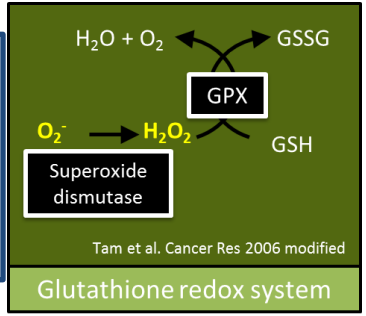
In the previous study, Glutathione peroxidase 2 (GPX2) was overexpressed in several cancers and the expression was correlated with histological malignancy, or invasiveness (Naiki-Ito A et al. Cancer Res 2007, Naiki T et al. Carcinogenesis 2014). ROS induced long term of smoking, or diet consumption was correlated in bladder carcinogenesis, therefore in this study, we investigated the role of GPX2 in bladder carcinogenesis and proliferation.



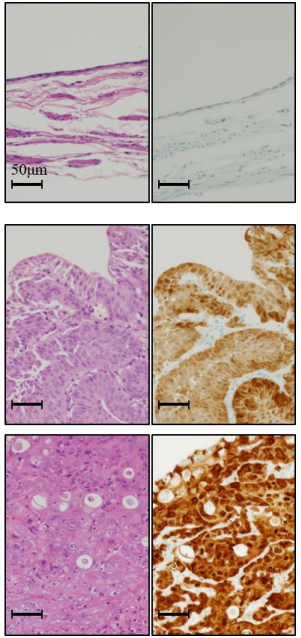
(i) Male F344 rats were given either 0.05% BBN in drinking water PEITC (Phenyl isothiocyanate) in their diet for 36 weeks, and GPX2 immunohistochemical analyses was performed.

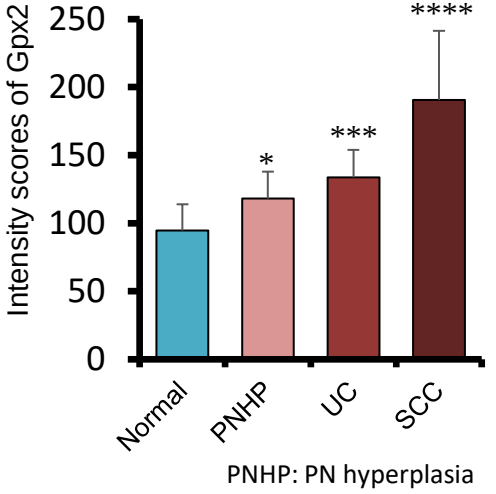
(ii) GPX2 was analyzed by immunohistochemistry in human radical cystectomy specimens.

(iii) The bladder cancer cell line were used, and GPX2 siRNA were transfected, and investigated the proliferation rates and ROS levels.

(iv) siRNA transfected BC31 cells were subcutaneously implanted into nude mice.

(i)

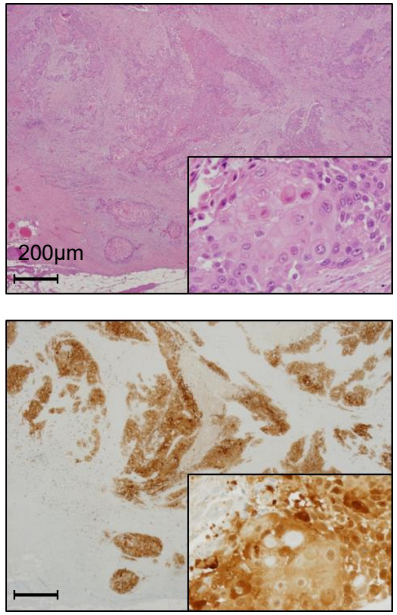




PNHP: PN hyperplasia

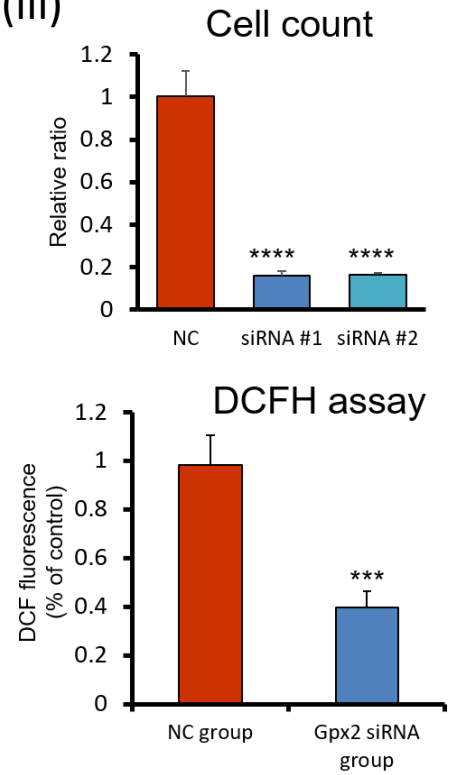
(ii)

UC with SqD

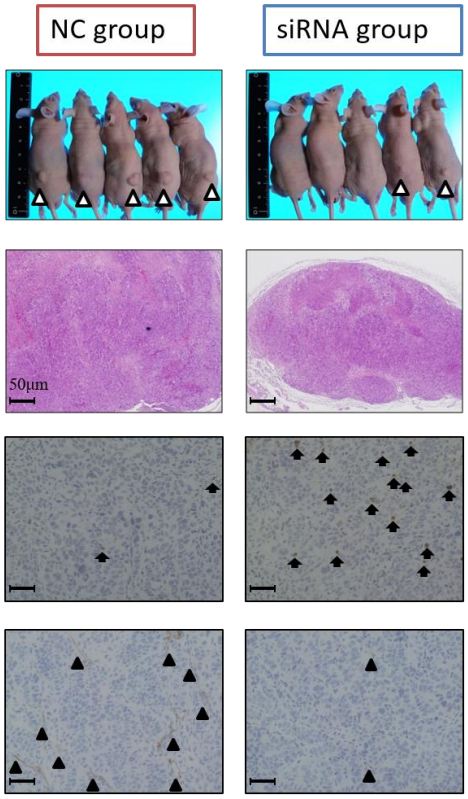


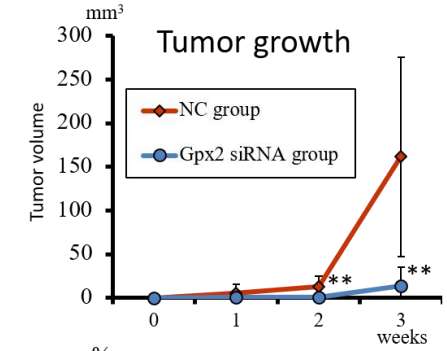
SqD: squamous cell differentiation

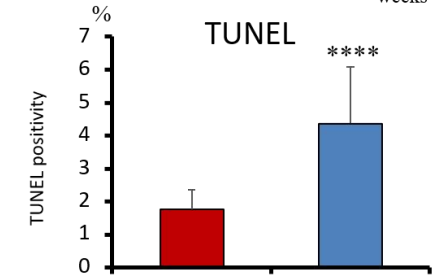
(iii)

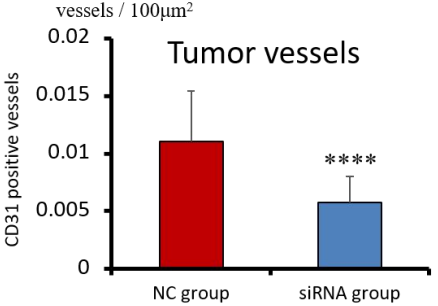


(iv)









GPX2 plays an important role in cell growth in the presence of ROS in bladder cancer, and the glutathione redox system can regulate cancer cell growth via the regulation of ROS, especially in SqD.