



Published in final edited form as:

Cell. 2022 August 04; 185(16): 3058. doi:10.1016/j.cell.2022.07.008.

Retraction Notice to: Allosteric Activators of Protein Phosphatase 2A Display Broad Antitumor Activity Mediated by Dephosphorylation of MYBL2

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This article has been retracted at the request of the authors. Their study showed that different PP2A holoenzymes, composed of distinct subunits and acting on unique substrates, can be specifically targeted by different small-molecule allosteric activators, including perphenazine. An important part of the study was the identification of a compound the authors called iHAP1 (improved heterocyclic activator of PP2A) that was suitable for *in vivo* studies in zebrafish and murine models because it did not interfere with dopamine signaling, which caused dose limiting off-target toxicity in the case of perphenazine. The authors also assessed iHAP1 for a second type of off-target activity involving inhibition of tubulin polymerization, and they reported that iHAP1 did not affect tubulin polymerization into microtubules (Figures S6D and S6E).

Recently, Vit and coworkers published an article in *The EMBO Journal* showing that iHAP1 does in fact significantly inhibit tubulin polymerization (Vit et al., 2022, EMBO J. 41, e110611, <https://doi.org/10.15252/embj.2022110611>). The authors have subsequently used the same tubulin polymerization kit that they originally used for determining the effects of small molecules on the rate of tubulin polymerization and carefully optimized the conditions with the control compounds provided with the kit. After recalibration of the plate reader based on these controls, the authors determined that their original results are not reproducible and that iHAP1 does in fact potently inhibit tubulin polymerization. They are uncertain why their original analysis yielded inaccurate results.

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Unfortunately, the fact that iHAP1 inhibits tubulin polymerization renders uninterpretable their *in vivo* studies showing anti-cancer cell activity in zebrafish and murine models, because they cannot tell how much of the activity is due to activation of PP2A and how much is contributed by the anti-tubulin activities of this molecule. Because these *in vivo* studies and other *in vitro* studies in the paper prominently include iHAP1, all of the authors have agreed that the most appropriate course of action is to retract the paper. Many *in vitro* experiments in the paper that address different aspects of PP2A activation were performed with both iHAP1 and perphenazine, which does not possess detectable anti-tubulin activity.

In view of the error involving iHAP1 and anti-tubulin polymerization, the authors are currently repeating each of these experiments in the original article in *Cell*; however, this will take a much longer time. They have decided the most responsible thing is to retract the paper now, so that others are not led to perform uninterpretable experiments with the iHAP1 compound. The authors regret and apologize for this mistake.

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