

CORRECTION

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Correction: Tobacco smoke induced hepatic cancer stem cell-like properties through IL-33/p38 pathway

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Correction: *J Exp Clin Cancer Res* 38, 39 (2019)
<https://doi.org/10.1186/s13046-019-1052-z>

Following publication of the original article [1], an overlapping of images was identified Fig. 1e and Fig. 5b.

The images of Oct4 in TS and TS+DMSO groups were replaced and the correct Fig. 5 is given as below:

The correction does not affect the overall results or conclusion of the article.

Reference

1. Xie C, Zhu J, Wang X, et al. Tobacco smoke induced hepatic cancer stem cell-like properties through IL-33/p38 pathway. *J Exp Clin Cancer Res*. 2019;38:39. <https://doi.org/10.1186/s13046-019-1052-z>.

Published online: 06 June 2023

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The original article can be found online at <https://doi.org/10.1186/s13046-019-1052-z>.

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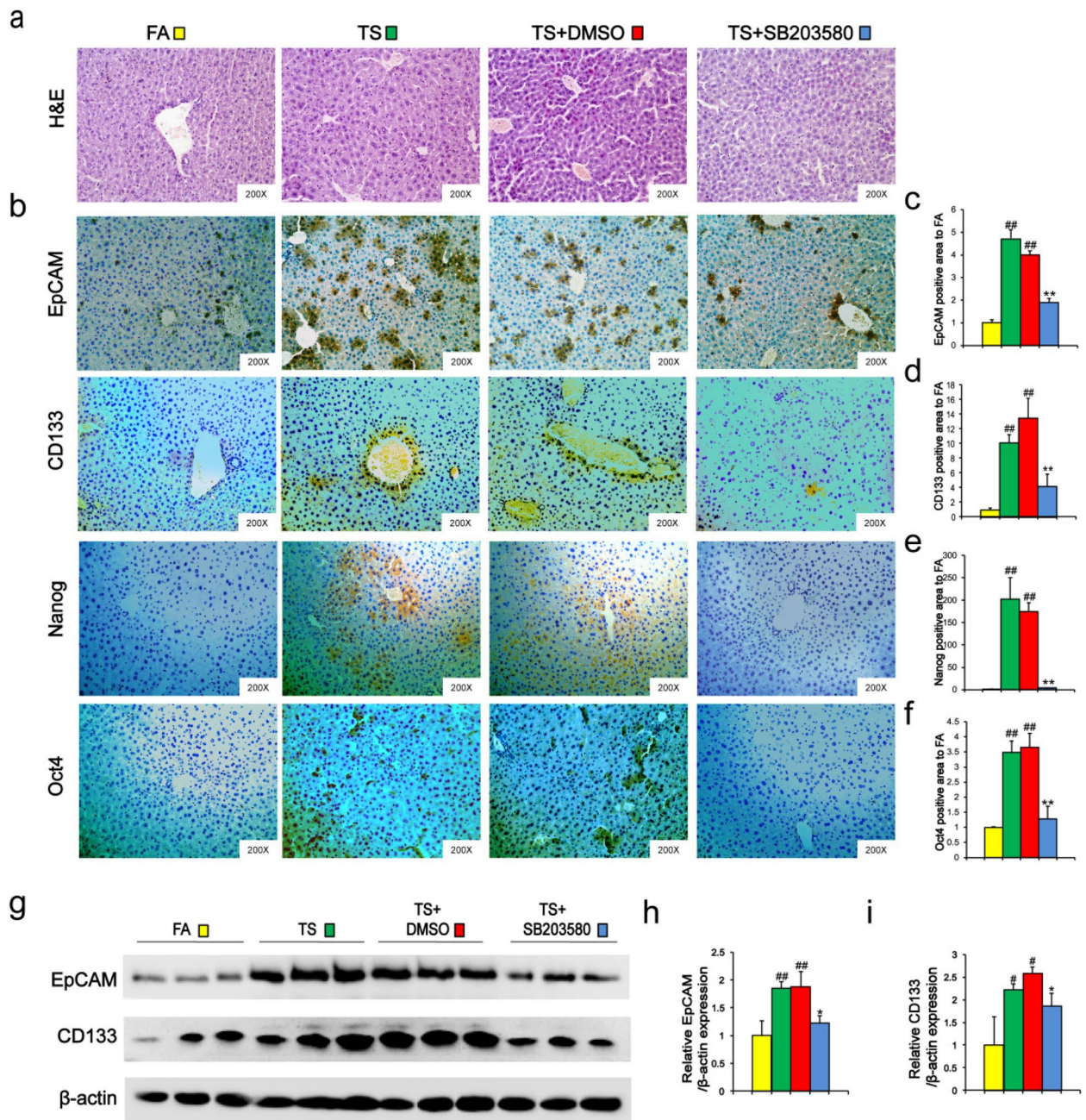


Fig. 5 p38 suppression reversed long term TS exposure-triggered CSC-like properties. Mice exposed to TS were treated with or without p38 MAPK inhibitor (SB 203580) for 12 weeks, and representative micrographs of liver tissue were stained with H&E (a). b Immunohistochemical staining for EpCAM, CD133 and Nanog, Oct4 in liver tissues. c-f Fold changes of EpCAM (c), CD133 (d), Nanog (e) and Oct4 (f)—positive area in TS group compared with FA group. g Western blotting of EpCAM and CD133 in liver tissues. β -actin was served as the loading control. h-i The indicated proteins relative to β -actin were assessed by densitometric analysis; six animal samples per group were used for the densitometric analysis. Data are expressed as mean \pm SD. The significance was assessed with one-way ANOVA test. # $P < 0.05$, ## $P < 0.01$, compared with FA control; * $P < 0.05$, ** $P < 0.01$, compared with TS + DMSO group. FA = filtered air; TS = tobacco smoke