

Rajagopalan and Long. Vol. 189, No. 7, April 5, 1999. Pages 1093–1099.

The authors wish to report the following two corrections.

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Most likely because of allelic polymorphism, the KIR2DL4 used here differs by three amino acids from the sequence deposited in EMBL/GenBank/DDBJ (under accession no. U7119): a valine for the leucine at position 87, a glycine for the glutamate 124, and an asparagine for the histidine 371. Second, the expression system in the cell line NK-92 that was used to test functional recognition of HLA-G by KIR2DL4 has been unreliable. Additional experiments have revealed that the inhibition of lysis of 721.221 cells expressing HLA-G (221-G) occurs sometimes in NK-92 cells infected with recombinant vaccinia viruses that do not encode KIR2DL4. Therefore, the data reporting inhibition in NK-92 cells are inconclusive. Although this correction invalidates several of the results obtained with the NK-92 cell line, the main conclusion of the paper that KIR2DL4 binds HLA-G remains valid. Specific binding of soluble KIR2DL4-Ig fusion protein to 221-G cells has been confirmed. KIR2DL4-Ig bound also to the HLA-G-expressing trophoblast cell line JEG-3. Binding was blocked by an anti-KIR2DL4 mAb, and was inhibited partially by the HLA-G-specific mAb G233.