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Journal of Genetic Syndromes & Gene Therapy

Commentary

An Evolutionary Change Caused By Alu-Gene

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DESCRIPTION

Alu-Genes must have been a massive number of other insertion events that were not fixed in order to create the more than 1 million Alu elements fixed in the human genome today. Elks elements have thus been a significant contributor to genomic instability and evolution throughout primate evolution. One such event was an insertion-mediated deletion that only in humans inactivated the CMP-N-acetyl neuraminic acid hydroxylase gene. This resulted in altered protein glycosylation, which may have been a significant change in the evolution of humans from chimps. Alu-Alu recombination events have also been implicated in chromosomal evolution and possibly speciation. Airs AM-mediated recombination in the gulonolactone oxidase gene appears to have occurred after prosimians diverged from the other primates.

This enzyme is a critical late step in the synthesis of vitamin C, and its absence resulted in primates' inability to synthesize this vitamin, potentially leading to scurvy. Alu elements appear to have been involved in a number of recombination events that contributed to segmental duplications on human chromosomes. Through the instability of the segmental duplications, these segmental duplications have been linked to a variety of different syndromes. Thus, Alu elements appear to have contributed to an overall rearrangement of the genome and chromosome that has resulted in extra copies of genes, which may be advantageous for evolution but can have negative consequences for long-term genomic stability and function.

Gene regulation and stability

Alu elements have been proposed to cause changes in gene structure and regulation, in addition to the evolutionary changes associated with their role in genetic instability. Transcriptional regulatory elements have been mapped to Alu elements near gene promoters, and Alu elements have been demonstrated in several reporter systems to be capable of contributing transcription factor binding sites to stimulate gene expression, as well as insulator sequences to isolate genes from other nearby elements. Thus, Alu elements have most likely influenced gene expression by insertion near their promoters. It has also been proposed that the expression of Mu elements may contribute to the selective regulation of translation initiation.

Because viral infection, transformation chemotherapeutic DNA-damaging agents, and a variety of cellular stresses stimulate the expression of Alu elements, there is speculation that this may help regulate the translation process in those situations. Alu elements may constitute a significant portion of the intronic sequence in RNAs, as well as appearing in three non-coding regions. It was recently discovered that human cells have high levels of adenine-to-inosine RNA editing, with more than 90% of it occurring within Alu elements. This is most likely due to Alu elements in various orientations in the RNA's ability to form duplex structures, which are excellent substrates for the editing enzyme.

It is currently unknown whether this editing of Alu elements serves a purpose or merely competes with other RNA substrates. Alu elements may influence the processing and stability of numerous cellular transcripts *via* RNA editing, differential splicing, and other mechanisms.

Although our understanding of the mechanisms of Ala element amplification and their role in human disease has grown significantly, a number of critical issues remain unresolved. One of the current issues in identifying human disease mutations is that many polymerase chain reaction-based strategies are biassed against detecting large sequence insertions or deletions. As a result, we are unlikely to detect a significant proportion of Ala clement-induced damage in human disease.

As we learn more about the mechanism of Alu amplification, it will be critical to pay attention to which cell types are involved in the amplification: germ line, somatic, and tumour cells. Questions about how Alu elements interact with cellular gene products that may modulate or respond to their amplification are also important. This will help determine whether different people are more or less vulnerable to the effects of these elements. Similarly, with evidence that numerous environmental factors stimulate the expression of Alu elements. Finally, Alu elements appear to use the LI machinery for amplification more efficiently than LI does.

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Received: 13-May-2022, Manuscript No. JGSGT-22-17935; **Editor assigned:** 18-May-2022, PreQC No. JGSGT-22-17935 (PQ); **Reviewed:** 1-Jun-2022, QC No. JGSGT-22-17935; **Revised:** 6- Jun-2022, Manuscript No. JGSGT-22-17935 (R); **Published:** 14-Jun-2022, DOI: 10.35248/2157-7412.22.13.361.

Citation: Pandey J (2022) An Evolutionary Change Caused By Alu-Gene. 13:361.

To fully assess the roles, these elements play in human genetic instability, it will be necessary to delineate both the similarities and differences in the actual mechanism of amplification of these elements, as well as their respective impacts once inserted in the genie. Finally, we need to figure out if there is anything we can do to reduce the genetic instability caused by these elements.