

A major chromatin regulator determines resistance of tumor cells to T cell-mediated killing

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SNF-ing out antitumor immunity

Immune checkpoint inhibitors induce durable tumor regressions in some, but not all, cancer patients. Understanding the mechanisms that determine tumor sensitivity to these drugs could potentially expand the number of patients who benefit (see the Perspective by Ghorani and Quezada). Pan *et al.* discovered that tumor cells in which a specific SWI/SNF chromatin remodeling complex had been experimentally inactivated were more sensitive to T cell-mediated killing. The cells were more responsive to interferon- γ , leading to increased secretion of cytokines that promote antitumor immunity. Miao *et al.* examined the genomic features of tumors from patients with metastatic renal cell carcinoma who had been treated with immune checkpoint inhibitors. Tumors harboring inactivating mutations in *PBRM1*, which encodes a subunit of the same SWI/SNF complex, were more likely to respond to the drugs.

Science, this issue p. 770, p. 801; see also p. 745

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