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PIT1 Gene Action in Mouse

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Commentary

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ABSTRACT

PIT1 gene lead to increased weaning weight, without the birth weight improvement. However, the distinction in PIT1 behavior observed between the two genetic groups denotes the need to test the effects of this polymorphism on different populations before using it in marker-assisted choice. It conjointly indicates the necessity for a much better understanding of the genetic mechanisms involved in the physiology of this pituitary transcription factor in the process of growth in cattle.

MAIN HEADING

In mouse, the first spontaneous Pit-1 mutant, named Snell dwarf mouse, was already defined in pituitary hormone deficiencies (CPHD) were shown to be caused by mutations in the human Pit-1 homolog. In our recent research the Genomic DNA isolated from Patients sample and the Exon 1, 2 and 3 were amplified by PCR. The amplified Exons 1, 2 and 3 were sequenced. From the Sequence the Multiple sclerosis is because of mutation in Exon 3. The mutants were originate in a large-scale screen for N-ethyl-N-nitrosourea-induced recessive mutations causing altered Growth hormone gene expression Juvenile and adult mutants display severe dwarfism, which appears even more dramatic than in mouse mutants and human patients. The analysis of the role of pit1 during zebrafish development turned out to be very helpful in further illuminating the mechanisms of pituitary patterning in fish. 1929. With its molecular cloning in 1990, Pit-1 was the first gene proven to be important for mammalian pituitary development. Solely 2 years later, many cases of human combined. Due mutation in Exon 3, leads to Prolactin, Growth Hormone and Thyroid Stimulating Hormone deficiency. Mutation at this partuclar exon may be due to several Factors [1-10].

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