

## Special Issue on

## Noncoding RNAs and Base Modifications: Epigenomic Players Implicated in Neurological Disorders and Tumorigenesis

# CALL FOR PAPERS

Epigenetics is defined to investigate heritable alterations in gene expression caused by mechanisms other than DNA sequence variations. This nongenetic but heritable cellular memory, including dynamic DNA methylation/demethylation, histone modification, nucleosome location, and noncoding RNAs including small RNAs and long noncoding RNAs (lncRNAs), maintains all the biological processes in the programmed track. Any aberrant alterations could lead to development of abnormality and initiation of many diseases including neurological disorders and cancers.

Before 2009, DNA methylation of 5-cytosine (5-mC) was the best characterized epigenetic marker. In recent years, 5-hydroxycytosine (5-hmC), the sixth modified base, has been identified as an important epigenetic marker in mammalian epigenome. 5-hmC, converted from 5mC and catalyzed by enzymes of ten-eleven translocation protein (TET) family, has become a hallmark of cancer cells, and deletions and inactivating mutations in the TET2 gene account for almost 30% of all myeloid malignancies, suggesting the essential roles of 5-hmC and TET proteins in tumorigenesis. In addition, increasingly accumulated evidences are supporting the roles of 5-hmC in almost all the known metabolic pathways that are involved in embryogenesis, cell reprogramming, stem cell self-renewal, proliferation and differentiation, central nervous system (CNS) development, and aging.

The regulation mechanisms of 5-hmC in the metabolic pathways have been acknowledged to serve as the intermediate in the DNA demethylation process as well as the stable and independent marker to regulate gene expression by altering the epigenetic landscapes.

Among the base modifications, besides markers located in genomic DNA, RNA markers, particularly noncoding RNAs including small RNAs and lncRNAs, have been characterized as important epigenetic players in pathogenesis of many kinds of diseases including cancers and neurological diseases. In addition to base modifications, histone modifications based chromatin remodeling has been implicated in pathogenesis of numerous diseases as well.

Given the significant progress made in recent decades, this special issue will specifically focus on the research advances in the link between the epigenetic alteration and pathogenesis of neurological diseases and cancers.

We invite investigators in the field of epigenetics and genomics, particularly in the study of neurological disorders and cancers, to contribute original research articles as well as review articles that address the role of base modifications, noncoding RNAs, and chromatin remodeling in the development of CNS, pathogenesis of neurological disorders, and tumorigenesis.

Potential topics include but are not limited to the following:

- ▶ Breakthrough technologies/data analysis methodologies for single base resolution of DNA and RNA modification markers
- ▶ Application of next generation sequencing in genome-wide mapping of epigenetic markers and transcriptomes and proteomics in study of neurological disorders and tumorigenesis
- ▶ Small molecule drug library screening to identify the compounds that could regulate the epigenetic modifications involved in CNS development, neurological disorders, and tumorigenesis
- ▶ Dynamic alteration of base modification markers and epigenetic landscapes in mammalian genomes during CNS development and in response to environment stimuli such as natural and artificial stresses, as well as aging
- ▶ Base modification markers in DNA and RNA in animal models and clinical patient samples for the neurological disorders and cancers particularly in brain cancer, breast cancer, colon cancer, liver cancer, lung cancer, prostate cancer, and pancreatic cancer
- ▶ Small RNAs and other noncoding RNAs including lncRNA mediated epigenetic regulation in CNS development, neuron stem cell aging, neurological disorders, and tumorigenesis
- ▶ Chromatin remodeling mediated interactomes among the metabolic pathways involved in neurological disorders or tumorigenesis, such as interactions of methylases/demethylases/epigenetic modifiers and other tumor suppressors and oncogenes

Authors can submit their manuscripts through the Manuscript Tracking System at <https://mts.hindawi.com/submit/journals/ijg/hlap/>.

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