting makes it possible to model complex and various environmental change scenarios. Our modeling results indicate that TEs can be maintained in asexuals for a wide range of scenarios involving environmental change, as suggested in Edwards and Brookfield (2003) and McGraw and Brookfield (2006). However, we conclude that intra-genomic competition tends to affect the stability of the host-TE system, and our simulations repeatedly report the loss of the transposition activity when non-autonomous copies are present. If confirmed, this observation precludes the persistence of long-term TE-host symbiosis, restraining TE-genome cooperation to short periods.

As for any model, our framework remains a simplification of reality, and many details were not accounted for. For instance, even asexual organisms are known to share episodically DNA sequences, through e.g. bacterial conjugation or horizontal transfers (HTs). However, a preliminary study we have performed suggests that these do not alter significantly the dynamics of TE families, as long as their frequency remains reasonable when compared to mutation rates, transposition rates, and selection coefficients, which is likely. Very high (unreasonably high) incidence of HTs has, however, the potential to gradually shut down transposition. See the Supplementary Information for details. In any case, sporadic horizontal transfers remain essential for TE invasion, since they constitute the likeliest explanation for the occurrence of the initial copy of the TE family, and for the persistence of TEs in spite of their unstable dynamics when intra-genomic selection is introduced in the models.

For computational reasons, population size remained limited to 10,000 in most runs, and we could not simulate the evolution of realistic prokaryotic populations, which size often reaches 10^9 or beyond. We have performed one study in which we let the population size vary between 10^2 and 10^7, and found that the impact of genetic drift is small with population sizes >1000, and is not likely to alter significantly the conclusions of this study, at least with the range of parameters considered. See the Supplementary Information for details. According to previous studies, genetic drift in small populations tends to (i) increase the number of copies (by limiting the efficiency of natural selection against deleterious insertions), and (ii) increase the risk of TE loss or population extinction (Edwards and Brookfield, 2003; Le Rouzic et al., 2007). In contrast, in extremely large populations, TE dynamics are expected to be smoother and more deterministic.

One of the most challenging aspect of TE modeling is the way to introduce the impact of a changing environment in the model. Here, we considered that environmental stochasticity corresponds to a change in the fitness function: when the environment changes, the population is no longer close to the phenotypic optimum, and thus it has to accumulate genetic changes to improve its fitness. In this regard, our model is comparable to the mutator system (Giraud et al., 2001; Taddei et al., 1997). Mutators are clones characterized

by high mutation rates, several orders of magnitude above the base mutation rate of the species. Theoretical models suggest that a long-term coexistence of mutators and non-mutators is possible when the environment changes regularly (Gillespie, 1981; Tanaka et al., 2003; Travis and Travis, 2002), and their dynamical properties was confirmed experimentally (Giraud et al., 2001). Our model thus confirms that TEs could play the role of mutator-like factors (as observed empirically by Fehér et al., 2012), by increasing the mutation rate in a flexible way when the environment changes. Yet, their capacity to amplify exponentially can also lead to lineage extinction (Rankin et al., 2010; Vinogradov, 2003), making TEs efficient, but dangerous, evolutionary helpers.

Here, we did not consider any direct effect of stress on transposable elements or on mutation rates: in our model, the transposition rate increases solely as a consequence of the accumulation of active copies. Some empirical results suggest that TE mobility might also be directly induced by stress (Capy et al., 2000; Grandbastien et al., 2005; Ogasawara et al., 2009), opening the way towards models considering epigenetic regulation of transposable elements. Therefore, it cannot be excluded that stress-induced transposition might be adaptive if environmental change generates physiological stress (e.g. by threatening the survival of the population). This setting could be similar to the 'SOS' system in bacteria (Janion, 2008; Radman, 1974), involving a stress-induced epigenetic increase in the mutation rate.

3.2. Impact of TEs on genome evolution

Transposable elements are generally considered as universal, and they may represent most of the genomic DNA, especially in multicellular eukaryotes: 45% in human (Lander et al., 2001), and up to 85% in maize (Schnable et al., 2009). In other eukaryotic phyla, TEs may be less overwhelming, as they constitute around 2% of the genome of *Caenorhabditis elegans* and 3% of the yeast *Saccharomyces cerevisiae* Kidwell and Lisch (2000). Even prokaryotes, with their tiny optimized genomes, are not devoid of TEs, called 'insertion sequences' (IS) (Chandler and Mahillon, 2002).

Although population genetic models generally focus on sexual, random-mating species, most lineages of living organisms harbor asexual reproduction regimes, with only rare and sporadic gene transfers. Members of two out of three kingdoms of life, Eubacteria and Archaea, reproduce clonally. Eukarya are featured by a higher diversity of reproduction regimes, including perfect asexuality, parthenogenesis, self-fertilization, and sexual mating. Asexuality can be found in multicellular eukaryotes, including fungi, plants, and even some animals.

Our model considers strictly asexual organisms and could correspond to any clonal prokaryotic or eukaryotic species. Even in asexuals, genetic transfer events might occur, but theoretical models predict that exchange rates need to be very large for the population to behave as sexual species in terms of TE content (Condit et al., 1988), which excludes the vast majority of asexuals. Mobile DNA content in genomes differs greatly between asexual clades. Eubacteria are generally thought to have a very small number of TEs, most of them being active and recent (Wagner, 2006). Nevertheless, the situation is not homogeneous, and the genome of some strains harbors up to 20% of TE-derived sequences (Newton and Bordenstein, 2011). Archaea do not appear as fundamentally different, although they might contain more copies in average, including non-autonomous insertions (Filée et al., 2007). In contrast, the genome of eukaryotes is much larger and contain many more TEs. Some asexual animals (such as bdelloid rotifers) tend to have fewer TE copies than sexual relatives (Arkhipova and Meselson, 2000), but the pattern is less clear for plants and fungi (Dufresne et al., 2011; Lockton and Gaut, 2010).

Even if plant, animal, and prokaryotic TEs are not exactly identical, large differences in the TE content across organisms do not necessarily reflect different TE properties. Indeed, ecological or environmental factors can also interact with TE dynamics and condition their evolution. It is suspected that the population size could explain some of the differences in genome size and TE content: the efficiency of natural selection at eliminating slightly deleterious insertions being higher at large population sizes, the accumulation of TEs is much faster in low-population size species (such as multicellular eukaryotes) than in very large population-size prokaryotes (Lynch, 2007; Lynch and Conery, 2003, but see Daubin and Moran, 2004; Charlesworth and Barton, 2004; Whitney and Garland, 2010). Our results suggest that TE accumulation is also more likely in asexual populations subject to frequent environmental change than in populations living in constant environment, with little evolutionary challenge. This hypothesis is supported by the observation that the TE genomic content in bacteria might be influenced by environmental factors (Newton and Bordenstein, 2011).

3.3. Conclusions

In this paper, we developed a model of TE evolution in asexual organisms that is sufficiently realistic for analysis of real-world phenomena. This model allows the evolution of TE copies and implements an explicit effect of TE mobility on phenotypic traits which, in conjunction to the environment, determines individual fitness. These simulations evidence that, contrary to what is generally assumed, TE dynamics in asexuals can be extremely rich and complex, featuring losses, re-invasions, bursts of non-autonomous copies, and lineage extinctions. These results show that environment remains a major factor conditioning the genomic content of mobile DNA, through the carrying capacity of the habitat, the frequency at which new evolutionary challenges occur, and the size of the corresponding evolutionary steps. The interplay between intragenomic competition between TE copies and natural selection at the individual level illustrate the rich and complex coevolutionary nature of the TE-host relationship.

The most obvious direction of further research would be to extend the model to sexual organisms. This, however is non-trivial, as it would require the introduction of a genomic model (which we have avoided so far—as the purely phenotypic model is enough for asexuals). However, with sexuals we would need to account for insertion site polymorphism, various modes of ploidy, the possibility of TE-induced mutations being dominant or recessive, etc. Another direction would be to introduce horizontal transfer into the model—we present some very preliminary information on the behavior of the model with horizontal transfer enabled in the Supplementary Information. A different direction is to add spatial modeling, which would allow us to study whether transposons can assist organisms in colonization of new niches.

3.4. Supplementary Information

The Supplementary Information for this article is attached to the article and hosted by the journal's website (see http://dx.doi.org/10.1016/j.tpb.2013.07.004). It contains, among others, a study of the impact of horizontal transfers on the behavior of the model, a study of impact of different proliferation dynamics (Michaelis–Menten vs. mass action), and a study of the impact of population size on the behavior of the model. In addition to that, we invite those interested to have a look at the webpage: http://bioputer.mimuw.edu.pl/transp. It contains some additional informations, several sets of additional simulations, as well as a browser software for reviewing them in a structured way.

Acknowledgments

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