INSTRUCTIONS FOR 2026 MALTO ABSTRACTS

Please submit your final abstract in MS Word format following the formatting guidelines below using the following link:

2025 MALTO Abstract Submission Form

Presenter's status must be indicated (i.e., Graduate student (GS) or Postdoctoral Fellow (PF))
Presentation type: Podium or Poster
Type your abstract in MS Word using the following format.
Graphics or Tables may be included in the abstract, but the 1-page limit cannot be exceeded.

1. Page Setup

Margins:	1" on all sides.
Font:	Times New Roman
Font Size:	12
Line spacing:	Single

2. Abstract Template

TITLE OF THE ABSTRACT (ALL CAPITAL LETTERS, BOLD, WITH JUSTIFIED MARGINS)
Leave one blank line after the title.
Author(s) name and affiliation. Underline the presenting author.
Leave one blank line after the author(s) name(s).
Include the institutional affiliation of each author.
Leave one blank line before the text of the abstract.

Text of the abstract: No indentation, left and right margins must be justified. *250 word limit.*

Page Limit: One.

See the Abstract example below.

GS/PF (Presentation: poster or podium)

TARGETS THE COLCHICINE BINDING SITE ON TUBULIN AND OVERCOMES TAXANE RESISTANCE

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Antimitotics that target tubulin are among the most useful chemotherapeutic drugs, but their clinical activity is often limited by the development of multidrug resistance. We recently discovered the novel small molecule 2-(1H-indol-4-yl)-4-(3,4,5-trimethoxyphenyl)-1Himidazo[4,5-c]pyridine (DJ101) as a potent and metabolically stable tubulin inhibitor that can circumvent the drug efflux pumps responsible for multidrug resistance of existing tubulin inhibitors. In this study, we further evaluated the mechanism of action of this drug. The basis for its activity was illuminated by solving the crystal structure of DJ101 in complex with tubulin at a resolution of 2.8Å (PDB 5H7O). Investigations of the potency of DJ101 in a panel of human metastatic melanoma cell lines harboring major clinically relevant mutations demonstrated IC_{50} values of 7-10 nM. Additional *in vitro* studies revealed DJ101 disrupted microtubule networks, suppressed anchorage-dependent melanoma colony formation and impaired cancer cell migration. Administration of DJ101 significantly inhibited A375 melanoma tumor growth and B16F10 melanoma metastasis in xenograft and lung metastasis models in mice. DJ101 also completely inhibited tumor growth in a paclitaxel-resistant xenograft mouse model of human prostate cancer (PC-3/TxR), where paclitaxel was minimally effective. Pharmacological screening data showed negligible interactions with physiologically important targets and observable toxicity was not apparent in animal studies, suggesting a good safety profile for DJ101. Our findings offer preclinical proof of concept for the continued development of DJ101 an improved generation of tubulin inhibitors for cancer therapy.