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The human CD77⁻ B cell population represents a heterogeneous subset of cells comprising centroblasts, centrocytes, and plasmablasts, prompting phenotypical revision

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Högerkorp, C.-M., and C. A. K. Borrebaeck. 2006. The human CD77$^-$ 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. 4*A*).

Under the heading *CD77$^-$ cells share the CD77$^-$ cell proliferation program*, in sentence four, six, and seven, *cyclin D3, E1, E2, A2, B1, B2, p27^{Kip}, 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 G_1-S, S, and G_2-M phase transitions, were expressed in both of these subsets. . . . Furthermore, the inhibitors of CDK2, *p21^{Cip}* (*CDKN1A*) and *p27^{Kip}* (*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. 4*C*) 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. 6*C*), as well as for the BER enzyme *UNG* (Fig. 6*D*), 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. 6*G*), 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. 6*E*).

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.

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