CellPress

Opinion

Adaptation to Global Change: A Transposable Element–Epigenetics Perspective

Olivier Rey,[1,2,*] Etienne Danchin,[3,4] Marie Mirouze,[5] Céline Loot,[6,7] and Simon Blanchet[1,3,*]

Understanding how organisms cope with global change is a major scientific challenge. The molecular pathways underlying rapid adaptive phenotypic responses to global change remain poorly understood. Here, we highlight the relevance of two environment-sensitive molecular elements: transposable elements (TEs) and epigenetic components (ECs). We first outline the sensitivity of these elements to global change stressors and review how they interact with each other. We then propose an integrative molecular engine coupling TEs and ECs and allowing organisms to fine-tune phenotypes in a real-time fashion, adjust the production of phenotypic and genetic variation, and produce heritable phenotypes with different levels of transmission fidelity. We finally discuss the implications of this molecular engine in the context of global change.

A Molecular View of Responses to Global Change

Understanding the molecular mechanisms underpinning phenotypic responses of organisms to stress is central to evolutionary biology [1,2]. In the last decades, major advances have been made in this field, notably by highlighting several environment-sensitive molecular elements potentially guiding and accelerating phenotypic and genetic responses to stress [3]. These elements include ECs and TEs.

Epigenetic components (ECs) constitute a molecular network (Box 1) that can adjust phenotypes instantaneously (i.e., during development) and/or generate new phenotypes – sometimes transmitted across generations – without modifying the DNA sequence [4]. They strongly connect the surrounding environment with the genome and the phenotype, hence playing a central role in organisms' responses to stress [5,6]. Transposable elements (TEs) are stretches of DNA sequences that can move and amplify their copy number within a host genome [7]. Their activity can be triggered by environmental cues, accelerate mutation rates, and rewire regulatory networks (Box 2) [8,9]. As first claimed by Barbara McClintock [10], TEs constitute a significant adaptive response of the genome to (unanticipated) environmental challenges. Interestingly, TEs and ECs are intimately linked, potentially amplifying their actions on phenotypes and genotypes [11–14].

While TEs and ECs are increasingly acknowledged as main actors of organisms' phenotypic responses to various stressors [5,6,15,16], their combined actions in promoting such responses have rarely been considered explicitly in the context of **global change** (see Glossary) (but see [12,13]). Although apparently slow at the human scale, global change

Trends

A major recent observation is that populations can rapidly and lastingly adapt to global change.

Understanding the molecular pathways underpinning rapid phenotypic responses to global change is central to evolutionary and conservation biology.

Epigenetic components (ECs) and transposable elements (TEs) are environment-sensitive molecular mechanisms enabling organisms to rapidly cope with environmental stressors in the short and long term.

ECs and TEs strongly interact with each other, hence constituting an environment-sensitive molecular engine rapidly producing new phenotypes and genotypes in response to stress.

This TE–EC engine represents an overlooked molecular engine of rapid adaptation to global change.

[1]CNRS, UPS, Station d'Écologie Théorique et Expérimentale, UMR 5321, 09200 Moulis, France
[2]Department of Biosciences, College of Science, Swansea University, Swansea SA2 8PP, UK
[3]CNRS, UPS, ENFA, Évolution & Diversité Biologique (EDB) UMR 5174, 118 Route de Narbonne, 31062 Toulouse, Cedex 9, France
[4]Université Paul Sabatier, Évolution & Diversité Biologique (EDB), 31062 Toulouse, Cedex 9, France
[5]Institut de Recherche pour le Développement, UMR232 DIADE

 http://dx.doi.org/10.1016/j.tree.2016.03.013

is fast and drastic at the evolutionary scale, affecting most living species and initiating the ongoing sixth mass extinction [17]. Recent observations, however, showed that adaptive phenotypic responses of populations to global change can be extremely rapid [2,18]. This recurrent observation was surprising because the pace of evolutionary responses is predicted to be not rapid enough to cope with the current rate of environmental change [19]. This has renewed interest in the evolutionary role of **phenotypic plasticity** [20] and promoted the idea that evolution by natural selection regularly unfolds over (short) ecological timescales (i.e., **microevolution**) [18,21]. More recently, fundamental questions were raised concerning the molecular mechanisms allowing organisms to react so rapidly (and adaptively) to these drastic environmental changes [22].

Here we argue that TEs and ECs might jointly constitute a powerful molecular engine triggering rapid adaptive phenotypic responses to global change. We first review evidence that TEs and ECs are sensitive to environmental stressors related to global change and that these activities can promote rapid phenotypic and genetic changes in organisms. We then describe how TEs and ECs mechanistically interact with each other to form a complex molecular network. We build on these findings to propose that TEs and ECs can be integrated into a single mechanistic engine potentially permitting organisms to rapidly and lastingly cope with global change. We finally suggest research avenues to incorporate this engine into the reasoning of evolutionary ecologists concerned with global change.

Diversité Adaptation et Développement des Plantes, Laboratoire Génome et Développement des Plantes, 58 avenue Paul Alduy, 66860 Perpignan, France
[6]Institut Pasteur, Unité de Plasticité du Génome Bactérien, Paris, France
[7]CNRS UMR3525, Paris, France

*Correspondence:
olivier.rey.1@gmail.com (O. Rey) and
simon.blanchet@sete.cnrs.fr
(S. Blanchet).

Box 1. The Most Common ECs that Promote Phenotypic and Genetic Variations (Figure I)

DNA Methylation

DNA methylation, the addition of a methyl radical to a DNA nucleotide, modifies the accessibility of DNA to binding proteins (e.g., transcription factors) hence modifying (most often inhibiting) gene expression [71]. When occurring in the germline, some patterns of DNA methylation, usually thought to be reset during meiosis, nonetheless persist across generations leading to heritable phenotypic changes [41]. Although less acknowledged, DNA methylation is also mutagenic as it favors deamination leading to C-to-T transitions [58] and can locally affect meiotic recombination rates [72]. Thus, as for TEs, DNA methylation also has the potential to lastingly affect the genome architecture.

Histone Modifications

Histones are central molecules in chromatin formation [73]. Post-translational modifications of histone tails (about 100 types are identified) can alter the affinity of histone complexes for DNA, thereby changing the spatial configuration of chromatin and affecting the accessibility of DNA sequences to transcription enzymes, ultimately affecting gene expression [73]. Some of these histone tail modifications are heritable over several generations and are associated with changes in key life-history traits [63].

Non-coding RNAs

Recent advances in transcriptomics revealed an amazing diversity of non-protein-coding RNAs (i.e., ncRNAs) participating in the transcriptional and post-transcriptional control of gene expression [74]. As such, many ncRNAs are integral parts of the epigenetic regulatory network [74]. Some functional ncRNAs are transmitted across generations through the nourishing tissues of parental gametes, providing another source of nongenetic inheritance [75].

Intricate Epigenetic Cross-talk

DNA methylation and histone tail modifications can interact through their respective enzymatic machinery, hence reinforcing their phenotypic influence [76,77]. DNA methylation can dictate histone tail modifications [76] by guiding the reproduction of histone-based chromatin spatial conformation after DNA replication. Conversely, the establishment of DNA methylation during early development can be mediated through modifications at histone tails [76]. Moreover, some ncRNAs partly control *de novo* DNA methylation and histone tail modifications [74]. In particular, they contribute to the reestablishment of epigenetic patterns after meiosis and are thus likely to be involved in the fidelity of some epigenetic patterns across generations [41].

The synergy between these mechanisms constitutes a self-reinforcing and self-perpetuating cycle of ECs leading to long-term transcriptional repression [74,76,77]. As a result, epigenetic regulations not only can modulate gene expression but also ensure the fidelity of gene expression states over generations [76].