

ABSTRACT

The definition of 11 GCNF coding exons on human chromosome 9 can be achieved by homology with the mouse gene, and all known cDNAs of human GCNNF are derived from mRNAs that are generated by splicing the fourth to the second exontium, despite the conservation of genomic sequences.

INTRODUCTION

The human GCNF gene was first identified in 1990 by the scientists from the University of California Berkeley (UBC), who in turn isolated the human gene from the mouse genome and showed that the mouse GCNF gene is the closest gene to the human gene GCNF. In the 1990s, the scientists of the University of Southern California (USC) and the University of California San Diego (UCSD) discovered that the human GCNF gene is the closest gene to the mouse gene GCNF and that human GCNF is the only human gene that can be expressed in the mouse.

The human GCNF gene was also found to be expressed in a mouse model of human breast cancer. The researchers found that the human GCNF gene is expressed GCNF is the first member of the sixth subfamily, and it has been assigned the name NR6A1 based on homology. Its transcriptional regulator functions as a functionally independent gene for various biological processes, including embryonic development, differentiation, homeostasis, estrogen/thyroid hormone (STAT), and vitamin D regulation, but its function as an orphan receptor remains unknown due to the presence of several known ligands. The isolation of a human cDNA that encoded hGCNF, similar to the mouse protein of about 98.7%, had similar regulation in mouse P19 cells and in the human embryonal carcinoma cell line NT2/D1, and two mRNAs of approximately 7.5 and 2.2 kb in human testis suggested that both functions were likely to have been fulfilled by the same human proteins (although it has been shown that humans may have found more complex ancestries within these different chromosomes than the clone as an experiment with four different sequence of By examining our understanding of the genomic structure of mammalian GCNF, we have discovered that alternative splicing generates at least three different isoforms of CGNF. We compare the exon/intron structure and molecular biology of mouse gene expression with that of human ortholog.

CONCLUSION

In conclusion, our investigation demonstrates that GCNF has a conserved structure, enables the confirmation and systematic review of splice variants, and could potentially enhance our understanding of human CGNF. The intron-exon boundaries of the human gene are consistent with the high level of amino-acid conservation between the mouse and human proteins, while alternative coding methods like cIF-26 enable the expression of several protein regulators.