20th International p53 Workshop- Abstract Submission Template

Submission Deadline: January 31st 2026 (5pm EDT)
Notification of acceptance: By or before February 27, 2026

Title of study/project: Mechanisms of p53-mediated transcriptional regulation of transposable elements

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Abstract:

The transcription factor and tumor suppressor p53, also known as the guardian of the genome, has been suggested to restrict transposable element (TE) expression. Tumors harboring mutant p53 show increased TE expression levels compared to wild-type p53 tumors. Mechanistically, it has been suggested that p53 directly represses the expression of TEs, particularly LINE-1 retrotransposons. However, most studies of p53-regulated TE expression have been limited to family-level analyses, a few specific TE loci, or exogenous consensus versions of TE families. These approaches have naturally hampered systematic in-depth expression analyses of individual TE instances. Here, we investigated the occurrence of p53 binding sites in TEs and the locus-specific regulation of TEs by p53 on a genome-wide scale. We used p53 binding sites derived from ChIP-seq data with predicted p53 response elements. Our data show that approximately half of all p53 binding sites are located within TEs and that several of these binding sites contribute to promoters and enhancers. Using a locus-specific TE expression analysis strategy, we identified more than 18,000 significantly differentially regulated individual TEs upon p53 activation by Nutlin-3a, with a similar number of up and down-regulated TEs. Importantly, our data suggest that the direct binding of p53 is strongly associated with upregulation of TEs, including LINE-1 instances. This finding is consistent with the general transactivator function of p53, although it contradicts previous studies on TE regulation by p53. We used ATAC-seq and CUT&Tag to identify p53-mediated changes in the chromatin structure at TEs. Our data reveal that p53 makes the DNA accessible and establishes active chromatin states at TEs. Using CAGE-seq data, we discovered that some transcription start sites (TSSs) drive the

expression of entire islands of TEs, enabling transcription factors such as p53 to regulate multiple TEs through one shared promoter.

Taken together, our study maps a comprehensive landscape of p53-regulated TEs in the human genome and highlights the function of p53 as a transactivator of TEs through chromatin remodeling.

Please email your submission to us at p53workshop2026@uhn.ca . Please use the following subject heading: Abstract- p53 International workshop