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On the cover: Isocitrate dehydrogenase 1 (IDH1) mutation is observed in nearly all secondary glioblastomas and suppresses the biochemical ability of IDH1 to convert isocitrate into α-Ketoglutarate (α-KG) by further converting α-KG into 2-hydroxyglutarate (2-HG). As a result, the 2-HG oncometabolite accumulates at high levels in IDH1 mutant tumors and inhibits α-KG-dependent histone and DNA demethylases, affecting epigenetic regulation and associated gene expression. Using targeted mass spectrometry the article by Doll *et al.* (This Issue) reports the quantification of significant histone methylation (Me), acetylation (Ac), and butyrylation (Bu) site occupancy changes related to IDH1 mutation in a cellular model of secondary glioblastoma. For more details, see the article by Sophia Doll *et al.*, pages 39–56.

[S] Online version of this article contains supplemental material. ⌘ Author's Choice

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