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Bahcall and colleagues report the case of a patient with recurrent non–small cell lung cancer (NSCLC) harboring an \textit{EGFR} exon 19 deletion mutation and high-level \textit{MET} amplification who initially responded to a type I \textit{MET} inhibitor combined with an \textit{EGFR} inhibitor but acquired a \textit{MET}D1228V mutation that promoted resistance. Protein modeling predicted that \textit{MET}D1228V would not alter sensitivity to type II \textit{MET} inhibitors, which bind the inactive conformation of \textit{MET}. Consequently, the patient was treated with a type II \textit{MET} inhibitor combined with an \textit{EGFR} inhibitor and achieved an ongoing response. These results indicate that \textit{MET} may be therapeutically targeted in NSCLC, and type II \textit{MET} inhibitor sensitivity may be maintained even in cells resistant to type I \textit{MET} inhibitors. Therefore, determining \textit{MET} inhibitor resistance mechanisms may guide drug selection in patients. For details, please see the article by Bahcall and colleagues on page 1334.