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Bahcall and colleagues report the case of a patient with recurrent non–small cell lung cancer (NSCLC) harboring an EGFR exon 19 deletion mutation and high-level MET amplification who initially responded to a type I MET inhibitor combined with an EGFR inhibitor but acquired a METD1228V mutation that promoted resistance. Protein modeling predicted that METD1228V would not alter sensitivity to type II MET inhibitors, which bind the inactive conformation of MET. Consequently, the patient was treated with a type II MET inhibitor combined with an EGFR inhibitor and achieved an ongoing response. These results indicate that MET may be therapeutically targeted in NSCLC, and type II MET inhibitor sensitivity may be maintained even in cells resistant to type I MET inhibitors. Therefore, determining MET inhibitor resistance mechanisms may guide drug selection in patients. For details, please see the article by Bahcall and colleagues on page 1334.

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