Supplemental Figure Legends

Figure S1: Stat5 expression correlates with the expression of anti-apoptotic genes. (A) RNA was isolated of MACS-purified and IL-2 expanded *Stat5*$^{fl/+}$ and *Stat5*$^{\Delta/+}$*Ncr1*-iCre^{Tg} NK cells and transcribed into cDNA used for qPCR expression analysis of *Stat5a, Stat5b*, *Bcl-xl, Bcl-2* and *Mcl-1*. Data represent means±SEM of two independent experiments. Expression levels were calculated relative to the housekeeping gene *Rplp0* and all values were normalized to *Stat5*$^{fl/+}$ NK cells. Unpaired t-test was applied for statistical analysis. (B) Protein lysates of *ex vivo*-derived MACS-purified and sorted *Stat5*$^{fl/+}$ and *Stat5*$^{\Delta/+}$*Ncr1*-iCre^{Tg} NK cells were used for western blotting and the detection of Stat5, Bcl-2, Bcl-xL and Mcl-1. ß-actin was used as loading control. (C) NK cell number of liver, lymph node, lung and bone marrow (1 femur and tibiae) of *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* mice. Single cell suspensions of the respective tissue were analysed by flow cytometry and total cell numbers were calculated. Bar graphs depict means±SEM of two individual experiments (n≥6) Number of bone marrow NK cells in *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice is not reduced as Cre deletion starts in NKp46+ cells as previously published (1). Tukey's post-hoc test was applied for statistical analysis. (D) A *Stat5*$^{\Delta/\Delta}$ PCR was performed with MACS-purified and IL-2 cultured *vav-bcl-2*, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *Stat5*$^{fl/fl}$ NK cells. One representative experiment out of three is shown. (E) *Stat5*$^{fl/fl}$*Mx1*-Cre mice were crossed with *vav-bcl-2* mice. *Stat5*$^{fl/fl}$*Mx1*-Cre-*vav-bcl-2* mice and respective controls were treated with Poly(I:C) to induce STAT5 deletion. Splenocytes were analyzed by flow cytometry for NK cells and total cell numbers of CD3$^-$NKp46$^+$ NK cells of *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre, *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre-*vav-bcl-2* and *vav-bcl-2* mice were calculated. One representative experiment out of two independent experiments with n≥6 per genotype is shown. Tukey's post-hoc test was applied for statistical analysis. (F) Protein lysates of IL-2 cultured and interferon-β treated *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/+}$*Mx1*-Cre, *vav-bcl-2* and *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre-*vav-bcl-2* NK

cells were used for western blotting to detect STAT5, Bcl-2 and Bcl-xL levels. ß-Actin was used as loading control. (G) Quantification of Bcl-2 protein levels of three individual western blots. (H) RNA was isolated of MACS-purified and IL-2 expanded *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* NK cells and transcribed into cDNA used for qPCR expression analysis of *Bcl-2*. Data represent means±SEM of two independent experiments. Expression levels were calculated relative to the housekeeping gene *Rplp0* and all values were normalized to *Stat5*$^{fl/fl}$ NK cells.

Figure S2: STAT5-deficient NK cells possess aberrant transcription factors expression. (A) Protein lysates of IL-2 cultured and interferon-β treated *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/+}$*Mx1*-Cre, *vav-bcl-2* and *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre-*vav-bcl-2* NK cells were used for western blotting to detect Id2 levels. ß-actin was used as loading control. (B+C) Splenocytes of *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* mice were stained for CD3 and NKp46, followed by fixation, permeabilization and intracellular staining of T-bet and Eomes. Expression levels were analyzed by flow cytometry. Mean fluorescence intensities and % positive cells are depicted. Data represent one representative experiment out of two with similar outcome (n≥3). Tukey's post-hoc test was applied for statistical analysis. (D) Splenocytes of *Stat5*$^{fl/+}$, and *Stat5*$^{\Delta/+}$*Ncr1*-iCre^{Tg} mice were stained for CD3 and NKp46, followed by fixation, permeabilization and intracellular staining of T-bet and Eomes. Expression levels were analyzed by flow cytometry. Mean fluorescence intensities and % positive cells are depicted. Data represent one representative experiment out of two with similar outcome (n≥4). Unpaired t-test was applied for statistical analysis. (E) IL-2 expanded and interferon-β treated *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre-*vav-bcl-2* and *vav-bcl-2* NK cells were stained for CD3 and NKp46, followed by fixation, permeabilization and intracellular staining of T-bet and Eomes. Expression levels were analyzed by flow cytometry. Percent positive cells are depicted and data represent means±SEM of two independent experiments (n≥4). Tukey's post-hoc test was

applied for statistical analysis. (F) NK cells of *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* mice were purified and IL-2 expanded and apoptosis was analyzed by flow cytometry using 7-AAD and Annexin-V. Bar graphs depict living cells, early apoptotic, late apoptotic and dead cells on day 4 after purification. Data illustrate one representative experiment out of two (n≥4 per genotype) and Tukey's post-hoc test was applied for statistical analysis.

Figure S3: Loss of STAT5 alters granzyme, perforin and IFN-γ production. (A) Splenocytes of *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* mice were stimulated with IL-2 (or IL-2+IL-12 for IFN-γ production) and stained for CD3 and NKp46, followed by fixation, permeabilization and intracellular staining of Granzyme A, Granzyme B and IFN-γ. Expression levels were analyzed by flow cytometry. % positive cells (means±SEM) are depicted in the representative FACS plots with n≥4 per genotype. Tukey's post-hoc test was applied for statistical analysis. (B) Protein lysates of IL-2 cultured and interferon-β treated *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/+}$*Mx1*-Cre, *vav-bcl-2* and *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre-*vav-bcl-2* NK cells were used for western blotting to detect Perforin levels. ß-Actin was used as loading control. (C) MACS purified and IL-2 expanded and interferon-β treated *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre-*vav-bcl-2* and *vav-bcl-2* NK cells were stained for CD3 and NKp46, followed by fixation, permeabilization and intracellular staining of Granzyme B and Perforin. Expression levels were analyzed by flow cytometry. Percent positive cells are depicted and data represent means±SEM of two independent experiments. Tukey's post-hoc test was applied for statistical analysis. (D) MACS purified and IL-2 expanded *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* NK cells were injected i.v. into *Rag2*$^{-/-}$*gc*$^{-/-}$ recipient mice. On the next day 10^6 RMA-S cells were s.c. injected into the flanks of the mice and tumor weight was determined after 10 days. Tukey's post-hoc test was applied for statistical analysis.

Figure S4: STAT5-deficient NK cells induce tumor promotion. (A) 10^6 RMA-S cells were injected s.c. into the flanks of the mice. After 9 days tumor weight was determined. Statistics

represent means±SEM of two independent experiments (n≥8 per genotype). Tukey's post-hoc test was applied for statistical analysis. (B) Tumor growth curve of s.c. RMA-S tumors injected into *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* mice. (C) 5x10^6 RMA-S cells were injected s.c. into the flanks of *Stat5*$^{fl/fl}$ and *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice. Tumor weight was determined after 10 days. Statistics represent means±SEM of two independent experiments (n≥10 per genotype). Unpaired t-test was used for statistical analysis. (D) Tumor growth curve of s.c. *v-abl*$^+$ tumors injected into NK cell-depleted and PBS-treated *Stat5*$^{fl/fl}$ and *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* mice. Tukey's post-hoc test was applied for statistical analysis of each individual time point. (E) Representative CD31 stainings of *v-abl*$^+$ tumors derived from *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* and *vav-bcl-2* mice and number of CD31$^+$ cells per specific field of different tumor sections (tumors were photographed at random representative areas) was counted by two independent researchers in a blinded manner. Tukey's post hoc test was applied for statistical analysis.

Figure S5: Loss of STAT5 increases expression of the pro-angiogenic factor VEGF-A. (A) Mice were Poly(I:C)-treated and RNA was isolated of MACS-purified and IL-2 cultured *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Mx1*-Cre-*vav-bcl-2* and *vav-bcl-2* NK cells and transcribed into cDNA used for qPCR expression analysis of *Vegf-A*. Data represent means±SEM of two independent experiments. Expression levels were calculated relative to the housekeeping gene *Rplp0* and all values were normalized to *Stat5*$^{fl/fl}$ NK cells. Tukey's post-hoc test was applied for statistical analysis. (B) Spheroids of endothelial cells were embedded into a collagen matrix. Supernantant of IL-2 cultured *Stat5*$^{fl/fl}$, *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* NK cells or mVEGF-A (positive control) was neutralized with an anti-VEGF-A antibody for 1 hour. The collagen gels were topped with neutralized or untreated supernatant and sprouting was documented after 24 hours. Average sprout number and cumulative sprouting length was quantified by

ImageJ software. Tukey's post-hoc test was applied for statistical analysis. (C) Mouse aortic rings were embedded into a collagen matrix and IL-2 media (control) or supernatant of IL-2 cultured *Stat5*$^{fl/fl}$, *vav-bcl-2* and *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* NK cells were added and sprouting was documented after 8 days. Representative pictures are depicted showing enhanced sprouting after addition of *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* NK cell supernatant compared to controls.

Figure S6: Peripheral splenic NK cells produce VEGF-A which does not impact NK cell numbers, proliferation or maturation but enhances tumor progression in lymphoid tumor models. (A) Blood, spleen and lymph nodes of *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice (n≥4 per genotype) were stained for CD3 and NKp46 and NK cell number was analyzed by flow cytometry. One independent experiment out of three with similar outcome is shown. Unpaired t-test was used for statistical analysis. (B) *Ex vivo* derived MACS-purified and sorted NK cells of *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice were used for RNA isolation. RNA was transcribed into cDNA for qPCR analysis of *Vegf-A* mRNA levels. Expression levels were calculated relative to the housekeeping gene *Rplp0*. *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} NK cells possess 85% reduction in *Vegf-A* mRNA expression compared to controls. One independent experiment out of three with similar outcome is shown. Unpaired t-test was used for statistical analysis. (C) MACS purified *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} NK cells were IL-2 cultured and living cells were counted every day for a growth curve analysis. One independent experiment out of two with similar outcome is shown Unpaired t-test was used for statistical analysis of each indicated time point. (D) For the analysis of NK cell maturation stages, splenic CD3^{-}NKp46^{+} NK cells were analyzed for CD27 and CD11b expression by flow cytometry. Data are representative for at least 2 independent experiments with n≥7 per genotype. Unpaired t-test was used for statistical analysis of each maturation stage and failed

to reach statistical significance for each stage (bar graphs not shown). (E) Spheroids of endothelial cells were embedded into a collagen matrix and IL-2 media (control), supernantant of IL-2 cultured *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} NK cells or mVEGF-A (positive control) were added and sprouting was documented after 24 hours. Average sprout number and cumulative sprouting length was quantified by ImageJ software. Tukey's post-hoc test was applied for statistical analysis. (F) NK cells were depleted twice before and after s.c. injections of 10^6 *v-abl*$^+$ cells into *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice. After 11 days the tumor weight (n≥10 per group) was determined. Tukey's post-hoc test was applied for statistical analysis. (G) Tumor growth curve of *v-abl*$^+$tumors of NK cell-depleted or PBS-treated *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice. (H) 10^6 RMA-S cells were injected s.c. into the flanks of *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice. After 12 days the tumor weight (n≥10 per group) was determined. Unpaired t-test was used for statistical analysis. (I) $5x10^4$ B16F10 melanoma cells were injected i.v. into *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice (n≥5 per genotype) and the number of tumor nodules in the lung was assessed after 27 days. Unpaired t-test was used for statistical analysis.

Figure S7: Tumor-infiltrating NK cells and macrophages are localized around blood vessels.

(A) CD31 (red) and F4/80 (green) co-stainings of *v-abl*$^+$ tumors (random representative areas) derived from *Vegf-A*$^{fl/fl}$ and *Vegf-A*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg} mice. Number of CD31$^+$ and F4/80$^+$ cells per specific field of different tumor sections wascounted by two independent researchers in a blinded manner. Number of infiltrating macrophages is unchanged and macrophage/CD31$^+$ cell ratio is shown. Unpaired t-test was applied for statistical analysis. (B) CD31 (green) and NKp46 (red) co-stainings of *v-abl*$^+$ tumors (random representative areas) derived from *Stat5*$^{fl/fl}$, *vav-bcl-2* and *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* mice. Number of CD31$^+$ and NKp46$^+$ cells per specific field of different tumor sections was counted by two independent researchers in a blinded manner. Tumors from *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* show a significant increase

in CD31 cells, the number of infiltrating NK cells was unchanged between the different genotypes. NKp46/CD31$^+$ cell ratio is shown. Tukey's post-hoc test was applied for statistical analysis. (C) CD31 (red) and F4/80 (green) co-stainings of *v-abl*$^+$ tumors (random representative areas) derived from *Stat5*$^{fl/fl}$, *vav-bcl-2* and *Stat5*$^{\Delta/\Delta}$*Ncr1*-iCre^{Tg}-*vav-bcl-2* mice. Number of CD31$^+$ and F4/80$^+$ cells per specific field of different tumor sections was counted by two independent researchers in a blinded manner. Number of infiltrating macrophages is unchanged and macrophage/CD31$^+$ cell ratio is shown. Tukey's post-hoc test was applied for statistical analysis.

Figure S8: IL-2 stimulated human NK cells mirror the murine data

(A) NK cells were stimulated with IL-2+ IL-10, IL-12, IL-18, IL-21 or IFN-ß for 20 or 60 minutes. RNA was isolated and transcribed into cDNA used for qPCR expression analysis of *Vegf-A*. (B) Lymphocytes of healthy human blood donors (n=8) were enriched and CD16$^-$ CD56^{bright} and CD16$^+$CD56$^+$ NK cells were sorted. RNA was isolated and transcribed into cDNA for qPCR analysis. *STAT5B* and *VEGF-A* expression levels were calculated relative to the housekeeping gene *Rplp0* and are ploted showing that CD16$^+$CD56$^+$cells have higher expression levels of *STAT5B* but lower expression of *VEGF-A* compared to CD16$^-$CD56^{bright} cells. Statistical analysis was performed in Figure 6E. (C) Lymphocytes of healthy human blood donors were enriched and CD16$^-$CD56^{bright} NK cells were sorted. RNA was isolated and transcribed into cDNA for qPCR analysis. *STAT5B* and *VEGF-A* expression levels were calculated relative to the housekeeping gene *Rplp0* (n=16, 8 donors in technical duplicates) and show an inverse correlation in their expression levels. R software was used to calculate Pearson correlation and to generate scatter plots. (D) Heatmap summarizing expression values of *VEGFA*, *CCND2*, *EOMES* and *CISH* in human naïve natural killer (NK) cells (n=4) and in IL-2 activated NK cells (n=4). Data were extracted from ArrayExpress dataset E-GEOD-50838 (Affymetrix Human Gene 1.1 ST Arrays). Colours range from high expression (red) to low expression (blue).The heatmap was generated using *ClustVis* software (1). FDR, false

discovery rate. (E) MACS-purified and IL-2 expanded C57/BL6 WT NK cells were treated with 1µM or 5µM of STAT5 inhibitor or DMSO as vehicle control. RNA was isolated and transcribed into cDNA used for qPCR expression analysis of *Vegf-A* and the STAT5 downstream targets *Bcl-2*, *Mcl-1* and *Cish*. . Expression levels were calculated relative to the housekeeping gene *ß-Actin* and all values were normalized to DMSO treated NK cells.

Supplemental Experimental Procedures

Genotyping PCR

For the detection of the $Stat5^{\Delta}$ band following primers were used Primer1: CCCATTATCACCTTCTTTACAG and Primer2: AGCAGCAACCAGAGGACTAC. Deletion of STAT5 gives rise to a 620bp product.

qRT-PCR

RNA was prepared from purified NK cells using peqGOLD TriFast reagent (PEQLAB) or the RNeasy Mikro Kit (Qiagen). 1 µg of RNA was reversely transcribed with the iSCRIPT cDNA synthesis kit (Bio-Rad). Real-time PCR was performed on a MyiQ2 cycler (Bio-Rad) with SsoFastTM EvaGreen®Supermix (Bio-Rad). In addition to previously published primers (1–3), following primers were used: *mVegf-A(1)*: Fw:5'-GGACCCTGGCTTTACTGC-3' and Rv:5'-CGGGCTTGGCGATTTAG-3', *mVegf-A(2)*: 5'-GCACAGCAGATGTGAATGCAG-3' and Rv:5'- CGCTCTGAACAAGGCTCACA-3', *Ccnd2*: Fw:5'-GGGAACTGGTAGTGTTGGG-3' and Rv:5'-GGGCATCACACGTGAGTGTG-3', *Bcl-2*: Fw:5'-ACTGAGTACCTGAACCGGCATC-3'and Rv:5'-GGAGAAATCAAACAGAGGTCGC-3', *Bcl-xl*: Fw:5'-GCAGCAGTTTGGATGCGCGG-3'and Rv:5'AAAGTGTCCCAGCCGCCGTTC-3', *Mcl-1*: Fw:5'-CGCAACCACGAGACGGCCTT-3' and Rv:5'- ACTCCACAAACCCATCCCAGCCT-3', *Id2*: Fw:5'-CCCGGTGGACGACCCGATGA-3'and Rv:5'-

GGGAATTCAGATGCCTGCAAGGACA-3', *Myc*: Fw:5'-GTGCTGCATGAGGAGACACCG-3' and Rv:5'- ATGGAGATGAGCCCGACTCCG-3', *ß-Actin:* Fw:5'-CTCTGGCTCCTAGCACCATGAAGA-3' and Rv:5'-GTAAAACGCAGCTCAGTAACAGTCCG-3', *hVEGF-A*: Fw:5'-GTCGGGCCTCCGAAACCATG-3'and Rv:5'-CGTGATGATTCTGCCCTCCTCCTTC-3', *hSTAT5A*: Fw:5'-GGCTCCCTATAACATGTACCC-3' and Rv:5'-AAGACTGTCCATTGGTCGGCG-3', *hSTAT5B* Fw:5'-GAACGCATCTGCAGATGCCG-3' and Rv:5'- TCAAGGACTGAGTCAGGG-3' and *hRPLP0*: Fw:5'-GGCGACCTGGAAGTCCAACT-3' and Rv:5'- CCATCAGCACCACAGCCTTC-3'. mStat5a primers were purchased from Qiagen. Target gene expression was normalized to *Rplp0 and ß-Actin*.

Mouse aortic ring assay

Aoartas from 6-10 weeks old mice were isolated, periaortic fibroadipose tissue was removed and blood was flushed out of the lumen using serum free media. Aortas were cut with the blade of a scalpel in 1mm long rings and embedded into a collagen matrix. Fresh supernatant of IL-2 expanded NK cells or 30ng/ml mVegf-A (PeproTech) were added during the first four days and micro-vessel formation was documented after 8 days with an Olympus IX71 microscope using cellSens Dimension Software (Olympus).

Western Blotting

Preparation of protein lysates and western blot was done as previously reported(2). Following antibodies were used: pSTAT5-Y694 (BD-611964), ß-actin (sc-69879), Bcl-2 (sc-7382), Id2 (sc-489), Mcl-1 (sc-819), Stat5 (C-17, sc-835), Stat5a (L-20, sc-1081), (Santa Cruz), Bcl-xl (CS#2764), Id2 (CS#3431) and Perforin (CS#3693) (Cell Signaling). Immunoreactive bands

were visualized by chemiluminescence detection (LumiGLO®, Cell Signaling) by the ChemiDoc MP Imaging System (Bio-Rad).

References

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