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Neonatal diabetes mellitus and a review of a specific mechanism for mutation in the INS gene for human insulin on the genome using a computer model. Evidence is presented in support of further research and study of this variant. ROBERT GOSHEN, HARRIET PAPERNICK, Goshen Papernick Incorporated, Pittsburgh, PA — A variety of gene mutations are known to play a role in the development of diabetes mellitus in patients from the neonatal to the older adult. This study focuses on a DNA triplet involved in the translation of the preproinsulin precursor peptide (messenger) mRNA into the mature bioactive insulin protein. A successful translation of the mRNA creates a peptide chain with a length of 110 amino acids, but the failure of this triplet called the AUG start codon to properly initiate the translation could potentially yield a mutant chain that damages the pancreatic beta cells, leading to permanent neonatal diabetes disease. Our computer (MS C sharp) simulation model allows both novel and well reported genetic mutation patterns to be tested and evaluated. The results for the case of this AUG start codon are shown to merit further investigation by means of both in vitro processing and expanded collection and analysis of patient genetic information along with the data from medical records.

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