OPEN ACCESS Freely available online

ISSN: 2329-6682
Gene Technology

Commentary

DNA Mutation Sequences and Algorithms in the Genes Linked to Inherited Disorders

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DESCRIPTION

Any living thing needs a healthy genome with stable genetic material. Nucleotide Excision Repair (NER), Base Excision Repair (BER), Mismatch Repair (MMR), Homologous Recombination Repair (HRR), and post-replication repair are a few of the repair pathways that cells have developed over time to ensure that genetic information is transmitted correctly to the next generation. *De novo* mutations, which can result in a variety of inherited illnesses, are mutations that develop in the parent's germ cells. Environmental factors can cause DNA errors to occur. Both transcription and replication processes result in a mutational strand asymmetry in human genes. Transcription Coupled Repair (TCR), a sub-pathway of NER, is started when RNA polymerase notices DNA damage on a transcribed DNA strand. The differing synthesis and proofreading processes used by the leading and lagging strands during replication are connected to strand asymmetry. These locations, known as hotspots, show a greater mutation frequency than would be anticipated by chance.

Every gene carries the instructions necessary to generate a protein, according to the central notion of gene expression. Multiple protein factors, referred to as transcription factors, bind to enhancer and promoter regions to start the expression of genes. By activating or inhibiting the transcription machinery, transcription factors control the expression of genes. A significant effort in addressing this difficulty is identifying regulatory elements, particularly the DNA regions where transcription factors bind. One of the most difficult challenges in molecular biology and computer science is finding patterns in DNA sequences. The issue can be expressed as follows in its most basic form unknown pattern that regularly appears from a series of sequences. A straightforward enumeration of all m-letter patterns that exist in the sequences yields the answer if a pattern precisely m letters long appears in every iteration. It is more complicated when working with DNA sequences because patterns contain nucleotide mutations, insertions, and deletions.

A DNA motif is a pattern of nucleic acids that has biological relevance, such as acting as DNA binding sites for a transcription factor or other regulatory protein. The pattern typically occurs numerous times inside a gene or across several genes and is known to be fairly brief. DNA patterns are frequently linked to structural patterns in proteins. Both strands of DNA can include motifs. In fact, transcription factors bind to double-stranded DNA directly. A motif could appear once, many times or not at all in a sequence. Palindromic motifs and spaced dyad motifs are two unique types of DNA motifs that are recognised in addition to the typical forms. A subsequence that exactly matches its own reverse complement is known as a palindromic motif. Two smaller conserved sites are connected by a spacer to form a spaced dyad motif. The transcription factors bind as a dimer, which causes the spacer to appear in the midst of the motif. Thus, the transcription factor is composed of two subunits with two different locations of interaction with the DNA sequence.

The aim of the motif discovery challenge is to identify overrepresented motifs as well as conserved motifs from orthologous sequences that are strong candidates for being transcription factor binding sites, given a set of DNA sequences (promoter region). There are numerous algorithms available for locating DNA motifs. Numerous coregulated genes from a single genome's regulatory area are taken into account by the majority of these algorithms in order to determine motifs. Gene coexpression is thought to result mostly through transcriptional coregulation. As coregulated genes are known to have some regulatory mechanism overlap, presumably at the transcriptional level, their promoter regions may have some shared motifs that serve as transcription factor binding sites. The promoter region of such a group of coexpressed genes can be searched for statistically overrepresented patterns as a practical method to identify these regulatory elements. Therefore, in this set of promoter sequences, these algorithms look for motifs that are overrepresented. However, it has been demonstrated that the majority of these motif searching algorithms function substantially worse in higher species than they do in yeast and other lower organisms. Recent algorithms for motif discovery are

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Received: 23-Nov-2022, Manuscript No. RDT-22-19234; **Editor assigned:** 28-Nov-2022, PreQC No. RDT-22-19234 (PQ); **Reviewed:** 13-Dec-2022, QC No. RDT-22-19234; **Revised:** 21-Dec-2022, Manuscript No. RDT-22-19234 (R); **Published:** 29-Dec-2022, DOI: 10.35248/2329-6682.22.11.206

Citation: Penn T (2022) DNA Mutation Sequences and Algorithms in the Genes Linked to Inherited Disorders. Gene Technol. 11:206.

use phylogenetic foot printing or cross-species genome comparison to get around this problem.

Humans identified periodically occurring 5-nucleotide sequences in these genes, which are both frequently and rarely linked to mutations. Second, we used sophisticated computational methods to examine the bending characteristics of two hotspots and two cold spots. Although the parameters describing trinucleotide bending with regard to the nucleosome and DNase I have been calculated, their use for our purpose is constrained since we concentrate on specific DNA deformation caused by a mismatch base pair and caused by the MutS protein. One sequence can respond differently in relation to various deformations, as demonstrated in a prior work that concentrated on DNA tracts. We specifically used free energy calculations utilising the adaptive biassing method improved by the multiple walker methodology and Molecular Dynamics (MD) simulations implemented in the Amber software package. We were able to determine the free energy shift required to bend a straight DNA duplex with a cold spot or hotspot in the direction of the bent geometry of DNA present in the MutS/DNA complex using our models.