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: Unraveling mechanisms of RFX7 regulation

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Training program first author is enrolled in: *First Author is required to be enrolled in a training program during 2025-2026. Example: Radiation oncology Residency Program*

In Germany, it is not common to be enrolled in dedicated training programs.

Year of training: *Example: PGY 3*
Early Postdoc (first year)

Abstract: *Approx. 300 words*
Suggested format: Purpose, Materials and Methods, Results, Conclusions

Purpose: We previously showed that the tumor suppressor p53 activates the transcription factor RFX7 in response to p53-activating stress signals and that this activation is associated with an unknown post-translational modification (PTM) of RFX7. Here, we elucidate the PTM underlying RFX7 regulation.

Materials and Methods: U2OS *RFX7*-knockout cells were reconstituted with doxycycline-inducible FLAG–RFX7, and p53 was activated using Nutlin-3a. RFX7 phosphorylation was assessed by electrophoretic mobility shifts on immunoblots and validated using a dephosphorylation assay. To identify differentially phosphorylated amino acids, phosphoproteomics was performed comparing Nutlin-3a and control-treated cells. To identify proteins that interact with hypo- or hyper-phosphorylated RFX7, co-immunoprecipitation was combined with mass

spectrometry analysis (coIP–MS). RFX7 activity was inferred by analyzing RFX7 target gene expression by RT–qPCR and immunoblotting (e.g., PDCD4, PIK3IP1, MXD4, and DDIT4) in response to established kinase inhibitors.
Results: We find that the lower-migrating form of RFX7 reflects a hypo-phosphorylated state. Thus, RFX7 is kept inactive by phosphorylation. Upon p53 activation, RFX7 becomes hypophosphorylated. Phosphoproteomics reveals at least a dozen RFX7 sites that are phosphorylated when RFX7 is inactive. CoIP–MS identified candidate RFX7 interacting proteins. Importantly, we identified kinases that enriched for binding to inactive, hyperphosphorylated RFX7. Using established kinase inhibitors, we validated a kinase as being critical for RFX7 phosphorylation and inactivation.
Conclusions: These data suggest a model whereby RFX7 activity is largely regulated by phosphorylation. p53 signaling impairs RFX7 phosphorylation and thereby activates RFX7 and RFX7 target gene expression. The kinase that inactivates RFX7 reveals a potential role of RFX7 in multiple cancer-relevant pathways.

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