Drugs
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CORRECTION

Correction: Targeting KRASG12C in Non-Small-Cell Lung Cancer: Current Standards and Developments

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In this article the legend for Fig. 1 was inadvertently truncated; the Fig. 1 should have appeared as shown below.

Fig. 1 Oncogenic KRAS signaling. The KRAS protein usually acts as the inactive KRAS-GDP. Mitogenic stimulation produces the activaction of GEFs (guanine nucleotide exchange factors) to the plasma membrane and to the binding of KRAS. It generates the destabilization of the nucleotide-binding capacity of KRAS and the transient generation of KRAS nucleotide free (NF). The KRAS protein loads with GTP due to high levels of GTP in the cells, generating a switch to the active KRAS-GTP state. GTPase-activating proteins (GAPs) catalyse hydrolysis of GTP to GDP, so the KRAS signaling finishes. When KRAS is mutated, it is constitutively bound to GPT and GAPs cannot bind, resulting in activation downstream signaling pathways

The original article has been corrected.

The original article can be found online at https://doi.org/10.1007/s40265-024-02030-7.

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