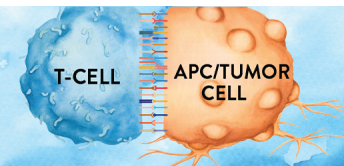


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## The human CD77<sup>-</sup> B cell population represents a heterogeneous subset of cells comprising centroblasts, centrocytes, and plasmablasts, prompting phenotypical revision

C.-M. Högerkorp and C. A. K. Borrebaeck

*J Immunol* 2006; 177:8877; ;

doi: 10.4049/jimmunol.177.12.8877

<http://www.jimmunol.org/content/177/12/8877.1>

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*The Journal of Immunology* is published twice each month by  
The American Association of Immunologists, Inc.,  
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Print ISSN: 0022-1767 Online ISSN: 1550-6606.



Högerkorp, C.-M., and C. A. K. Borrebaeck. 2006. The human CD77<sup>-</sup> B cell population represents a heterogeneous subset of cells comprising centroblasts, centrocytes, and plasmablasts, prompting phenotypical revision. *J. Immunol.* 177: 4341–4349.

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<sup>-</sup> cells share the CD77<sup>-</sup> cell proliferation program*, in sentence four, six, and seven, *cyclin D3*, *E1*, *E2*, *A2*, *B1*, *B2*, *p27<sup>Kip</sup>*, *p18*, and *BM11* 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 G<sub>1</sub>-S, S, and G<sub>2</sub>-M phase transitions, were expressed in both of these subsets. . . . Furthermore, the inhibitors of CDK2, *p21<sup>Cip</sup>* (*CDKN1A*) and *p27<sup>Kip</sup>* (*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 *BM11* 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<sup>-</sup> and CD77<sup>-</sup> 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<sup>-</sup> and CD77<sup>-</sup>*, 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).

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Kawasaki, T., W. J. Hubbard, M. A. Choudhry, M. G. Schwacha, K. I. Bland, and I. H. Chaudry. 2006. Trauma-hemorrhage induces depressed splenic dendritic cell functions in mice. *J. Immunol.* 177: 4514–4520.

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

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