Mycobacterium tuberculosis-induced CXCR4 and chemokine expression leads to preferential X4 HIV-1 replication in human macrophages


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http://www.jimmunol.org/content/177/12/8874
CORRECTIONS


In the Abstract, in the seventh sentence, there are errors in punctuation. The corrected sentence is shown below.

We found that *M. tuberculosis* infection of macrophages in vitro increased viral entry and RT of CXCR4-using HIV-1, but not of CCR5-using HIV-1.

In Fig. 5B, one of the V3 sequences of the HIV-1 primary isolates (BAL fluid 2 CD14) was a contaminant derived from the positive control virus, the laboratory strain NL4-3. The authors have corrected the GenBank sequence (AY289013) for the BAL fluid 2 CD14 isolate. The corrected Fig. 5, containing this sequence, is shown below.

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### A HIV-1 Source in BAL

![Graph showing percent of anti-CD14 captured virus](image)

### B V3 Sequences of Captured Virus

<table>
<thead>
<tr>
<th>BAL fluid #1</th>
<th>Net Charge</th>
<th>Pos. Charge</th>
<th>No. of Seq</th>
</tr>
</thead>
<tbody>
<tr>
<td>Input: CTRENNTTRK SHHSQGRAF YAT-ETUJGNI RQAVC</td>
<td>+5</td>
<td>+6</td>
<td>1/1</td>
</tr>
<tr>
<td>CD14: CTRENNTTRK SHHSQGRAF YAT-DIUDID RQAVC</td>
<td>+6</td>
<td>+8</td>
<td>9/9</td>
</tr>
<tr>
<td>CD26: CTRENNTTRK SHHSQGRAF YTQQKRDGNI RQAVC</td>
<td>+6</td>
<td>+7</td>
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<table>
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<th>BAL fluid #2</th>
<th>Net Charge</th>
<th>Pos. Charge</th>
<th>No. of Seq</th>
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<tr>
<td>Input: CTRENNTTRK GSHHSQGRAF YTDKIANI RQAVC</td>
<td>+5</td>
<td>+6</td>
<td>1/1</td>
</tr>
<tr>
<td>CD14: CTRENNTTRK GSHHSQGRAF YTDKIANI RQAVC</td>
<td>+4</td>
<td>+6</td>
<td>20/20</td>
</tr>
<tr>
<td>CD26: CTRENNTTRK GSHHSQGRAF YTDKIANI RQAVC</td>
<td>+5</td>
<td>+6</td>
<td>1/1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>BAL fluid #3</th>
<th>Net Charge</th>
<th>Pos. Charge</th>
<th>No. of Seq</th>
</tr>
</thead>
<tbody>
<tr>
<td>Input: CTRENNTTRK SISIGGRAF YTTG-QIDGDI RQAVC</td>
<td>+4</td>
<td>+5</td>
<td>1/1</td>
</tr>
<tr>
<td>CD14: CTRENNTTRK SISIGGRAF YTTG-QIDGDI RQAVC</td>
<td>+6</td>
<td>+8</td>
<td>1/6</td>
</tr>
<tr>
<td>CD26: CTRENNTTRK SISIGGRAF YTTG-QIDGDI RQAVC</td>
<td>+4</td>
<td>+6</td>
<td>5/6</td>
</tr>
<tr>
<td>CD26: N.A.</td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

The flow cytometry panels of Figs. 1B and 4B (CD4<sup>FITC</sup> panels) were mistakenly duplicated. The corrected panels are presented below as dot plots instead of histograms. In the Fig. 1 legend, the penultimate sentence is therefore incorrect and in Materials and Methods, in the fifth sentence under the heading Flow cytometry of gene-modified T lymphocytes, the words “or histograms” should be deleted. The errors do not change the message of either the figures or the paper. The corrected sentence, Fig. 1 and legend, and Fig. 4, are shown below.

The dot plots represent viable T cells gated on forward and sideward light scatter signals.

**FIGURE 1.** The gp100/A2-specific T cells do not require CD8α for binding peptide/MHC I ligand. CD8-positive and -negative fractions of primary human T cells were transduced with TCR genes derived from a gp100/A2-specific CTL-296 and, in case of CD8-negative T cells (i.e., CD4<sup>+</sup> T cells), also a CD8α<sup>+</sup> gene. T cells were subsequently sorted into three populations with the use of PE-labeled gp100/A2 tetramer and FITC-labeled CD8α mAb: 1) CD8<sup>+</sup> T cells expressing the TCRαβ transgenes; 2) CD4<sup>+</sup> T cells expressing the TCRαβ transgenes; and 3) CD4<sup>+</sup> T cells expressing the TCRαβ and CD8α transgenes. These three T cell populations were validated by flow cytometry for the expression of CD4 and CD8 (a) and binding with tetramers (b). See Materials and Methods for details on flow cytometry and sorting of T cells. Dot plot ordinates are set using stainings with a nonrelated HLA-A2-binding tetramer. Data represent one of two donors with similar results.
FIGURE 4.

(a) Ig fold

Extracellular hinge region

p56Lck

SH1 SH2 SH3

CD8α wt

CD8α mutant

(b) No CD8α

CD8α C2157A

CD4 PE

CD4 FITC

gp100/A2 PE

FL-1

(c) p56Lck

IP: CD8α

Blot: Lck

97

68

43

FIGURE 4.

In Results, under the heading Phenotypic B cell markers correspond to mRNA expression, in the second paragraph, sentence four, the designation “B cell line (BCL)6” is incorrect. The corrected sentence is shown below.

As expected, BCL6 was found to be typically linked to GC B cell subsets accompanied by a strong down-regulation of BCL2 (Fig. 4A).

Under the heading CD77− cells share the CD77− cell proliferation program, in sentence four, six, and seven, cyclin D3, E1, E2, A2, B1, B2, p27Kip, p18, and BMI1 should be italicized. The corrected sentences are shown below.

Genes, including cyclin D3 (CCND3), E1 (CCNE1), E2 (CCNE2), A2 (CCNA2), B1 (CCNB1), and B2 (CCNB2), all regulators of the G1-S, S, and G2-M phase transitions, were expressed in both of these subsets. . . . Furthermore, the inhibitors of CDK2, p21Cip (CDKN1A) and p27Kip (CDKN1B), were effectively down-regulated, and among the inhibitors of CDK4 class of proteins (INK4) only p18 (CDKN2C) displayed an increased expression in the GC B cell subsets. The members of the polycomb group of genes, ENX and EED, involved in proliferation (12) were also highly up-regulated in the GC B subsets, whereas BMI1 was equally significantly down-regulated (Fig. 4C) in both subsets (13).

Under the heading The GC genomic integrity and DNA maintenance programs are active in both the CD77− and CD77− population, in sentence five, p53 (TP53) should be italicized. Notable was that p53 (TP53), another target of ATM, displayed a baseline expression pattern across all B cell subsets.

Under the heading Transcriptional regulation of SHM and CSR does not separate CD77− and CD77−, in the first paragraph, sentence five, “MutS homologue 2 (MSH2), MutS homologue 6 (MSH6)” are incorrect; and EXO1 and UNG should be italicized. In the first sentence of the third paragraph, H2AX, XRCC4 DDB2, and XPG should be italicized. The corrected sentences are shown below.

This transcriptional regulation was seen also among components participating in MMR, such as the MSH2, MSH6, and EXO1 (Fig. 6C), as well as for the BER enzyme UNG (Fig. 6D), which is noteworthy considering the specific implication of these particular MMR and BER members in SHM (19).

As for the regulation of the repair pathways implicated in CSR, the nonhomologous end joining members H2AX (H2AFX) and DNA-PKcs (PRKDC) (14) together with XRCC4 demonstrated an activation-induced expression seen in both GC B cell subsets (Fig. 6G), and the only members of the nucleotide excision repair pathway that changed were the DDB2, which increased, and the XPG (ERCC5), which surprisingly decreased in the GC subsets (Fig. 6E).


Two authors’ names were inadvertently omitted from the article. The corrected author and affiliation lines are shown below.

Takashi Kawasaki,* Satoshi Fujimi, † James A. Lederer, † William J. Hubbard,* Mashkoor A. Choudhry,* Martin G. Schwacha,* Kirby I. Bland,* and Irshad H. Chaudry*

*Center for Surgical Research and Department of Surgery, University of Alabama, Birmingham, AL 35294; and †Department of Surgery (Immunology), Brigham and Women’s Hospital and Harvard Medical School, Boston, MA 02115

In Table III, the second column heading was inadvertently duplicated in the fifth column. “MRL/lpr mouse strain (10-wk-old)” is the correct heading. The corrected table is shown below.

<table>
<thead>
<tr>
<th>CD4⁺ T Cell Subsets</th>
<th>Percentages (±SD)</th>
<th>isoAsp Residue (pmol/mg protein ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B10.BR mouse strain (10-wk-old)</td>
<td>MRL/lpr mouse strain (10-wk-old)</td>
</tr>
<tr>
<td>Naive</td>
<td>77.9 ± 3.7%</td>
<td>71.75 ± 2.89</td>
</tr>
<tr>
<td>Activated</td>
<td>3.5 ± 0.1%</td>
<td>ND</td>
</tr>
<tr>
<td>Memory</td>
<td>10.9 ± 0.9%</td>
<td>ND</td>
</tr>
</tbody>
</table>

*a* Cell percentages were obtained via FACS analysis for the naive (CD4⁺CD62Lhigh), activated (CD4⁺CD62Lhigh), and memory (CD4⁺CD62Llow) subsets as shown in Fig. 5. After sorting by the expression profile of CD4 and CD62L, cell lysates from naive, activated, and memory CD4⁺ T cells were measured for intracellular isoAsp content. *n* = 3.


The twelfth author’s last name is misspelled. The correct name is Christine M. Rousseau.


In the first paragraph, fifth sentence of the Introduction and Materials and Methods, and in References, IKKE and IKKe should be IKKε. The corrected sentences and Reference 9 are shown below.

These infectious particles activate several kinases in the host including the recently described IκB kinase (IKK) homologs, IKKe (9), also called IKKi (10), and Tank-binding kinase 1 (TBK1) (11).

Commercial Abs were from the following suppliers: anti-IRF-3 Abs specific for human and rodent species were from Immuno-Biological Laboratories (IBL) and Zymed Laboratories, respectively; anti-IKKε Ab (IMG-270A) (that recognize as well TBK1) was from Imgenex; anti-ubiquitin mAb (clone P4D1) and mAb to MYC were from Santa Cruz Biotechnology; mAbs to hemagglutinin (HA) (clone HA-7) and Flag epitopes and β-actin (clone AC-74) were from Sigma-Aldrich.

In Results, in sentence 16 under the heading A Cullin-based ubiquitin ligase pathway is involved in host cell-mediated IRF-3 degradation following SeV infection, reference to CREB coactivator is incorrect. In sentence ten, under the heading Degradation of IRF-3 is dependent of the TBK1/IKKs-signaling pathway; reference to RNA interference silencing technology is incorrect. The corrected sentences are shown below.

Interestingly, this increase in the stability of the hyperphosphorylated forms of IRF-3 was also associated with a sustained activation of IRF-3 as verified by the presence of dimers or its association to CREB binding protein (CBP) coactivator after infection with SeV (Fig. 3E).

We next directly examined the contribution of the IKK-related kinases in IRF-3 degradation by first using RNA interference (RNAi) technology.


The second author’s last name is misspelled. The correct name is Jobin Eslahpazir.