

人线粒体tRNA^{Leu}(UUR)基因A3243G点突变对其亮氨酰化活性的影响 Effects of A3243G Point Mutation on Aminoacylation of Human Mitochondrial tRNA^{Leu}(UUR)

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摘要 化学法合成人线粒体野生型与A3243G点突变型tRNA^{Leu}(UUR)基因,体外转录生成相应的tRNA^{Leu}(UUR),表达并纯化人线粒体亮氨酰tRNA合成酶(mtLeuRS),用mtLeuRS催化野生型与突变型tRNA^{Leu}(UUR)与亮氨酸结合,分别检测两种类型tRNA^{Leu}(UUR)的氨酰化动力学常数。结果表明,野生型tRNA^{Leu}(UUR)的Km/Kcat仅为突变型tRNA^{Leu}(UUR)的63.9%,A3243G点突变使tRNA^{Leu}(UUR)接受亮氨酸的能力明显下降,提示此为A3243G点突变致病机制之一。
Abstract: The wild-type and mutant-type human mitochondrial tRNA^{Leu}(UUR) genes were synthesized and transcribed in vitro with T7 RNA polymerase. The kinetic parameters of human mitochondrial leucyl-tRNA synthetase(mtLeuRS) were determined with wild-type and mutant-type human mitochondrial tRNA^{Leu}(UUR) respectively. The results show that the value of Km/Kcat of mtLeuRS for the mutant-type tRNA^{Leu}(UUR) is 63.9% as compared with the wild-type. Human mitochondrial tRNA^{Leu}(UUR) gene A3243G point mutant can remarkably reduce its aminoacylation activity, suggesting it would be one of the mechanisms that the mutation could produce such clinical phenotypes.

关键词 [人线粒体tRNA^{Leu}\(UUR\)](#) [A3243G点突变](#) [氨酰化](#) [体外转录](#) [人线粒体亮氨酰tRNA结合酶](#) **Key words** [human mitochondrial tRNA^{Leu}\(UUR\)](#) [A3243G point mutant](#) [aminoacylation](#) [transcription in vitro](#) [human mitochondrial leucyl-tRNA synthetase](#)

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