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Computational analysis of single-nucleotide polymorphisms in proximal promoter region of rice (*Oryza sativa* L.)

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The term promoter is used to designate a region in the genome sequence upstream of a gene transcription start site (TSS). Most promoter elements regulating TSS are localized in the proximal promoter, is a region of several hundreds nucleotides around the TSS. If the SNP occurs within coding sequence, which may or may not alter amino acid sequence. Where as, SNPs occur within the proximal promoter region has more impact on gene expression. The publication of whole genome sequences for the japonica (Nipponbare) and indica (93-11) types of rice enables the determination of common SNPs occurring between them. In present study we used 31 non-redundant proximal promoter sequences in rice, from previous studies which are experimentally determined transcription start site for RNA polymerase II. When proximal promoters of japonica were compared with the corresponding proximal promoters of indica using BLAST alignment programme, a total of 69 SNPs were identified. Out of these 32 SNPs (46.3%) were transitions (A/G, T/C) and 37 SNPs (53.6 %) were transversions (A/C, A/T, G/C, G/T). Maximum frequency of SNPs was found in the region -50 to -150 (61.2 %). Minimum frequency found in region -151 to -201(9.6 %). It demonstrate that functional cis-regulatory polymorphisms segregate within sub species of rice and there is abundant SNP present in proximal promoter region. SNPs in proximal promoter may one of the causes of fictional variation. In addition, study indicates that SNPs are not evenly distributed. Selection pressure is always more for a region having more fictional impact and theoretically should have conserved, in contrast nonfunctional regions acting as reservoir of mutation.

Key words: Cis acting elements, Proximal promoter, SNP

INTRODUCTION

In most cases the genetic variation comprises single base changes in the DNA sequence, known as single nucleotide polymorphisms (SNPs), where this may occurs in coding DNA, or non coding regulatory sequences. The implications are readily apparent, if the changes occur in coding DNA in the form of modified amino acid sequence. Even where the polymorphism remains silent at the protein level, as in the case of synonymous mutations, the effect of the polymorphism can be assayed at the level of mRNA. This is based on the fact that when a polymorphism occurs in coding DNA it will be present in the transcribed mRNA, allowing the relative abundance of the two alleles in cells from an individual heterozygous for a given SNP to be assayed (Knight 2003). In contrast, assaying the functional effect of polymorphisms occurring in non-coding DNA is more problematic. Non-coding regions are interspersed throughout genome and most of them are junk. Promoter is non-coding regulatory sequence upstream of a gene transcription start site (TSS). Promoter elements decide the transcription initiation point, specificity and the rate. Promoters include sets of various elements participating in the complex process of cell, tissue, organ, developmental stage and environmental factors-specific regulation of transcription. Most promoter elements regulating TSS selection are localized in the proximal promoter **(**Shahmuradov**)**, is a region several hundreds nucleotides around the TSS. If the SNP occurs within coding sequence, that may alter amino acid sequence and if the SNP occurs within the proximal promoter region, it may affect the potential alteration in transcription factor binding.The cis-regulatory regions have been hypothesized to facilitate adaptive innovations, because subtle nucleotide changes may generate novel phenotypes while preserving existing functions (Wray et al., 2003). Promoters and other cis-regulatory regions form a protein/ DNA complex with trans-regulatory proteins (transcription factors), thereby promoting interactive and integrative control of the expression. The functional architecture of these regions consists of short and often redundant transcription factor binding sites interspersed within a background sequence of apparently nonfunctional regions. In some cases, binding site loss through point mutation may be easily complimented by remaining other binding sites (Piano *et*

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