

CORRIGENDUM

Pleiotropic regulation of macrophage polarization and tumorigenesis by formyl peptide receptor-2

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Correction to: *Oncogene* (2011) 30, 3887–3899; doi:10.1038/onc.2011.112; published online 18 April 2011

The original version of this article contained some errors and mistakes. The corrections are below:

1. In the last paragraph of ‘Effects of FPR2 agonists on macrophage polarization’ in the ‘Results’ section: After the sentence ‘To further confirm these results, we then compared the mRNA expression levels of these phenotypes in tumor-infiltrating macrophages (TIMs) from *in situ* tumor tissue using real-time PCR (Supplementary Figure S2)’ add one sentence ‘As CXCL8 is not expressed in mouse, we detected another TAM phenotype TGF- β 1 instead.’ and the related reference ‘Ma YY, He XJ, Wang HJ, Xia YJ, Wang SL, Ye ZY, Tao HQ *et al.* (2011). Interaction of coagulation factors and tumor-associated macrophages mediates migration and invasion of gastric cancer. *Cancer Sci* 102: 336–342.’

2. The two sentences of the above same paragraph: ‘Consistent with the *in vitro* study, the macrophages in the H22 group showed an M2d-like profile (high levels of IL-10, IL-23p19, IL-6, IL-1 β , TNF α , CCL17, CCL1 and CXCL8; low expressions of IL-12p35 and CXCL13)’ and ‘IL-23p19, IL-6, IL-1 β , TNF α , CCL1 and CXCL8 were inhibited by ANXA1’
Replace ‘CXCL8’ with ‘TGF- β 1’.

3. In the last sentence of ‘STAT3 phosphorylation is responsible for FPR2-mediated M2 subset differentiation’ in the ‘Results’ section: ‘however, it was not M-CSF + involved in the FPR2 agonist SAA-mediated CCL1 secretion.’
Delete ‘M-CSF +’.

4. In Figure 6b, ‘CXCL18’ should be replaced by ‘CXCL8’. The modified Figure 6 is shown below.

5. In the first sentence of the fourth paragraph in ‘Discussion’ section: ‘In many solid tumors, overexpression of M-CSF and MCP correlates’.
‘MCP’ should be replaced with ‘MCP-1’.

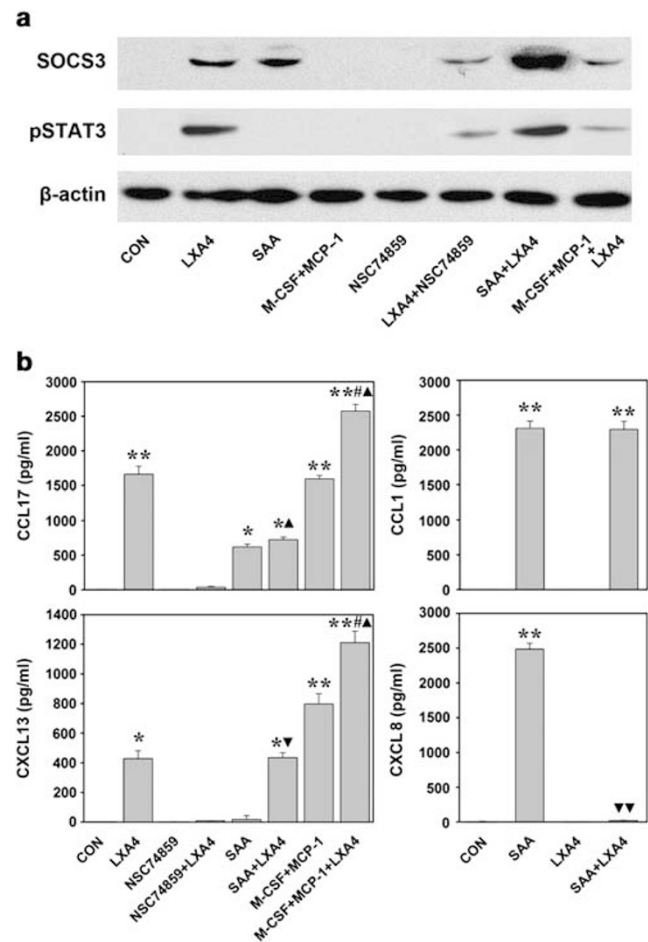


Figure 6 STAT3 signaling is involved in FPR2-mediated macrophage polarization. U937 cells were treated with LXA₄ (100 nM), SAA (100 nM), recombinant human M-CSF (10 ng/ml) + MCP-1 (10 ng/ml) and NSC74859 (100 μ M) for 72 h. (a) The expressions of SOCS3 and pSTAT3 were analyzed by western blotting. (b) The secretions of CCL17, CCL1, CXCL13 and CXCL8 were detected by ELISA. * P <0.05, ** P <0.01 versus control; \blacktriangle P <0.05 versus the LXA₄ group; \blacktriangledown P <0.05, $\blacktriangledown\blacktriangledown$ P <0.01 versus the SAA group; # P <0.01 versus the M-CSF + MCP-1 group. CCL, chemokine (C-C motif) ligand; CXCL, chemokine (C-X-C motif) ligand; FPR2, formyl peptide receptor-2; LXA₄, lipoxin-A₄; MCP-1, monocyte chemoattractant protein-1; M-CSF, macrophage colony-stimulating factor; SAA, serum amyloid-A; SOCS3, cytokine signaling-3; STAT, signal transducer and activator of transcription; pSTAT, phosphorylated STAT.

6. In the third sentence of the last paragraph in the 'Discussion' section:

'However, before that, important questions such as a more molecular basis'

Replace 'a more' with 'the exact'.

7. Use 'TGF- β 1' primers in 'Supplementary table' instead of 'CXCL8'

Forward: 5'-GTGTGGAGCAACATGTGGAACCTA-3'

Reverse: 5'-TTGGTTCAGCCACTGCCGTA-3'

8. In Figure S2C, change 'CXCL8' to 'TGF- β 1'. We were previously confused and used the human data of CXCL8. We apologize for that. Mouse does not have CXCL8. We have now provided the modified Figure S2.

RETRACTION

Requirement for chromatin-remodeling complex in novel tumor suppressor HIC1-mediated transcriptional repression and growth control

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Retraction to: *Oncogene* (2009) **28**, 651–661; doi:10.1038/onc.2008.419; published online 17 November 2008

The authors wish to retract this article after examination of the publication raised concerns that the primary

real-time PCR data, which underlie several of the figures in the article, may not be accurate. The integrity of all of the data presented therefore cannot be assured.