

## Heeyoun Bunch

Department of Applied Biosciences, Kyungpook National University

**Monday, July 1, 2024**

14:00-15:00

2F C210-212, Central Building, Yokohama / Broadcast online via Zoom

Zoom meeting URL will be announced on the event day by e-mail.

※Non-BDR members: Please register from the following link.

<https://krs2.riken.jp/m/bdrseminarregistration> (Registration deadline June 26)

## Crosstalk between transcriptional elongation by RNA polymerase II and DNA topological regulation and repair in human stress-inducible genes

### Summary

During the early transcriptional elongation stage of stress-inducible genes in metazoan cells, RNA polymerase II (Pol II) is paused in the promoter-proximal site, approximately 25–100 bp from the transcription start site, a phenomenon called Pol II pausing. This Pol II pausing is mediated and stabilized by multiple regulatory elements and must be released upon the reception of transcriptional activating signals. In 2014, our study discovered that TRIM28 regulates Pol II pausing at human *HSP70* gene. In fact, TRIM28 not only stabilizes Pol II pausing but also is required to activate transcription of *HSP70*. Importantly, this finding led to further discoveries that DNA repair factors such as phosphatidylinositol 3-kinase-related kinases function as transcriptional activators in stress-inducible genes including *HSP70* and immediate early genes. Why, then, is the function of DNA repair factors needed for transcriptional activation and elongation? For the question, we have been achieving important, on-going mechanistic understanding involving DNA topoisomerase II, suggesting that the regulation of DNA topology takes place and is necessary to activate these genes. In this presentation, I will summarize and discuss our findings in the past decade, regarding the crosstalk between Pol II pausing and DNA topological regulations and the important function of DNA repair factors in human gene regulation.