Conclusion

Conclusion

Our study investigated the role of TRIM34 in non-small cell lung cancer (NSCLC). Expression analyses, particularly in response to various interferons, revealed substantial upregulation, implicating TRIM34 in the intricate network of cellular responses to interferon treatments. The study delved into the impact of IFN-γ on NCI-H23 cells, revealing increased TRIM34 protein levels and providing visual confirmation of its presence post-treatment. Functional consequences, including pro-apoptotic effects, altered expression of apoptosis-related genes, and inhibition of cell migration, suggest a potential therapeutic avenue for IFN-γ induced TRIM34 overexpression in lung adenocarcinoma.

The exploration of TRIM34's role in NSCLC extended to CRISPR/Cas9 gene KO editing, showcasing its impact on cell viability, apoptosis, and cell cycle dynamics. RNA-seq analysis uncovered significant alterations in expression of genes involved in ubiquitylation pathway upon TRIM34 knockout. Protein-Protein interaction network analysis shed light on potential molecular mechanisms affected by TRIM34. Enrichment studies pointed towards TRIM34's influence on ubiquitylation processes in NSCLC.

Epigenetic studies revealed the impact of DNA methylation on TRIM34 expression, with 5-Aza-2'-deoxycytidine (AZA) treatment demonstrating a reduction in methylation levels and a subsequent upregulation of TRIM34 expression. Validation through qRT-PCR and sanger sequencing of bisulfite converted TRIM34 promoter supported the potential of combining DNA methylation inhibitors with IFN as a strategy in cancer therapeutics.

The comprehensive exploration of TRIM34 in NSCLC not only advances our understanding of its functional roles but also unveils potential therapeutic avenues, emphasizing the complex interplay between genetics and epigenetics in lung cancer pathogenesis and treatment strategies.