**Supplementary tables and figures legends**

**Supplementary Table 1.** A list of all molecules detected by antibody microarray (Multiplex analysis) with their UniPort entry name. Repeated listing of some molecules indicates using different antibody clones targeting the same molecule.

**Supplementary Table 2.** GSEA raw data generated by Bubble GUM software.

**Supplementary Figure 1.** The gating strategy applied to isolate and determine the frequency of BDCA1+ DCs, BDCA1+ CD14+ cells, monocytes and MDSCsfrom PBMCS of melanoma patients (**A**), or healthy donors (**B**)by FACS.

**Supplementary Figure 2.** The distribution of potentially “missed genes” over the RPKM value spectrumin all samples that underwent RNA sequencing.

**Supplementary Figure 3.** A Venn diagram displaying the overlap between the four gene sets used in GSEA analysis: Mono high, infDC high, MoDC sig and BDCA1+ DC high.

**Supplementary Figure 4.** The T cell suppressive capacity of BDCA1+ DCs, BDCA1+ CD14+ cells, monocytes and MDSCs.(A) These subsets were isolated from healthy donors (left panel) or melanoma patients (right panel) and T cell suppression was determined in an antigen none specific setting. CD4+ T cells were stimulated with anti-CD3/anti-CD28 coated bead (1 bead per T cell) in the presence or absence of either one of the subsets that were isolated from the same donor (autologous setting). (B) The KLH-specific CD4+ T cell suppressive capacity of melanoma-derived BDCA1+CD14+ cells, using different BDCA1+CD14+ cell:CD4+ T cell ratios. The results are the mean±SEM of 3 (A left panel) or 5 (A right panel), or representative of 3 (B) independent experiments. \**P* < 0.05.

**Supplementary Figure 5.** Quantification of immunofluorescence stainings performed on sections of paraffin-embedded resection specimens of healthy and melanoma metastasized lymph nodes (A), or healthy and melanoma metastasized colon (B). BDCA1+ cells (circle), CD14+ cells (rhombus) and BDCA1+CD14+ cells (square).

**Supplementary Figure 6.** Large view, showing multiple cells, ofMay-Grünwald/Giemsa-stained healthy donor-derived, FACS-sorted BDCA1+ DCs, BDCA1+CD14+ cells and monocytes. Scale bar represents 20 µm.

**Supplementary Figure 7.** GSEA enrichment plots.

**Supplementary Figure 8.** BDCA1+ DCs, BDCA1+CD14+ cells and monocytes respond to TLR2 and TLR8 stimulation. Healthy donor-derived, FACS-sorted BDCA1+ DCs, BDCA1+CD14+ cells and monocytes were activated by either the TLR2 ligand Pam3CSK4 (1 µg/ml) or the TLR8 ligand R848 (4 µg/ml). TNF-α and IL-10 levels were determined by ELISA in 24 hours supernatants. Results are shown as mean±SEM of 3 independent experiments. \**P* < 0.05.

**Supplementary Figure 9.** IL-12 levels determined in overnight supernatants of BDCA1+ DCs, BDCA1+ CD14+ cells and monocytes cultures following stimulation with pIC and R848. Results are shown as mean±SEM of triplicates of 1 representative experiment (n=3).

**Supplementary Figure 10.** The T cell polarizing capacity of BDCA1+ DCs, BDCA1+ CD14+ cells and monocytes. (A) 10,000 BDCA1+ DCs, BDCA1+ CD14+ cells or monocytes, isolated from healthy donor PBMCs, were cultured with 40,000 allogenic naïve CD4+ T cells in the presence of 10 pg/ml SEB. T cell cultures were maintained in the presence of IL-2 till T cells were resting around day 11. Resting T cells were restimulated by PMA/Ionomycin in the presence of brefeldin .The percentage of IFN-γ+ and IL-4+ cells was determined by intracellular staining. Depicted figure is a representative of 3 independent experiments. (B) 50,000 CD4+ T cells, isolated from melanoma patient PBMCs, were cultured without or with 20,000 autologous BDCA1+ DCs, BDCA1+ CD14+ cells or monocytes, in the presence of 0.5 µg/ml SEB for 18 hours with the additional presence of brefeldin for the last 3 hours. The percentage of IFN-γ+ and IL-17+ cells was determined by intracellular staining. A representative figure is depicted.

**Supplementary Figure 11.** The effect of IL-10 on BDCA1+ CD14+ cell-induced T cell proliferation was determined by performing an MLR with allogenic naïve CD4+ T cells in the presence or absence of anit-IL-10 and anti-IL10 receptor or the matching isotype controls. Results are shown as mean±SEM of triplicates of 1 representative experiment (n=3).

**Supplementary Figure 12.** The suppressive capacity of BDCA1+ CD14+ cells is not reversed by PD-L1 blocking. KLH-specific assay was performed as described in Figure 1, with melanoma patient-derived BDCA1+ CD14+ cells without or with the additional presence of anti-PD-L1 or an isotype control. Results are shown as mean±SEM of duplicates of a representative experiment (n=2).