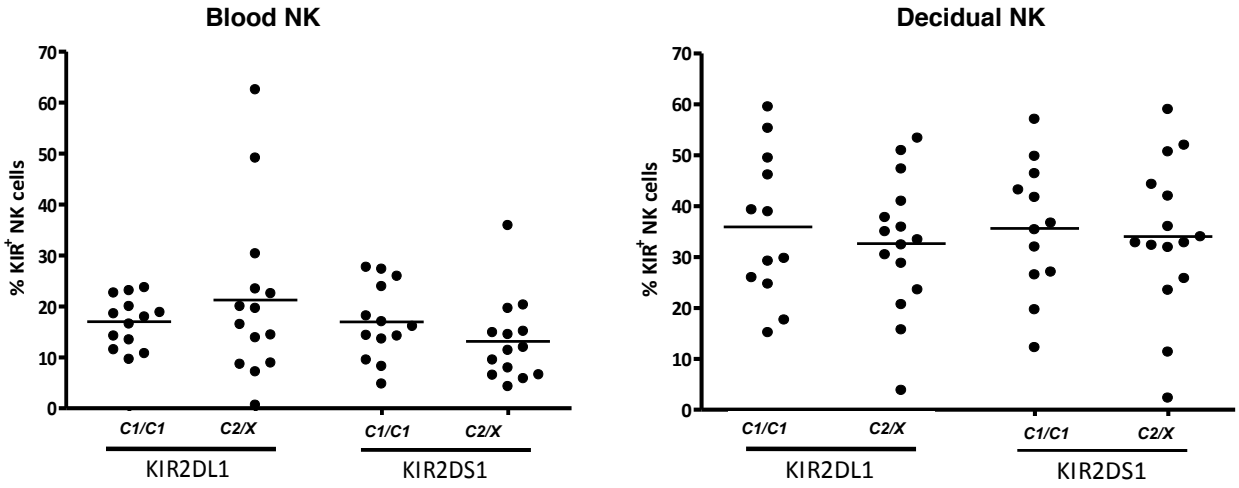
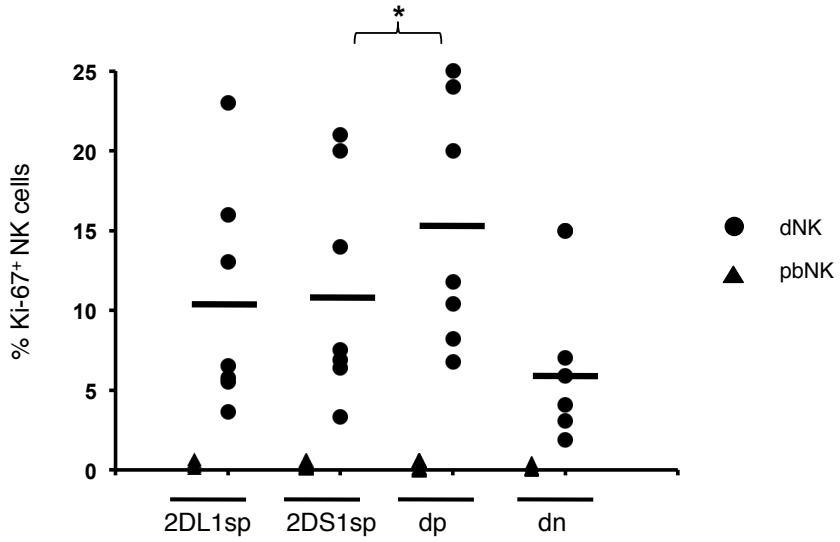


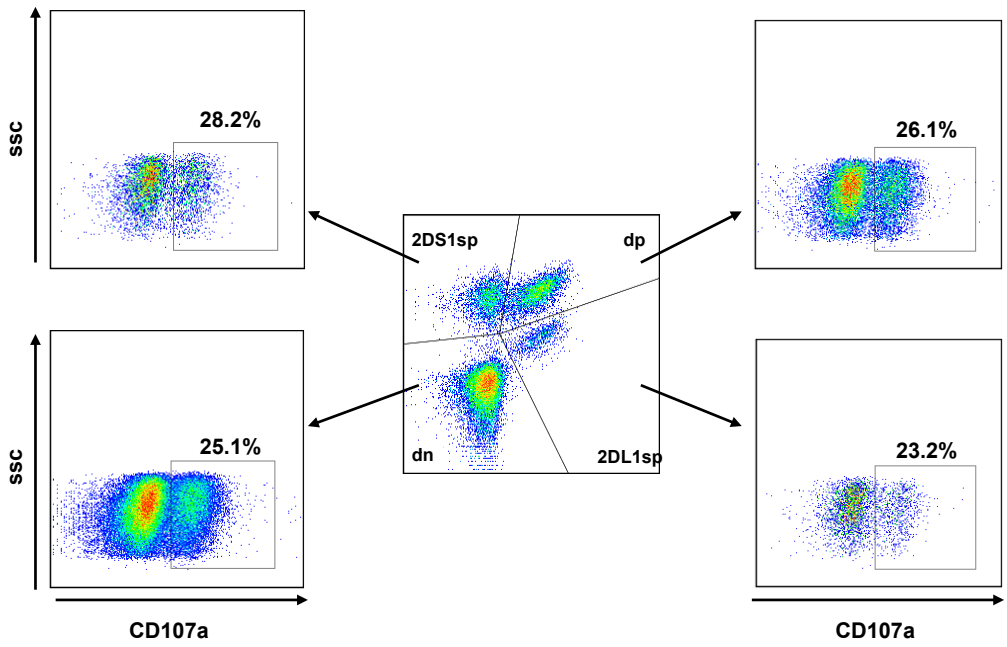
Supplementary Figure S1.
Maternal *HLA-C* type does not significantly affect the overall frequency of expression of KIR2DL1 or KIR2DS1 in maternal pbNK or dNK.

Overall frequency of KIR2DL1+ or KIR2DS1+ NK cells was determined by FACS as described in Figure 1B and stratified according to the maternal *HLA-C* type. *C1/C1* indicates donor had both alleles of *C1* group, *C2/X* indicates either *C2/C1* or *C2/C2*. No significant differences were observed in KIR2DS1 or KIR2DL1 frequencies relative to maternal *HLA-C* type in pbNK or dNK.



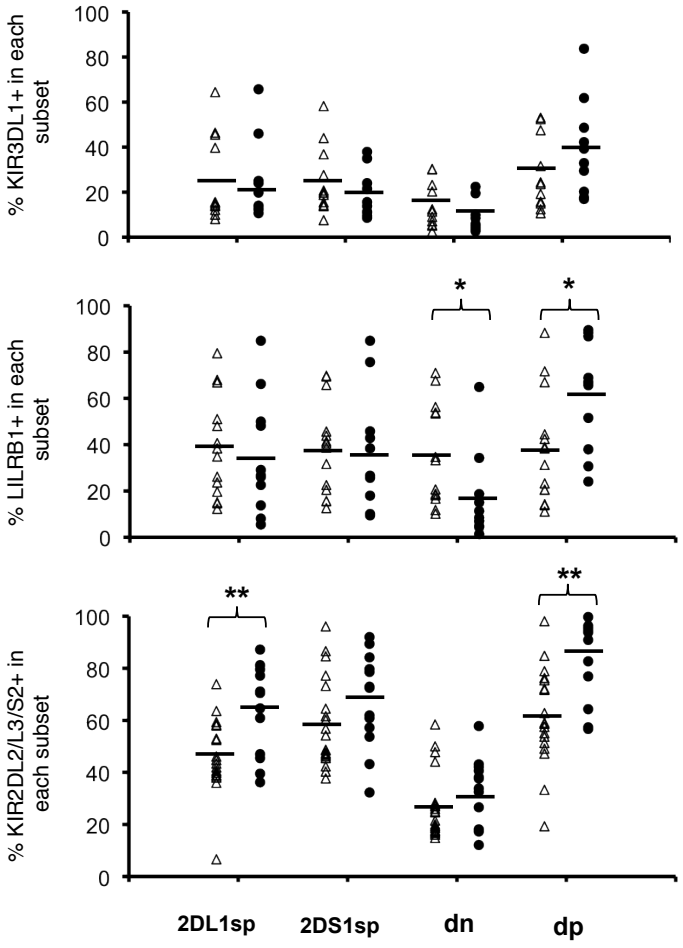
Supplementary Figure S2
KIR2DS1+ and KIR2DL1+ dNK subsets show increased staining for Ki-67 compared to their blood counterparts.

The percentage of NK cells that are positive for Ki-67 by intracellular FACS was determined in the four KIR2DL1/S1 subsets in matched pairs of blood or decidual NK cells gated as shown in Figure 1B (n=7). Horizontal bars represent the mean frequency of Ki-67+ NK cells within each subset. * indicates a statistically significant difference between 2DS1sp and dp subsets, * $p<0.05$, Wilcoxon signed Rank Test.



Supplementary Figure S3
KIR2DL1sp and KIR2DS1sp dNK subsets all degranulate to the same extent in response to cross-linking using mAb to NKp46.

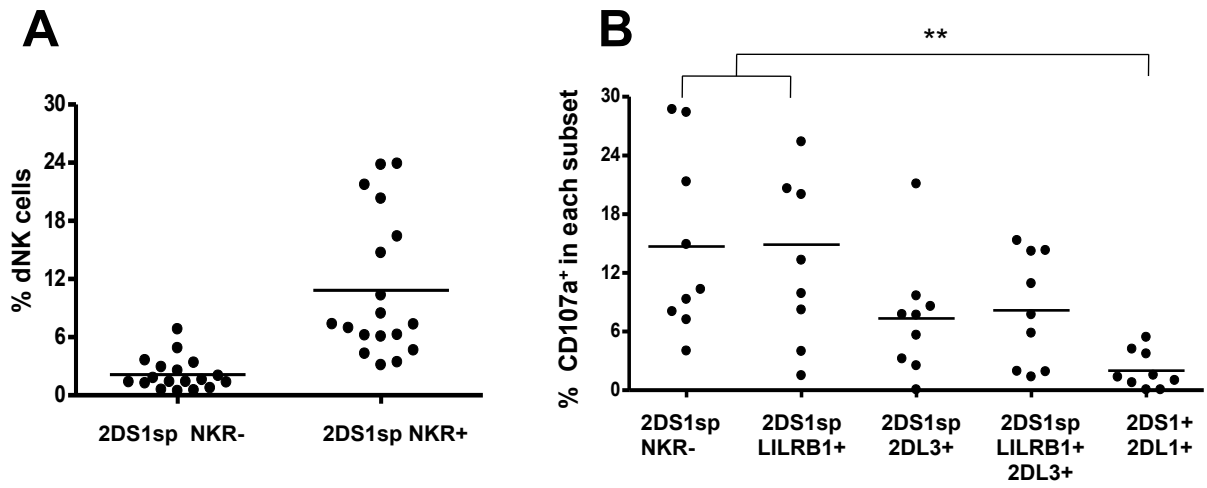
Decidual NK cells were activated by culture for 5 hours in wells coated with mAb 195314 which cross-links NKp46. Control wells were coated with isotype matched IgG. dNK were stained and gated as shown in Figure 1A and degranulation of KIR2DS1 and KIR2DL1 subsets was assessed using CD107a cell surface staining. All subsets incubated with control IgG showed similar mean degranulation of <5% (data not shown). Results shown are representative of three separate experiments. dNK expressing other NKRs (LILRB1, KIR2DL3/L2, 3DL1, 2DS4) were not gated out in this experiment.



Supplementary Figure S4.
The KIR2DL1/S1 subsets in pb and dNK differ in expression of other NK receptors (NKR) that recognize MHC.

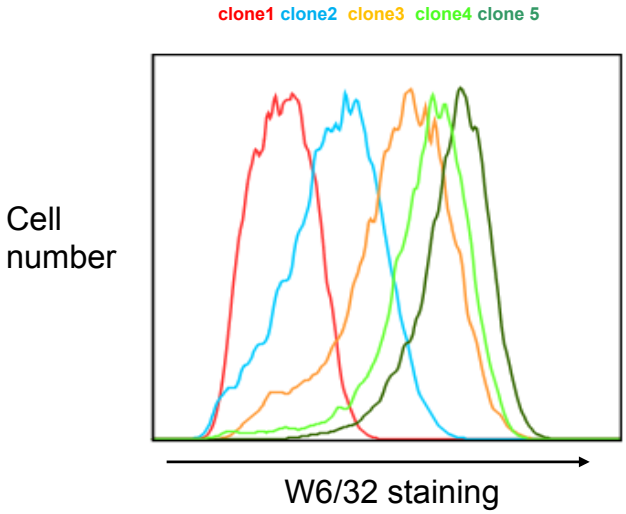
Matched pairs of decidual (dNK) and peripheral blood (pbNK) cells were gated as shown in Figure 1A and expression of other NKR determined by FACS in the four subsets: KIR2DS1sp, KIR2DL1sp, dp and dn.

* p<0.05, **p<0.01, Wilcoxon Signed Rank Test was used to compare frequency of these NKR in each KIR2DL1/S1 subset from blood and decidua

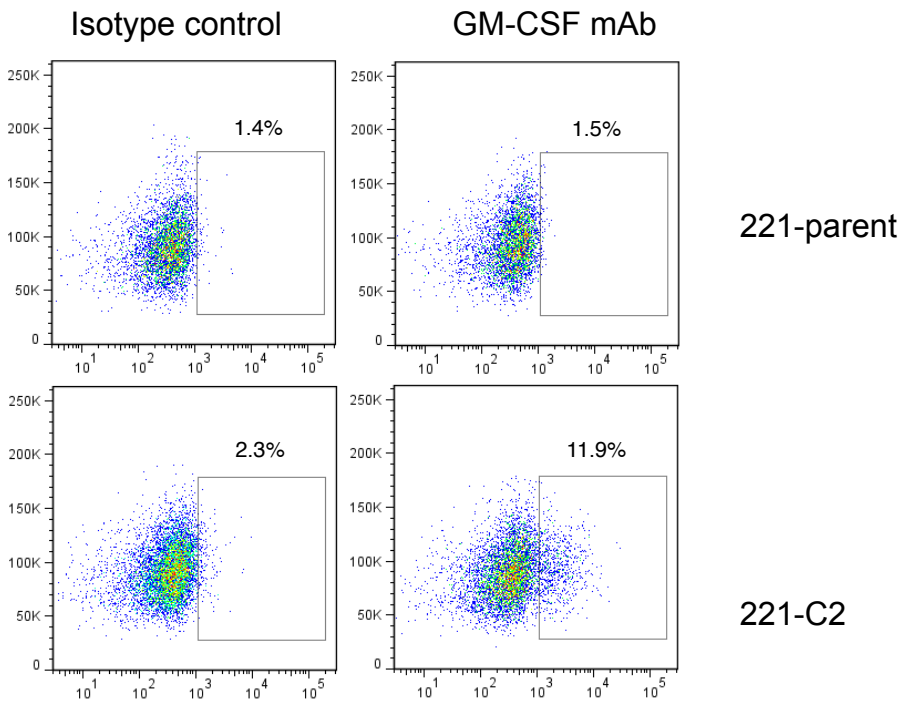


Supplementary Figure S5.
Effect of other NKRs on responses of KIR2DS1+ dNK cells.

(A) Frequency of KIR2DS1sp cells that are NKR- compared to KIR2DS1sp cells that co-express other NKR, expressed as a percentage of total dNK cells.
(B) Co-expression of specific NKRs affects the degranulation response of KIR2DS1sp dNK cells following co-culture with 221-C2 target cells. The response of KIR2DS1+ cells is abolished if KIR2DL1 is co-expressed but reduced to different extents by co-expression of other NKRs. Horizontal bars represent the mean frequency of CD107a+ NK cells within each subset. ** indicates $p<0.01$, Friedman test with Dunn's Multiple comparison post-test.

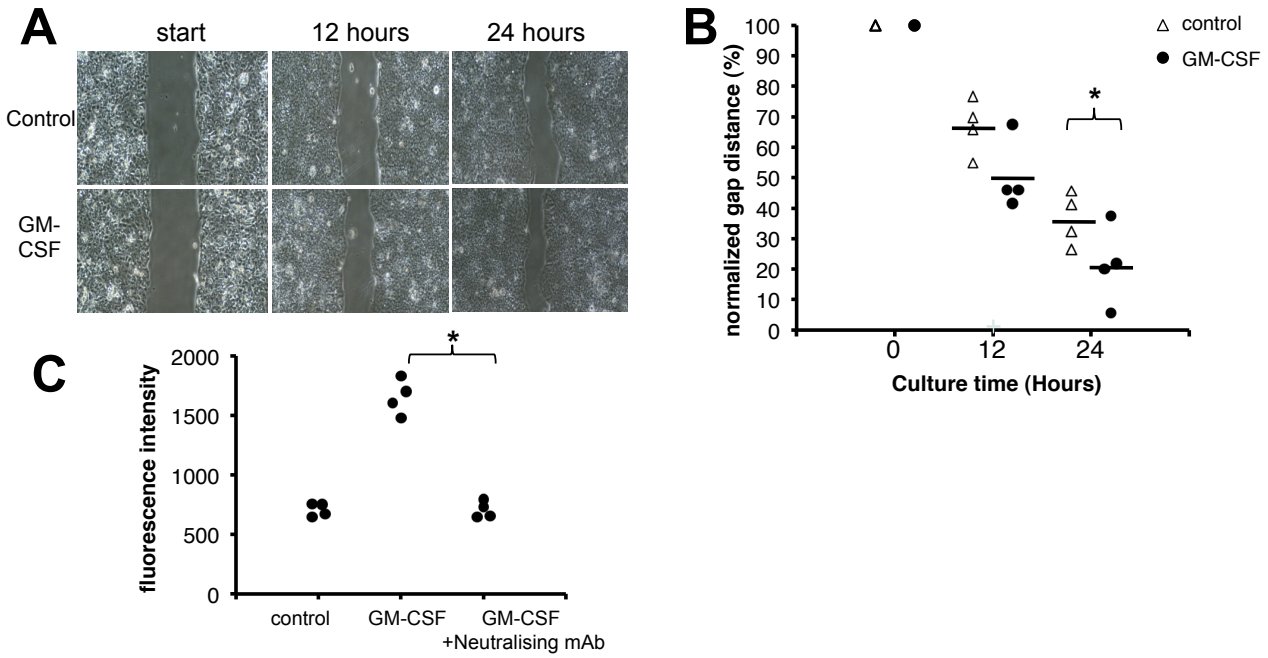


Supplementary Figure S6.
Clones of 721.221 cells transfected with HLA-Cw*0602 were selected that express different levels of surface HLA-C2.
Red trace indicates fluorescence of 721.221-parent cells (untransfected) after staining with mAb W6/32 against HLA-class I. Clones of 721.221 cells transfected with cDNA encoding Cw*0602 were selected with differing levels of HLA class I surface expression assessed by the mean fluorescent intensity (mfi). These clones were then used as targets for pbNK and dNK as shown in Figure 3E.



Supplementary Figure S7.
GM-CSF secretion is stimulated in KIR2DS1+ cells in response to HLA-C2.

Intracellular staining for GM-CSF in KIR2DS1spNKR+ decidual NK cells after co-culture with 221-parent or 221-C2 targets for 12 hours. Control staining used isotype-matched mAb. The NK cells were gated as shown in Figure 1A to include all KIR2DS1+NKR+ cells except those that co-expressed KIR2DL1.



Supplementary Figure S8.
GM-CSF increases motility of JEG-3 choriocarcinoma cells and migration in a Transwell assay.
(A) JEG-3 cells were seeded in a culture-insert dish and the insert was then removed to create a defined 'wound' of 500µm in width at the start of the experiment. GM-CSF (10ng/ml) or control medium was added and cells photographed at 12 and 24 hours to monitor cell movement into the gap.
(B) Average distance migrated by JEG-3 cells in (A) was evaluated by measuring the distance between the two sides of the gap at six random points and expressing this as a percentage of the initial 500µm separation. Cell treated with GM-CSF showed significantly greater migration into the gap at 24 hours. * $p<0.05$ (n=4) Student's t-test.
(C) Recombinant GM-CSF promotes migration of the trophoblast cell line JEG-3 in a Transwell assay. JEG-3 cells were seeded on the upper surface of the Transwell chamber with 10ng/ml GM-CSF added to the medium in the lower chamber. Increased migration in response to GM-CSF was abolished by neutralizing antibody to GM-CSF. * $p<0.05$, (n=4) Student's t-test

Supplementary Table 2

Antibodies used for multi-colour flow cytometry

mAbs specificity/dye	Supplier	Clones
CD3-APCcy7	Biolegend	UCHT-1
Near IR fixable live/dead cell dye	Invitrogen	-
CD56-Brilliant violet 421	Biolegend	HCD56
KIR2DL1-FITC	R&D	143211
KIR2DL1/S1-APC	Beckman Coulter	EB6
KIR2DL2/L3/S2-PE	Beckman Coulter	GL183
LILRB1-PE	Beckman Coulter	HP-F1
KIR3DL1-PE	Beckman Coulter	DX9
KIR2DS4-PE	Beckman Coulter	FES 172
CD107a-PerCP-Cy5.5	Biolegend	H4A3
HLA-G-FITC	AbD Serotec	MEM-G/9
CD14-PerCP-Cy5.5	Biolegend	HCD14
GM-CSF-PerCP-Cy5.5	Becton Dickinson	BVD2-21C11
GM-CSFRα-PE	Biolegend	4H1