frontiers in Immunology

CORRECTION
published: 13 February 2020
doi: 10.3389/fimmu.2019.03153

Corrigendum: Critical Role of Alternative M2 Skewing in miR-155 Deletion-Mediated Protection of Colitis

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Edited and reviewed by:
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Specialty section:
This article was submitted to Microbial Immunology, a section of the journal Frontiers in Immunology

Received: 28 November 2019
Accepted: 30 December 2019
Published: 13 February 2020

Citation:
Li J, Zhang J, Guo H, Yang S, Fan W, Ye N, Tian Z, Yu T, Ai G, Shen Z, He H, Yan P, Lin H, Luo X, Li H and Wu Y (2020) Corrigendum: Critical Role of Alternative M2 Skewing in miR-155 Deletion-Mediated Protection of Colitis. Front. Immunol. 10:3153. doi: 10.3389/fimmu.2019.03153

Keywords: M2 macrophages, miR-155, colitis, C/EBPβ, SOCS1

A Corrigendum on

Critical Role of Alternative M2 Skewing in miR-155 Deletion-Mediated Protection of Colitis
by Li, J., Zhang, J., Guo, H., Yang, S., Fan, W., Ye, N., et al. (2018). Front. Immunol. 9:904. doi: 10.3389/fimmu.2018.00904

In the original article, there were mistakes in **Figure 1J** and **Figure 6E** as published. **Figure 1J** (representative FACS plot of $CD4^+IFN\text{-}\gamma^+$ cells) was mistakenly duplicated from **Figure 1I** (representative FACS plot of $CD4^+IL\text{-}17^+$ cells), and the tubulin band of **Figure 6E** was inadvertently covered by the band of that in **Figure 6C**. The corrected **Figure 1** and **Figure 6** appear below.

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

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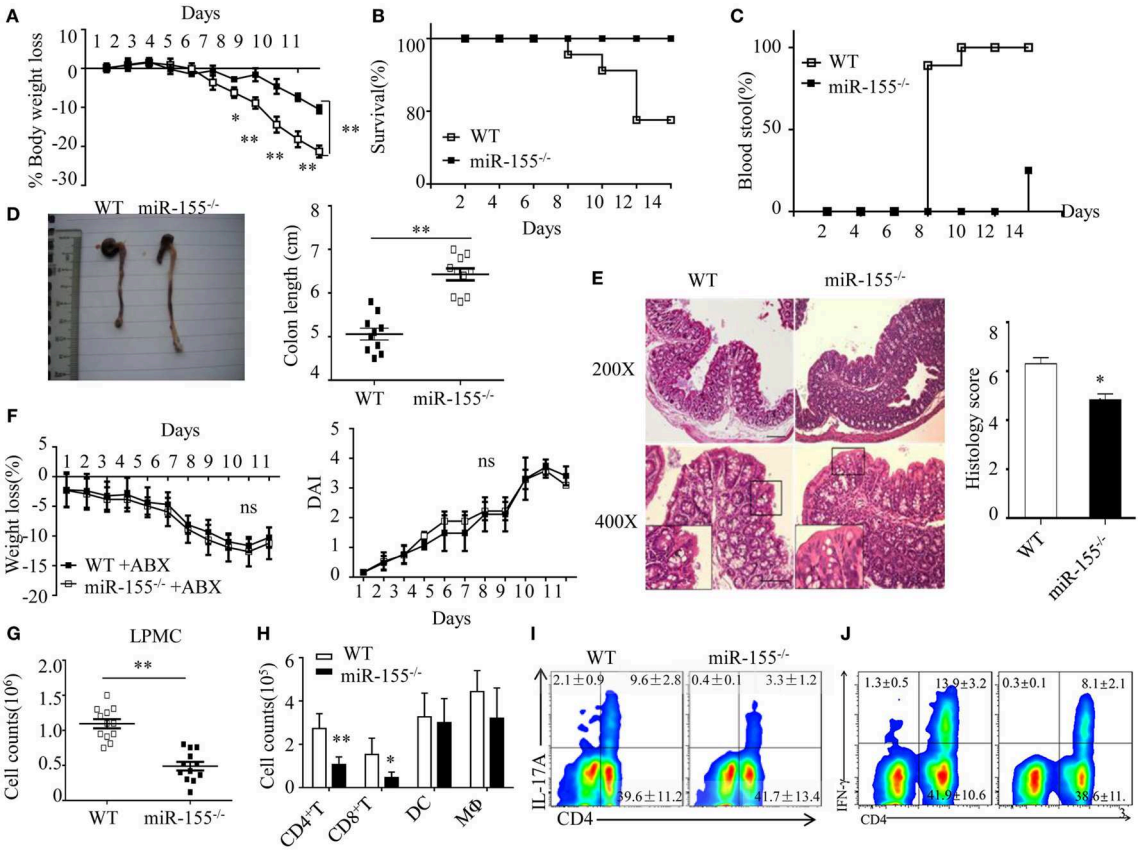

FIGURE 1 | Attenuated dextran sulfate sodium (DSS)-induced colitis in miR-155$^{-/-}$ mice is dependent on commensal bacteria. WT or miR-155$^{-/-}$ mice were given 3% DSS in drinking water for 5 days, followed by regular drinking water for 6 days. **(A–E)** Weight change **(A)**, Kaplan–Meier plot of survival rate **(B)**, stool blood **(C)**, representative gross colon appearance [**(D)** left] and colon length [**(D)** right], and representative H&E-stained colon cross-sections [**(E)** left, original magnification, 200× or 400×] and semi-quantitative histopathology score [**(E)** right]. **(F)** WT and miR-155$^{-/-}$ mice were treated with broad-spectrum antibiotics cocktail (ABX) for 4 weeks and then given 3% DSS, the body weight change (left) and DAI (right) were monitored daily. Ns vs WT control **(G,H)**. The LPMCs were isolated from colon tissues of DSS-treated WT ($n = 12$) and miR-155$^{-/-}$ ($n = 15$) mice, then the total number of LPMCs (CD45$^+$) **(G)**, T cells (CD4$^+$ and CD8$^+$), DCs (CD11c$^+$CD11b$^-$) and macrophages (CD11b$^+$CD11c$^{-/low}$) **(H)** were counted by flow cytometry. **(I,J)** Representative FACS showing CD4$^+$IL-17$^+$ cells **(I)** and CD4$^+$IFN-γ$^+$ cells **(J)** in isolated LPMCs of DSS-treated WT ($n = 12$) and miR-155$^{-/-}$ ($n = 15$) mice. $^*P < 0.05$, $^{**}P < 0.01$ vs WT control [Student's *t*-test in **(A,D,E,G,H)** and Kaplan–Meier analysis in **(B,C)**]. ns vs WT control. Data are representative of three independent experiments (mean and SD in A-D); n = 12–15 mice per group in **(A–F)** and n = 5–6 mice per group in **(G)**. ns, not significant. WT, wild-type.

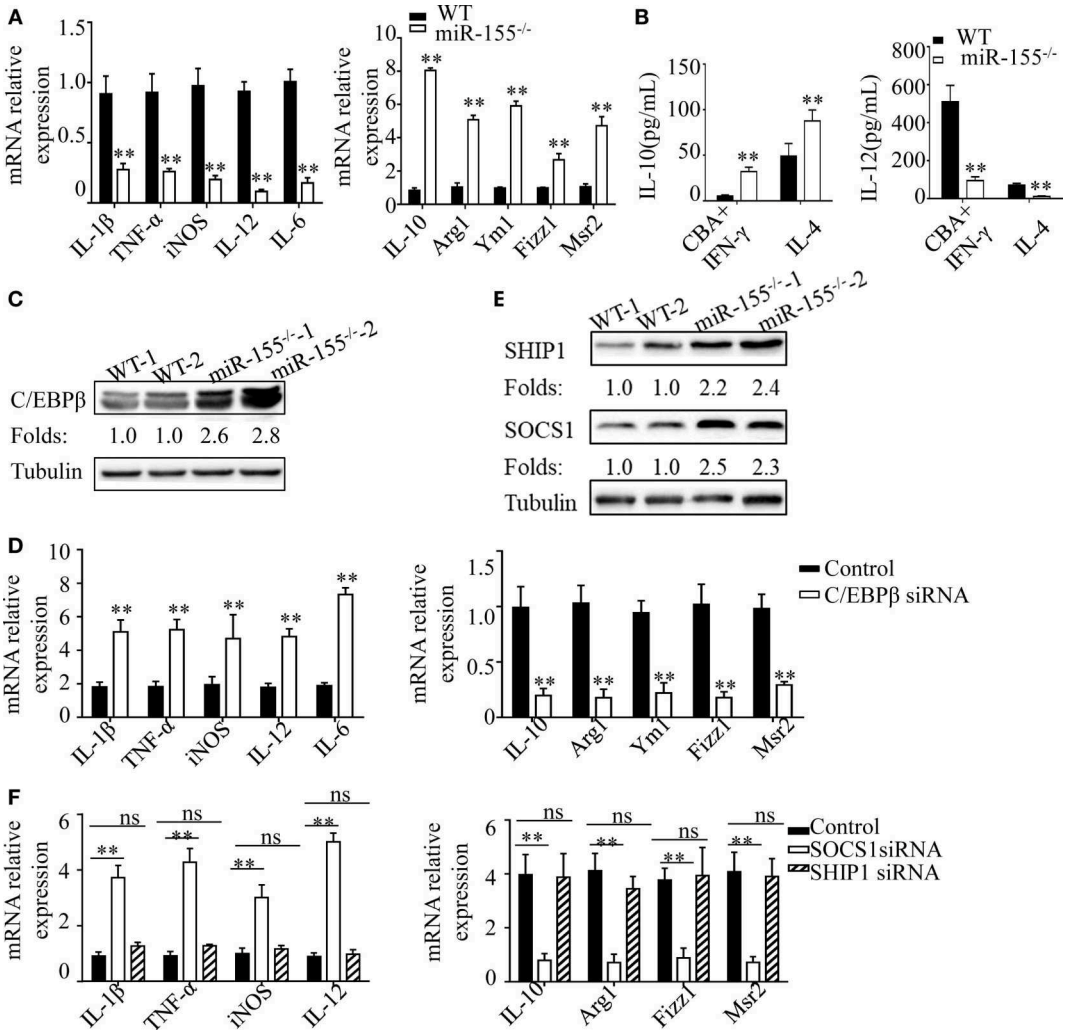

FIGURE 6 | C/EBPβ and SOCS1 are key functional targets in intestinal M2 polarization. **(A)** BMDMs isolated from WT and miR-155$^{-/-}$ mice were treated with CBA (10 μg/mL) and IFN-γ (20 ng/mL), and the relative expression of M1genes and M2 genes were determined by Q-PCR. **(B)** The absolute amounts of secreted cytokines IL-10 and IL-12 (as representative of M2 and M1 gene products, respectively) in the supernatants of WT or miR-155$^{-/-}$ BMDMs that had been treated with M1 condition (CBA + IFN-γ) and M2 condition (IL-4) were measured by ELISA. **(C)** The protein expression level of C/EBPβ in macrophages (CD11b^{+}CD11c$^{-/low}$) isolated from LPMCs of dextran sulfate sodium colitis mice were determined by western blotting. **(D)** miR-155$^{-/-}$ BMDMs were transferred with C/EBPβ siRNA or control and then stimulated with CBA (10 μg/mL) and IFN-γ (20 ng/mL), and the relative expression of M1genes and M2 genes were determined by Q-PCR. **(E)** The protein expression level of SOCS1 and SHIP1 in macrophages, as described in **(C)**, was determined by western blotting. **(F)** miR-155$^{-/-}$ BMDMs were transferred with SOCS1 and SHIP1 siRNA and treated as described in **(D)**, and the relative expressions of M1genes and M2 genes were determined by Q-PCR. $^{*}P < 0.05$, $^{**}P < 0.01$ vs WT control or siRNA control [Student's *t*-test in **(A,B,D)**]. $^{*}P < 0.05$, ns > 0.05 vs. siRNA control (ANOVA with Bonferroni's posttest correction for multiple comparisons in **(F)**. Data are representative of three independent experiments (mean and SD). Ns, not significant. BMDMs, bone marrow-derived macrophage. WT, wild-type. CBA, cecal bacterial antigen.