CORRIGENDUM

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Corrigendum: Identifying a missing lineage driver in a subset of lung neuroendocrine tumors

Karine Pozo, John D. Minna, and Jane E. Johnson

In the above-mentioned Outlook article, there was an error in Figure 1. INSM1 was mistakenly listed in the panel with the POU2F3^{high} tumors. As is clearly reported in the primary study (Huang et al. 2018), INSM1 is notably absent in the POU2F3^{high} subtype of lung neuroendocrine tumors. The corrected figure is provided below and has been corrected online.

The authors apologize for any confusion this may have caused.

Figure 1. Heterogeneity in SCLC suggests distinct cells of origin, vulnerabilities, and responses to treatment. At least three subtypes of SCLC can be distinguished, including ASCL1^{high}, NEUROD1^{high}, and POU2F3^{high}, with signatures for neuroendocrine gene expression going from high to low, respectively. This heterogeneity may result from genetic inactivation of the tumor suppressors RB1 and TP53 in distinct cells in the lung, as just reported (Huang et al. 2018). Rare neuroendocrine and chemosensory cells are found in the bronchial epithelium in the adult lung that share dependency on ASCL1 (neuroendocrine cells; ASCL1^{high} tumors) or POU2F3 (chemosensory tuft cells; POU2F3^{high} tumors). It is not clear whether the NEUROD1^{high} SCLCs arise from yet a different cell lineage or whether they reflect an ASCL1^{high} tumor that has acquired additional genetic alterations such as MYC amplification. Nevertheless, the distinct chromatin landscape and gene expression seen in these different SCLC subtypes suggest that they will have distinct vulnerabilities and should inform precision medicine strategies (Saunders et al. 2015; Mollaoglu et al. 2017; Huang et al. 2018).

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