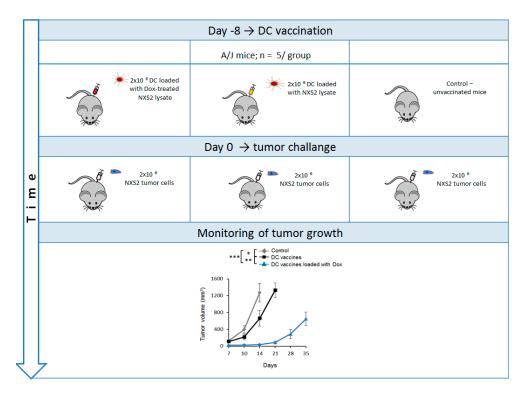
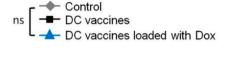
## Supplementary Materials.



**Figure S1.** Testing the immunogenicity of DC vaccines – prophylactic setting. The tumor lysatespulsed murine DCs (2 x 10<sup>6</sup> DCs/dose) or untreated DCs (2 x 10<sup>6</sup> DCs/dose) were injected intradermally into A/J mice (n = 5/group). Next, each mouse was injected subcutaneously (SC) in the lateral flank with 2 x 10<sup>6</sup> NXS2 cells on day 8 after vaccination (day 0). Unvaccinated mice (n = 5) served as a contol group. Tumor growth was monitored by measuring SC tumors once to thrice a week with a microcaliper until control mice were euthanized due to extensive tumor burden. \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001.



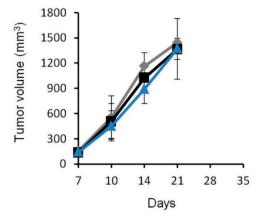
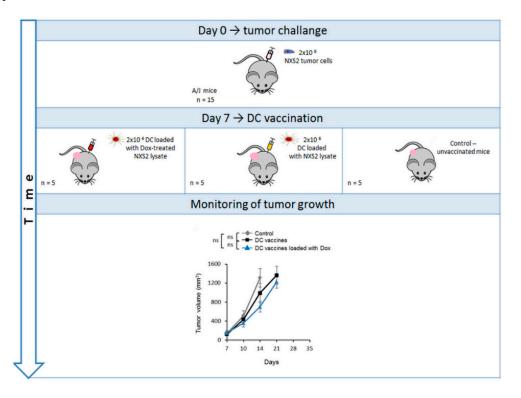
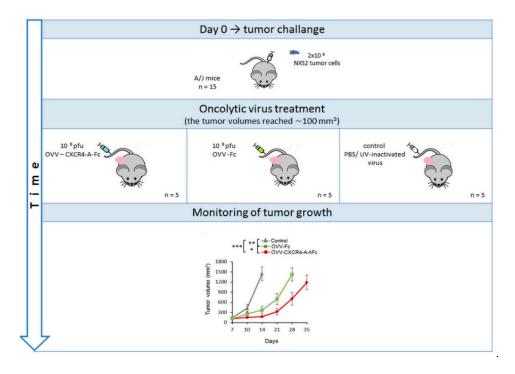


Figure S2. Effect of DC vaccines on tumor growth of NXS2 tumor-challenged SCID mice. BM-derived DCs were loaded with Dox-treated NXS2 tumor cell lysates or untreated NXS2 tumor cells and injected intradermally ( $2 \times 10^6$  DCs/dose) into SCID mice. Mice were challenged SC in the lateral flank

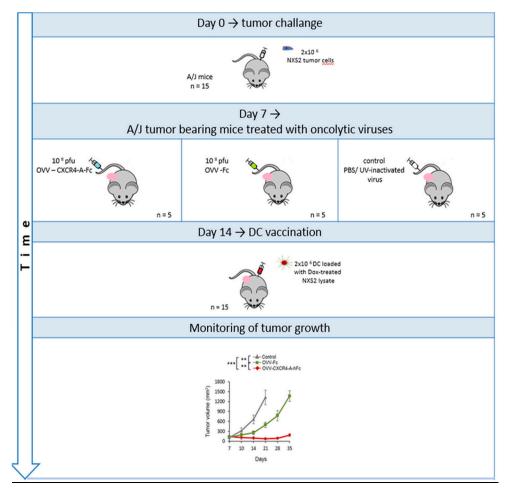
with 2 x  $10^6$  NXS2 cells on day 8 after vaccination. Control mice were unvaccinated. Tumor growth was monitored by measuring SC tumor growth with a microcaliper until control mice were euthanized due to extensive tumor burden. Results are presented as mean  $\pm$  SD of three independent experiments.



**Figure S3.** Testing the immunogenicity of DC vaccines—therapeutic setting. A/J mice (n= 15) were first injected SC with 2 x 10 $^6$  NXS2 cells in the lateral flank and 7 days later DC vaccines (DCs loaded with NXS2 lysates preapared from untreated or Dox-treated tumor cells) were injected intradermally into the opposite site (n = 5/group). Unvaccinated A/J mice served as controls (n = 5). Tumor growth was monitored by measuring SC tumors once to thrice a week with a microcaliper until control mice were euthanized due to extensive tumor burden.



**Figure S2.** Treatment of established tumors with oncolytic viruses. A/J mice (n = 15) were injected SC with 2 x 10<sup>6</sup> NXS2 cells and treated with OVV-CXCR4-A-Fc or OVV-Fc (10<sup>8</sup> PFU delivered intravenously, IV; n = 5 mice per group) once the tumor volumes reached ~100 mm<sup>3</sup> (on day 7). Control mice (n = 5) received PBS or UV-inactivated virus. Tumor progression was monitored by measuring SC tumor growth with a microcaliper until control mice were euthanized due to extensive tumor burden. \*p < 0.05, \*\*p < 0.01, \*\*\*p < 0.001.



**Figure S3.** The enhancement of oncolytic viruses treatment of established tumors with DC vaccines. NXS2 tumor bearing mice were treated with OVV-CXCR4-A-Fc or OVV-Fc ( $10^8$  PFU delivered IV) once the tumor volumes reached ~100 mm³ (on day 7). Control group was injected with PBS or UV inactivated virus (n = 5 mice/group). The DC vaccines were delivered after the cessation of viral replication (on day 14). Tumor growth was monitored by measuring SC tumors once to thrice a week with a microcaliper until control mice were euthanized due to extensive tumor burden. \*\*p < 0.01, \*\*\*p < 0.001.