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Editorial

Gene Regulation and Signaling: A Platform Integrating Chromatin Dynamics, RNA Biology, and Cellular Communication

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The past three decades have witnessed extraordinary progress in our understanding of intracellular signaling and gene regulation. Molecular cloning and dissection of receptors at the cell membrane and within the nucleus, followed by characterization of their downstream mediators, revealed a diverse array of signaling pathways that govern cell growth, differentiation, metabolism, and survival. In parallel, the molecular identification of transcription factors and regulatory elements illuminated the core processes of transcriptional control. The subsequent emergence of epigenetics—catalyzed by the histone code concept—transformed our view of how chromatin structure and histone modifications shape transcriptional outcomes. Together, these advances established gene expression, chromatin regulation, and cellular signaling as interdependent layers of biological control.

Despite remarkable mechanistic insights, major questions remain. Most signaling pathways ultimately converge on the nucleus to modulate gene expression, yet the molecular logic linking extracellular stimuli to chromatin dynamics and transcriptional programs is still incompletely understood. Signaling-dependent chromatin remodeling, higher-order genome organization, and nuclear events that coordinate enhancer–promoter communication represent key frontiers. Addressing these challenges requires approaches that integrate molecular biology, genomics, structural biology, and systems-level analysis. Understanding how signaling and chromatin reorganization are coupled is not only fundamental biology—it is also central to deciphering disease mechanisms.

Disease research has also advanced rapidly, driven by large-scale cohort studies, epidemiology, and, most notably, next-generation sequencing (NGS) combined with bioinformatics. These technologies have broadened our ability to identify genetic and epigenetic drivers of disease, and the emergence of artificial intelligence promises to accelerate interpretation of complex datasets. NGS has revealed that much of the human genome is transcribed, producing vast repertoires of non-coding RNAs (ncRNAs). Yet only a small fraction of ncRNAs have been functionally characterized. This gap is particularly important because many hereditary disorders remain unresolved when analysis is confined to protein-coding exons. Increasing evidence suggests that pathogenic mechanisms may involve disruptions in non-coding regulatory elements and ncRNA-mediated networks, including enhancer RNAs, long non-coding RNAs, and microRNAs.

Moreover, the landscape of human variation is far richer than previously appreciated. Beyond single-nucleotide changes, NGS has enabled detection of small insertions and deletions, structural variants, copy number variations, and somatic mosaicism—forms of variation that conventional sequencing approaches often failed to capture. These genetic architectures add additional layers of complexity to gene regulation, chromatin state, and ncRNA function, and they may help explain variable penetrance, heterogeneity in clinical phenotypes, and differential therapeutic responses.

At the cellular level, ncRNAs are increasingly recognized as active regulators of transcription, chromatin modification, and post-transcriptional gene control. Many ncRNAs are transcribed by RNA polymerase II and appear to be regulated by enhancer-binding transcription factors, responding dynamically to extracellular cues and environmental changes in ways that resemble messenger RNAs. Thus, aberrant expression of ncRNAs—like

dysregulated mRNA programs—may contribute directly to disease initiation and progression. At the organismal level, recent studies have revealed complex signaling cross-talk between organs mediated by humoral factors, many of which remain functionally uncharacterized. Understanding how these systemic signals modulate gene expression and epigenetic states in target tissues represents another major frontier with high relevance to metabolic, immune, cardiovascular, and aging-related disorders.

A further emerging dimension is the role of chemical modifications on RNA, such as m^6A and related marks, which modulate RNA stability, localization, and translation. These epitranscriptomic mechanisms influence gene regulatory networks and intersect with chromatin regulation and signaling pathways, opening promising avenues for both mechanistic discovery and therapeutic intervention.

Against this backdrop, the journal *Gene Regulation and Signaling* (*GRS*) aims to serve as an international platform for high-quality research spanning gene expression, epigenetic regulation, chromatin dynamics, signaling pathways, and cross-talk between cells and organs in health and disease. We welcome contributions ranging from basic mechanisms to translational and clinical science, and we also encourage reports from industrial research that advance technology development, drug discovery, and clinical intervention. By bringing together diverse disciplines and communities, we hope that *GRS* will accelerate progress in understanding the regulatory logic of life and transforming that knowledge into improved strategies for prevention, diagnosis, and therapy.

Conflicts of Interest

Given the role as the Editor-in-Chief of the journal, Shigeaki Kato had no involvement in the peer review of this paper and had no access to information regarding its peer-review process. Full responsibility for the editorial process of this paper was delegated to another editor of the journal.

Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.