Hypothesis of the conjunct expression gene: can random mutation explain the phenotypic variability?

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It is argued how the factors that regulate the gene expression can increased the mutation rate for the genes involved in a behavior.

It is proposed how a probabilistic view of the random mutation can explain the evolution of the phenotypic variability.

The behavior of an organism, and the environment in where it development are factors important of the evolutive process.

If the environmental conditions can or not influence in the phenotype evolution, is a topic that has been discussed during the last ten years. The epigenome regulates the gene expression of all cells and indicates which specific genes must be transcribed. It is argued that the expression factors that act in specific genes of the somatic cells involved in a behavior also act in the partial transcription of the same genes in the most undifferentiated cells of the germ line. The functional role of the isochores as an epigenetic determinant regulator of the transcription and therefore of gene expression levels is discussed. It is proposed how a probabilistic view of the random mutation can integrate all the evidence pointing to a conjunct phenotype evolution with the environment. This work provides a new point of view on how the environment can influence in adaptation through natural selection considering the theory currently accepted.

KEYWORDS: evolution, gametogenesis, transcription, mutation, epigenome, isochores.

The conjunct expression gene

INTRODUCTION

In the last ten years a discussion has been developed on whether evolutionary theory needs to be rethought, and the cause has been the evidence suggesting that phenotypic variability cannot be due solely to the random mutation (Laland et al. 2014, 2015; Futuyma 2017). For that a phenotype can to expressed it is necessary for the cell to transcribed only a specific portion of your DNA. The epigenome is responsible for regulating the expression of the specific genes that the cell needs to perform its functions in the presence of the factors that regulate transcription (Jeltsch & Rots 2018; Carlberg & Molnár 2018).

Here is proposed that the expression of specific genes in a phenotype due to the high demand of gene resources as consequence of behavior of an organism, increases the probability of a random mutation for these genes in the most undifferentiated cells of the germ line during gametogenesis; and that the evolution of a phenotype associated with the environment in where it develops is the result of the probability of a random mutation linked to the partial transcription of the high demand genes occur during replication meiotic; considering for all the processes involved the probability that this mutation it is not repaired, adding to this the probability of that the mutation have consequences on the phenotype, the likelihood that the resulting phenotype will provide an advantage for survival or reproduction; plus the likelihood of that the natural selection will act in favor of the phenotype (Fig. 1B).

DISCUSSION

There is evidence that exposure to specific stimulus can regulate the gene expression of the somatic cells involved (Gunaratne et al. 2011). This is notorious in birds of the order Passeriformes in where of singing behavior is sexually dimorphic, implying that during embryogenesis, oogenesis and spermatogenesis, different genes involved in behavior are expressed (Luo et al. 2012). A similar epigenetic mechanism biased by sex has recently been documented in mammals (Warnefors et al. 2017). It has been proposed that the type of mechanism that regulates a behavior can influence the probability that phenotypic plasticity evolve (Rittschof & Hughes 2018). Here it is suggested that the factors that regulate the gene expression of somatic cells involved in a high frequency behavior can induce the partial transcription of the high demand genes in the most undifferentiated cells of the germ line.

In birds of the order Passeriformes, platelet phosphofructokinase is a tissue enzyme that should not be expressed during gametogenesis, and yet it does partial so in one or several steps of this process. It has been suggested that the gene of the enzyme tissue phosphofructokinase in birds of this order, has raised its GC content intragenic not only by mutation pressure by replication but also by mutation pressure associated to transcription (Khrustalev et al. 2014). The autonomous transcription of genes that should not be expressed during gametogenesis can be explained with the hypothesis here present (Fig. 1A).

Isochores are regions in the genome with rich in GC contend. This high of GC contend also may be intragenic (intrachores) (Khrustalev et al. 2014). There is evidence that associates the content of GC with the levels of expression (Barton et al. 2016). If the transcription rate is directly proportional to the genic demand, the exons located in isochores rich in GC they must have been in the past under strong mutation pressure, as in the case of the enzyme tissue phosphofructokinase that could have increased their intragenic GC contend, probably due to changes in the metabolic rate in birds of the order Passeriformes during the ultimate 91.4 - 47.1 million years (Khrustalev et al. 2014). Also, empirical evidence in studies on pre-implantation in human and mouse indicated that of levels of expression increased for those genes that were in regions of the DNA with high GC contend, from early to late stages; while than those that were in regions with low of GC contend, decreased their levels of expression (Barton et al. 2016). As well, it is had reported evidence that in the hexaploid genome of wheat, the levels of expression and the presence of isochores, indicate that these last act as an epigenetic determinant regulator of the transcription (Thomas et al. 2014). Is necessary realized more studies that contribute with evidence empirical on the evolutive function of the isochores.

Factors that regulate gene expression in the somatic cells can increase the mutation rate during replication mitotic by mutation pressure associate to transcription depending on the frequency of stimulus, but these mutations cannot be inherited; and this has been the barrier that prevents accepting the fact that the environment does can influence the evolution of the phenotypes (Laland et al. 2014, 2015; Futuyma 2017; Rittschof & Hughes 2018). Although not directly, gene expression of the somatic cells due a behavior, for example the construction of the niche (Saltz & Nuzhdin 2014; Gilbert et al. 2015), can increase the probability

that of a random mutation on spermatogonia and oogonia (that are undifferentiated cells and that give rise to the gametes that does transmitted the genetic information to the next generation) due to presence of the factors that regulate the transcription of the genes involved in the behavior, raising of this way the mutation rate.

A statistical correlation of the genic demand, the transcription rate of specific genes involved in a behavior in the somatic cells; and the transcription rate of the same genes in the most undifferentiated cells of the germ line, would be evidence of the conjunct expression of gene resources. It should be clarified that the function is independent of the structure, and it cannot be affirmed that a phenotype evolved to perform a specific function, due to that the mutation was a random event. A probabilistic view of the random mutation can integrate all the evidence pointing to a conjunct phenotype evolution with the environment and accordingly, the evolutionary theory does not need to be rethought.

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FIGURES

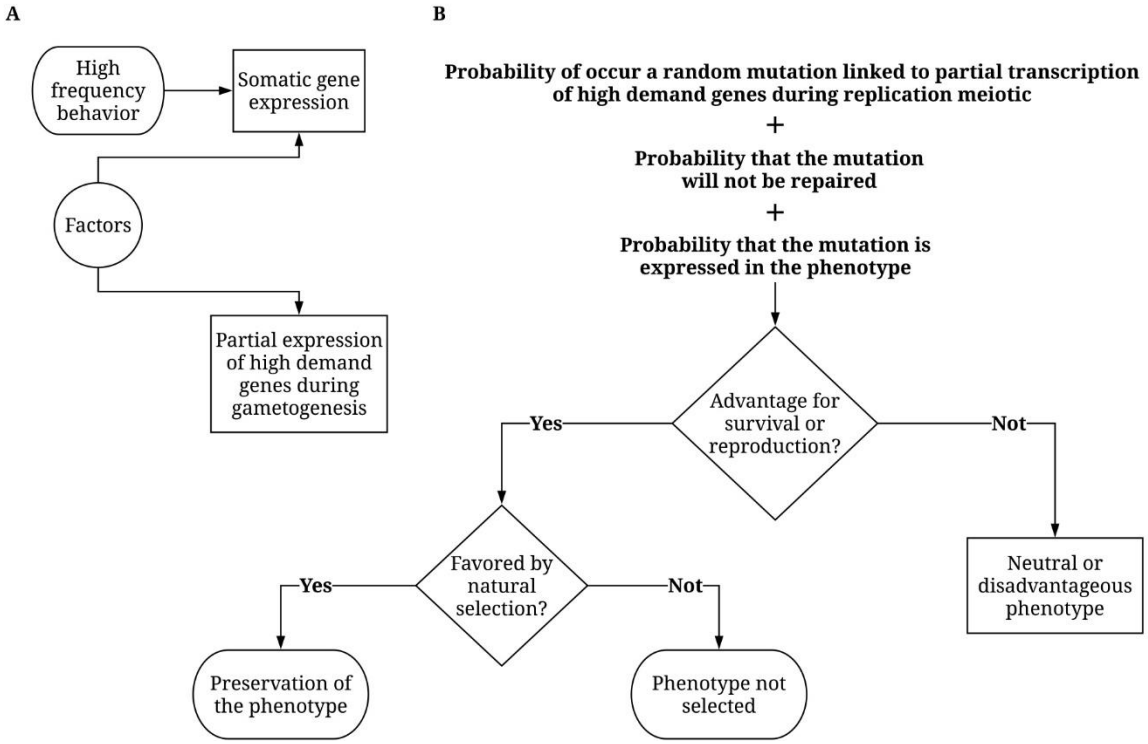

Fig. 1. — Hypothesis of the conjunct expression gene.

FIGURE CAPTIONS

Fig. 1. — Hypothesis of the conjunct expression gene. A) The factors that regulate gene expression in the somatic cells also act in the most undifferentiated cells of the germ line during gametogenesis. B) Probabilistic view of the random mutation that increases the mutation rate for the high demand genes. Natural selection is the mechanism responsible for the conservation of the phenotype.