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

The authors wish to report the following two corrections.

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.