

Identifying [genomic markers](#) of response to [immune checkpoint blockade](#) (for example, [PD-1](#) blockade) may benefit [cancer](#) patients by providing predictive [biomarkers](#) for patient stratification and identifying resistance mechanisms for therapeutic targeting. [Gliomas](#) typically have a low [tumor mutational burden](#) (TMB) and a highly immunosuppressive microenvironment—two features associated with immunotherapy resistance. Nevertheless, recent work has suggested that a subset of patients with high-TMB (hypermutated) gliomas might benefit from PD-1 blockade ¹⁾

Koyama S, Akbay EA, Li YY, Herter-Sprie GS, Buczkowski KA, Richards WG, Gandhi L, Redig AJ, Rodig SJ, Asahina H, Jones RE, Kulkarni MM, Kuraguchi M, Palakurthi S, Fecci PE, Johnson BE, Janne PA, Engelman JA, Gangadharan SP, Costa DB, Freeman GJ, Bueno R, Hodi FS, Dranoff G, Wong KK, Hammerman PS. Adaptive resistance to therapeutic [PD-1 blockade](#) is associated with upregulation of alternative immune checkpoints. *Nat Commun.* 2016 Feb 17;7:10501. doi: 10.1038/ncomms10501. PubMed PMID: 26883990; PubMed Central PMCID: PMC4757784.

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Bouffet, E. et al. Immune checkpoint inhibition for hypermutant glioblastoma multiforme resulting from germline biallelic mismatch repair deficiency. *J. Clin. Oncol.* 34, 2206–2211 (2016).

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