



OPEN ACCESS

EDITED AND REVIEWED BY

Michael E. Symonds,
University of Nottingham,
United Kingdom

*CORRESPONDENCE

Xiang Wang,
✉ xiangwangbio@gmail.com

RECEIVED 07 July 2023

ACCEPTED 17 July 2023

PUBLISHED 18 August 2023

CITATION

Wang X (2023), Editorial: Chromatin architecture in gene regulation and disease.

Front. Genet. 14:1254865.

doi: 10.3389/fgene.2023.1254865

COPYRIGHT

© 2023 Wang. This is an open-access article distributed under the terms of the [Creative Commons Attribution License \(CC BY\)](https://creativecommons.org/licenses/by/4.0/). The use, distribution or reproduction in other forums is permitted, provided the original author(s) and the copyright owner(s) are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.

Editorial: Chromatin architecture in gene regulation and disease

Xiang Wang*

Children's Hospital of Philadelphia, Philadelphia, PA, United States

KEYWORDS

chromatin architecture, human disease, genome organization, gene expression, enhancer-promoter interaction

Editorial on the Research Topic
[Chromatin architecture in gene regulation and disease](#)

Introduction

Chromatin architecture, the three-dimensional organization of DNA and its associated proteins, plays a pivotal role in gene regulation and disease. Recent advances in genomics and molecular biology have shed light on the intricate relationship between chromatin architecture and various biological processes. This Research Topic aims to highlight the significance of understanding chromatin architecture in unraveling the complexities of gene regulation and its implications for human health and disease.

Insights into the genome organization

Chromatin architecture influences gene regulation by regulating the accessibility of DNA to transcriptional machinery. It orchestrates the interactions between regulatory elements, such as enhancers, promoters, and insulators, and their target genes. Elucidating the mechanisms by which chromatin architecture modulates gene expression holds the key to understanding fundamental biological processes, including development, cellular differentiation, and environmental responses. [Kyrchanova et al.](#) review the enhancer-promoter interaction and chromosomal architecture. They discuss the difference of chromosomal architecture between mammals and *Drosophila*. [Scott et al.](#) review how changes in the mechanical environment induce alterations in chromatin architecture. They discuss the critical questions need to be addressed to give insights into the mechanically induced disease.

Uncovering chromatin architecture during DNA replication

DNA replication is a highly regulated process that ensures the accurate duplication of genome prior to cell division. The replication timing (RT) of different regions of the genome is not uniform and has been found to correlate with chromatin architecture ([Marchal et al., 2019](#); [Pope et al., 2014](#)). [Yu et al.](#) examine the 3D genome characteristics at the RT-switching

regions. This study expands our understanding of the relationship between RT, histone modification and 3D chromatin structure in cell differentiation.

Chromatin architecture in cancer

Aberrations in chromatin architecture have been implicated in various human diseases, including cancer, neurodegenerative disorders, and developmental abnormalities. Dysregulation of gene expression due to altered chromatin structure can disrupt delicate cellular homeostasis, leading to disease initiation and progression. [Gridina and Fishman](#) review how the organization of the cancer cell genome differs from the healthy genome at various levels. They discuss how these changes in genome organization contribute to cancer development.

Unraveling the connections between chromatin architecture and disease provides crucial insights for the development of novel diagnostic, prognostic, and therapeutic strategies. [Wang et al.](#) examine the alterations of chromatin organization in doxorubicin-resistant breast cancer and identify potential diagnostic or therapeutic targets associated with the reorganization of chromatin architecture. This study highlights the connection between 3D genome reorganization, chromatin accessibility, and gene transcription and provides valuable insights into the epigenomic mechanisms underlying doxorubicin resistance and offers potential targets for breast cancer treatment.

The study of chromatin architecture in gene regulation and disease represents a rapidly advancing field that offers unprecedented insights into the fundamental mechanisms governing cellular processes and

disease pathology. By understanding the principles underlying the intricate orchestration of chromatin architecture and gene regulation, researchers can pave the way for innovative approaches to diagnose, prevent, and treat a wide range of diseases. Continued exploration of this Research Topic has the potential to redefine our understanding of human health and open new avenues for personalized medicine in the future.

Author contributions

The author confirms being the sole contributor of this work and has approved it for publication.

Conflict of interest

The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article, or claim that may be made by its manufacturer, is not guaranteed or endorsed by the publisher.

References

Marchal, C., Sima, J., and Gilbert, D. M. (2019). Control of DNA replication timing in the 3D genome. *Nat. Rev. Mol. Cell Biol.* 20 (12), 721–737. doi:10.1038/s41580-019-0162-y

Pope, B. D., Ryba, T., Dileep, V., Yue, F., Wu, W., Denas, O., et al. (2014). Topologically associating domains are stable units of replication-timing regulation. *Nature* 515 (7527), 402–405. doi:10.1038/nature13986