EDITORIAL

Preliminary analysis of whole-genome methylation and transcriptome-related genes in thyroid carcinoma

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ABSTRACT

Thyroid cancer is the most common endocrine cancer in the world and the leading cause of cancer-related death. Epigenetic changes are increasingly being related to metastasis. This work used a MethylationEPIC BeadChip (850K), RNA sequencing, and a targeted bisulfite sequencing technique to analyse whole-genome DNA methylation patterns and gene expression profiles in thyroid cancer tissue samples. The Illumina Infinium human methylation kit (850K) analysis found differentially methylated CpG sites (DMPs) and differentially methylated CpG regions (DMRs) across almost the whole genome with great resolution and depth. Gene ontology and KEGG pathway analysis found that DMR-associated genes belonged to a variety of domain-specific ontologies, including cell adhesion, molecule binding, and proliferation. The researchers discovered 1627 differentially expressed genes, 1174 of which were up-regulated and 453 of which were down-regulated, using RNA-Seq. The targeted bisulfite sequencing experiment demonstrated that methylation levels of CHST2, DPP4, DUSP6, ITGA2, SLC1A5, TIAM1, TNIK, and ABTB2 were considerably reduced in thyroid cancer patients compared to controls, although GALNTL6, HTR7, SPOCD1, and GRM5 were significantly higher. Our findings show that whole-genome DNA methylation patterns and gene expression levels in thyroid cancer provide fresh insight on thyroid cancer carcinogenesis.

Keywords: *thyroid carcinoma; targeted bisulfite sequencing assay; DNA methylation; MethylationEPIC*

BeadChip (850K); RNA-Seq

INTRODUCTION

Thyroid carcinoma is thought to be caused by a multi-step process of carcinogenesis in which cancer cells grow from thyroid follicular cells (thyroid epithelial cells) as a result of several instances of genomic insult. These lesions are most commonly seen in oncogenes and anti-oncogenes that encourage proliferation or the development of malignant characteristics, such as the capacity to infiltrate surrounding tissue or metastasis to distant organs. Thyroid carcinomas are a common kind of endocrine cancer with a wide range of phenotypes, ranging from benign to the most malignant forms of human cancer. Thyroid carcinomas are categorized into four types: well-differentiated thyroid carcinoma (WDTC), undifferentiated thyroid carcinoma (UTC), poorly differentiated thyroid carcinoma (PDTC), anaplastic thyroid carcinoma (ATC), and medullary thyroid carcinoma (MTC) (MTC). WDTC, UTC, PDTC, and ATC are all formed from thyrocytes, while MTC is derived from C cells. Furthermore, differentiated thyroid cancer is classified into three major subtypes: papillary thyroid carcinoma (PTC), follicular thyroid carcinoma (FTC), and Hürthle cell carcinoma. Differentiated thyroid carcinomas account for 95% of all thyroid cancers worldwide. Numerous epidemiological studies have discovered that the prevalence of differentiated thyroid cancer has dramatically grown during the previous few centuries. The great majority of thyroid carcinomas manifest as thyroid nodules, which are discovered via a physical examination or during neck imaging for other illnesses. In a tiny percentage of instances, thyroid nodules can be malignant. Thyroid nodules seen in the general population have a 5–10% probability of becoming malignant, while men and individuals at the ages of extremes are at a higher risk. A significant proportion of patients with well-differentiated thyroid carcinoma are treated with a total thyroidectomy, which includes excision of the anterior or central compartment lymph nodes, radioactive iodine therapy for metastasis, thyroid remnant abscission, and TSH suppression with l-thyroxin.

A better understanding of the molecular processes behind thyroid cancer development may be necessary for personalizing therapy. Significant progress has been achieved in this field during the previous three decades. Epigenetic processes include DNA methylation, chromatin remodeling, and post-translational histone changes. These mechanisms have been examined in the past. DNA methylation is a long-term epigenetic alteration that has been linked to cancer for more than three decades. DNA methylation is required for the normal development and functioning of various structures and cellular processes, including embryogenesis, transcription, X-inactivation, and genomic imprinting, as a gene silencing mechanism. In humans, polymer methylation happens virtually utterly within CpG dinucleotides, which are underrepresented, which means they occur less ofttimes than expected supported by the gigacycle per second content of polymer and aren't uniformly distributed throughout the order. The overwhelming majority of the human order is an alkyl group, with more or less 60–80 % of CG sites alkyl group, except selected CpG-rich sections called CpG islands or CG

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islands (CGIs), that ar are ofttimes unmethylated and contain promoters for about an hour of all protein-coding genes.

Although previous research has given insight on the role of gene regulation and DNA methylation in thyroid cancer development, the entire knowledge base remains exceedingly restricted. Several studies have shown that abnormal DNA methylation patterns in malignant cells can mutate tumor suppressor genes while activating oncogenes through hypermethylation/hypomethylation. However, hypermethylation is more commonly found in malignancies than hypomethylation. Some genes, such as MLH1 and p16INK4A, are often hypermethylated in cancers, including thyroid carcinoma. NIS (also known as SLC5A5), a tumor-specific sodium iodide symporter gene, is also expressed. Among all epigenetic modifications, DNA methylation on CpG islands has received the most attention. It is widely understood that hypermethylation of CpG islands in the promoter region of a gene lowers its expression. Furthermore, alterations in DNA methylation have been reported to occur early in oncogenesis, suggesting that they might be used as a potential biomarker for cancer diagnosis. Several DNA methylation-based indicators have been identified in malignancies ranging from stomach cancer to prostate cancer to bronchial carcinoma and bowel cancer. The first investigations on the impact of DNA methylation in thyroid cancer used a candidate gene technique to examine the DNA methylation level of specific gene promoters.

CONCLUSION

Finally, our integrative analysis provides a new perspective that gene expression regulated by DNA methylation alterations located primarily in the gene body may play a critical role in tumor progression and that DNA methylation levels of critical genes could be restored to normal by methylation or demethylation drugs for cancer treatment. Furthermore, these newly found genes might be employed as biomarkers to predict the development of thyroid cancer.