

Resistance is futile: Researchers identify gene that mediates cisplatin resistance in ovarian cancer

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Platinum compounds, such as cisplatin and carboplatin, induce DNA cross-linking, prohibiting DNA synthesis and repair in rapidly dividing cells. They are first line therapeutics in the treatment of many solid tumors, but cancer cells frequently develop resistance to these drugs.

Mechanisms of resistance typically include reduced platinum uptake and increased platinum export. In this issue of the *Journal of Clinical Investigation*, Anil Sood and colleagues at M.D. Anderson Cancer Center identified a cellular membrane protein, ATP11B, that mediates cisplatin resistance in [ovarian cancer cells](#).

They found that ATP11B expression was correlated with higher tumor grade in human ovarian cancer samples and with cisplatin-resistance in human ovarian cancer cell lines. Further, loss of ATP11B restored the sensitivity of ovarian cancer cell lines to cisplatin and reduced ovarian tumor growth in mice.

These findings suggest that ATP11B could serve as a [therapeutic target](#) to overcome cisplatin resistance.

More information: ATP11B mediates platinum resistance in ovarian cancer, *J Clin Invest*.
[doi:10.1172/JCI65425](https://doi.org/10.1172/JCI65425)

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