

The authors reply

Levi A. Garraway* || and William R. Sellers*#

*Department of Medical Oncology, Dana-Farber Cancer Institute, Boston, Massachusetts 02115, USA.

Melanoma Program in Medical Oncology, Dana-Farber Cancer Institute, Boston, Massachusetts 02115, USA.

Center for Cancer Genome Discovery, Dana-Farber Cancer Institute, Boston, Massachusetts 02115, USA.

||Department of Medicine, Brigham and Womens Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.

The Broad Institute of Harvard and MIT, Cambridge, Massachusetts 02141, USA.

#Novartis Institutes for Biomedical Research Inc., Cambridge, Massachusetts 02139, USA.

Correspondence to L.A.G. e-mail: levi_garraway@dfci.harvard.edu

Osada and Takahashi raise the intriguing notion that the ASH1 transcription factor might function as a lineage survival oncogene in pulmonary neuroendocrine cells (PNEC). Indeed, *ASH1* seems to have a crucial role in the development of PNECs, has persistent expression in tumours from the PNEC lineage (lung tumours with neuroendocrine features) and might also be required for the survival of such tumours. Therefore, *ASH1* fulfills four of the five criteria we proposed for lineage survival oncogenes in our recent review. As such, we would agree that *ASH1* is a ‘predicted’ lineage survival oncogene. The demonstration that *ASH1* is targeted by genetic alterations in PNEC tumours (or subsets therein) would provide a formal confirmation of its lineage-survival oncogenic function *in vivo*.