Figure S5

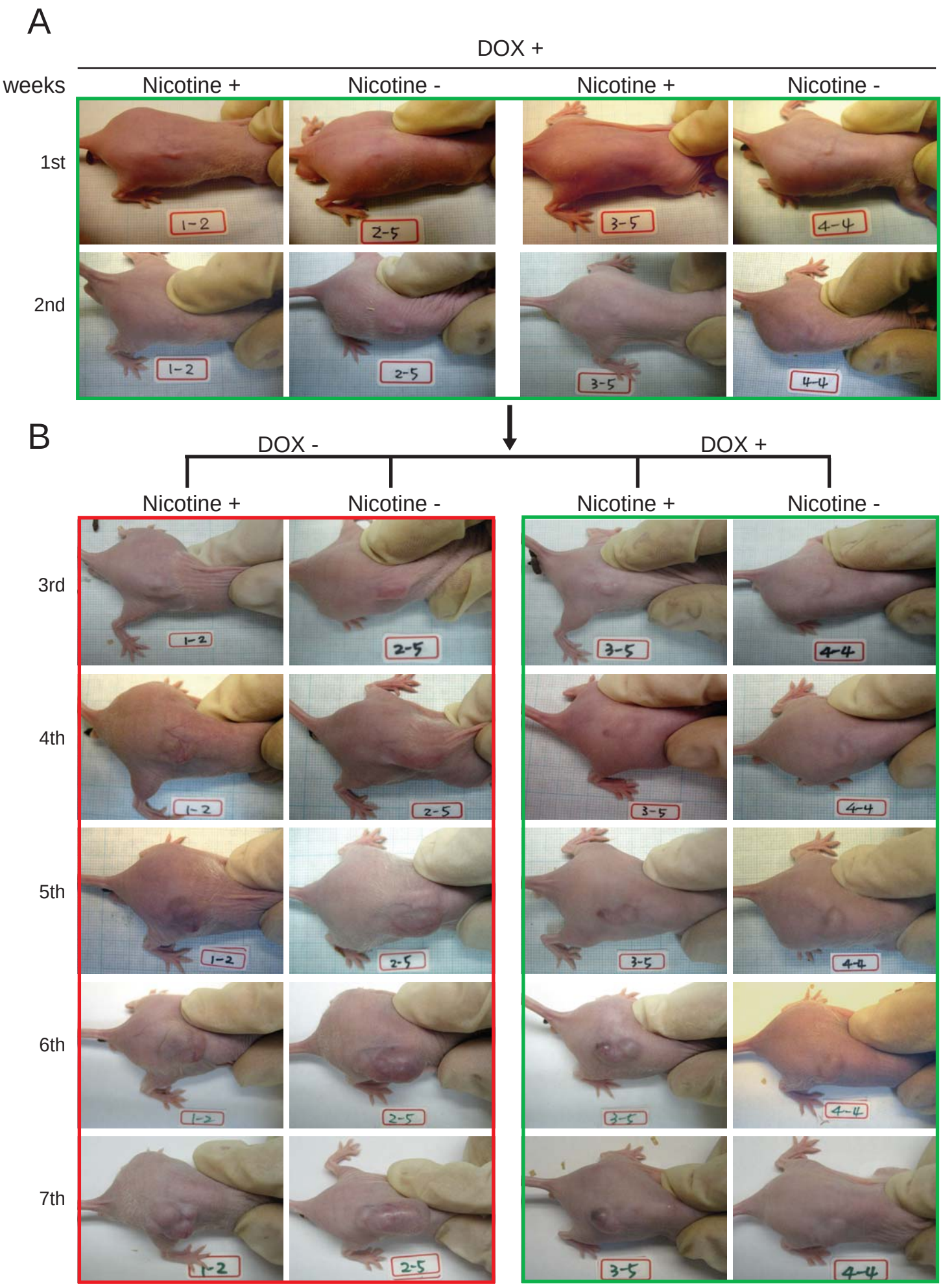
A
DOX +
weeks
Nicotine +
Nicotine -
Nicotine +
Nicotine -
1st
2nd
B
DOX -
DOX +
Nicotine +
Nicotine -
Nicotine +
Nicotine -
3rd
4th
5th
6th
7th

Figure S5. In vivo treatment of mice bearing nicotine-transformed MCF-10A cell xenografts and overexpression of the α9-nicotinic acetylcholine receptor (α9-nAChR) in response to doxycycline (DOX) removal. In the present study, the MCF-10A-Nic (DOX) transformed cell line was generated from soft agar colonies treated long-term with nicotine. The .α9-nAChR gene expression was induced by removal of DOX (DOX-) (Figure 4, B, bars 5 and 6, and Supplementary Figure S4, available online). **A)** Four representative female BALB/c-nu/nu mice (green square frame) from each group were injected subcutaneously with MCF-10A-Nic (DOX) (5×10^6) cells. Adenovirus vector-transfected MCF-10A (neo) cells were also injected into mice as a negative control (data not shown). Mice with established tumors were treated with DOX (0.5 mg/mL) in the presence or absence of nicotine (10 mg/mL) in their drinking water until the tumors reached a mean size of 200 mm^3. **B)** The mice described above were subdivided into two groups, designated DOX- and DOX+ (represented by the red and green square frames, respectively) according to the regulation of α9-nAChR mRNA expression. The mice were consecutively treated with or without nicotine in their drinking water (nicotine+, 10 mg/mL, vs nicotine-) for an additional 5 weeks.