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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.

1. Introduction

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. The mechanisms underlying the evolution of the capacity to evolve, 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