

Review of: "Increased Protein and Transcript Expression Levels of Lysine-Specific Demethylase 1 (LSD1) Signify Worse Prognosis in Triple-Negative Breast Cancer"

Xiaoshuang Wang¹

1 New York University

Potential competing interests: No potential competing interests to declare.

The authors aim at the recruitment of LSD1 expression as a prognosis marker for triple-negative breast cancer. By analyzing the samples from 389 TNBC cases, which is a reliable resource for clinical investigation, the author provided evidence showing the high expression of LSD1 in TCBA cases and how it is related to survival; then the authors identified several differentially expressed genes from LSD1 high-low expression patients groups, and showing their prognosis prediction potential by the KM analysis. Overlap differentially expressed genes from LSD1 high-low expression TNBCs and KDM1A high-low expression TNBCs were found to be related to cell cycle and DNA replication, and two of the top ten cell cycle-associated hub genes, CDC6 and PLK1, were noted significantly poorer TNBC survival rates.

It is meaningful to identify more prognosis predictors for cancer therapy. However, for this article, some points still need to be included in the draft for an ideal publication.

- 1. Fig1A: Are the 400x images apart from the 200x ones? If so, the regions should be marked in the images.
- 2. The author shared space to display the difference for LSD1 expression in different age groups, histological grades, lymph node statuses, patient races, tumor sizes, etc. (Fig 2), as well as for the KDM1A (Fig 6). However, the meaning inside or the further detailed analysis based on the hints from those results were not included in the manuscript.
- 3. While 1 different expression gene was found to overlap between all 3 cohorts in KDM1A high-low TNBCs, CDC6 and PLK1 were noted to have significantly poorer TNBC survival rates from the top 10 high-expressed cell cycle-associated genes in KDM1A-expressing TNBCs—however, those three critical like genes didn't been involved into the clinical samples analysis. For example, KDM1A, CDC6, and PLK1 can be detected by IHC in TNBC samples.
- 4. A proposed schematic diagram of the KDM1A-mediated oncogenic shift in TNBCs was drawn in Figure 10. However, there is no direct evidence in the paper showing that CDC6 and PLK1 can be regulated by KDM1A. As shown in the diagram, phosphorylation PLK1 is the functional form of a tumor suppressor; unfortunately, the author should also confirm that with the clinical samples to draw this conclusion.
- 5. There are apparent inconsistencies in some fonts in the manuscript, such as the third paragraph on page 6.

