



HHS Public Access

Author manuscript

Cell. Author manuscript; available in PMC 2020 November 14.

Published in final edited form as:

Cell. 2019 November 14; 179(5): 1239. doi:10.1016/j.cell.2019.10.037.

Yap1 Activation Enables Bypass of Oncogenic Kras Addiction in Pancreatic Cancer

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During the assembly of the images for Figure 4C of this article, we inadvertently used different regions from a single delta 60–89 well to represent the GFP and delta 60–89 cells. Since the quantitation shown in the graph in Figure 4C was done independently of the figure assembly, this error had no impact on the data presented in the graph in Figure 4C or on the conclusions presented in the article. In addition, the home institution's Research Integrity office conducted an independent review of one of the authors' (A.K.) raw data and notebooks to ensure the validity of the findings. Here, we replaced the previous GFP image with the corrected image in Figure 4C.

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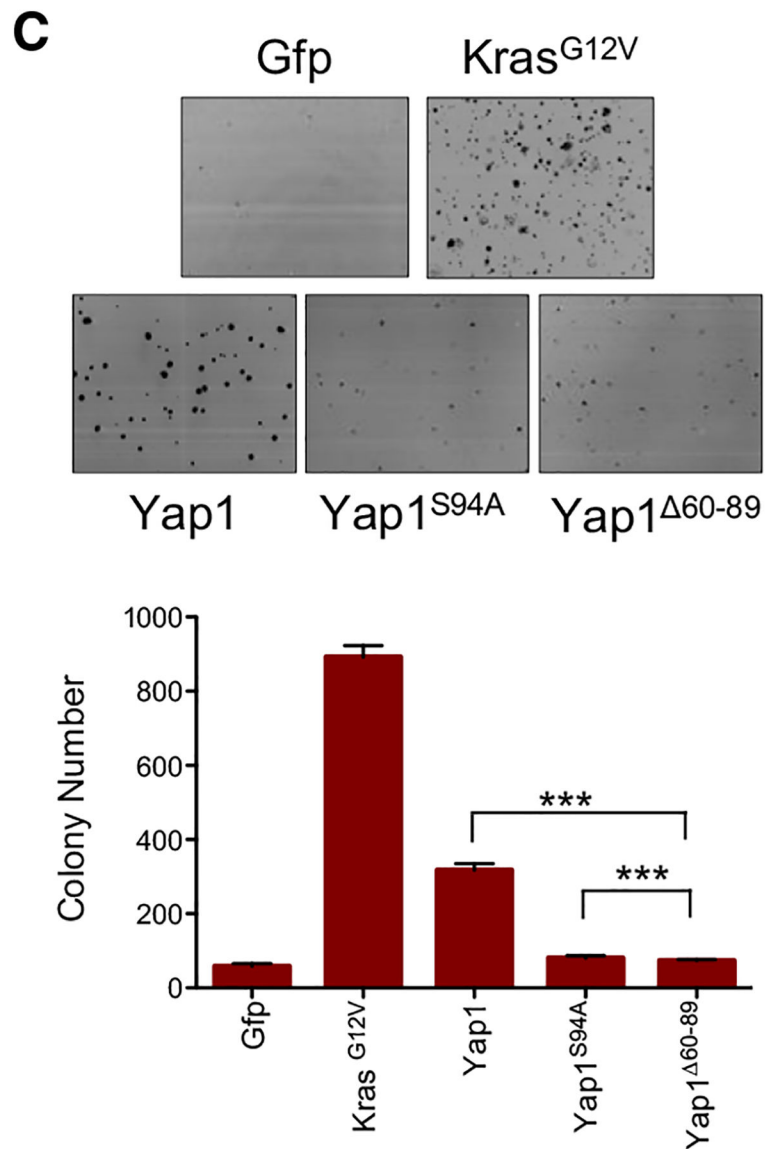


Figure 4. Interaction of Yap1 with Tead2 Is Critical for Its Ability to Bypass Kras^{G12D} Dependence

(C) Sustained expression of wild-type Yap1, but not TEAD-binding-defective Yap1 mutants (Yap^{S94A} and Yap1⁶⁰⁻⁸⁹), can promote anchorage-independent growth of IKras cells off doxy. For each condition, five random fields were counted. Error bars represent SD of the mean. ***p < 0.001.