Correction

Correction: Discovery of a small molecule targeting SET-PP2A interaction to overcome BCR-ABLT315I mutation of chronic myeloid leukemia

Shuzhen Wang[1,*], Weiquan Xie[1,*], Duowei Wang[2,*], Zhigang Peng[1,*], Yan Zheng[2], Nan Liu[1], Wen Dai[1], Yang Wang[1], Zongqiang Wang[1], Yong Yang[2] and Yijun Chen[1]

[1]State Key Laboratory of Natural Medicines and Laboratory of Chemical Biology, China Pharmaceutical University, Nanjing, China

[2]State Key Laboratory of Natural Medicines and Institute of Pharmaceutical Science, China Pharmaceutical University, Nanjing, China

[*]These authors contributed equally to this work

Published:

This article has been corrected: Due to errors during figure assembly, the western blot image for actin in Figure 2B is an accidental duplicate of the "input SET" image in Figure 4A. In addition, the image for SET in the "IP: SET" group and actin in Figure 4A were all linked to the wrong image files. The corrected Figure 2B and Figure 4A, obtained using the original data, appear below. The authors declare that these corrections do not change the results or conclusions of this paper.

Original article: Oncotarget. Oncotarget. 2015; 6:12128–12140. https://doi.org/10.18632/oncotarget.3665

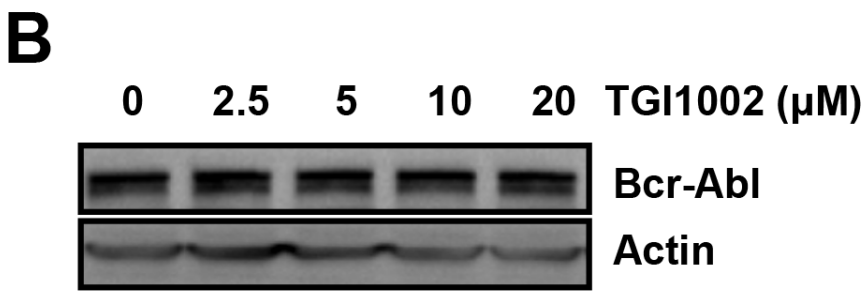

Figure 2: Identification of cellular targets of TGI1002. (**A**) SDS-PAGE for serial affinity chromatography by silver staining. Up panel: blank matrices; down panel: TGI1002-affinity probe. Lane M, protein molecular weight markers. Lane 1-3, Resin-bound proteins from the first, second and third series. Specifically bound proteins are indicated as BP1-BP4. (**B**) Effects of TGI1002 on the expression of BCR-ABL. (**C**) Effects of TGI1002 on microtubules dynamics in K562 cells. Tubulin(S), depolymerized tubulin; Tubulin (P), polymerized tubulin. (**D**) SET protein levels in K562 cells (control) and K562 cells expressing negative control shRNA or SET shRNA. (**E**) TGI1002 sensitivity in K562 cells (control) and K562 cells expressing negative control shRNA or SET shRNA. Data are presented as mean ± s.d. (n = 3).

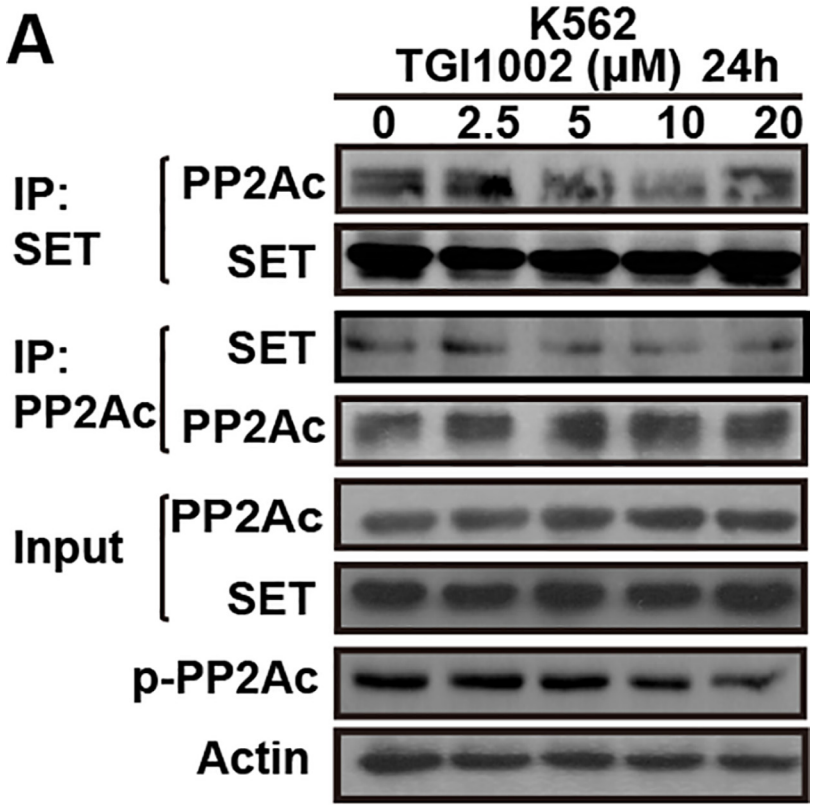

Figure 4: TGI1002 disrupts SET-PP2Ac association and activates PP2A *in vitro*. (**A**) TGI1002 inhibited SET-PP2Ac interaction in K562 cells. K562 cells were incubated with TGI1002 for 24 h. SET and PP2Ac protein complexes were immunoprecipitated and analyzed for SET and PP2Ac co-immunoprecipitation. TGI1002 showed inhibition of SET-PP2Ac complex formation. (**B**) TGI1002 treatment activated PP2A in K562 cells. K562 cells were treated with TGI1002 and the activity of PP2A was measured using an immunoprecipitation phosphatase assay. PP2A activity was significantly increased compared to untreated control. (**C**) TGI1002 inhibited SET-PP2Ac interaction in murine BaF3-p210T315I cells. SET and PP2Ac protein complexes were immunoprecipitated and analyzed for SET and PP2Ac co-immunoprecipitation. TGI1002 showed inhibition of SET-PP2Ac complex formation. (**D**) TGI1002 treatment activated PP2A in murine BaF3-p210T315I cells. Data in (B) and (D) are presented as mean ± s.d. (n = 3). *, $P < 0.05$ and **, $P < 0.01$ compared to untreated; #, $P < 0.05$ and ##, $P < 0.01$ compared to OA treatment.