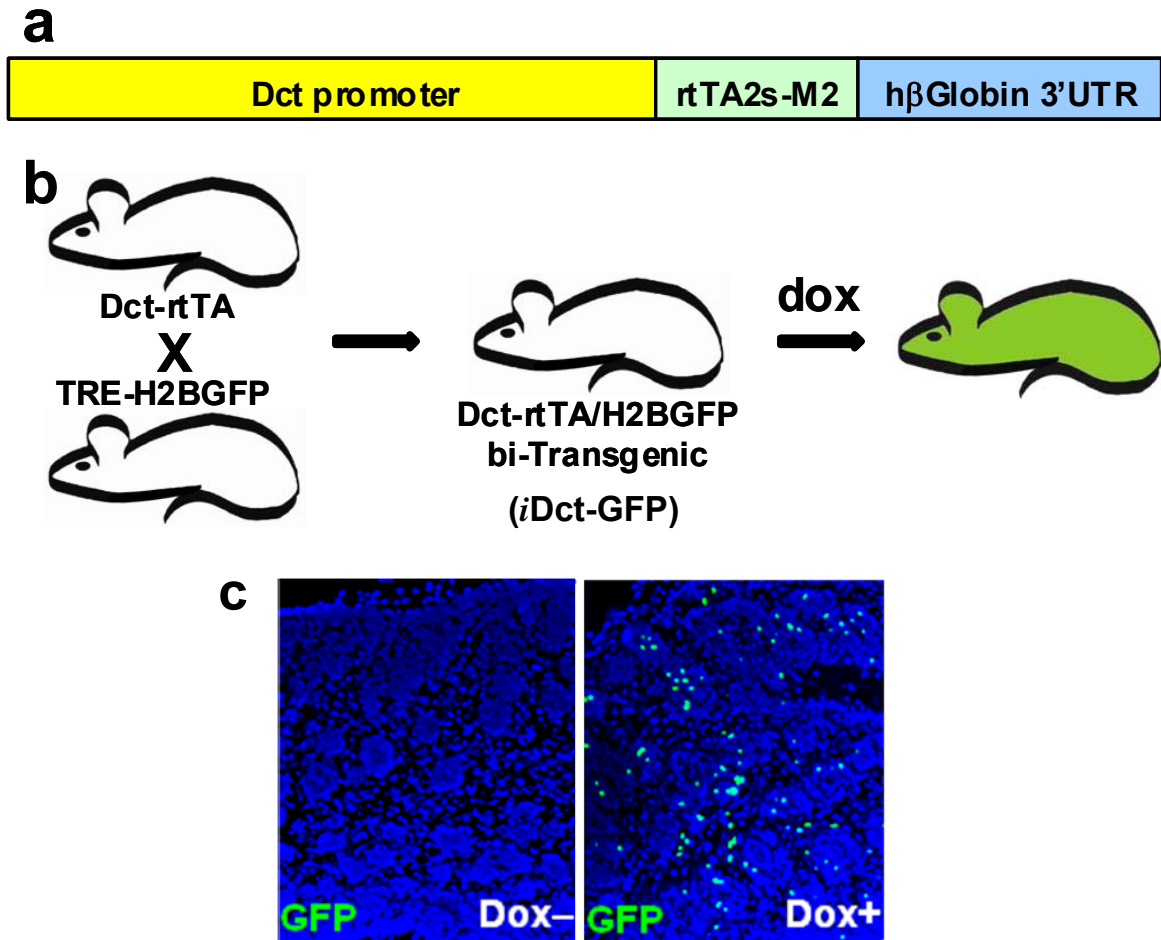
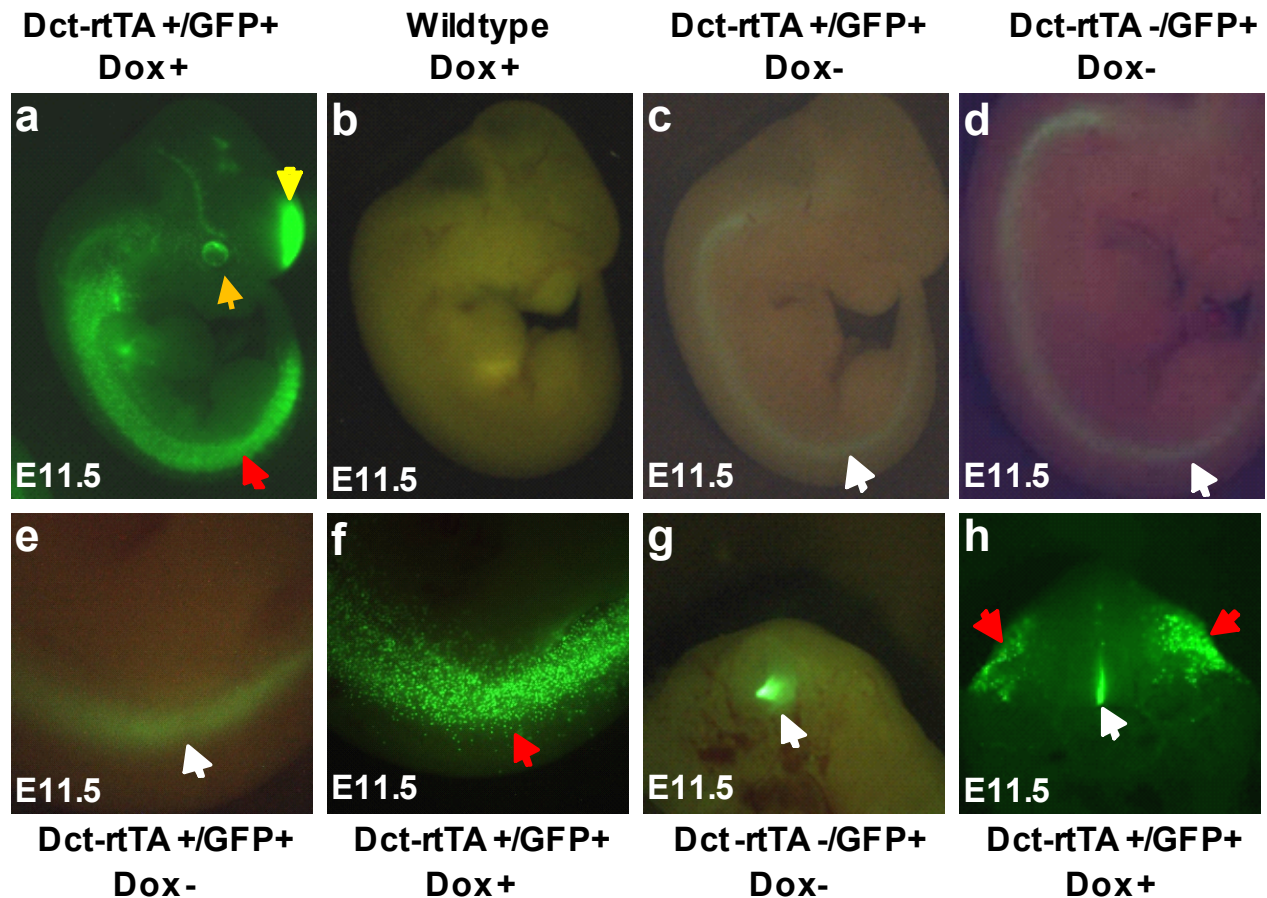


Supplementary Figure 1



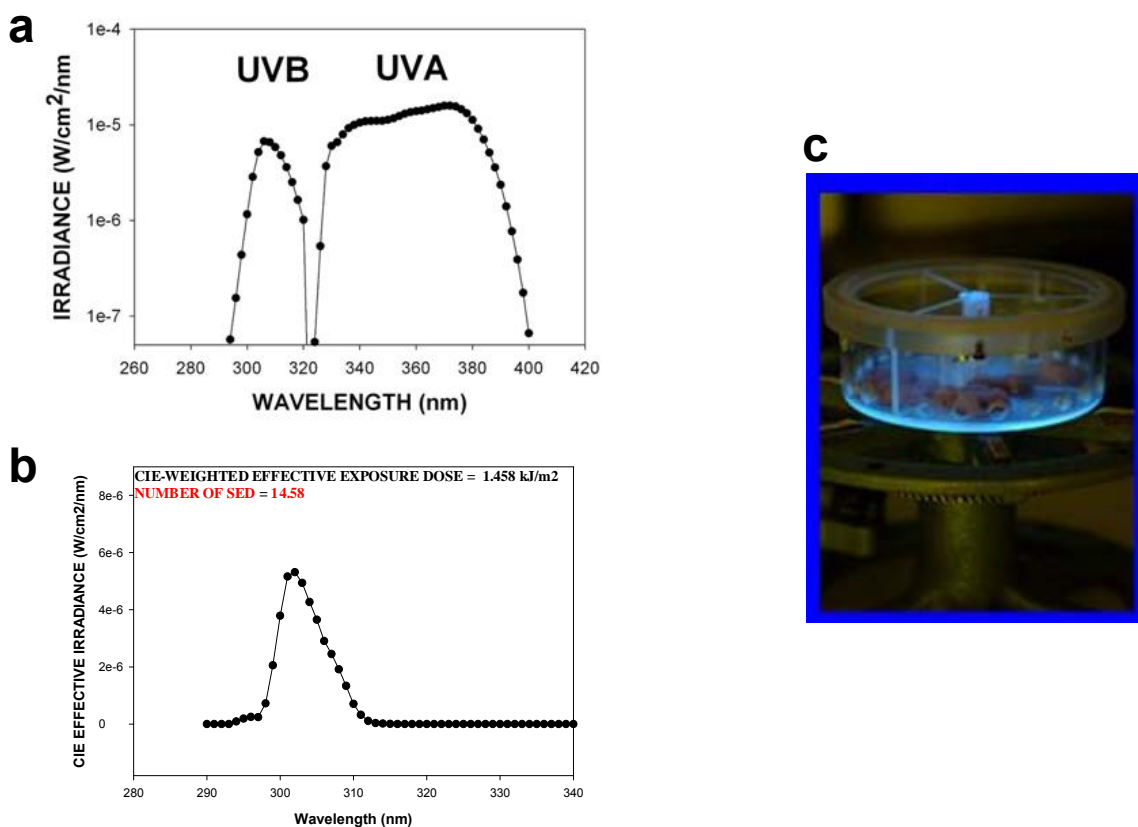
Supplementary Fig. 1. a, The transgene construct for making the Dct-rtTA mice. The 3.4 kb dopachrome tautomerase (*Dct*) gene promoter is melanocyte-specific. The rtTA2s-M2 version of the reverse tetracycline-activated transactivator provides leak-free expression and no background. The 3'UTR consists of partial exon 2, full intron 2 and exon 3 of human beta globin gene, and provides splice sites and an enhancer sequence for optimal transgene expression. Thirty-one transgenic founders were generated, of which one was selected for further studies. **b**, Dct-rtTA mice were bred with TRE-H2BGFP transgenic mice to produce *inducible* Dct-rtTA/TRE-H2BGFP double transgenic mice (*iDct-GFP*), which expressed GFP exclusively in melanocytes when injected intraperitoneally with doxycycline (dox). **c**, *iDct-GFP* mice show tight regulation of GFP expression in skin. No GFP expression is detectable in the absence of dox (left), but robust expression is seen within 12-18 hours of a single intraperitoneal injection of non-toxic dose of dox (right).

Supplementary Figure 2



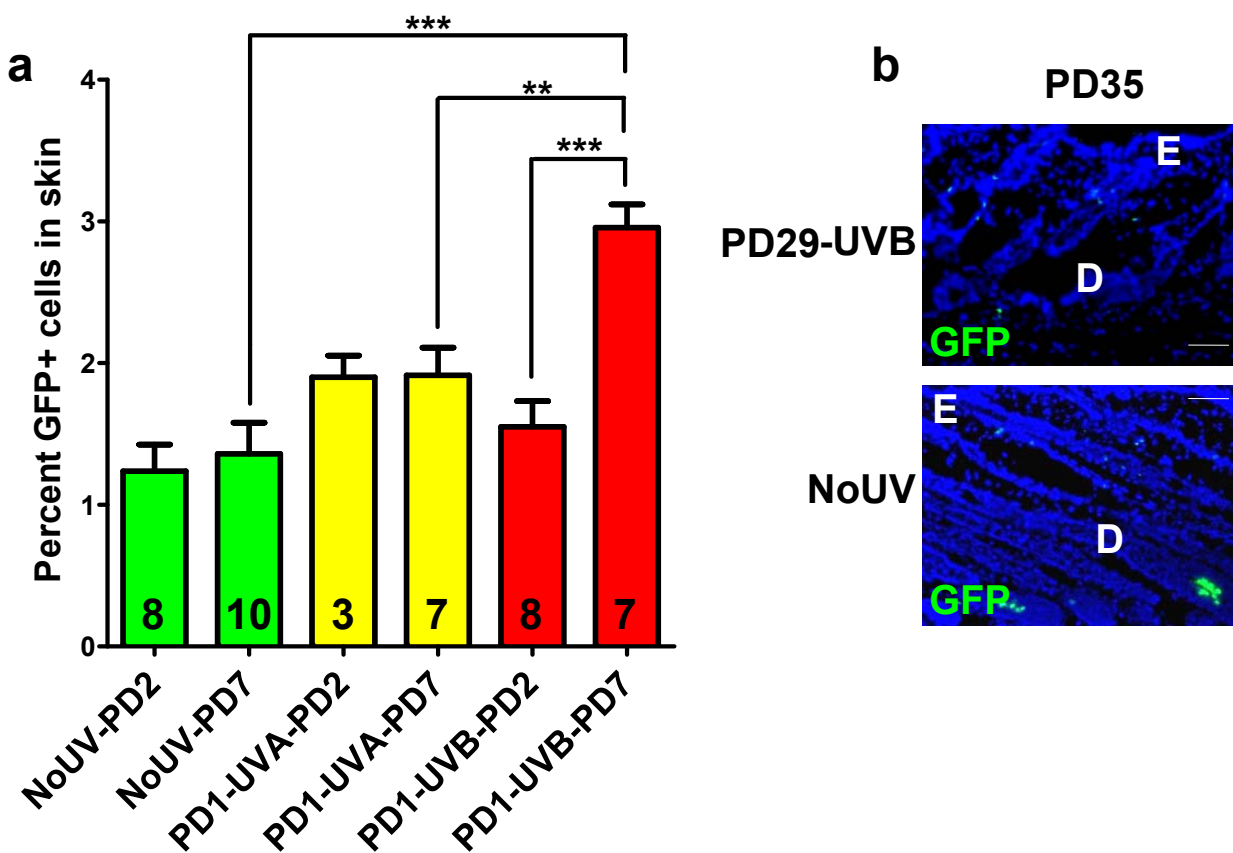
Supplementary Fig. 2. **a**, An E11.5 *iDct*-GFP embryo showing GFP expression in the neural crest (red arrow), telencephalon (yellow arrow), and retinal pigment epithelium (orange arrow) after doxycycline (dox) administration to the mother. This expression pattern is in congruence with that of *Dct* as expected (Silver *et al.*, *Adv Exp Med Biol* 589:155-169). **b**, A wildtype control embryo shows no GFP expression. **c**, *iDct*-GFP embryos without dox administration exhibit no GFP expression in neural crest, telencephalon or retinal pigment epithelium. However, there is a non-specific faint fluorescence in the floorplate (white arrow), which is also visible in the TRE-H2BGFP mice in the absence of dox (**d**). **e-f**, Floorplate shows non-specific faint fluorescence in the absence of dox (**e**), but dox induces GFP expression in the neural crest cells (**f**). **g**, A dorso-ventrally cut TRE-H2BGFP embryo shows non-specific fluorescence in the floorplate (arrow) in the absence of dox. **h**, A dorso-ventrally cut *iDct*-GFP embryo after dox administration shows GFP+ melanoblasts moving dorso-laterally (red arrows), and non-specific floorplate fluorescence (white arrow).

Supplementary Figure 3



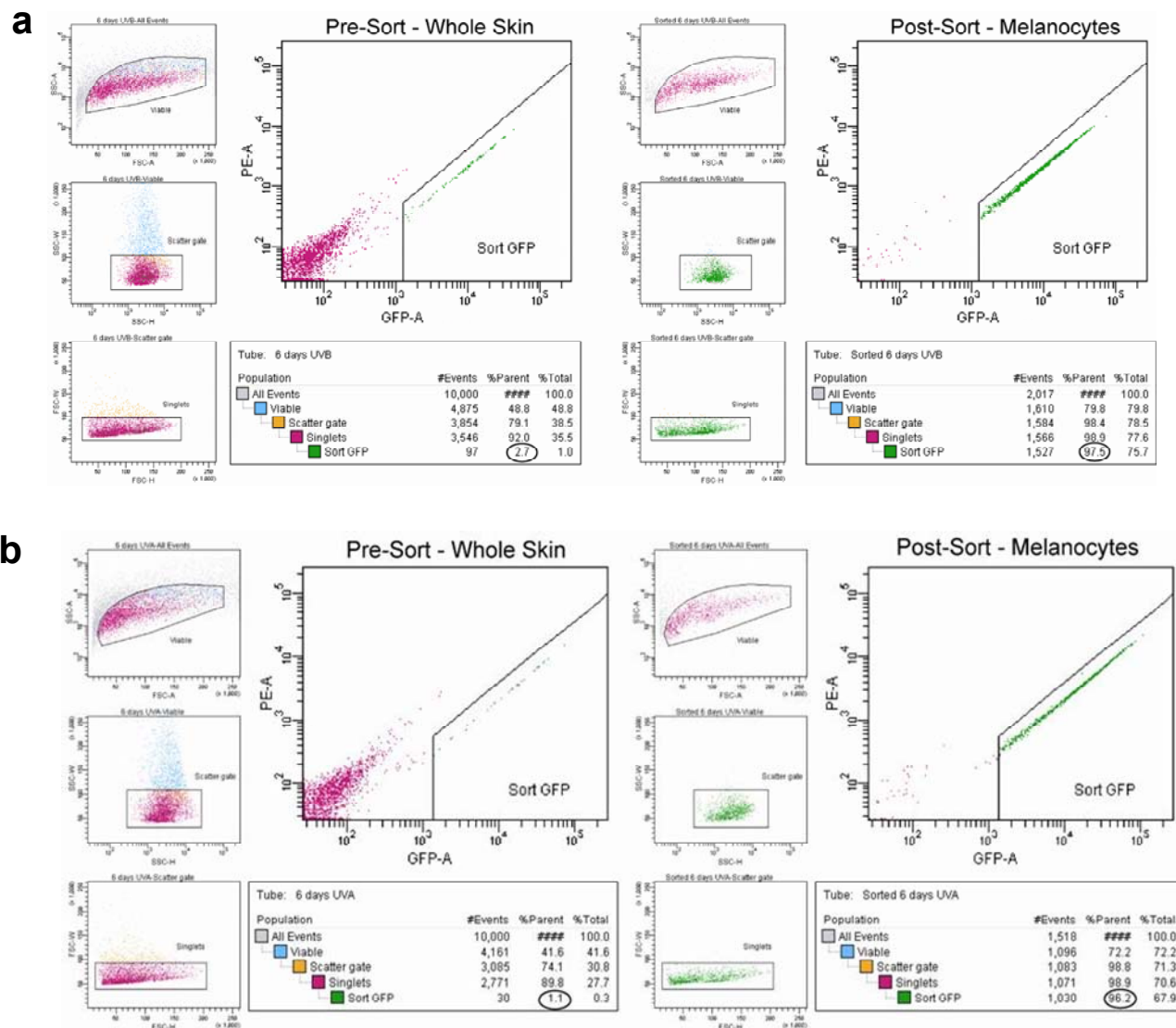
Supplementary Fig. 3. a, Interference filters with precisely defined wavebands of UVA (320-400 nm) or UVB (280-320 nm) radiation coupled to a 2.5 kW xenon arc created ~ 100 cm² of exposure area sufficient to expose all mice. As there is no mammalian melanoma “action” or wavelength dependent spectrum available for use in measuring melanoma-effective doses, we chose to use the CIE (Commission Internationale de l’Eclairage) erythemal or sunburn action spectrum (see Methods). **b**, The erythemal-effective dose delivered to neonatal mice from the UV source was calculated to be 15 Standard Erythemal Effective doses (SED). The absolute UVB dose delivered was 6 kJ/m² (output peak = 308 nm). In contrast, for UVA exposure, it was necessary to use the absolute rather than an erythemal-effective dose because UVA is far less effective than UVB in producing erythema. A dose of 150 kJ/m² (output peak = 375 nm) was delivered to the mice from the isolated UVA waveband. To put this in perspective, in sunlight, in mid-summer at Northern Hemisphere mid-latitudes between 11 am and 3 pm, 150 kJ/m² of UVA would have been received in approximately 50 minutes of sunlight exposure. In that time, an erythemal dose of about 7 SED would have been delivered, a dose we have previously determined to be melanomagenic (data not shown). **c**, Mice were irradiated in a specially designed module with a quartz lid, and three separate compartments capable of accommodating up to 12 neonates or 3 adult mice and evenly spaced air inlets around the module. The module sits on top of a rotating platform (3 RPM) to evenly distribute the circular UV beam to all compartments.

Supplementary Figure 4



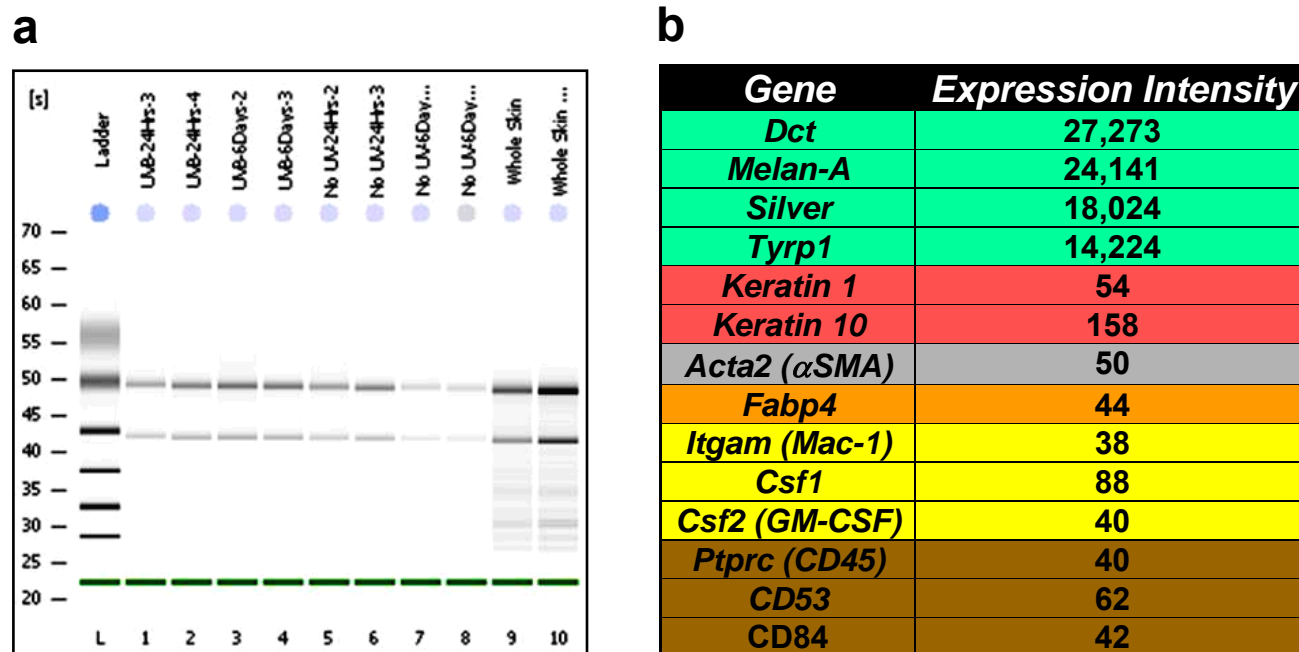
Supplementary Fig. 4. **a**, Percentage of GFP⁺ melanocytes in single cell suspensions made from back skins of neonatal *iDct-GFP* mice at ages of 2 days (PD2) and 7 days (PD7), following irradiation at PD1 with either UVA or UVB. No statistical differences in the GFP⁺ melanocytic populations were seen among the unirradiated and UVA-irradiated pups at either PD2 or PD7. However, UVB-irradiated pups exhibited statistically significant increase (~2-fold) in GFP⁺ melanocytes at PD7, as compared with the unirradiated control and UVA-irradiated PD7 pups. The numbers on each bar represent the number of biological replicates examined. All samples were quantified via flow cytometry. Every replicate consisted of back skins pooled from 6-8 pups. ** $P < 0.01$; *** $P < 0.001$; Student's t-test. **b**, UVB did not activate melanocytes in adult mice irradiated at PD29 and examined at PD35. Blue, DAPI. E, epidermis; D, dermis. Scale bars = 40 μm .

Supplementary Figure 5



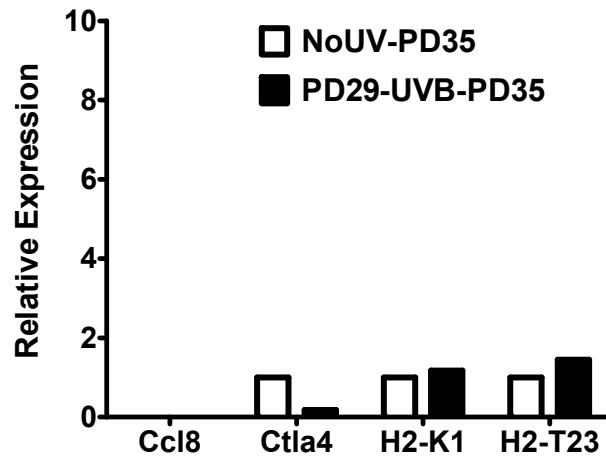
Supplementary Fig. 5. Representative FACS sorting schemes are shown for isolation of GFP+ melanocytes from UVB (a) and UVA (b) irradiated *iDct-GFP* pup skins at 6 days post irradiation (PD7). Pre-sort analyses of the whole skin cell suspensions (left panels) showed 1-2% GFP+ cells in the UVA irradiated skins (equivalent to unirradiated controls), but an elevated 2.5-3.5% GFP+ cells in the UVB-irradiated pups (circled). Post-sort analyses (right panels) showed high enrichment (>95%, circled) of GFP+ melanocytes.

Supplementary Figure 6



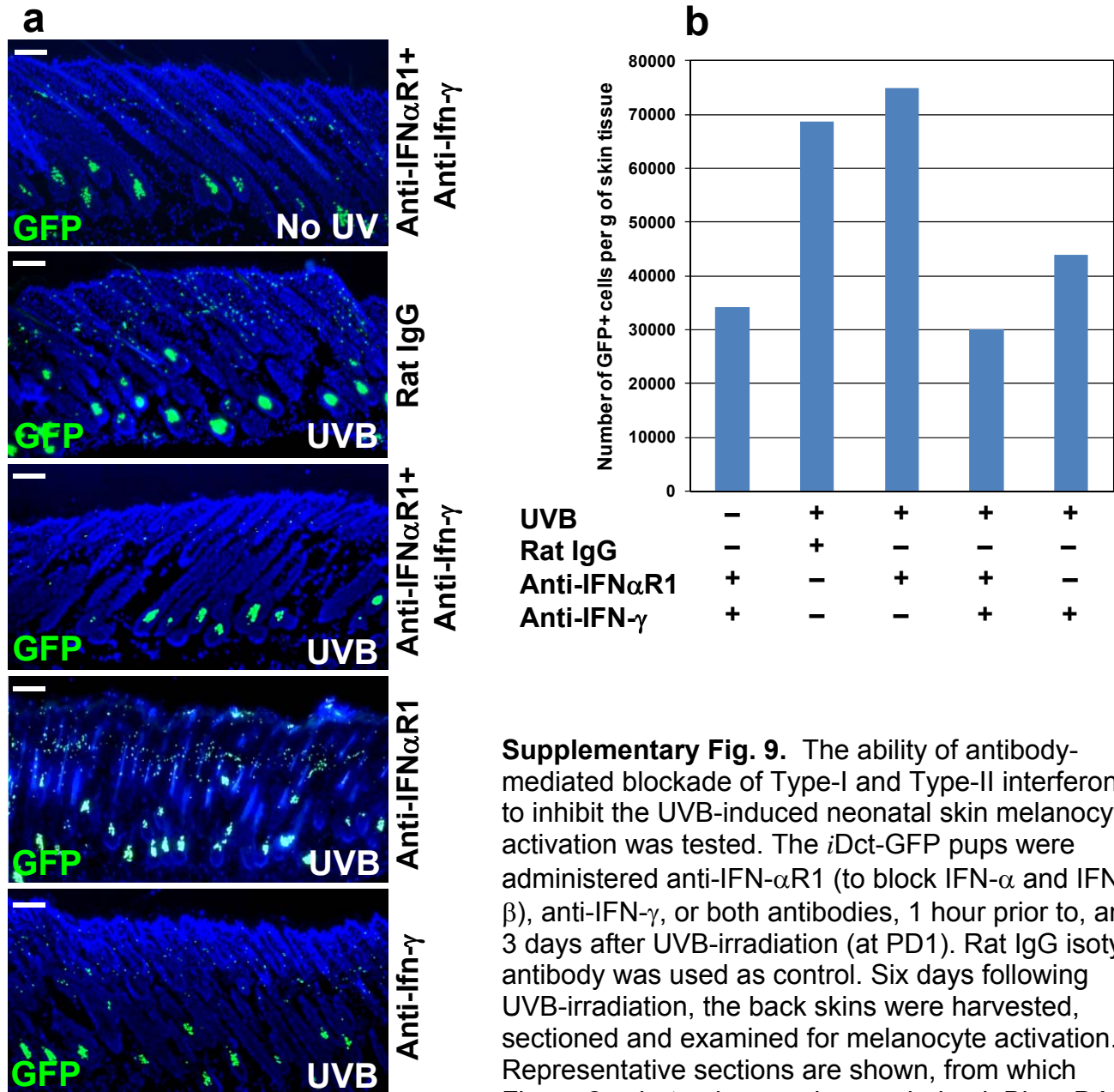
Supplementary Fig. 6. **a**, Bioanalyzer analysis of the total RNA extracted from the FACS-sorted GFP⁺ melanocytes showed high quality RNA. **b**, cDNA microarray analysis of gene expression showed that the FACS-sorted GFP⁺ cells were indeed highly pure melanocytes. The mean expression intensities detected on the microarray for melanocyte-specific genes (green) were extremely high and represented among the highest detected gene intensities on the array; *Dct* was in fact the highest. In contrast, expression of genes specific to keratinocytes (red), fibroblasts (grey), adipocytes (orange), macrophages (yellow), and leukocytes (brown) were below the threshold of detection. This proved that the FACS-sorted GFP⁺ cells were highly pure melanocytes and were devoid of contamination from other cell types found in the cellular milieu of the mouse skin.

Supplementary Figure 8



Supplementary Fig. 8. In order to determine whether the adult skin melanocytes exhibit the same gene expression response to UVB as the neonatal skin melanocytes, adult *iDct-GFP* mice were UVB-irradiated at age PD29, and melanocytes were FACS-isolated 6 days later at PD35. qRT-PCR analysis of the indicated genes showed no upregulation in adult skin melanocytes in response to UVB irradiation, as compared to the unirradiated controls.

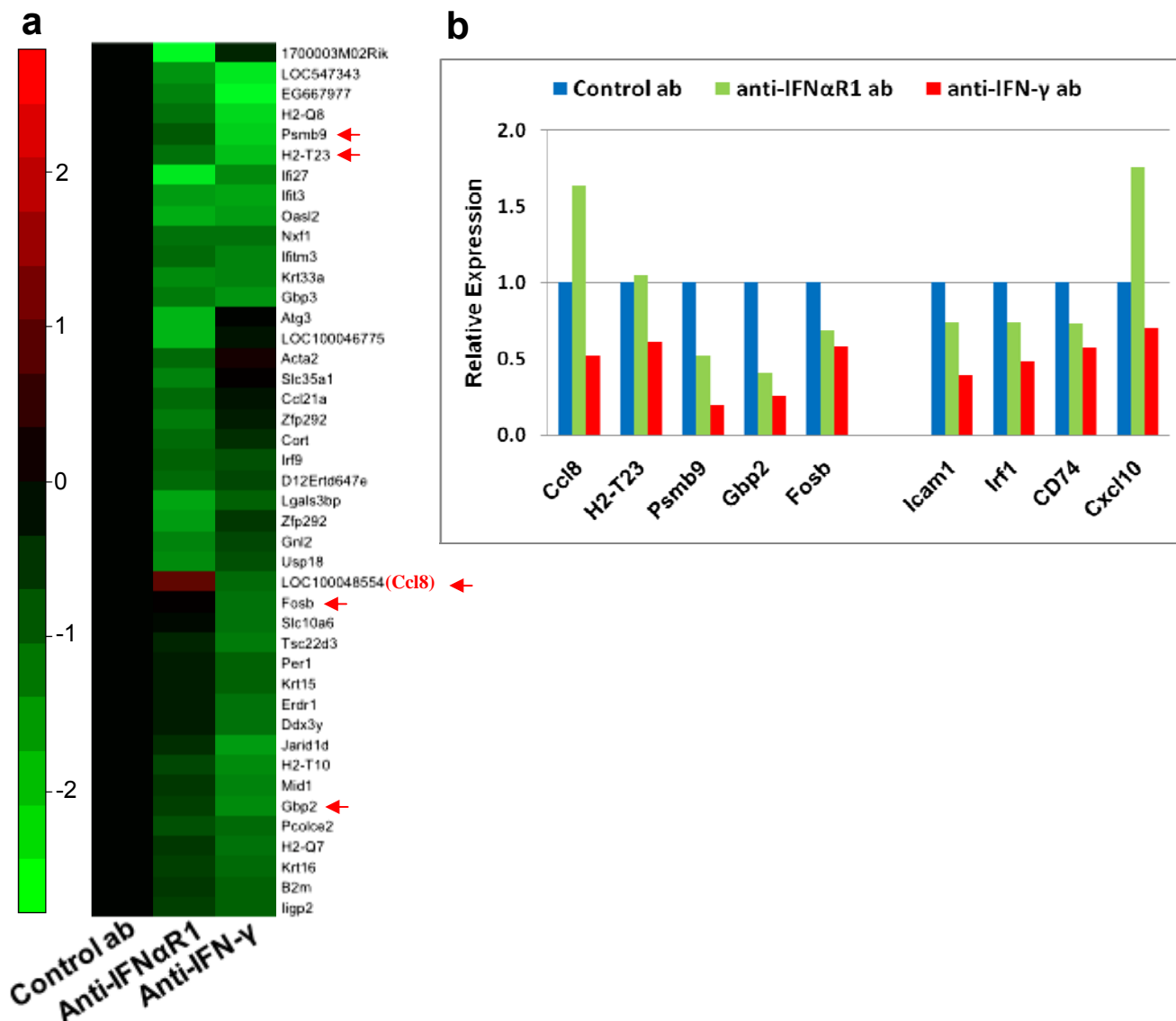
Supplementary Figure 9



Supplementary Fig. 9. The ability of antibody-mediated blockade of Type-I and Type-II interferons to inhibit the UVB-induced neonatal skin melanocyte activation was tested. The *iDct-GFP* pups were administered anti-IFN- α R1 (to block IFN- α and IFN- β), anti-IFN- γ , or both antibodies, 1 hour prior to, and 3 days after UVB-irradiation (at PD1). Rat IgG isotype antibody was used as control. Six days following UVB-irradiation, the back skins were harvested, sectioned and examined for melanocyte activation. **a**, Representative sections are shown, from which Figure 2c photomicrographs are derived. Blue, DAPI.

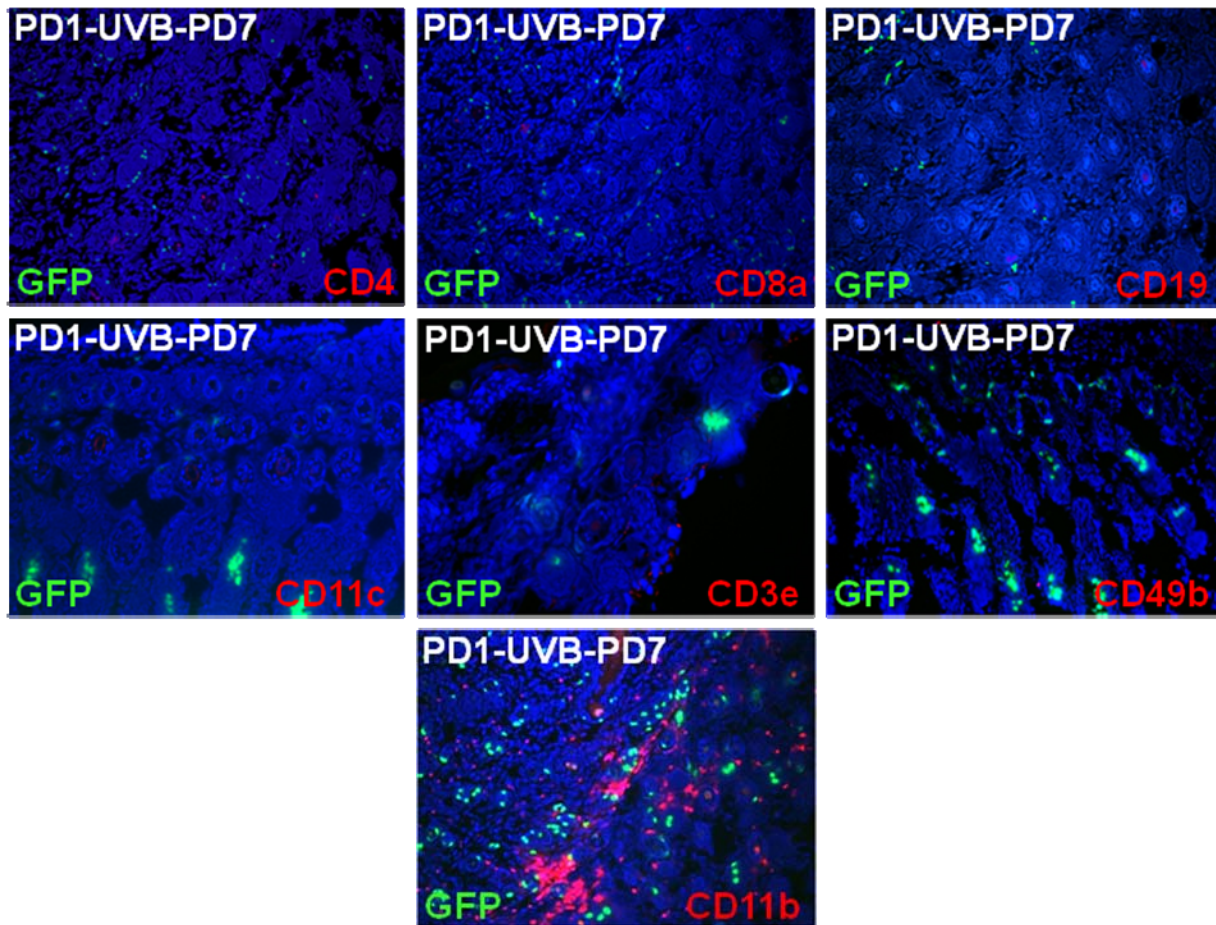
Scale bars, 40 μ m. **b**, Pooled back skins from 5-6 pups were weighed, single-cell suspensions were made, and GFP+ melanocytes were FACS-quantified per gram of skin tissue. As expected, the UVB-irradiated skins with Rat IgG isotype antibody treatment yielded twice the number of GFP+ cells as compared to the unirradiated controls with dual antibody treatment. Single antibody treatment with anti-IFN- α R1 antibody failed to affect the GFP+ cell yield. However, dual treatment with anti-IFN- α R1+anti-IFN- γ antibodies and single treatment with anti-IFN- γ antibody reduced the GFP+ cell yield by 44-57%, to levels comparable to the unirradiated skin. This showed that blockade of IFN- γ dramatically inhibited the UVB-induced melanocyte activation.

Supplementary Figure 10



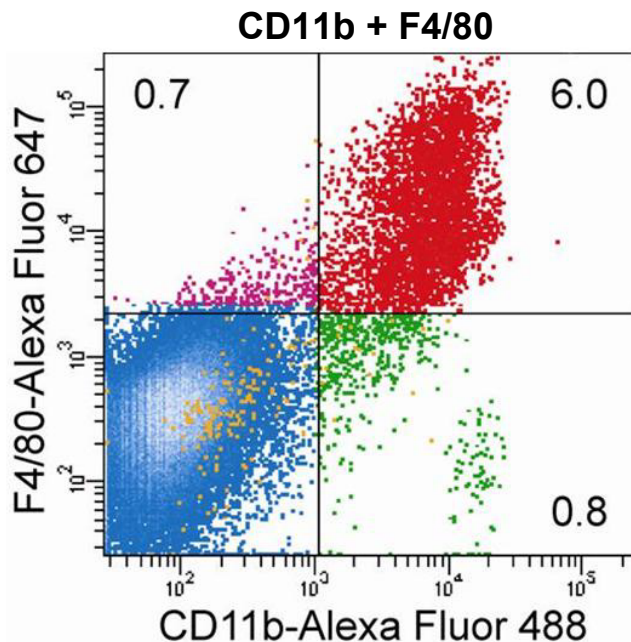
Supplementary Fig. 10. In order to delineate IFN Type-I and Type-II specific gene expression response to UVB, *iDct*-GFP pups were administered anti-IFN- α R1 (to block type-I IFN) or anti-IFN- γ antibodies, 1 hour prior to, and 3 days after UVB irradiation (at PD1). Rat IgG isotype (clone GL113) antibody was used as control. Six days following irradiation, GFP⁺ melanocytes from back skins (pooled from 6 pups for each sample) were FACS-isolated, and cDNA microarrays were performed. **a**, Heatmap of unsupervised clustering of genes that were >1.5-fold changed in either anti-IFN antibody-treated sample as compared to the control antibody-treated sample. As expected, a subset of genes was concurrently downregulated by both Type-I and Type-II IFN blockade. In addition, Type-I- and Type-II-specific genes were also evident. **b**, qRT-PCR validations were performed for 5 select genes indicated in the heatmap (red), as well as for four other known IFN-responsive genes (rightmost 4).

Supplementary Figure 11



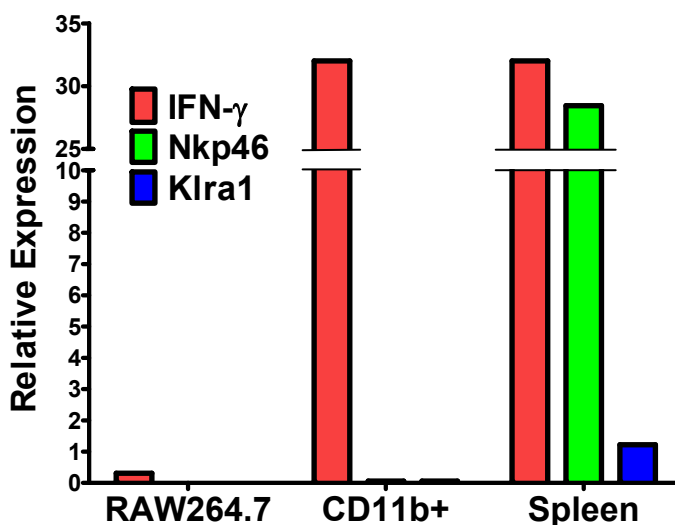
Supplementary Fig. 11. Immunohistochemical (IHC) analyses for a variety of immune cell types in *iDct*-GFP neonatal skins that were UVB-irradiated at PD1 and harvested at PD7. IHC with anti-CD4 (T cell-specific), anti-CD8a (T cells), anti-CD19 (B cells), anti-CD11c (dendritic cells), anti-CD3e (NK-T cells), and anti-CD49b (DX5; natural killer cells) showed no immunostaining. However, a substantial number of CD11b⁺ cells (myeloid cells) were readily identifiable.

Supplementary Figure 12



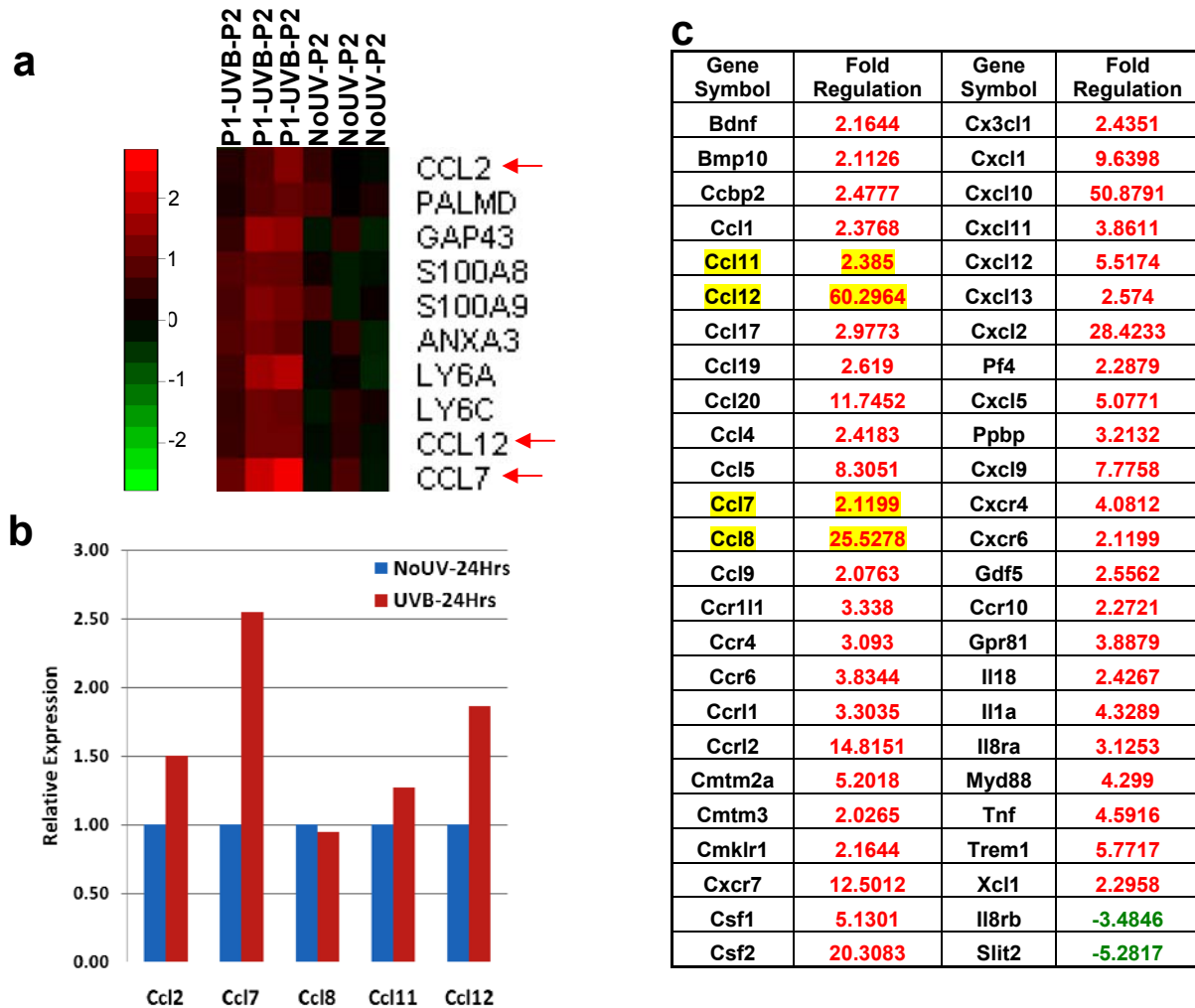
Supplementary Fig. 12. Dual fluorescence flow cytometry analysis of single-cell suspensions from back skins of *iDct-GFP* pups UVB-irradiated at PD1 and harvested at PD7 showed that 90% of the CD11b⁺ cells were also F4/80⁺, identifying them as macrophages. For this experiment, GFP expression was kept off by not administering dox to the *iDct-GFP* mice, which allowed use of Alexa Fluor 488-labeled anti-CD11b antibody.

Supplementary Figure 13



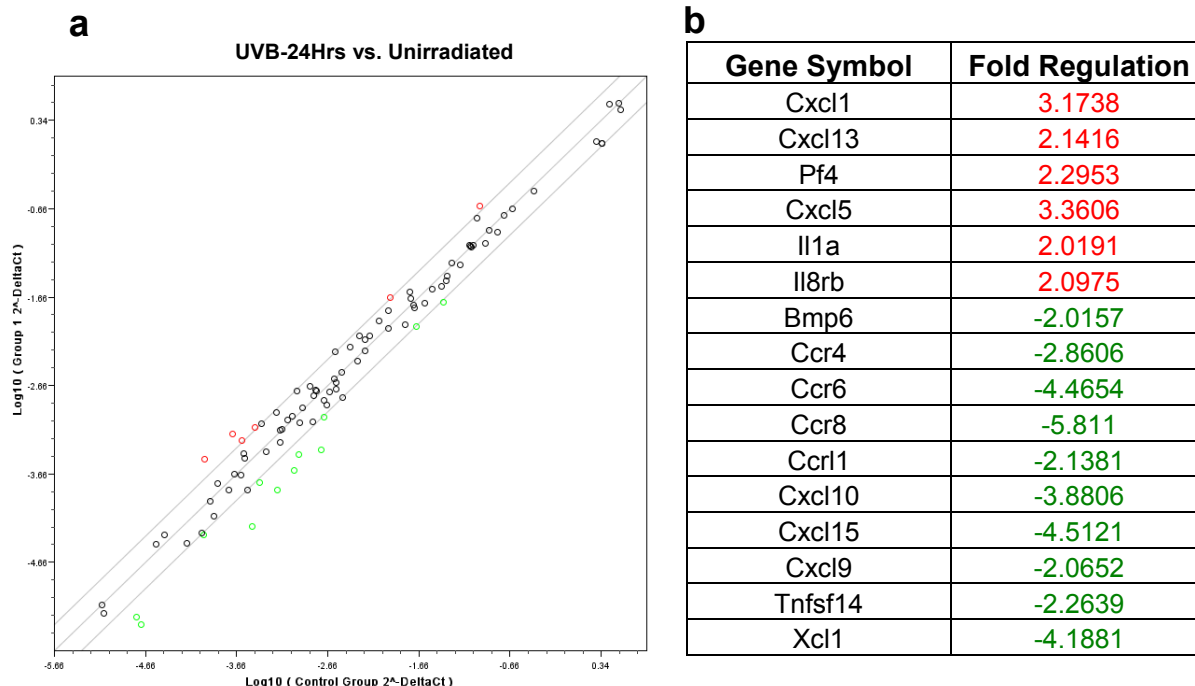
Supplementary Fig. 13. Natural killer (NK) cells are not detectable in the FACS-isolated, UVB-recruited skin CD11b⁺ population. In order to determine whether the CD11b⁺ population FACS-isolated from skin 6-days post UVB irradiation (irradiation at PD1, isolation at PD7), which show highly upregulated expression of IFN- γ , may be contaminated with NK cells, we performed qRT-PCR for two NK cell markers, Nkp46 and Klra1 on the CD11b⁺ population. We were unable to detect any expression of either of these two NK cell markers in the CD11b⁺ population. Whole spleen was used as a positive control.

Supplementary Figure 14



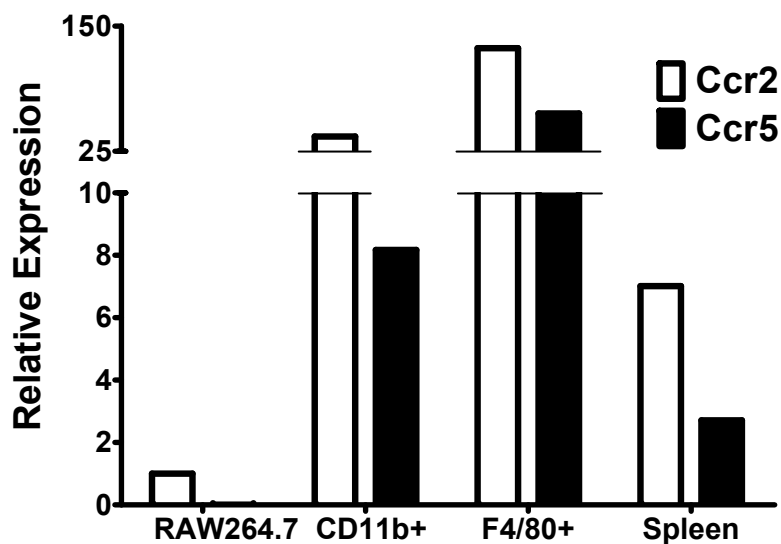
Supplementary Fig. 14. **a**, Partial heatmap depicting unsupervised hierarchical clustering of cDNA microarray data of 24 hrs post UVB irradiated and the unirradiated control samples. Three of the Ccr2/Ccr5 ligand chemokines (red arrows) show upregulated expression in the UVB-irradiated samples as compared to the unirradiated control samples. **b**, qRT-PCR validation for the Ccr2/Ccr5 ligands was performed on FACS purified melanocytes from back skins of pups UVB-irradiated at P1 and harvested 24 hrs later. Unirradiated pups of same age were used as controls. Means of two experiments are plotted, with each experiment containing pooled skins from 6-8 pups. **c**, RT² Profiler PCR Array (SABiosciences, Inc.) for mouse chemokines and receptors (PAMM-022A; 84 genes) was performed on total RNA isolated from the melan-c melanocyte cell line that was UV-irradiated by FS20 sunlamps and harvested 24 hrs later. 48 genes were upregulated (red) and only 2 downregulated (green) >2-fold as compared to the unirradiated controls. The ligands for Ccr2/Ccr5 are highlighted in yellow. It should be noted that Ccr2 can also bind at relatively low affinity human beta-defensin-2 and -3 (in mouse -4 and -14) (Rohrl *et al.*, *J Immunol* 184:6688-6694), which can be released following UV exposure (Glaser *et al.*, *J Allergy Clin Immunol* 123:1117-1123).

Supplementary Figure 15

RT² qPCR Array 24 Hrs post UVB irradiation in whole skin

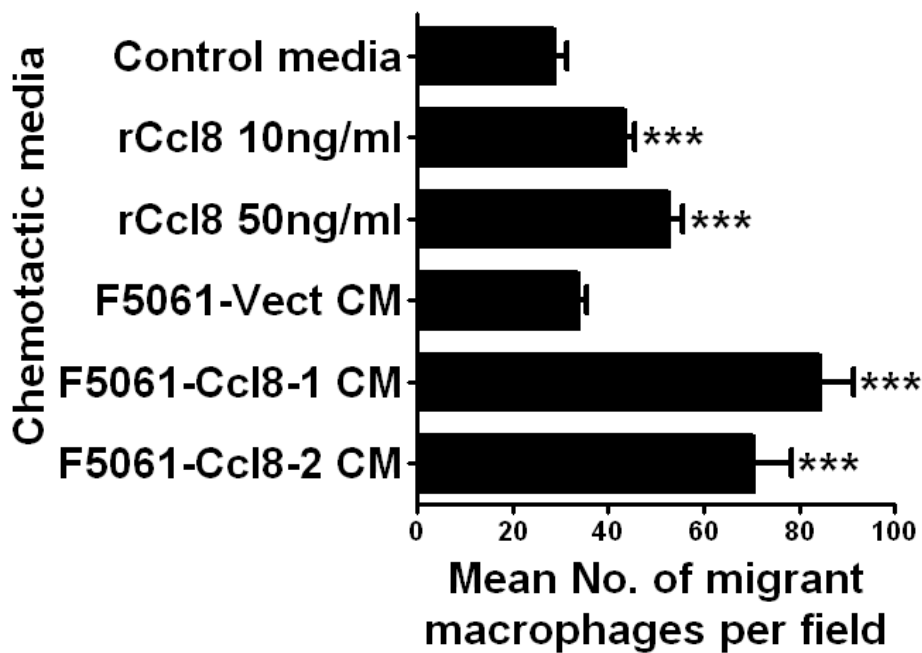
Supplementary Fig. 15. RT² Profiler PCR Array (SABiosciences, Inc.) for mouse chemokines and receptors (PAMM-022A; 84 genes) was performed on total RNA isolated from whole skin (~85% keratinocytes) samples from pups that were UVB-irradiated at PD1 and harvested 24 hours later (n=4). Unirradiated pups of same age were used as controls (n=4). **a**, Scatter plot depicting comparison of mean expression levels of the 84 genes profiled, with a 2-fold cut-off threshold. **b**, Six genes were upregulated (in red) and 10 were downregulated (green) >2-fold in the UVB-irradiated skin samples as compared to the unirradiated controls. None of the upregulated genes were known to be chemoattractants for macrophages.

Supplementary Figure 16



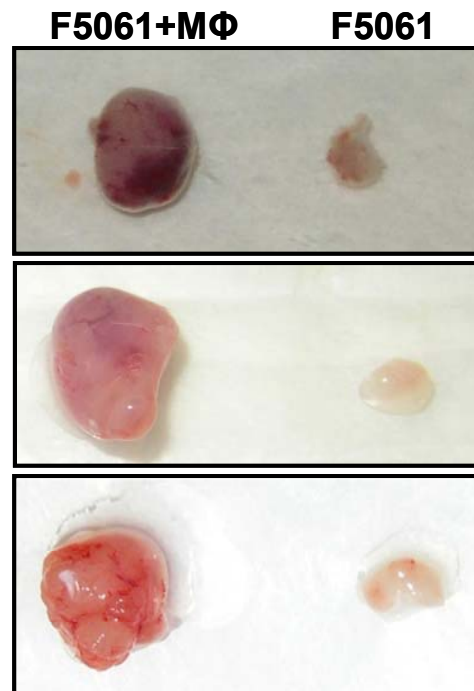
Supplementary Fig. 16. qRT-PCR analyses for Ccr2 and Ccr5 (receptors for Ccl8) were performed on CD11b+ and F4/80+ cells isolated from PD1-UVB-PD7 skin samples by immunofluorescence-FACS sorting. Non-activated RAW264.7 macrophage cell line was included as a positive control, as this cell line expresses Ccr2 and Ccr5 at baseline levels with Ccr2 being expressed at approximately 100-fold higher levels than Ccr5. Both CD11b+ and F4/80+ neonatal skin cells highly expressed Ccr2 and Ccr5 relative to non-activated RAW264.7 macrophages.

Supplementary Figure 17



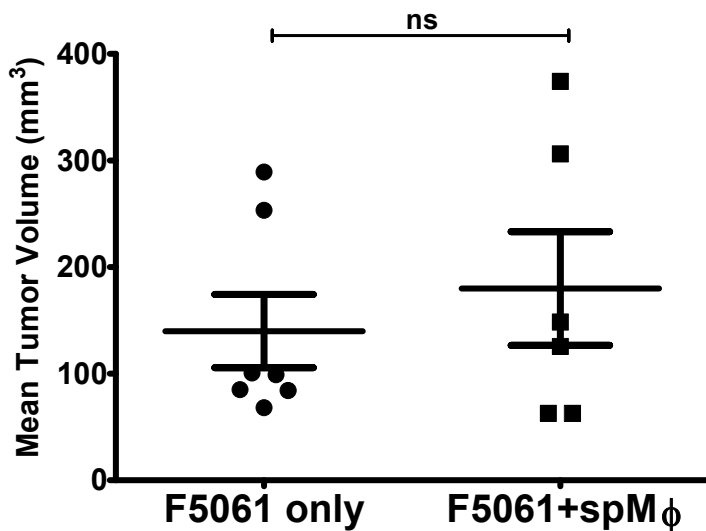
Supplementary Fig. 17. Boyden chamber chemotactic assay with conditioned media (CM) from Ccl8-transfected or vector-transfected F5061 melanoma cells (a cell line established from a UVB-induced melanoma in our HGF/SF transgenic mouse model). Recombinant mouse Ccl8 (rCcl8) in indicated concentrations was used as positive control. Migration of RAW264.7 macrophage cells was quantified in duplicate with 5 random fields observed per chamber. Mean number of migrant cells per field \pm s.e.m. are depicted. *** p <0.001, One-Way ANOVA test with post-hoc Tukey analysis. The results confirm that Ccl8 is a potent macrophage chemoattractant (Van Damme *et al.*, *J Exp Med* 176:59-65).

Supplementary Figure 18



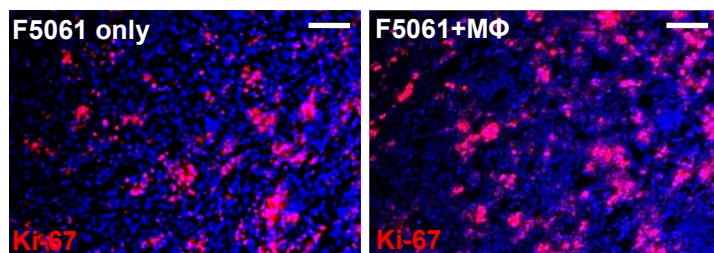
Supplementary Fig. 18. Macrophages (M Φ) recruited to the skin after UVB irradiation are pro-tumourigenic. F4/80⁺ macrophages were FACS-isolated from PD1-UVB-PD7 pup skins, and admixed with the F5061 cells (a cell line established from a UVB-induced melanoma in our HGF/SF transgenic mouse model) in a 1:5 ratio (50,000 M Φ + 250,000 F5061) and inoculated subcutaneously in syngeneic FVB/N mice (n=10). Control mice were inoculated with 250,000 F5061 cells only (n=10; one mouse died prematurely and was removed from the analysis). The experiment was performed in three different batches. The experiments were terminated 3 weeks after inoculation and tumour volumes were measured as described in Methods. Representative pairs of admixed and control tumours are shown from the three experiments, clearly demonstrating a growth advantage to the F5061+ M Φ admixed tumours.

Supplementary Figure 19



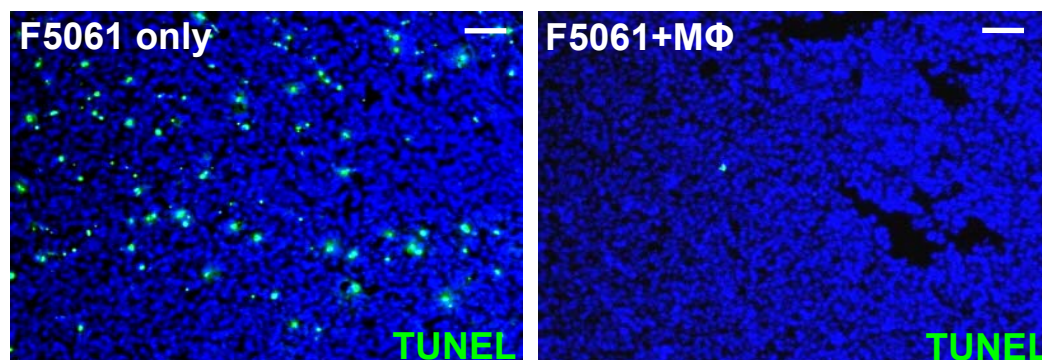
Supplementary Fig. 19. Macrophages isolated from spleens (spM Φ) of unirradiated pups did not significantly affect the growth of subcutaneously transplanted F5061 melanoma cells. F4/80+ splenic macrophages were isolated by FACS from single-cell suspensions made from spleens of unirradiated 7-day old pups. These spM Φ were admixed with F5061 cells in a 1:5 ratio (50,000 M Φ + 250,000 F5061) and inoculated subcutaneously in syngeneic FVB/N mice (n=6). Control mice were inoculated with 250,000 F5061 cells only (n=7). The experiment was terminated 3 weeks after inoculation, and tumor volumes were measured. $P = 0.52$, Student's t-test. ns = not significant.

Supplementary Figure 20



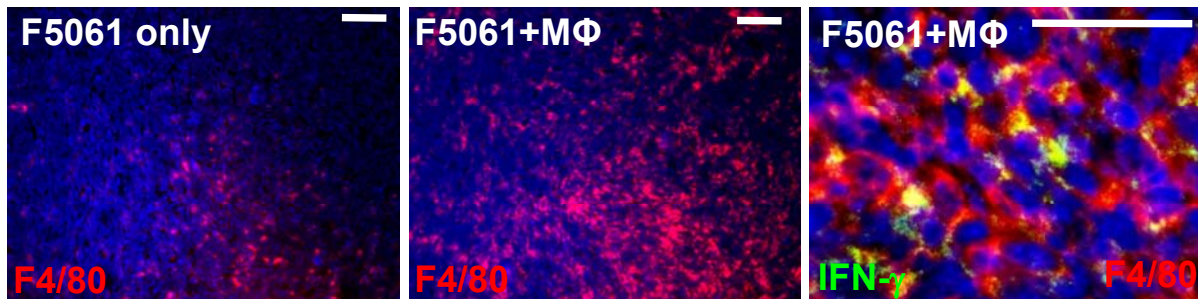
Supplementary Fig. 20. The F5061+ M Φ admixed tumours and the F5061 only control tumours exhibited equivalent Ki-67 immunohistochemical positivity (n=6 each group). Representative photomicrographs are shown. These results show that differential growth of these two tumour groups is not due to differences in cellular proliferation. Scale bars = 60 μ m.

Supplementary Figure 21



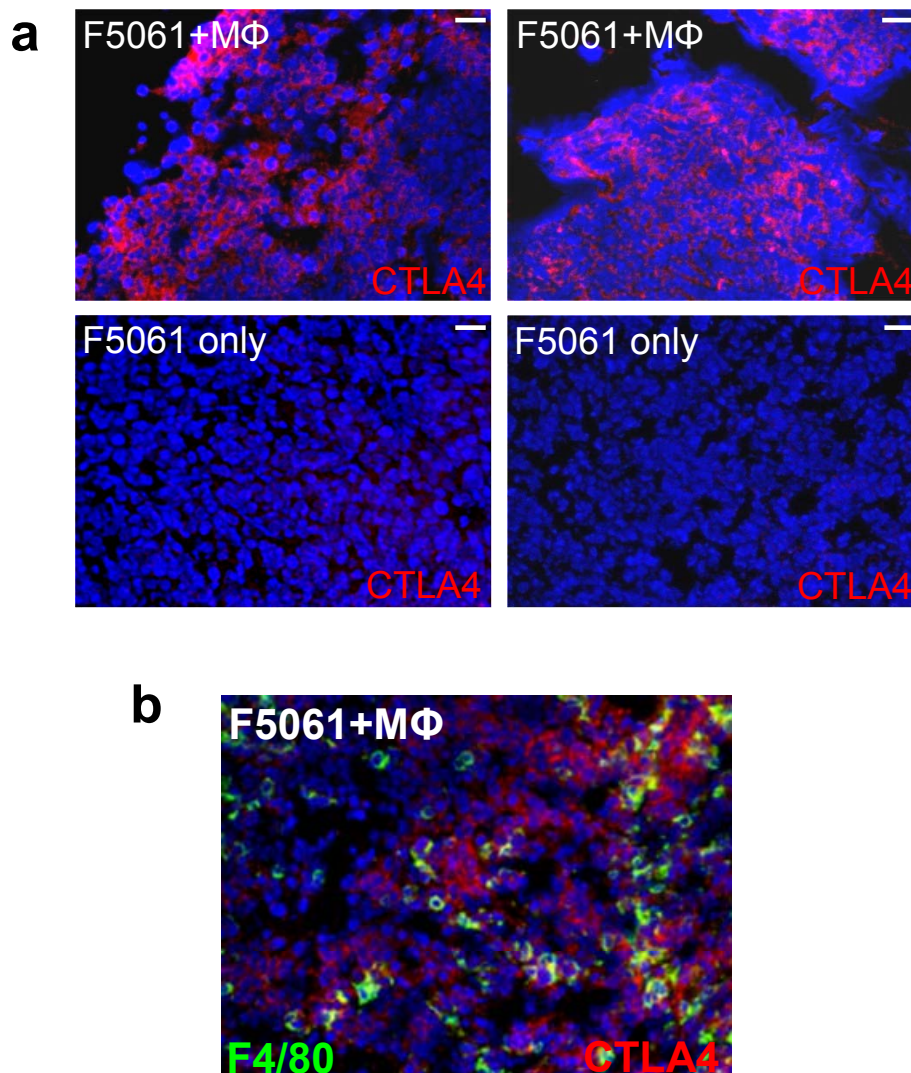
Supplementary Fig. 21. Fluorescence-based TUNEL assay showed dramatically different levels of apoptosis between F5016+M Φ admixed and control tumours (n=6 each). The apoptosis levels were significantly lower in the admixed tumours than in the control tumours. Representative photomicrographs are shown. Scale bars = 40 μ m.

Supplementary Figure 22



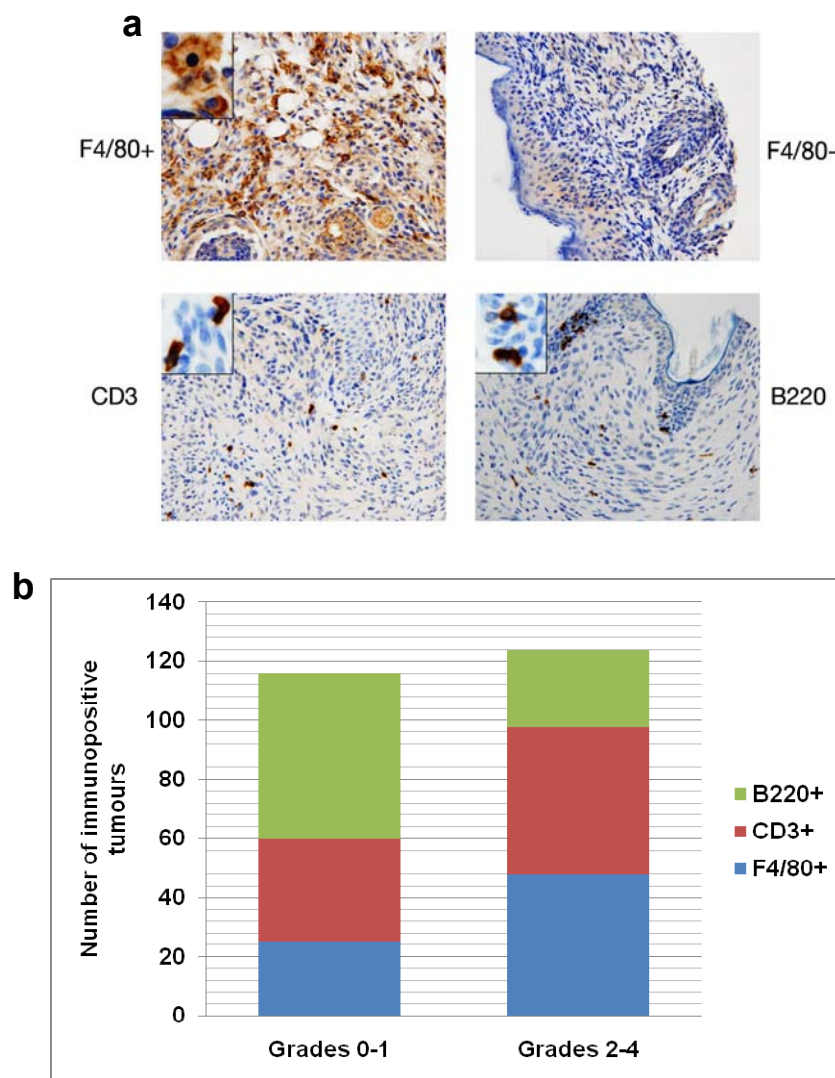
Supplementary Fig. 22. IHC analysis for presence of macrophages in control (F5061 only; left panel) and admixed (F5061+MΦ; middle panel) tumours. The survival advantage in the admixed tumours coincided well with the continued presence of macrophages in the admixed tumours; in fact, the number of tumour-associated macrophages was overtly greater than the original number of macrophages transplanted, indicating recruitment by positive feedback. Anti-F4/80 and anti-IFN- γ dual IHC showed that a subset of these macrophages expressed IFN- γ (right panel; yellow merged signal). Scale bars = 40 μ m and 130 μ m.

Supplementary Figure 23



Supplementary Fig. 23. a, Since CTLA4 has been shown to be positively regulated by IFN- γ (e.g. Wong *et al.*, Journal of Investigative Dermatology 126:212, 2006), we tested whether CTLA4 was upregulated in the tumours with admixed skin macrophages. The F5061+M Φ admixed tumours exhibited high levels of CTLA4 immunopositivity. All 6 of 6 admixed tumours examined were positive for CTLA4. In contrast, only 1 of 6 F5061 only control tumours (lower left panel) showed faint positivity for CTLA4, while all others were negative. Representative photomicrographs are shown. Scale bars = 40 μ m. **b**, Dual IHC with anti-F4/80 (green) and anti-CTLA4 (red) antibodies shows CTLA4 expression predominantly in melanoma cells but not in macrophages.

Supplementary Figure 24

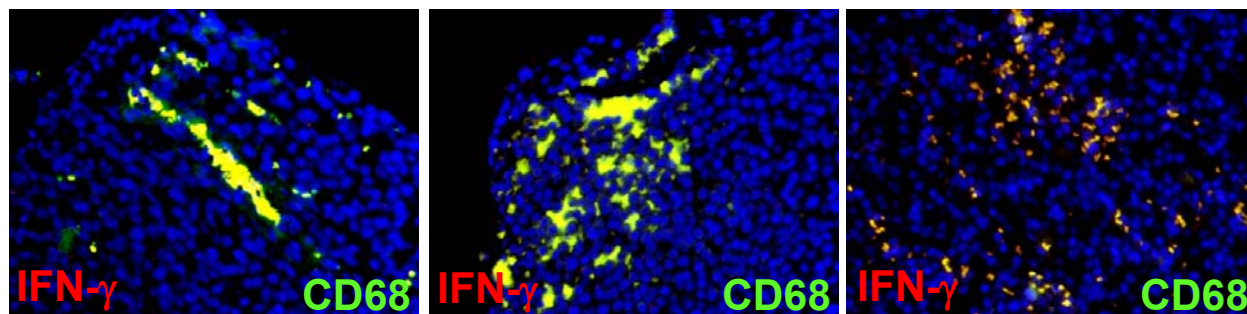


Supplementary Fig. 24. **a**, Immunohistochemical staining for F4/80 (macrophage marker), CD3 (T cell marker) and B220 (B cell marker) were performed on a tissue microarray (TMA) of UVB-induced melanomas from our albino HGF/SF transgenic mouse model. Immunostaining was evaluated according to the following grading system: 0=negative, 1=minimal positivity, 2=mild positivity, 3=moderate positivity, and 4=severe positivity. Representative photomicrographs (10x) are shown with high magnification insets (100x). **b**, Graphical representation of the number of tumours that were found to be either negative-minimally positive (grades 0-1) or mild-strongly positive (grades 2-4).

Supplementary Figure 25

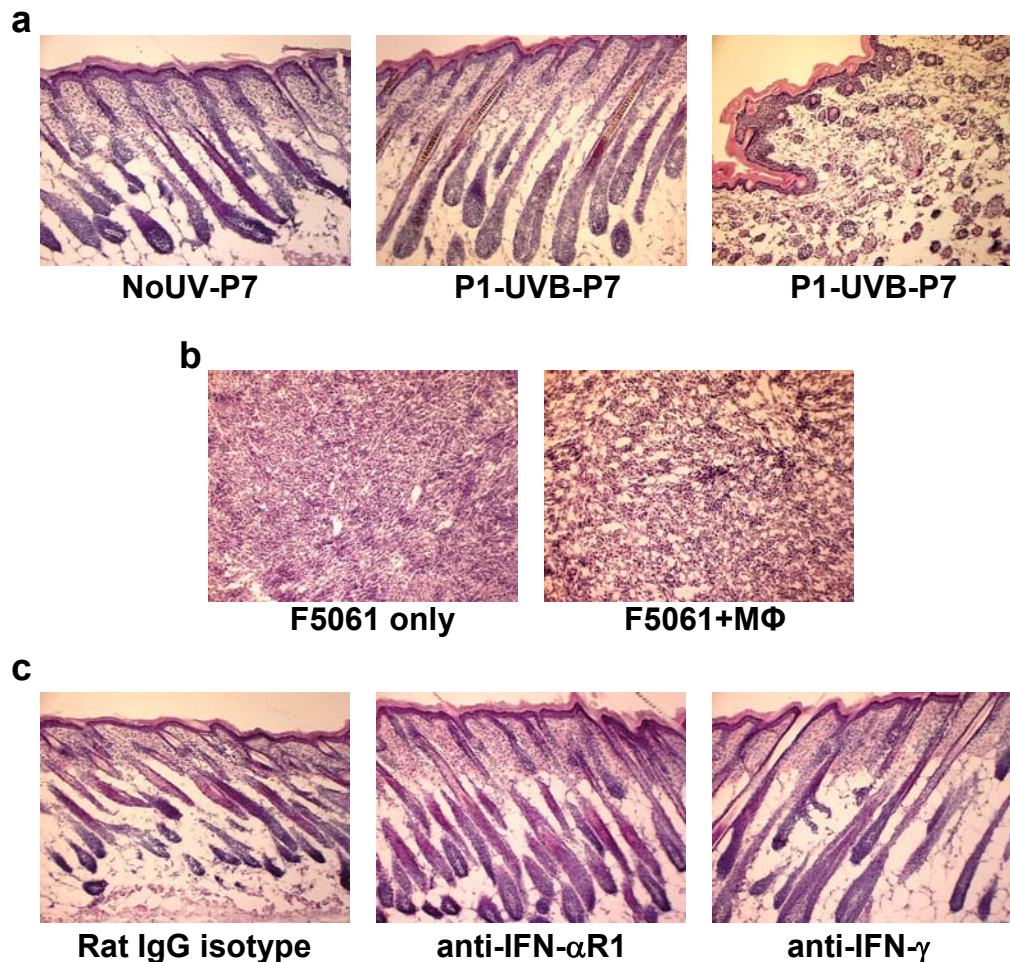
a

Human Melanoma Tissue Microarray	
Melanomas on the array	27
CD68-	8
CD68+	19
CD68+IFN-g+	19

b

Supplementary Fig. 25. Dual immunohistochemistry with anti-CD68 (green) and anti-IFN- γ (red) antibodies on a human melanoma tissue microarray (TMA) showed tumour-associated macrophages expressing IFN- γ . **a**, Details of the human melanoma TMA. The TMA included 11 primary, 7 lymph node metastases, 5 distant metastases, and 4 human melanoma cell line xenografts. **b**, Three different representative human melanomas from the TMA show co-immunostaining of CD68 and IFN- γ (yellow).

Supplementary Figure 26



Supplementary Fig. 26. Representative hematoxylin & eosin stained photomicrographs. **a**, Histological skin sections from the pups UVB-irradiated at PD1 and harvested at PD7, and age-matched unirradiated control (corresponding to Figure 1d). **b**, Histological sections from subcutaneously transplanted tumors from F5061 alone and F5061 with admixed macrophages. **c**, Histological skin sections from UVB-irradiated pups (irradiated at PD1, harvested at PD7), administered with the indicated antibodies (corresponding to Figure 2c). Magnification 10x for all panels.

Supplementary Table 1

List of IFN- γ -specific responsive genes* among the top 20% most significantly altered genes in melanocytes at 6 days post-UVB time point

<i>Gene Symbol</i>	<i>Gene Name</i>	<i>Regulation</i>
<i>Psmb8</i>	Proteasome subunit, beta type, 8	Up
<i>Psmb9</i>	Proteasome subunit, beta type, 9	Up
<i>Psmb10</i>	Proteasome subunit, beta type, 10	Up
<i>Ccl8</i>	Chemokine (C-C motif) ligand 8	Up
<i>Ciita</i>	Class II transactivator	Up
<i>Casp1</i>	Caspase 1	Up
<i>C3</i>	Complement component 3	Up
<i>C4a</i>	Complement component 4a	Up
<i>C4b</i>	Complement component 4b	Up
<i>Hira</i>	Histone cell cycle regulation defective homolog A	Up
<i>Tmod1</i>	Tropomodulin 1	Up
<i>Tap1</i>	Transporter 1, ATP-binding cassette, subfamily B	Up
<i>Tap2</i>	Transporter 2, ATP-binding cassette, subfamily B	Up
<i>Il7r</i>	Interleukin 7 receptor	Up
<i>Ctss</i>	Cathepsin S	Up
<i>Ccl5</i>	Chemokine (C-C) ligand 5	Up
<i>Aif1</i>	Allograft inflammatory factor 1	Up
<i>B2m</i>	Beta-2-microglobulin	Up
<i>Foxp3</i>	Forkhead box P3	Up
<i>Serpina3g</i>	serine peptidase inhibitor, clade A, member 3G	Up
<i>Slc6a6</i>	solute carrier family 6, member 6	Up
<i>Tnfrsf1b</i>	Tumor necrosis factor receptor superfamily, member 1B	Up
<i>Bach1</i>	BTB and CNC homology 1, basic leucine zipper transcription factor 1	Up

* These genes are known to be responsive to IFN- γ , but not to Type-I interferons (IFN- α and IFN- β). See ref. 7 and references therein.

Supplementary Table 2

List of primers

<i>Gene</i>	<i>Forward (F) and Reverse (R) Primers</i>	<i>Source</i>
<i>Real-Time RT-PCR primers</i>		
Klra1	F 5'-AAGATTGGGCATGGATTGAC-3' R 5'-GATAACAACATACATCCCCCA-3'	qPrimerDepot ^a
Ncr1	F 5'-GGCTCACAGAGGGACATACA-3' R 5'-GGCTGCTGTTCTCAACACCT-3'	qPrimerDepot
Ccr2	F 5'-AACAGTGCCCAGTTTTCTATAGG-3' R 5'-CGAGACCTCTTGCTCCCCA-3'	qPrimerDepot
Ccr5	F 5'-CAAGACAATCCTGATCGTGCAA-3' R 5'-TCCTACTCCCAAGCTGCATAGAA-3'	qPrimerDepot
H2-K1	F 5'-GAGCAGTGGTTCCGAGTGA-3' R 5'-GGTCTTCGTTCCAGGGCGATG-3'	Primer Express ^b
H2-T23	F 5'-GACTTGAAAGCCAGGGACAT-3' R 5'-CACGTCGCAGCCGTACATC-3'	Primer Express
18S rRNA	F 5'-CTTAGAGGGACAAGTGGCG-3' R 5'-ACGCTGAGCCAGTCAGTGTA-3'	Primer Express
Primers for Ccl8, Ifna1, Ifnb1, and Ifng were obtained from RealTimePrimers.com ^c		
<i>Genotyping primers</i>		
TRE-H2BGFP	F 5'-GCCACAAGTTCAGCGTGTCC-3' R 5'-GATGCCCTTCAGCTCGATCC-3'	
Dct-rtTA	F 5'-ACTAAGTAAGGATCAATTCAG-3' R 5'-TGTACTAGGCAGACTGTG-3'	

^aThese primer sequences were obtained from the qPrimerDepot database (Cui *et al.*, 2007, *Nucleic Acids Res.* **35**, D805-D809).

^bThese primers were designed using the Primer Express software (Applied Biosystems).

^cReal Time Primers, LLC., Elkins Park, PA, USA.

Supplementary Table 3

List of commercially available antibodies

Antibody	Host	Conjugate	Supplier
<i>Flow cytometry</i>			
CD11b	Rat	Alexa Fluor 488	BioLegend
F4/80	Rat	Alexa Fluor 647	BioLegend
IFN- γ	Rat	PE	BioLegend
NKp46	Rat	Alexa Fluor 647	eBioscience
IgG2b Isotype	Rat	Alexa Fluor 488	BioLegend
IgG1 Isotype	Rat	PE	BioLegend
IgG2a Isotype	Rat	Alexa Fluor 647	BioLegend
IgG Isotype	Hamster		Invitrogen CALTAG
IgG1 Isotype	Mouse		BD Pharmingen
<i>Immunohistochemistry</i>			
IFN- γ (human)	Rabbit		Lifespan Biosciences
IFN- γ (mouse)	Rabbit		Lifespan Biosciences
CD68	Mouse		Invitrogen
Ki-67	Rabbit		Epitomics
CD49b	Rat		BioLegend
CD11b	Rat		BD Pharmingen
F4/80	Rat		BioLegend
CD8a	Rat		BD Pharmingen
CD19	Rat		BD Pharmingen
CD4	Rat		BD Pharmingen
CD3e	Arm. Hamster		BD Pharmingen
CD11c	Arm. Hamster		BD Pharmingen
Gr-1	Rat		BD Pharmingen
CD16/32	Rat		BioLegend
Ctla4	Syr. Hamster		BioLegend
<i>Others</i>			
HRPN IgG1	Rat		BioXcell
LIVE/DEAD Fixable Dead cell stain kit			Invitrogen