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CORRECTION

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Correction: "Mouse mammary tumor virus is implicated in severity of colitis and dysbiosis in the IL-10-/- mouse model of inflammatory bowel disease"

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In the article titled "**Mouse mammary tumor virus is implicated in severity of colitis and dysbiosis in the IL-10$^{-/-}$ mouse model of inflammatory bowel disease**" [1], the results section subheading "**Characterization of MMTV infection in IL-10$^{-/-}$ mice",** paragraph 4 incorrectly assigned the T-cell receptor gene and protein names. The original paragraph is as follows:

"Active MMTV infection in mice can be demonstrated by evaluating superantigen activity and observing differences in the cognate TCR-Vβ subsets that react with the specific vSAG [14]. The IL-10$^{-/-}$ model has a mixed genetic background of several mouse strains including the C57BL/6 and sub strains of 129, each with different and partially characterized endogenous *mtv* loci [30]. For example, the C57BL mouse has three full length endogenous genomes, *mtv-8, mtv-9* and *mtv-17*, and sub strains of 129 mice contain combinations of *mtv-1, mtv-3, mtv-8, mtv-9, mtv-11, mtv-13,* and *mtv-17* [14-16]. Eleven of 12 clones were derived from *mtv-9 sag* encoding the vSAG9 (**Figure 2A**) that preferentially binds and expands TCR-Vβ5, TCR-Vβ11, and TCR-Vβ12 lymphocytes [14]. We then evaluated the TCR-Vβ subset distribution in the spleen and colon (**Supplemental Fig. 2**) and found that TCR-Vβ5 and TCR-Vβ12 were expressed in sufficient quantity for analysis, but TCR-Vβ11 constituted less than 0.5% of the population. Significant differences were observed in the colon that were not observed in the spleen (**Supplemental Fig. 2**). Consistent with vSAG9-induced activity, the percentage of TCR-Vβ12 was significantly increased (17.14 vs. 6.45, q = 0.012), and a trend was observed for increased TCR-Vβ5 (IL-10−/− vs. SvEv, 0.63% vs. 0.21%, q = 0.067) in the IL-10$^{-/-}$ versus the SvEv WT colon (**Fig. 2B**)."

†Heather Armstrong and Mandana Rahbari shared co-first authorship.

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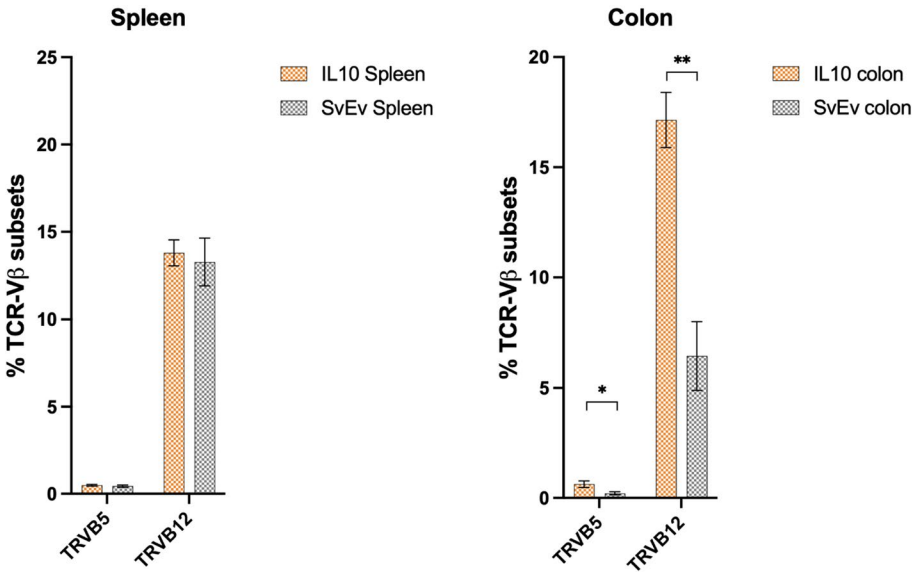
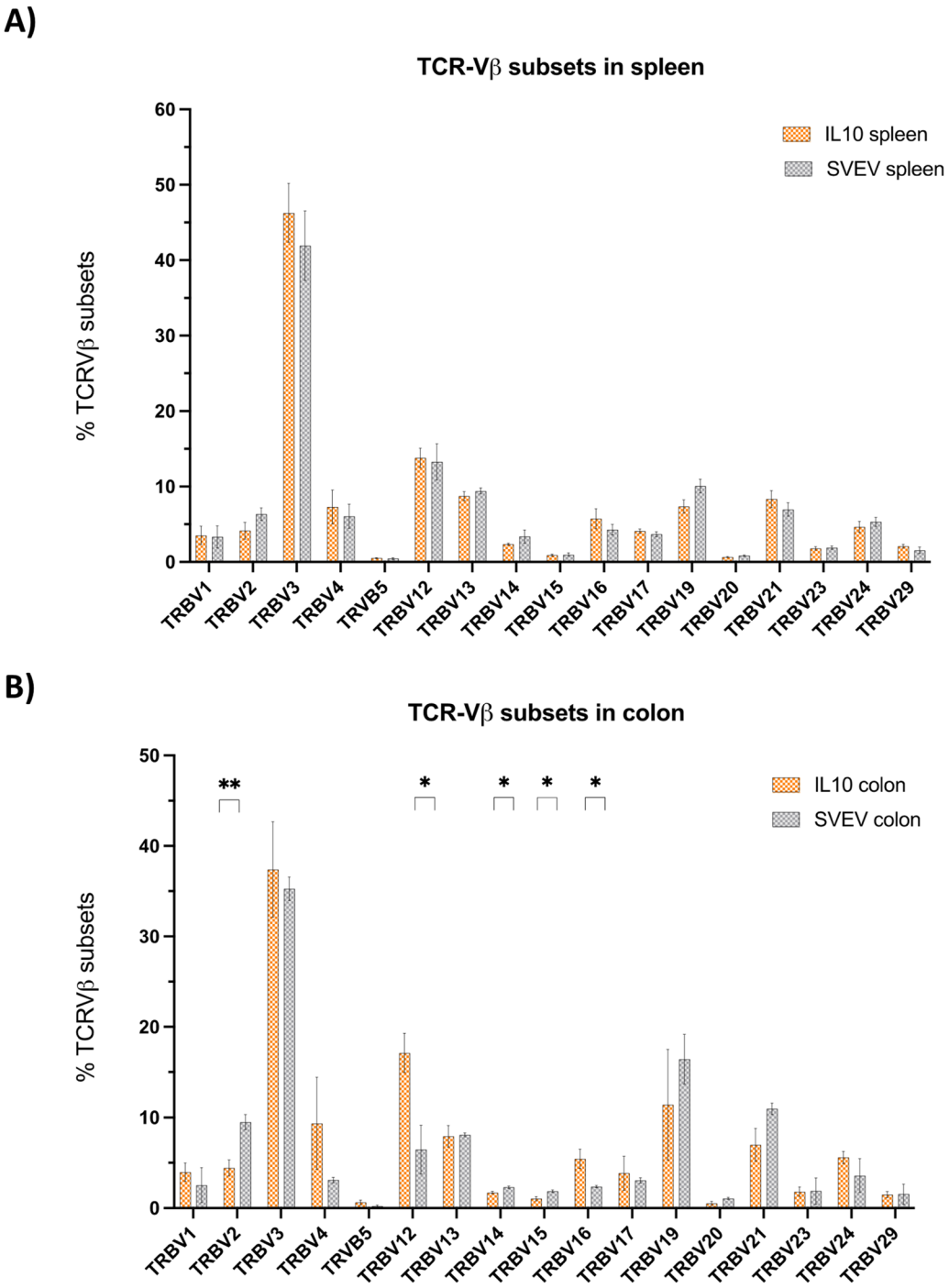


Legend Figure 2B: TCR-Vβ5 and TCR-Vβ12 subset distribution of read count assessed by Illumina sequencing in spleen and colon showed no significant differences between IL-10$^{-/-}$ vs. SvEv in the spleen, whereas IL-10$^{-/-}$ colon had more than twofold % increase in TCR-Vβ5 and TCR-Vβ12 (mean ± SEM, **p = 0.006, multiple unpaired t-test, Benjamini, Kreiger, and Yekutieli two-stage setup)

Supplementary Figure 2

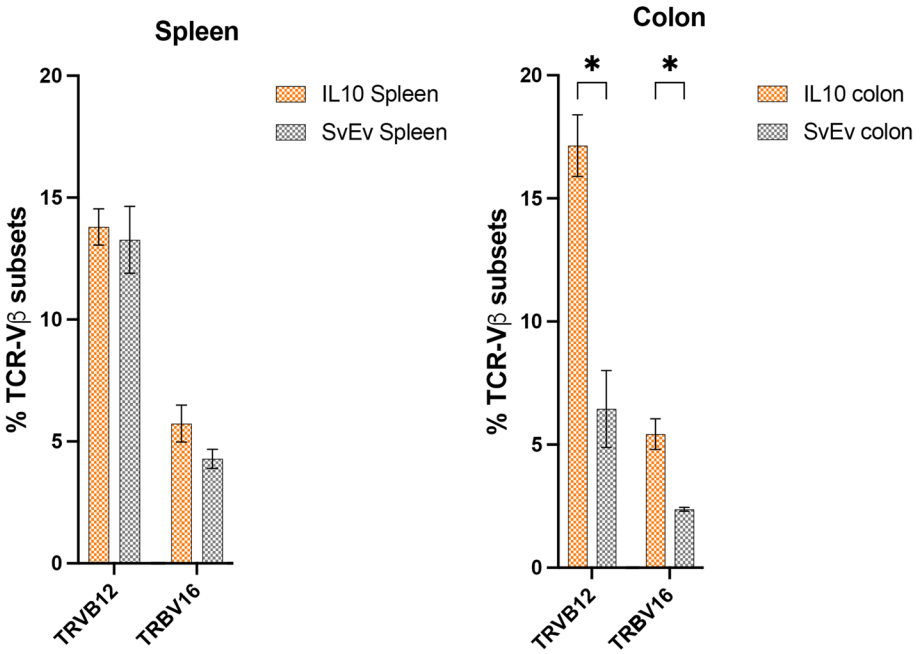


Supplementary Figure 2 legend

T cell receptor (TCR)-Vβ subset distribution in spleen and colon of IL-10-/- vs. SvEv mice assessed by Illumina sequencing. (A) No significant differences were observed between IL-10-/- vs SvEv in the spleen. (B) the IL-10-/- colon had increased TCR-Vβ12 and TCR-Vβ16 subsets with diminished TCR-Vβ2, TCR-Vβ14 and TCR-Vβ15 expression. [Mean ± SEM, TCR-Vβ subsets with percent less < 0.5% removed from the analyses. * $p < 0.01$, ** $p = 0.002$, Multiple unpaired t-test, Benjamini, Kreiger, and Yekutieli two stage set up, q value < 0.1].

The correct nomenclature is incorporated into the new paragraph and shown in the revised figure Fig. 2B:

"Active MMTV infection in mice can be demonstrated by evaluating superantigen activity and observing differences in the cognate TCR-Vβ subsets that react with the specific vSAG [14]. The IL-10$^{-/-}$ model has a mixed genetic background of several mouse strains including the C57BL/6 and sub strains of 129, each with different and partially characterized endogenous *mtv* loci [30]. For example, the C57BL mouse has three full length endogenous genomes, *mtv-8, mtv-9* and *mtv-17*, and sub strains of 129 mice contain combinations of *mtv-1, mtv-3, mtv-8, mtv-9, mtv-11, mtv-13,* and *mtv-17* [14-16]. Eleven of 12 clones were derived from *mtv-9 sag* encoding the vSAG9 (**Figure 2A**) that preferentially binds and predominantly expands lymphocytes subsets expressing TCR-Vβ5 and TCR-Vβ11 [14]. We then evaluated the *TRVB* gene distribution in the SvEv WT and IL-10$^{-/-}$ mice in the spleen and colon. We found that the corresponding *TRVB* genes were differentially expressed in sufficient quantity for analysis with significant differences in the colon that were not observed in the spleen (**Supplemental Fig. 2**). Consistent with vSAG9-induced superantigen stimulation, the percentage of *TRVB12* encoding TCR-Vβ5 (17.14 vs. 6.45, $p < 0.01$) and *TRVB16* encoding TCR-Vβ11 (5.43 vs. 2.37, $p < 0.01$) were significantly increased in the colon of IL-10$^{-/-}$ vs. SvEv mice."



Revised Figure 2B: Distribution of *TRVB12* and *TRVB16* gene read counts assessed by Illumina sequencing in spleen and colon. No significant differences were observed in TRBV gene expression between IL-10$^{-/-}$ vs. SvEv in the spleen, whereas in the colon, the IL-10$^{-/-}$ mice demonstrated more than twofold % increase in *TRVB12* and *TRVB16* genes encoding TCR-Vβ5 and TCR-Vβ11, respectively (*$p < 0.01$, multiple unpaired t-test, Benjamini, Kreiger, and Yekutieli two-stage setup)

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Reference

1. Armstrong H, Rahbari M, Park H, Sharon D, Thiesen A, Hotte N, Sun N, Syed H, Abofayed H, Wang W, Madsen K, Wine E, Mason A. Mouse mammary tumor virus is implicated in severity of colitis and dysbiosis in the IL-10-/- mouse model of inflammatory bowel disease. Microbiome. 2023;11(1):39. https://doi.org/10.1186/s40168-023-01483-4.