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Enhancers-Target Gene Pairs Identified Using Computational Biology

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Abstract

Threats to global food security have been widely publicised, including population growth, ageing farming populations, meat consumption trends, climate-change effects on abiotic and biotic stresses, and the environmental impacts of agriculture. Furthermore, with increasing pest, disease, and weed tolerance, traditional crop genetic and protective chemistry technologies of the 'Green Revolution' are under increasing strain. To alleviate the burden of these challenges, efforts have been made to automate and robotize aspects of the farming process. This drive has typically focused on higher-value sectors, such as horticulture and viticulture, which have historically relied on seasonal manual labour to maintain produce supply.

Keywords: Green revolution • Disease • Crop • Horticulture

Introduction

Enhancers are non-coding regulatory elements that are located outside of the target gene. Their characterization is still elusive, owing to difficulties in achieving a complete pairing of enhancers and target genes. To address this issue, a number of computational biology solutions have been proposed, taking advantage of the growing availability of functional genomics data and the improved mechanistic understanding of enhancer action. In this review, we will look at computational methods for defining enhancer-target gene pairs across the genome. We describe the various methods classes, as well as their main benefits and drawbacks. The types of information integrated by each method are presented and discussed, as well as details on their applicability [1].

Among these three challenges, the definition of enhancer-target gene pairs has received increasing attention in the fields of computational biology and genomics, owing to the increasing availability of genome-wide experimental data that can be used to address this problem. Indeed, high-throughput genome-wide methodologies for studying transcription factors (TFs) binding, core histone modifications, and RNA polymerase II (Pol2) association have drastically altered our understanding of how regulatory sequences are distributed in mammalian genomes [2]. Genome-wide chromatin mark profiles across various cell types are now available thanks to the efforts of large epigenomics consortia such as ENCODE and Roadmap Epigenomics.

Literature Review

To regulate their target genes, regulatory proteins bound at enhancers and those bound at their target gene promoters must be in close physical proximity. The formation of loops in chromatin, with the collaboration of various architectural proteins such as mediator or cohesin complexes, facilitates the physical interaction of enhancers and their distant target genes [3,4]. As a result, one key feature that can be used to refine ETG pairs is their localization within specific chromatin 3D structures, such as topological domains (TADs) [5].

Discussion

Specifically, p300 is a histone acetyltransferase protein that acts as a transcription co-activator and is known to bind at active enhancers. As a result, ChIP-seq experiments targeting p300 are frequently used for genome-wide enhancer annotation. However, ChIP-seq targeting specific histone marks has also been used to obtain a more comprehensive and unbiased list of enhancers [6]. In nucleosomes associated with active enhancers, chromatin marks such as high levels of histone H3 lysine 4 monomethylation (H3K4me1) accompanied by histone H3 lysine 27 acetylation (H3K27ac) are commonly found. H3K4me1 and H3K4me3 are commonly found in promoter regions, but the relative enrichment of the two marks is expected to differ between enhancers and promoters [7].

Conclusion

Active regulatory regions, such as enhancers and promoters, are distinguished by more accessible chromatin that is depleted of nucleosomes. As a result, genomics methods that probe chromatin accessibility, such as DNase-seq and ATAC-seq, can be used as alternatives for genome-wide identification of regulatory elements. Because these methods do not rely on antibodies, they avoid any issues with specificity or immunoprecipitation efficiency. DNase-seq is based on partial digestion with DNA nucleases such as DNase I, which will cut more frequently in more accessible positions that are not protected by histones or other DNA-associated proteins. DNase hypersensitivity sites are another name for these areas of increased accessibility (DHS). ATAC-seq, on the other hand, uses differences in transposase accessibility to identify open chromatin regions.

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Conflict of Interest

There are no conflicts of interest by author.

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