Title: Targeted genomic CRISPR-Cas9 screen identifies MAP4K4 as essential for glioblastoma invasion

Authors: Laura M Prolo¹, Amy Li², Scott F Owen³, Jonathon J Parker¹, Kara Foshay⁴, Ryan T Nitta¹, David W Morgens², Sara Bolin¹, Christy M Wilson¹, Johana C M Vega L¹, Emily J Luo¹, Gigi Nwagbo¹, Allen Waziri⁴, Gordon Li¹, Richard J Reimer⁵, Michael C Bassik², Gerald A Grant*¹

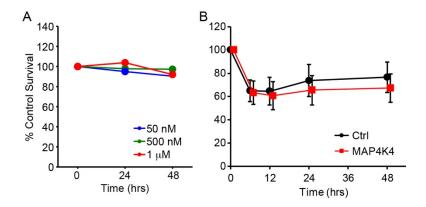
¹Stanford University School of Medicine, Department of Neurosurgery, 300 Pasteur Dr, Stanford, CA 94305

²Stanford University School of Medicine, Department of Genetics, 291 Campus Dr, Stanford, CA 94305

³J. David Gladstone Institute of Neurological Disease, 1650 Owens St, San Francisco, CA 94158

⁴ Inova Neuroscience and Spine Institute, Inova Health Systems, 8110 Gatehouse Rd, Falls Church, VA, 22042

⁵Stanford University School of Medicine, Department of Neurology, 300 Pasteur Dr, Stanford, CA 94305



Supplementary Figure 1: U138 cell viability in the presence of MAP4K4 inhibitor or MAP4K4 knock-out. (A) Quantification of U138 cell viability in presence of PF06260933 dihydrochloride normalized to control over 48 hours. Data displayed as mean with standard error, n=4 for each drug concentration and time point. (B) Quantification of U138 Cas9-control (black) or U138 Cas9-sgMAP4K4 (red) viability normalized to t=0. Data are mean with standard error, n=12 for each time point. There was no significant difference in viability between control and MAP4K4 knock-out lines, p>0.05.

Full size immunoblots are presented below:

