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: RUNX3-p53-MYC interplay determines metastatic outgrowth in gastric cancer
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Training program first author is enrolled in: Postdoctoral Fellowship
Year of training: Year 3

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

Purpose

Although RUNX3 suppresses early tumorigenesis, its frequent elevated expression in gastric cancer metastases suggests context-dependent functions. We therefore sought to determine how TP53 status governs a RUNX3-driven transcriptional switch between tumor suppression and metastatic reprogramming, and to establish proof-of-concept for TP53 genotype-guided therapeutic disruption of RUNX in metastasis.

Materials and Methods

RUNX family gene expression was quantified by immunohistochemistry in gastric cancer specimens including metastases. RUNX3 was deleted by CRISPR–Cas9 in metastatic gastric cancer cell lines with TP53 loss-of-function, gain-of-function mutation, or wild-type TP53. Analyses using RNA-seq, ChIP-seq/re-ChIP, co-immunoprecipitation, migration/invasion assays and mouse metastasis models were integrated. RUNX small-molecule inhibitors (Ro5-3335 or AI-10-104) were tested in vitro and in vivo.

Results

RUNX3 was the most frequently expressed RUNX paralog in metastatic lesions and was elevated in 57% of gastric cancer liver metastases. TP53 status dictated RUNX3 binding partners and transcriptional output in metastatic gastric cancer cell lines. With functional p53, RUNX3 reinforced p53-dependent cytostatic and pro-apoptotic pathways and constrained epithelial–mesenchymal transition (EMT) and metastatic outgrowth. With p53 loss or mutation, RUNX3 engaged MYC to drive TGFβ–dependent EMT programs and metastatic colonization. Accordingly, pharmacologic RUNX inhibition suppressed EMT-associated gene expression, migration/invasion and metastatic burden in TP53-defective contexts, but enhanced EMT and metastatic traits in TP53-wild-type cells.

Conclusion

TP53 status determines whether RUNX3 promotes p53-dependent tumor suppression or MYC-driven metastatic programs, thereby predicting benefit versus harm of RUNX inhibition during clinical intervention. Our work supports TP53 genotype–guided targeting of RUNX signaling in metastatic gastric cancer.

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