*Heredity* **85** (2000) 101–106 *Received 21 March 2000, accepted 25 May 2000*

Short Review

# Stress and transposable elements: co-evolution or useful parasites?

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The activity of transposable elements can be induced by environmental and population factors and in particular by stresses in various organisms. A consequence of the increase in transposable element mobility is the creation of new genetic variability that can be useful in the face of stressful conditions. In this review, results supporting this hypothesis are presented and discussed. The main question is how stress induces the activity of transposable elements. We discuss hypotheses based upon the existence of promoters or fixation sites of transcription activators in the untranslated regions of transposable elements, similar to those found in regulatory regions of host defence genes.

**Keywords:** adaptation, evolution, stress, transposable elements.

## Interaction of genome and environment

In the natural world, individuals, populations and species all have to cope with environmental change. Individual organisms and their cells have to adapt physiologically through responses that are immediate and reversible. At the population and species levels, selection may lead to genetic changes and to the evolution of the inherited characteristics of an individual organism. In such organisms this long-term response is irreversible.

During the last two decades several authors have reported that stress increases the genetic variability of many quantitative traits in a population, see for instance Imasheva *et al*., (1998), including life history and morphology (Hoffmann & Parsons, 1997). This genetic variability may have various origins. For example, different genes may be expressed in normal as opposed to stressful environments. It has also been shown recently that genetic variability can be hidden by buffering proteins such as *Hsp90* (Rutherford & Lindquist, 1998). This variability can be revealed by stress and then maintained when the protein function is restored. Finally, mutator mechanisms can be induced by stress. These are the origin of the genetic variability that allows selection to take place in response to environmental changes. At least two mechanisms are frequently described: those involving the SOS response (the activation of mutagenic activity) or the MRS response (inhibition of an antimutagenic system, the mismatch repair system) (Taddei *et al*., 1997) and those involving transposable elements (TEs) (Capy *et al*., 1997). Here we review and discuss findings of the impact of TEs on the host genome under stressful conditions and also summarize the various models put forward to account for these findings.

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## What is stress?

Before we discuss the relationship between TEs and their host genomes in a stressful environment, it is important to define stress. According to Koehn & Bayne (1989) stress reduces the fitness of an organism. Hoffmann & Parsons (1997) have proposed a definition which includes 'any environmental change that drastically reduces the fitness of an organism'. There are also several more precise definitions of stress, discussed by Bijlsma & Loeschcke (1997), which range over various biological levels from molecules to populations according to the intensity of the environmental changes and the organism or population involved. In addition, these authors identify two classes of stress defined according to the response of the organism: those evoking a physiological response (the individual context), and those evoking a phenotypic and/or a genetic response (the evolutionary context). In this review we will consider this last class of stress. Moreover, the genetic consequences of the relationships between transposable elements and stresses will be considered at individual and population levels.

## Transposable elements and genomes

In the late 1970s transposable elements were described as parasitic sequences because they use the host machinery for their own amplification (Doolittle & Sapienza, 1980; Orgel & Crick, 1980). Since the beginning of the 1980s detailed analysis has been carried out on their polymorphism (DNA and protein), copy numbers, chromosomal location, activation, regulation and transposition rates in natural and laboratory populations and between closely related species. In some cases they have been domesticated by the host genomes (Miller *et al*., 1997;

Nouaud *et al*., 1999). Several other findings supporting this view have also been published (see the special issues of *Genetica* on transposable elements in 1992, 1994, 1997 and 2000).

TEs are a major component of all genomes and represent between 3 to 50% of the content of the genome, depending on the species (Capy *et al*., 1997). While TE interactions with the host genome remain poorly understood there are several examples of their impact on host functioning, structure and evolution. For example, *Tn*, bacterial composite elements (elements flanked by Insertion Sequences), provide several examples in which a gene involved in antibiotic resistance can be horizontally transferred, see for instance Berg & Howe (1989) and more recently, Hall (1997) and Recchia & Hall (1997). In *Drosophila melanogaster*, *LINE*-like elements such as *TART* and *Het-A* are used as a 'cap' at the extremities of the chromosomes to prevent their degradation (Pardue *et al*., 1997). Also in this species, the *hobo* element is involved in chromosomal inversions (Lim, 1988; Lyttle & Haymer, 1992; Lim & Simmons, 1994; Ladeveze *et al*., 1998). Gene regulation under the partial or complete control of retrotransposon LTRs (long-terminal repeats) or solo LTRs left by retrotransposons are another illustration of TE effects on host genome evolution (White *et al*., 1994; Britten, 1996; McDonald *et al*., 1997). This is also seen in the human *α-amylase* gene (Ting *et al*., 1992). Similarly, in the *Saccharomyces cerevisiae* genome, 331 *Ty* insertions (85% of which are solo LTRs) have been detected (Kim *et al*., 1998). These sequences are frequently inserted in tRNA genes or other transcribed genes (Hani & Feldmann, 1998). Therefore, they may have an impact on the expression profile of these genes. Thus, many of the interactions between TEs and their host genomes can be seen as a domestication of the former by the latter, or as a coevolution between the two entities.

## Stresses and transposable elements

McClintock (1984) suggested that transposable element activity could be a response to challenges to the genome. Two approaches can be used to test this hypothesis. The first is the application of stresses to genetically controlled organisms. This is a direct investigation in which genetic modifications of the organism can be followed in terms of variability, structure and function. The second approach is the analysis of natural populations of the same species living in different conditions. This indirect investigation may also reveal the effect of environmental factors.

In adaptive mutagenesis in bacteria, discussed by Hall (1998), the *IS* elements may play an important role since these elements can transpose in 'response to external environmental signals that are related to the fitness function at the host level' (Hall, 2000). In such experiments, stresses correspond to deficient substrates. In *E. coli*, silent operons can be activated by *IS* insertions and then contribute to the fitness of the cell (Hall, 2000). For instance, *β*-glucoside operons are silent in wildtype strains and could be activated by *IS* elements to use *β*-glucoside sugar as sources of carbon and energy.

Wessler (1996) and Grandbastien (1998) have reviewed a number of elements in plants (mainly retrotransposons) that are turned on by stresses. For example, it was clearly shown that transcription of the *Tnt1* element of *Nicotiana tabacum* can be induced following several biotic and abiotic stresses (Grandbastien *et al*., 1997).

In *Drosophila simulans*, the copy number of the *412* element increases with latitude following the minimum temperature (Viera & Biémont, 1996). This phenomenon has been observed on various continents and on both sides of the equator. Independently, in the same species, Giraud & Capy (1996) have described a latitudinal cline between tropical Africa and Europe for the somatic activity of the *mariner* element, again suggesting an effect of factor(s) closely related to temperature. This is in agreement with laboratory results showing an effect of the developmental temperature on both somatic (Chakrani *et al*., 1993) and germline (Garza *et al*., 1991) excision rates.

The temperature effect is one of the most extensively tested stresses. While this factor is suspected of affecting several transposable elements, we must be cautious in generalising results obtained from one element in one population. In *Drosophila*, some findings seem to suggest the mobilisation of retrotransposon elements after heat shock (Ratner *et al*., 1992; Vasilyeva *et al*., 1999), while others show no effect of similar stresses (Arnault & Dufournel, 1994; Arnault *et al*., 1997). In addition, several transposable elements can be mobilized by the passage to cell culture (Di Franco *et al*., 1992), which may serve as a source of stress.

## What these observations tell us

The effect of stress on the behaviour of transposable elements prompts several comments. Firstly, the induction of TE activity has been demonstrated for only a few elements. Different populations and strains do not respond in the same way, as illustrated by the response of *copia* element transcription to temperature in *D. melanogaster* (Arnault & Dufournel, 1994). This may reflect not only the sensitivity of population/strain genomes to a given stress, but also the effect of the host genome on TE activity.

Secondly, TEs on which stresses may have some effect are generally of class I (elements moving by an RNA intermediate and using a reverse transcriptase). Are the class II elements (moving by a DNA intermediate) therefore insensitive to environmental stresses? Several results suggest not. For example, analysis of *mariner* elements in natural populations of *D. simulans* reveals the existence of a latitudinal cline in somatic activity (Giraud & Capy, 1996). This is also seen in the *P* and *hobo* elements in *D. melanogaster*, for which the hybrid dysgenesis syndrome is observed at 29°C and 25°C respectively (Kidwell, 1977; Blackman *et al*., 1987). In *Antirrhinum majus*, the excision rates of copies of the *Tam3* element inserted in the *pal*$^{rec}$-2 and *niv*$^{rec}$-98 alleles are higher at 15°C than at 25°C (Coen *et al*., 1989). Similar effects have also been reported for the *Tn3* element of *E. coli* (Kretschmer & Cohen, 1979). Finally, as previously mentioned, *IS* elements of bacteria are involved in the response to stress (Hall, 1998).

Thirdly, analyses of TE dynamics after stress remain difficult to interpret (Jouan-Dufournel *et al*., 1996). Marked elements with a reporter gene for detecting the transcriptional induction of the element (for Class I and II elements), or reporter genes with a TE insertion to detect excision events

(for Class II elements) can also be used. In these cases somatic events can easily be detected, see for instance Capy *et al*., (1990) and Grandbastien, (1998). Whether this somatic induction has effects on the next generation, both on somatic and germ lines, remains uncertain. To find out, the dynamics of TEs within a species will have to be considered. For several elements, at least in *Drosophila*, it is assumed that a product of TE activity in females can be transferred to the next generation through the cytoplasm of the eggs. This has been suggested for several Class II elements following the description of maternal effects for the *hobo* (Ho *et al*., 1993), *P* (Kidwell, 1981), *I* (Bucheton, 1979) and *mariner* (Bryan & Hartl, 1988) elements. For the last of these, analyses of two successive generations have shown higher activity in the offspring of females carrying active elements (Bryan & Hartl, 1988). In *A. majus*, *Tam1* and *Tam2* elements also show non-Mendelian inheritance, suggesting cytoplasmic transmission (Coen *et al*., 1989). Therefore, a relationship may exist between the somatic activity of an element in a given generation and its germline activity in the following generations.

It is also important to consider how the stresses are applied. In plants, specifically for the *Tnt1* element, the stresses were applied locally (one part of the plant like a sector of a leaf) and led to local responses. Moreover, in most cases the stresses led to cell death in the affected tissues. In *Drosophila*, stresses were generally applied to the entire organism and led to a general response. In such cases the existence of a general system called VAMOS, similar to the SOS response of bacteria, has been suggested (Brégliano *et al*., 1995; Laurençon & Brégliano, 1995; Laurençon *et al*., 1997). It has been shown that a repair-recombination system might exist in *D. melanogaster*, and that its efficiency could be modulated by endogenous and environmental factors. Moreover, the reactivity level of *R* strains of the *I-R* system seems to be related to the DNA repair system. Therefore, a general perturbation may lead to modifications of transposable element regulation.

Stressful conditions can be applied for short or long periods, for the whole life of an organism or even over several generations. This last situation is encountered when populations face long-lasting environmental change. So, the creation of new genetic variability induced by the activity of transposable elements may favour a more rapid adaptation.

Finally, there is the question of how stress can induce the activity of an element. It is possible that in most cases these effects are not direct. Indeed, when faced by stress, an organism may have to induce one or several responses at the physiological and/or genetic level. In the latter case, defence genes may be activated by transcriptional activators. The correlated response of the transposable elements could be due to the presence of fixation sites of transcription activators in

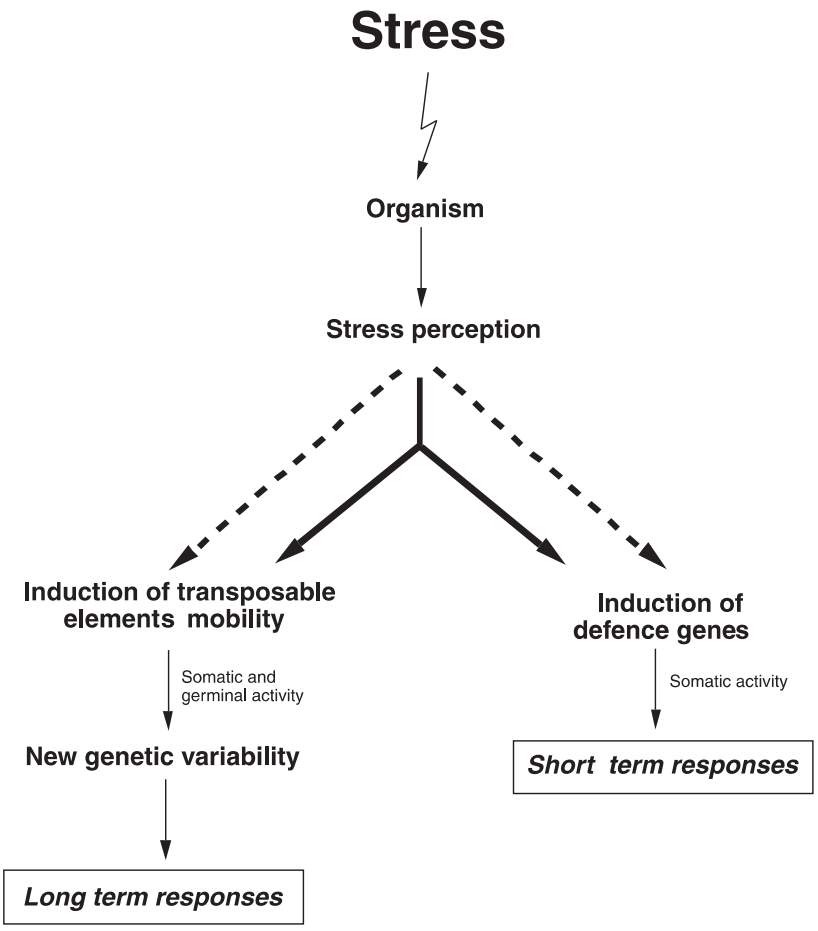


**Fig. 1** Induction of transposable element mobility after stress. The scheme is a working hypothesis. Independent (dashed lines) and common (solid lines) effects on host defence genes and transposable elements are shown.

their regulatory regions. This phenomenon is suspected for the *Tnt1* element in which the U3 region exhibits short sequences similar to those found in the regulatory motifs of the defence genes (Grandbastien *et al*., 1997). In *Drosophila*, detailed analyses of the *copia* and *mariner* regulatory sequences show the existence of motifs very similar to those of *hsp* promoters (Strand & McDonald, 1985; Chakrani *et al*., 1993; McDonald *et al*., 1997). This suggests that after stress, a proteolytic cascade leading to the release of transcription activators require for the induction of the host defence genes, could also induce the activity of some TEs (Fig. 1). Such phenomena at the individual level may also have some implications for the population level. Indeed, this leads to an increase in the genetic variability upon which selection can draw, so helping the population to confront stress (Fig. 1).

These observations raise the question of the origin of these fixation sites or promoters in transposable elements. Extensive comparisons of TE sequences during the past decade suggest that they are like construction sets or assemblages of different modules, each with a different history. This is supported by analysis of the variability of the structure of retrotransposons. For example, the similarities observed between the reverse transcriptase of retrotransposons and retroviruses, and also with those of retrons and group II introns, suggest that this domain comes from the same ancestral sequence (McClure, 1993; Xiong & Eickbush, 1990). Again, the similarities of the class I element integrases with transposases of class II elements also suggest evolutionary relationships (Capy *et al*., 1996). More recently, the description of elements in fungi, such as *Ant1* in *Aspergillus niger* (Glayser *et al*., 1995) and *hupfer* in *Beauvaria bassiana* (Maurer *et al*., 1997) in which foreign sequences were found, reinforce the idea that the acquisition of new functions is possible.

In this respect, the presence of fixation sites of host transcription factors or promoters could be an acquisition of TEs or an acquisition by the host defence genes of TE regions. This is suggested by the recently reported relationship between transposable elements and the immune repertoire of vertebrates (Agrawal *et al*., 1998). More specifically, the RAG1 and RAG2 (recombination-activating genes) can form a protein able to induce transposition. This similarity, initially mentioned by Dreyfus (1992), is strongly indicative of an ancient transposable element which has been tamed by the host genome (Plasterk, 1998).

## Conclusion

While the plasticity of the genome and TEs is now generally accepted, the interactions between these two entities are still not clearly understood. Stresses seem to induce the activity of some TEs but the underlying mechanisms remain hypothetical. One explanation can be sought in the similarity of the activation processes of host defence genes and transposable elements. In this hypothesis the question that remains to be solved is the origin of such similarities. Is it the result of a domestication of TEs by the host genome or of TEs acquiring host sequences? These two interpretations are not, of course, mutually exclusive.

Another explanation could be that stresses have induced a destabilization of the genome, leading to the malfunction of several genetic systems. In this case TE induction could be a secondary rather than a direct effect of the stress. For example, the SOS and MRS systems in bacteria and the putative VAMOS system in *Drosophila* could play a central role in the host response, and their induction may have several effects on the functioning of others systems, including TE regulation.

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