Investigating the physical interactions between HMGB2 and important players in genome organization, cohesin, and CTCF

DNA inside of a cell is organized in a precise manner and this organization is crucial for the control of gene expression. Cohesin is a ring-like protein complex that extrudes DNA to organize into DNA loops, brining linearly distant segments of DNA into close physical proximity in the three-dimensional space. Cohesin interacts with a protein known as CTCF in order to form most DNA loops. Cohesin and CTCF are essential for genome organization at the DNA loop level as loss of either eliminates loop domains[1]. However, not all DNA loops have CTCF at their boundary sites, suggesting that other proteins may play a structural role in DNA loop regulation with cohesin. One such protein that we are interested in studying is HMGB2. HMGB2 has been shown to localize to CTCF and non- CTCF loop boundary sites. Loss of HMGB2 has also been shown to decrease the number of DNA loops[2]. We hypothesize that HMGB2 may play a structural role in DNA loops by interacting with either cohesin and/or CTCF. We use co-immunoprecipitation to identify physical interactions between cohesin, CTCF, and HMGB2.

Reference:

1. Rao SSP, Huang S-C, Glenn St Hilaire B, Engreitz JM, Perez EM, Kieffer-Kwon K-R, et al. *Cohesin loss eliminates all loop domains.* Cell. 2017 Oct 5;*171*(2):305-320.e24.

2. Zirkel A, Nikolic M, Sofiadis K, Mallm J-P, Brackley CA, Gothe H, et al. *HMGB2 Loss upon Senescence Entry Disrupts Genomic Organization and Induces CTCF Clustering across Cell Types.* Mol Cell. 2018 May 17;*70*(4):730-744.e6.