CHANGES IN GENE EXPRESSION

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ABSTRACT
The process of converting deoxyribonucleic acid information into a functional gene product is called gene expression (GE). To synthesize ribonucleic acid (RNA) from DNA, the transcription process, which is part of the GE process, is used. As an organism matures or responds to changes in its environment, cells can adjust the kind and amount of GE produced. As a result, genetic engineering research can disclose knowledge regarding biological reactions at a certain moment. Discoveries have been made regarding the variations in gene expression due to changes in regulatory mechanisms. This attribute has been utilized in clinical studies to compare the GE of different groups of people to enhance the process of investigations and understand the prognosis of a disease.

KEYWORDS
Gene expression, Ribonucleic acid, Deoxy ribonucleic acid.

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Introduction

Gene expression (GE) is the process of converting deoxyribonucleic acid information into a functional gene GE process, is used to synthesize ribonucleic acid (RNA) from DNA. Cells can vary the type and amount of GE as an organism develops or responds to changes in its environment. As a result, GE research can reveal information about biological responses at a certain point in time. Over 40 years ago, King and Wilson (1975) demonstrated that modest changes in the regulatory mechanisms linked with changes in GE could explain substantial phenotypic differences between organisms. This feature has been used in clinical research to measure differences in GE between groups of people. These investigations have helped us better understand illness development,[2] as well as symptom severity disparities.

MECHANISM OF GENE EXPRESSION

Epigenetic Regulation

Changes in GE can be regulated by epigenetic control in response to the environment. DNA methylation, histone modifications, and non coding RNA expression can all affect GE epigenetic regulation.[3]

Methylation of DNA happens largely at the molecule's cytosine base, which is next to guanine (i.e., CpG site). DNA methyltransferase converts cytosine to 5-methylcytosine. Methylation of a CpG island in a gene's promoter region can suppress GE by preventing transcription factors from attaching to the methylated promoter site.[4] DNA is linked to histone proteins to create a nucleosome, which is another vehicle for epigenetic regulation of GE. In the nucleus of the cell, nucleosomes are arranged in a compact chromatin structure. Acetylation, phosphorylation, and methylation can all affect the amino acids in the histone protein. Histone changes can influence GE in two ways. For starters, histone changes can make

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DNA less compact, making it more accessible for transcription. Second, proteins can attach to the histone protein's changed amino acid and modify DNA transcription.[5] Finally, noncoding RNA expression can play a role in GE epigenetic control. Noncoding RNAs serve a crucial function in transcriptional and posttranscriptional regulation.

Post-transcriptional Processes

Noncoding RNA is converted into a functional gene product and mRNA is prepared for translation by posttranscriptional mechanisms. 50 capping of pre-mRNA, splicing out intron regions from RNA, alternative splicing of pre-mRNA, insertion of a poly(A) tail to the pre-mRNA, gene fusion transcript processing, and modulation of mRNA stability are all post transcriptional activities. Removal of intron sequences from RNA by splicing, alternative splicing, and gene fusion transcript processing events are some of the processes that can affect GE regulation.

CHANGES IN GENE EXPRESSION

1.Tissue Specificity

GE differs from one cell to the next and from one tissue to the next.[6] Perilipin1 (PLIN1), for example, is expressed in adipocytes but not fibroblasts, peripheral nerves, or chondrocytes.GE in embryonic stem cells is fundamentally different from GE in adult cells. Because these cells are actively proliferating, roughly 60% of the coding genes are translated into mRNA, and only a small percentage of the genes are cell-specific. Housekeeping genes are RNA

transcripts that are expressed nonspecifically at a consistent level in most tissues. Glyceraldehyde 3-phosphate dehydrogenase (GAPDH) is a housekeeping gene that is found in nearly all human tissues.[7] Housekeeping gene expression acts as a check for cell activity.

2.Host Age

The percentage of people who have GE fluctuates depending on their age. The authors of a meta-analysis [8] discovered that as humans grew older, 56 genes were consistently overexpressed and 17 genes were consistently underexpressed. DNA damage is most likely to blame for changes in GE over aging.[9] Changes in GE with age tend to be gradual. Researchers should keep track of the participants' ages in each GE study. When employing tissue banked samples, investigators should age match the specimens. Researchers should account for participants' age in statistical analyses if a study reveals variations in the expression of genes that are known to change their levels of expression as humans age.

3.Host Gender

Gender influences GE in whole blood. In one study that enrolled 41 healthy males and 36 healthy females, 46 genes (i.e., 35 in females and 11 in males) showed gender-associated differences in GE. In microarray experiments, researchers found significant differences in GE by gender for both autosomal and sex chromosome genes. For example, while investigators found no gender-associated differences in neutrophil counts in one study, they did find a small number of genes that were highly expressed in the neutrophils of females. Depending on the phenotype under investigation, if a study reveals differences in expression in genes whose expression is known to be influenced by gender, then researchers may need to control for the participants' gender in the statistical analyses.

4.Environment

Environmental conditions have an impact on GE. A core collection of 25 transcripts were differentially expressed in one research investigating alterations in GE as a result of exposure to traffic-related contaminants.[10] These 25 genes were found to be involved in cancer, heart disease, and chronic lung disease pathways. Depending on the phenotype under consideration, researchers may need to collect data on a variety of environmental factors (e.g., stress, air pollution, food) for disease susceptibility studies.[11]

5.Inherited Variation

Inherited (i.e. genetic) variables influence GE. A substantial amount of research has looked into how genetic variants affect GE and disease risk.[12] For example, researchers assessed changes in GE in adipose tissue and blood associated with obesity-related features in one major investigation of obesity attributes in a large population-based cohort.[13] While differences in GE in adipose tissue were shown to be strongly linked to obesity-related characteristics, the researchers found no such links in blood. In addition, the researchers looked into the genetic component of GE and discovered that there is a considerable heritable component to the observed GE variations in both tissue types. Changes in the expression of inflammatory and immune-response genes were found to be causally linked to obesity.

Conclusion

GE is a diverse, complex process with complex regulating processes. Changes in GE could be used to explain differences in certain phenotypes. For example, GE studies can be used to diagnose specific diseases,[14] assess differences in disease prognosis, assess the mechanisms that underpin differences in symptom severity,[15] aid in treatment decisions, and identify patients at increased risk for adverse events from specific treatments.[11]

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