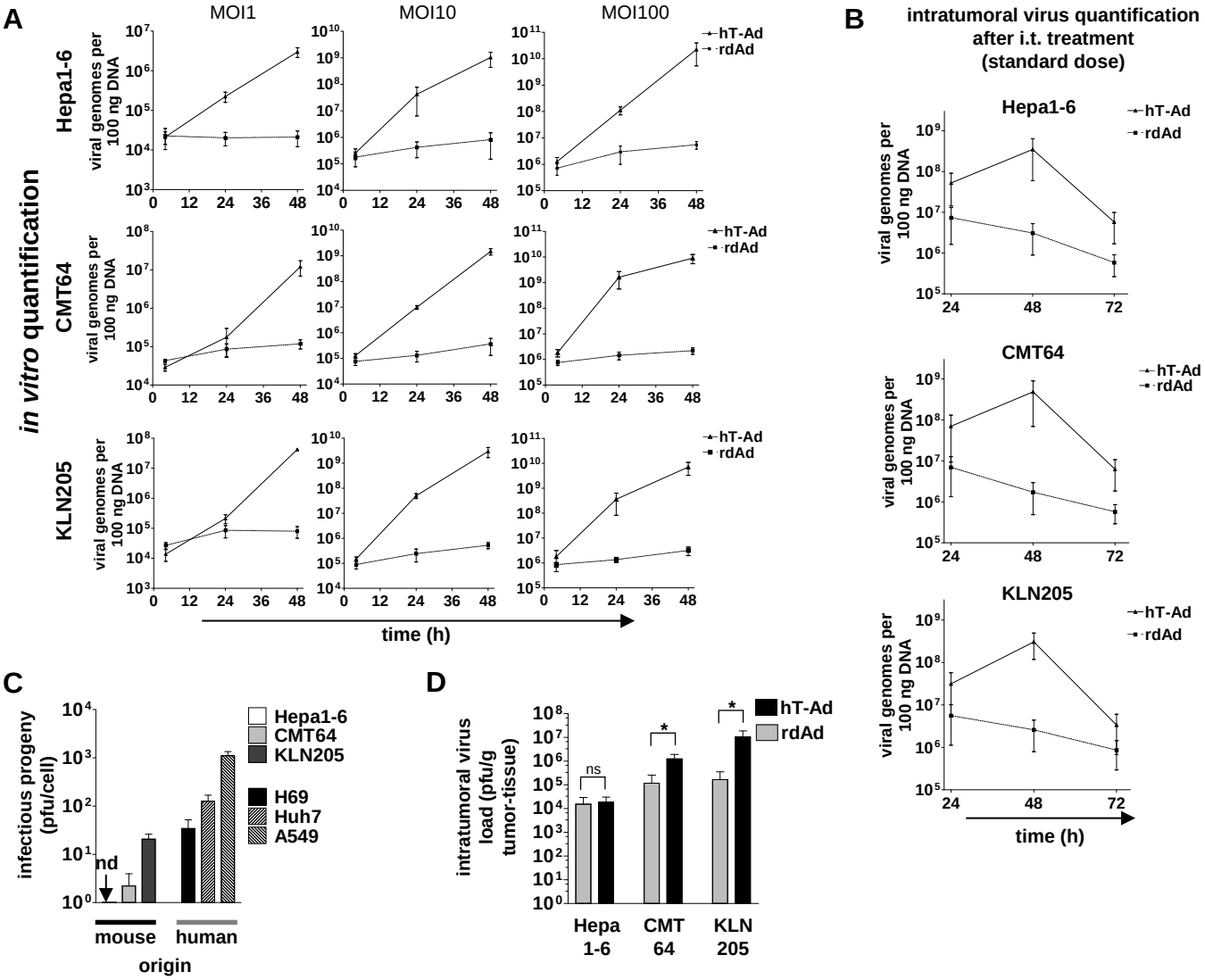
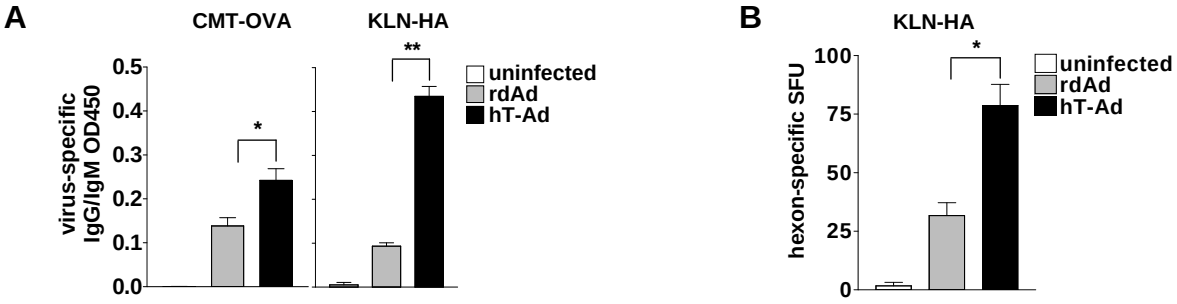
Figure S1



The murine cell lines Hepa1-6, CMT64, and KLN205 support DNA-replication of human adenovirus, but only KLN205 and CMT64 are able to produce infectious virus *in vitro* and *in vivo*

A KLN205, CMT 64 and Hepa1-6 cells were infected *in vitro* with hTert-Ad (hT-Ad) at different MOI. In all experiments a replication-deficient Ad was applied to determine the background of initial virus input. After 4h, 24h, and 48h cellular DNA was prepared and the viral genome content was quantified by hexon-qPCR. Values were normalized against the 18S genomic control. **B** KLN205-, CMT64-, or Hepa1-6-tumor bearing mice of corresponding syngeneic background received an intratumoral injection of 10^9 pfu of hTert-Ad. rdAd served as control. After 24h, 48h, and 72h tumors were explanted, three tissue-samples were prepared per tumor and total DNA was isolated for quantification of viral genomes by hexon-qPCR, normalized by genomic 18S (n=3 mice were used for each value). **C** Murine and human cell lines as indicated were infected with hTert-Ad at MOI 1. 48 h after infection, number of infected cells was determined by GFP-FACS (hTert-Ad contains a GFP-cassette), infectious progeny was released by 3x freeze/thaw cycles and titered. The content of infectious viral particles/cell was quantified (nd = not detectable). The cell lines Hepa1-6 (hepatoma), CMT64 (small cell lung cancer), and KLN205 (non-small cell lung cancer) are of mouse origin. Huh7, H69, and A549 are human cell lines from corresponding tumor species. **D** *In vivo* production of infectious viral progeny in subcutaneous CMT64-, KLN205- and Hepa1-6-tumors was determined after i.t. administration of hT-Ad. 48h post injection the tumors were explanted. Infectious viral particles were prepared from tumor-tissue slices by homogenization followed by three freeze/thaw cycles and the viral titer was assessed.

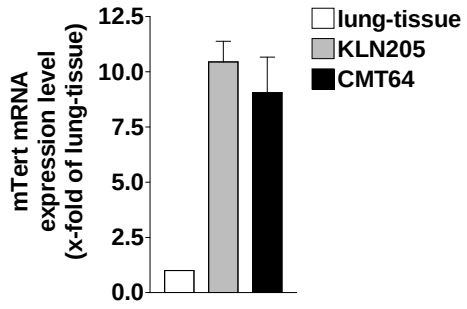
Figure S2



Replication of hTert-Ad in CMT-OVA and KLN-HA cells triggers an increased virus-specific immune response compared to a replication-defective control adenovirus

A 3 x 10^6 KLN-HA or CMT-OVA cells were infected with hTert-Ad (hT-Ad) or a replication-defective virus (rdAd) at MOI=25 and injected i.p. into syngeneic mice 24h following infection. Uninfected cells were injected as negative control. 14 days later, serum was analysed for antibodies against the viral capsid (total IgG and IgM) to investigate the effect of viral propagation on the humoral immune response. **B** KLN-HA cells were infected as described above with viruses as indicated. Infected cells were then delivered i.p. into DBA/2 mice. After one week splenocytes were harvested and subjected to ELISpot analyses to determine the cellular immune response against the viral hexon protein.

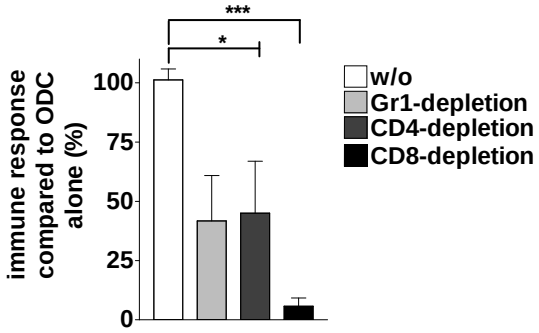
Figure S3



CMT64 and KLN205 cells express elevated mTert levels

To assess expression levels of mTert, RNA from murine lung-tissue, CMT64- and KLN205-tumors was isolated and then subjected to reverse transcription with random hexamer primer and TaqMan Reverse Transcription Reagents (Applied Biosystems). mTert cDNA-expression levels were quantified with the ΔΔCt-method using SYBR Green PCR Master Mix (Applied Biosystems). Primer for mTERT where designed with PrimerExpress-Software (Applied Biosystems): Fw-mTERT-1451 5'-GCGCCGCTTCTTTAAGAACTT-3', Rev-mTERT-1502 5'-GCCGTATTTCCCCAACGA-3'. Amplification of 5S was used as internal control. The expression levels of mTert are shown as x-fold expression of mTert-levels found in normal murine lung-tissue.

Figure S4

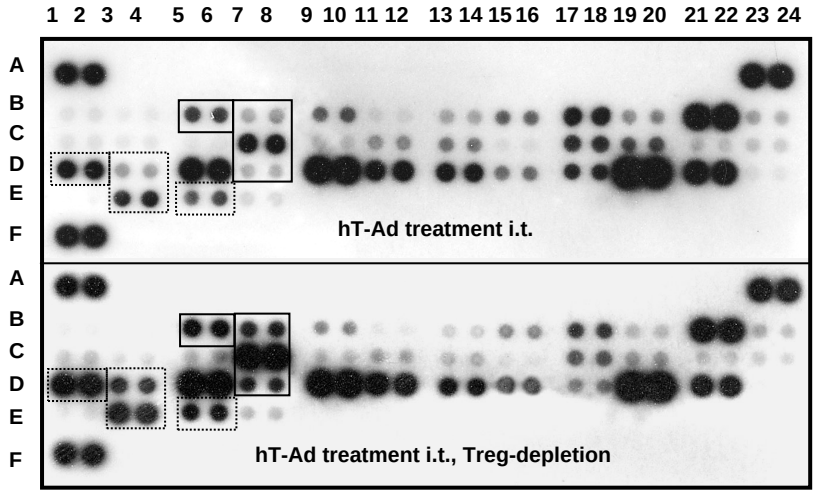


Depletion of myeloid cells or T cells results in significantly reduced effectivity of oncolysis-assisted DC-vaccination

To assess the relevance of different leukocyte populations in ODC, tumor bearing mice received depleting antibodies against leukocyte antigens as indicated in the figure. Mice without antibody-treatment served as control. After ODC-treatment, the HA-specific response was measured by ELISpot analysis (n = 6 mice used per group, HA-specific response is shown as mean ± SD).

Figure S5

	1,2	3,4	5,6	7,8	9,10	11,12	13,14	15,16	17,18	19,20	21,22	23,24
A	pos	-	-	-	-	-	-	-	-	-	-	pos
B	BLC	C5a	*G-CSF*	*GM-CSF*	CCL1	CCL11	CD54	IFN-γ	IL-1α	IL-1β	IL-1ra	IL-2
C	IL-3	IL-4	IL-5	*IL-6*	IL-7	IL-10	IL-13	IL-12p70	IL-16	IL-17	IL-23	IL-27
D	IP-10	CXCL11	KC	*M-CSF*	CCL2	CCL12	MIG	MIP-1α	*MIP-1β*	MIP-2	CCL5	CXCL12
E	CCL17	TIMP-1	TNF-α	TREM-1	-	-	-	-	-	-	-	-
F	pos	-	-	-	-	-	-	-	-	-	-	neg



Depletion of regulatory T cells during viral inflammation leads to an induction of pro-inflammatory cytokines and cytokines that can induce MDSC within the tumor microenvironment

Tumor bearing mice received an intratumoral virus infection with or without Treg depletion. Tumor-tissue was minced and tumor-associated lymphocytes were isolated as described in the methods section. Isolated lymphocytes were suspended in RPMI medium to a density of $3x10^6$ cells/ml. The cell suspension was incubated for 24h, cells were pelleted and the cytokine pattern was analysed in the supernatants using a cytokine array. For this purpose, the Proteome Profiler Mouse Cytokine Array Panel A Kit (R&D Systems) was used according to manufacturer's recommendations.

Tested cytokines are listed in the upper panel according to the array setup. The results of the array are shown below (n=3 animals per group, the experiment was repeated twice with similar results). Upregulation of proinflammatory cytokines are marked in dashed frames, whereas cytokines known to be involved in MDSC regulation are indicated by bolt frames.