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Studies on Junk-DNA: Where does Junk-DNA come from?

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Studies on Junk-DNA: Where does Junk-DNA come from?

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Abstract

Here we present a hypothesis that describes where junk DNA comes from in a pursuit of forming a well-established scientific theory. We suppose that junk DNA is added to our genomes by practical organisms via history (DNA fragments). The hypothesis is based on a number of facts such as oncoviruses and tumeor-causing bacteria. A set of experiments that aims to test our hypothesis is proposed as well. In addition to our main pursuit, we aspire to form a protocol that aims at discovering the "evolutional age" of various species.

Keywords

Junk-DNA; Genome; Ti Plasmid; *Agrobacterium tumefaciens* and Evolution.

Overview and background

In genetics, the term “Junk DNA” refers to regions of DNA that are non-coding (over 98% of human genome). Some of this non-coding DNA is used to produce non-coding RNA ingredients such as tRNA, rRNA and regulatory RNA. Other sequences of Junk DNA regulate gene expression (Regulatory genes). Other sequences are essential for chromosome structure such as centromeres and telomeres. However, most of these sequences remain with unknown specific importance. Such sequences have been a key focus in genetic research for many years, particularly in terms of evolution (Mandal 2023).

Objectives and Impact

1. Formation of a well-established scientific theory that explains where Junk-DNA comes from.
2. Finding out the mechanism(s) of how Junk-DNA be added to genomes.
3. Suggestion of a biological protocol that aims at discovering the "evolutional age" of various species on Earth.

Research Approach and Methodology

“Junk DNA are sequences inserted to host genome by practical organisms via history” is a hyporhesis suggested by the author based on a number of facts:

1. The quantitative relationship between the ratio of junk DNA to the whole genome and organism’s evolution (complexity): Non-coding sequences make up only a small fraction of DNA in prokaryotes. However, among eukaryotes, as their complexity increases, the proportion of junk DNA increases in their genomes (Tomkins 2013). The author explains this based on the fact that the more the complexity of a living organism is, the larger the number of practical organisms which attack it; therefore, the larger the percentage of junk DNA in its genome.
2. Tumor viruses (Oncoviruses): The viruses that cause tumors to a group of organisms including men by inserting a segment of their genome to host cell’s genome (Hunt 2016).
3. Tumor-causing bacteria: Some species of bacteria cause tumors to plants such as *Agrobacterium tumefaciens*, the causal agent of crown gall disease in over 140 species of eudicots. Symptoms are caused by the insertion of a small segment of DNA (Ti Plasmid) into the plant cell, which is incorporated at a semi-random location into plant genome (Zupan 2001).

Proposed experiments:

Experiment 1:

1. Ti Plasmid in *Agrobacterium tumefaciens* is cloned into the genome of zygotic embryos of eudicotic plants.
2. Seeds with Ti Plasmid are planted. It is predicted that symptoms of crown gall disease to be watched after growth.
3. Plants are kept and given special care to survive till producing seeds.
4. Seeds (F_2) are planted:

- 1st condition: Symptoms still observed: Result of the experiment is negative and the disease has been inherited.
- 2nd condition: Symptoms not observed: This means F_2 plants created some method(s) to stop gene expression of Ti Plasmid genes.

In the 2nd condition, the author suggests two mechanisms in which F_2 plants stops gene expression of foreign DNA:

- Sexual cells destroys most of foreign DNA using endonucleases in a recombinational repair proofreading mechanism.
- F_2 plants stops gene expression of Ti Plasmid genes through modifying or destroying regulatory genes and/or functional genes of foreign DNA.

In both conditions, foreign DNA (Ti Plasmid) is converted into "junk DNA" in the next generations. In this case, foreign sequences have become non-functional, non-coding sequences to stop the disease in the following generations.

Notes on experiment 1:

Notes:

1. The eudicotic plant used in this experiment should be chosen carefully; it should be able to survive despite infection by tumors till producing seeds.
2. It is possible that some F_2 plants grow with disease symptoms, so a large (suitable) number of plants should be planted. It is also possible that plants with no watched disease symptoms (in F_2) to be watched after more than two generations.

Experiment 2 (Performing this experiment depends on the success of experiment 1):

1. Ti Plasmid in *Agrobacterium tumefaciens* is marked with thymidine and cloned into the genome of zygotic embryos of eudicotic plants.
2. The genome of F_2 plants with no watched symptoms (in experiment 1) is examined to search for thymidine to discover how do F_2 plants stop the gene expression of foreign DNA:

- 1^{st} condition: Small fragments which contain thymidine be found: Restriction enzymes are used during meiotic division to destroy most of the harmful, foreign DNA.
- 2^{nd} condition: Most of the inserted DNA be found: Sexual cells have followed some techniques to stop regulatory genes and/or functional genes from doing their jobs.

Experiment 3:

Genome size is measured for two individuals of the same species:

1. Individual 1: Currently alive.
2. Individual 2: lived too long ago (fossil).

It is expected that – to author's hypothesis - genome size in "individual 1" to be larger than genome size in "individual 2". In addition, the difference in genome sizes between individuals 1 and 2 would refer to an approximate ratio of added nucleotides (junk DNA) to organism's genome via history. In this way, we can estimate the "evolutional age" of every organism on Earth, assuming that the genome of every organism did not contain any junk DNA in the beginning of its appearance taking evolution terms of the organism under study in mind.

Conflicts of interest

The authors have declared that no competing interests exist.

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