



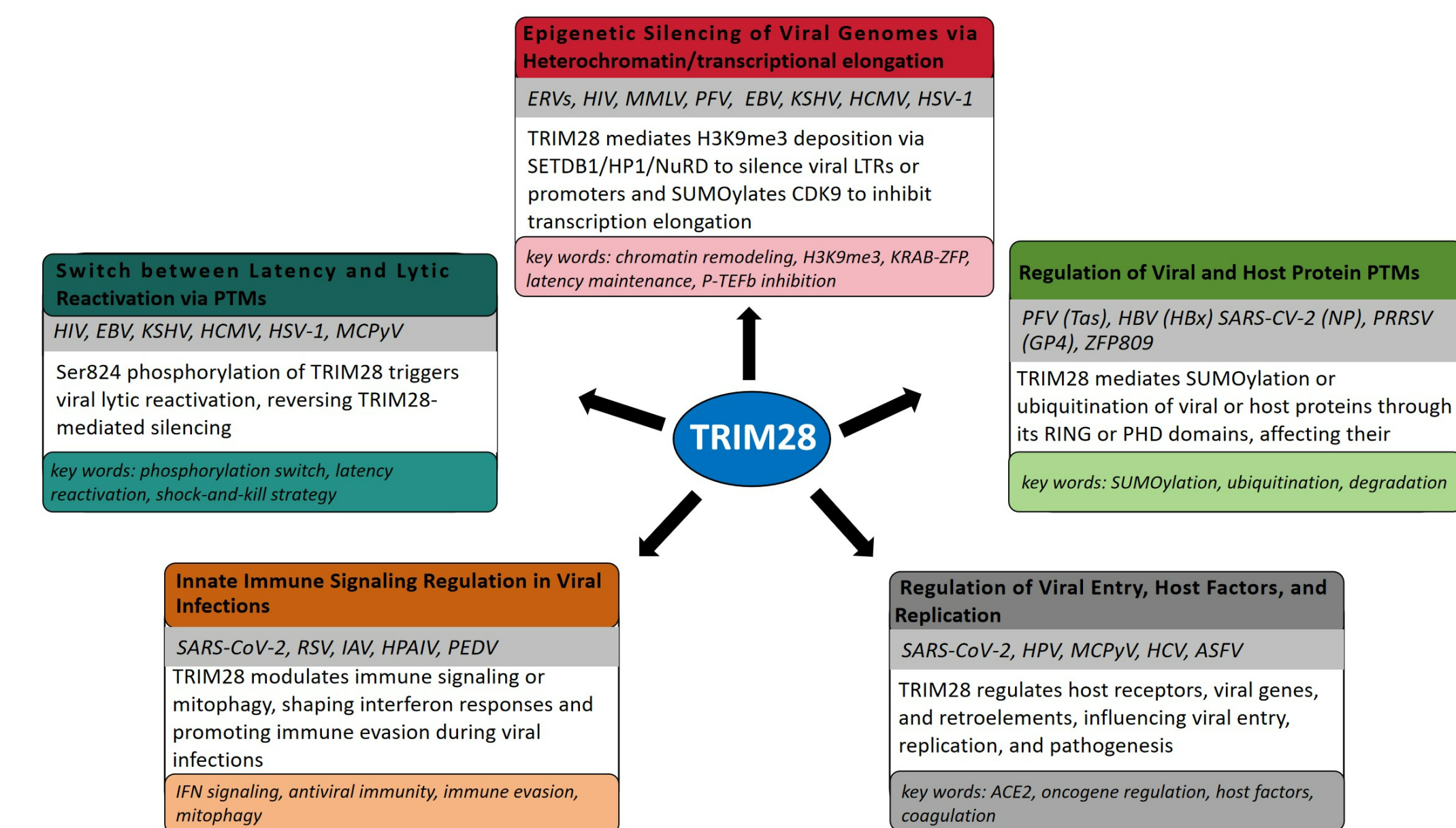
KAP1 in Antiviral Immunity: Dual Roles in Viral Silencing and Immune Regulation



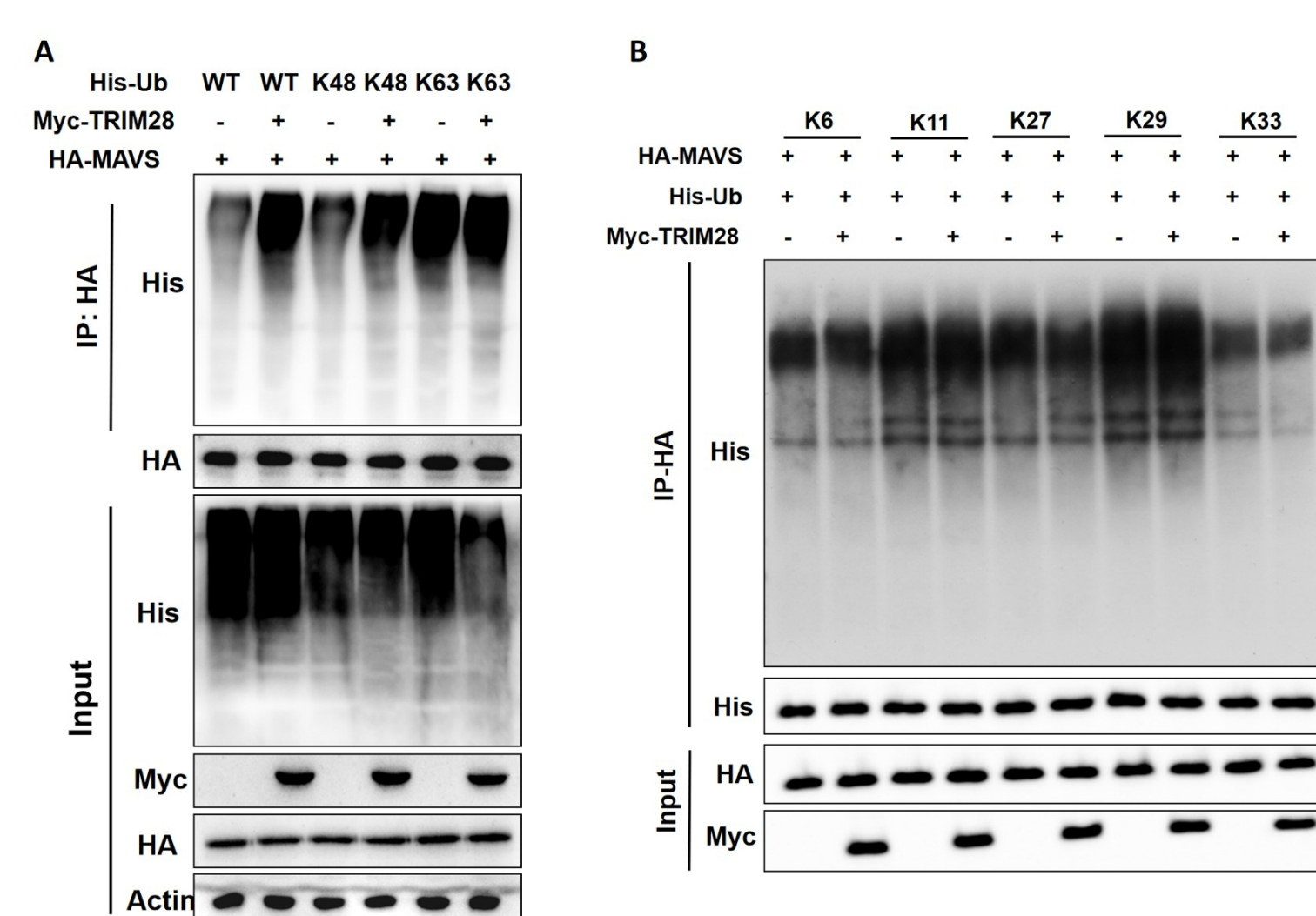
INTRODUCTION

This review provides a structured and up-to-date summary of KAP1/TRIM28, integrating its protein architecture, canonical physiological functions, and emerging regulatory roles in viral infection. Building upon established knowledge of KAP1's involvement in the transcriptional repression of endogenous retroviruses (ERVs) and its context-dependent regulation of HIV, we expand the scope to include its roles in herpesviruses, respiratory RNA viruses (such as SARS-CoV-2 and IAV), and DNA viruses (such as HBV and ASFV). By organizing these findings into five mechanistic modules—heterochromatin-based silencing, latency-lytic phase transition, regulation of protein post-translational modifications (PTMs), modulation of innate immune signaling, and control of host factors and viral entry—we aim to provide a comprehensive perspective on how KAP1's structural domains and PTMs contribute to its diverse roles in virus-host interactions.

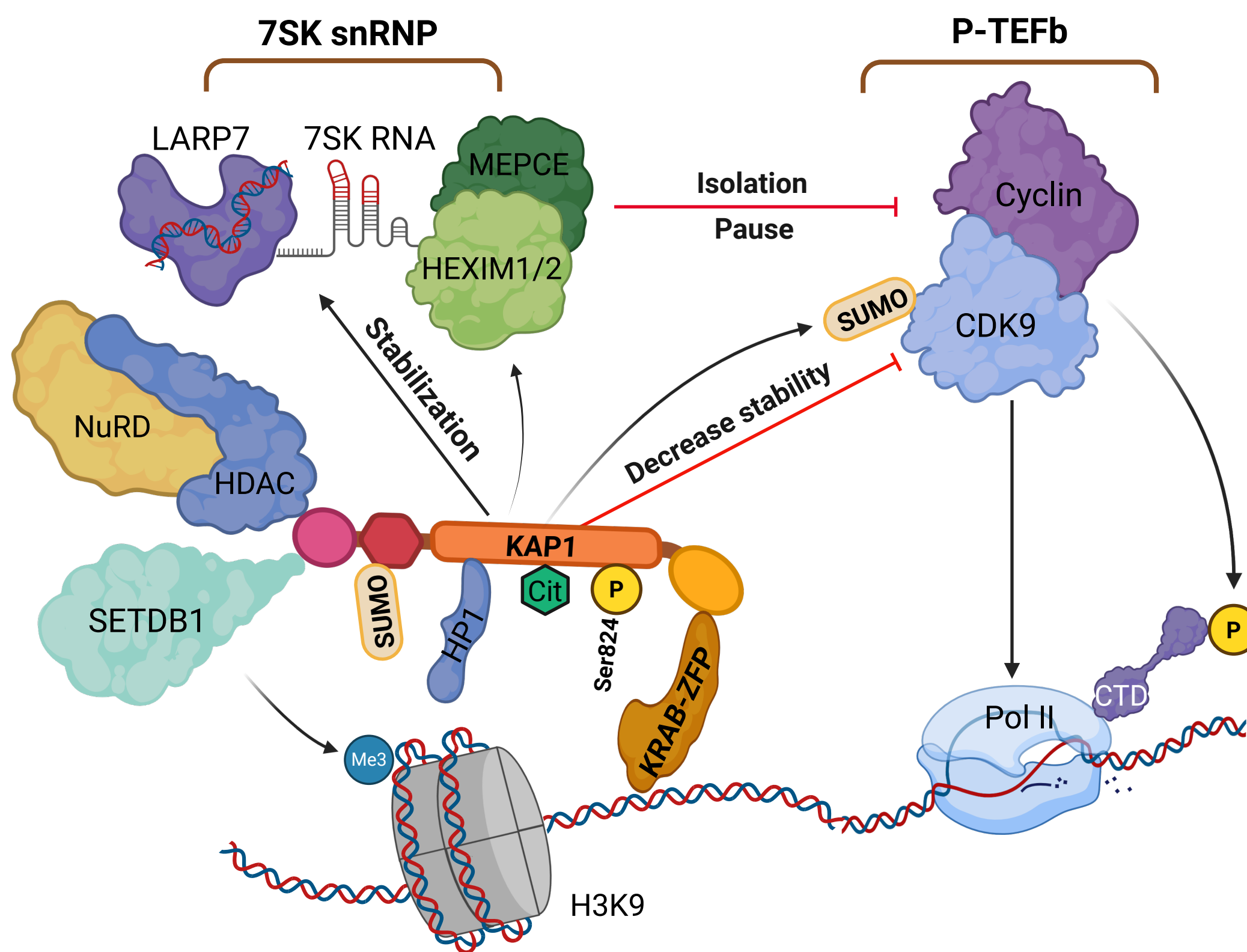
METHODS



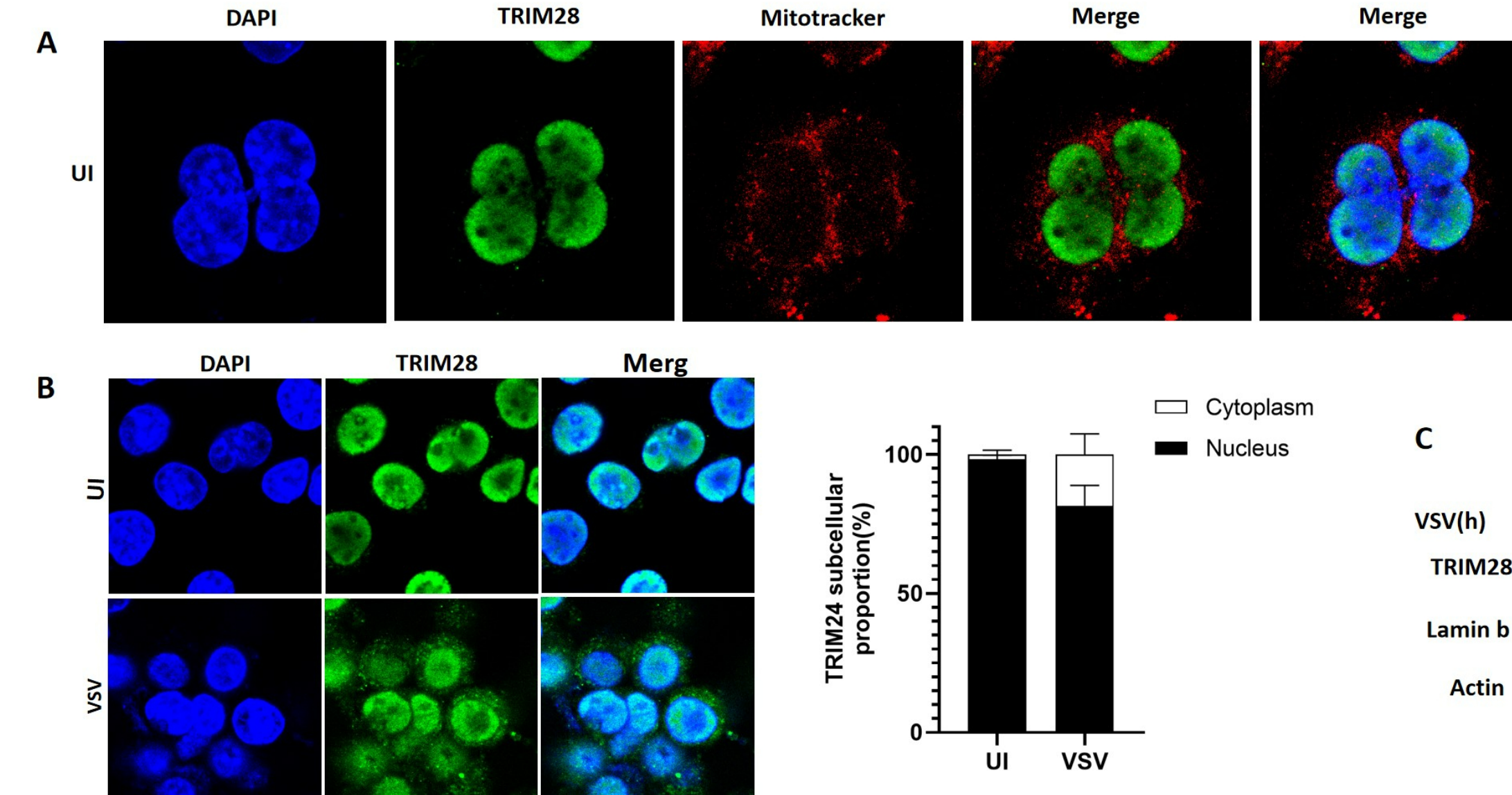
9. TRIM28 mediates K48-linked ubiquitination of MAVS



- Since TRIM28 acts as an E3 ligase and interacts with MAVS, we tested whether it mediates MAVS ubiquitination.
- We co-transfected MAVS with different ubiquitin mutants in 293T cells and found that TRIM28 specifically enhanced K48-linked ubiquitination. This suggests TRIM28 may promote MAVS degradation through K48-linked ubiquitination.

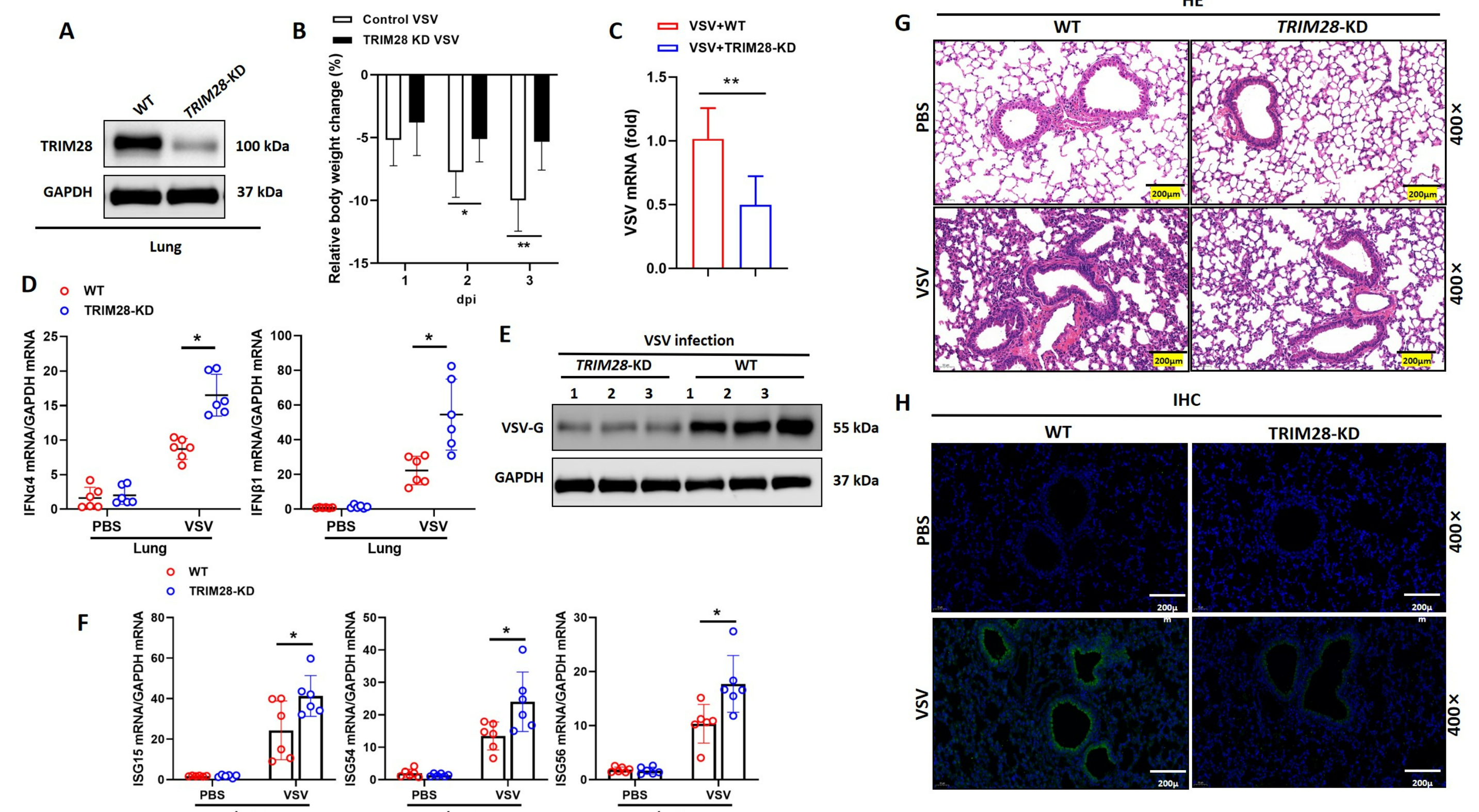
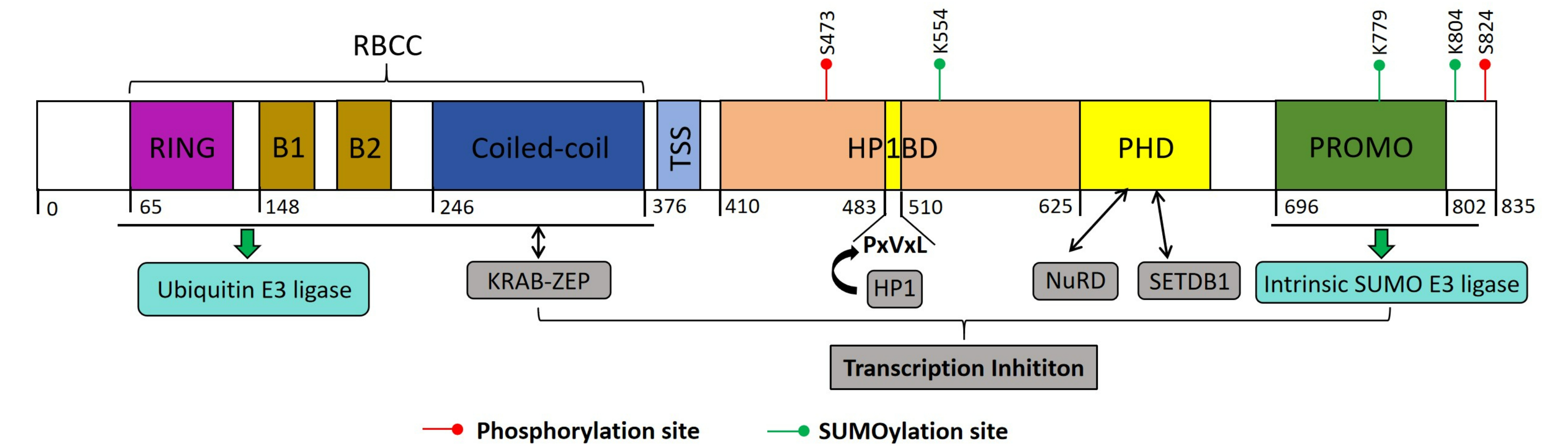


8. TRIM28 is transported out of the nucleus through Crm1 and co-localizes with MAVS



As expected, in unstimulated cells, TRIM28 was predominantly nuclear. However, after VSV infection, we observed that part of TRIM28 translocated from the nucleus to the cytoplasm.

RESULTS



ACKNOWLEDGEMENTS

- University of Liège
- Lanzhou Institute of Husbandry and Pharmaceutical Sciences, Chinese Academy of Agricultural Sciences(CAAS)