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Function of *SNHG12* in early response to boron neutron capture therapy (BNCT) in tumor cells

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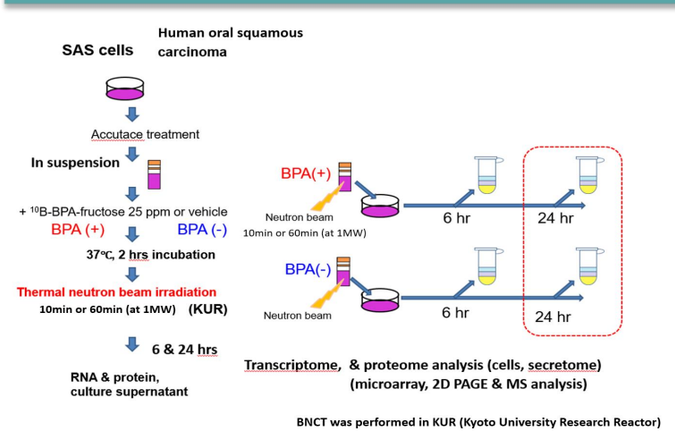
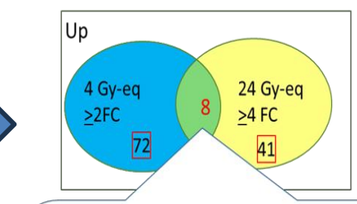
INTRODUCTION & AIM



RNA microarray analysis

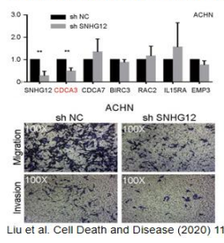
Dose-dependently induced genes 24 hours after BNCR in SAS cells



Gene	Signal intensity 4 Gy-eq 24 h BPA(+/−)	Signal intensity 24 Gy-eq 24 h BPA(+/−)	Increase rate	intracellular localization	Biological process
SNHG12	2.00	5.16	2.6	unknown	Unknown

Small Nucleolar Host Gene 12 (*SNHG12*), LNC04080

Ch 1. p35.3
1867 bases.
Encodes four small nucleolar RNAs through its splicing introns
SNORA66
SNORA61
SNORA16A
SNORD99
Binds to miRNAs similar to "sponges"



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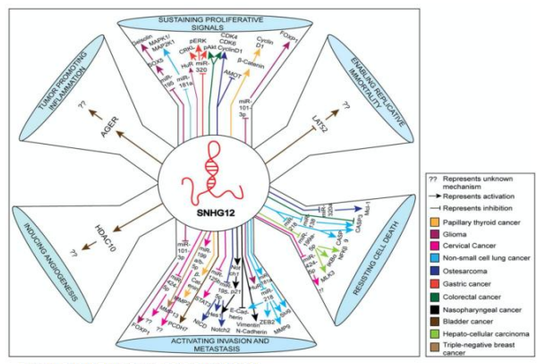

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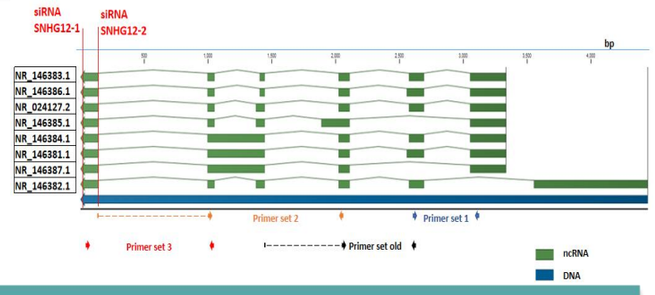
BNCT (boron neutron capture therapy) is a cancer therapy combining neutron irradiation and the administration of boron carrier drug, such as ^{10}B-boronophenylalanine (BPA). The clinical application of BNCT has recently been approved for oral and head-and-neck cancers in Japan. To evaluate the therapeutic efficacy and side effects of BNCT for various cancer stages, we evaluated factors related to early responses to BNCT in tumor cells. We showed that the expression of the long noncoding RNA *SNHG12*, a cancer-related molecule, is increased in oral cancer SAS cells after BPA-based BNCT. Here, the biological function of *SNHG12* during BNCT and early cell responses was investigated.

METHODS

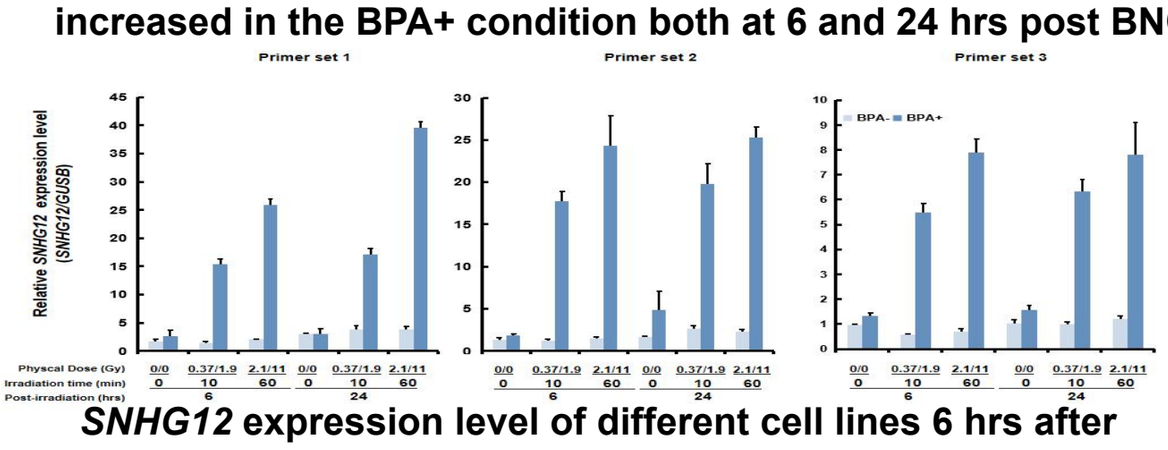
- SAS cell line:
  oral squamous cancer
- Transfection:s iRNA and plasmid
- Colony formation assay
- FACS: Propidium Iodide staining
- RT-PCR and qPCR
- Statistical analysis



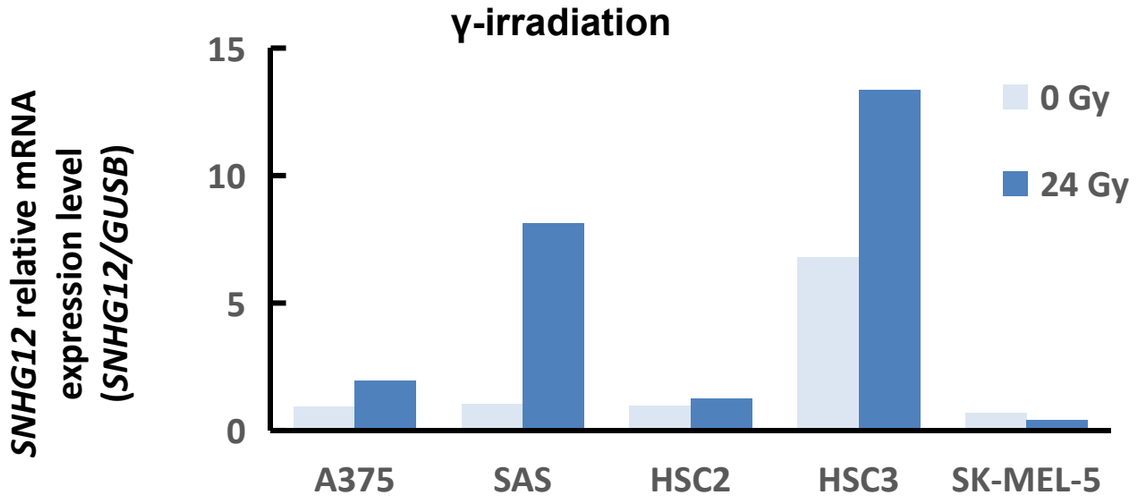
RESULTS & DISCUSSION

（Unpublished, do not post）

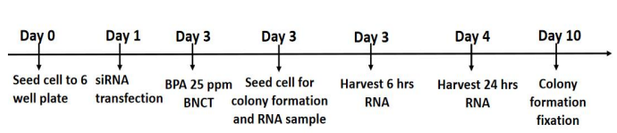
In SAS cells, *SNHG12* expression was dose-dependently increased in the BPA+ condition both at 6 and 24 hrs post BNCT

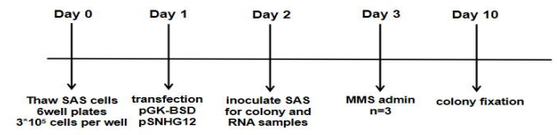


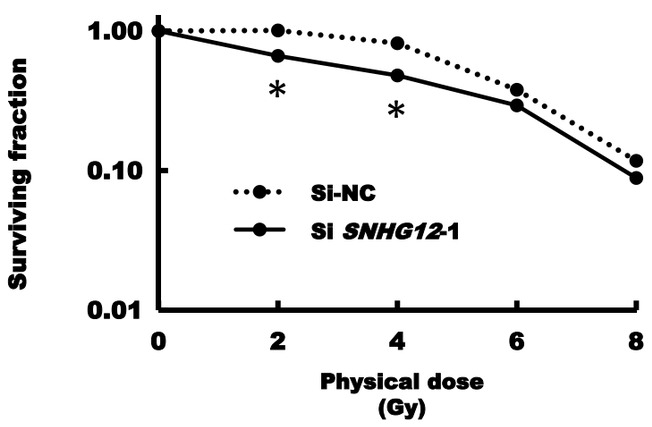
SNHG12 expression level of different cell lines 6 hrs after γ-irradiation

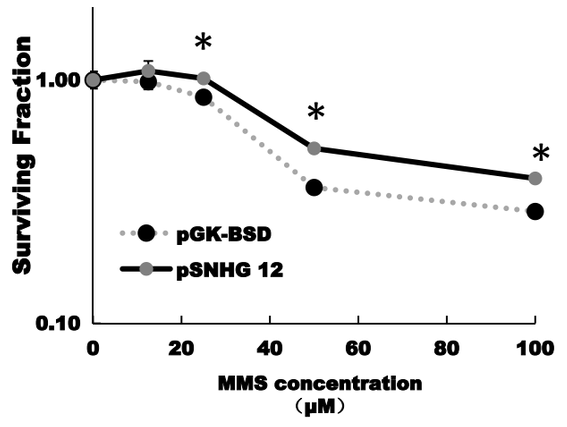


Knockdown of *SNHG12* in SAS cells after BNCT led to the decrease of the cell survival rate,and overexpression of SNHG12 after MMS treatment increased the survival rate

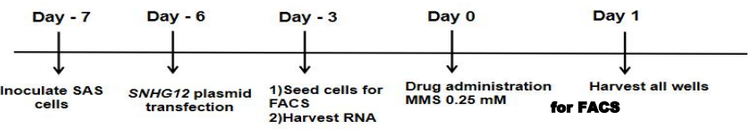


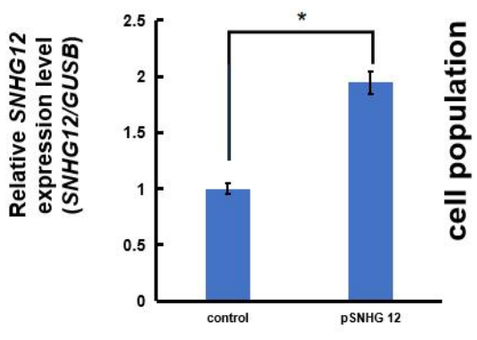


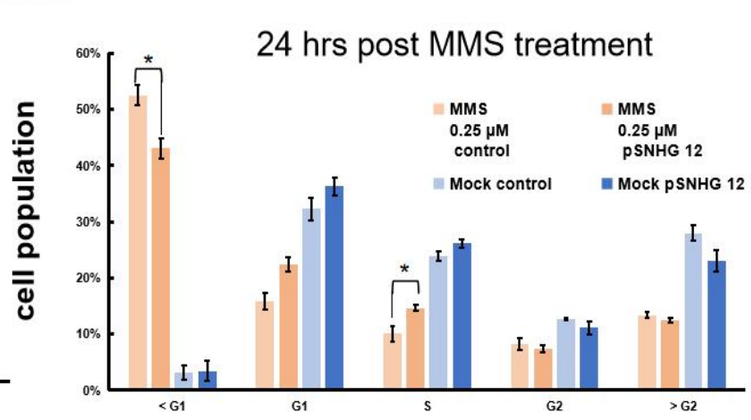




Overexpression of *SNHG12* in SAS cells reduced cell apoptosis after MMS treatment: Cell cycle 24 hours







CONCLUSION

- We have found that *SNHG12* expression in SAS cells also increased significantly after gamma-irradiation and the alkylating agent treatment. Therefore, we hypothesized *SNHG12* may play a role in particular types of DNA damage responses.
- In cancer cell lines such as SAS, A375, HSC3,SNHG12 expression is induced after γ-irradiation at 6 hours.
- Over-expression of *SNHG12* in SAS cells after MMS treatment reduced cell apoptosis at 24 hours as detected by FACS analysis,and increase the surviving rate of colony formation assay.
- Furthermore,we have found knockdown of *SNHG12* in SAS cells after BNCT decreased the surviving rate of SAS cells.

FUTURE WORK / REFERENCES

Exploration of the specific mechanisms by which *SNHG12* participates in DNA damage response after BNCT is ongoing.

COI： Lead Presenter: Zhongming Gao Principal Researcher: Mitsuko Masutani
We have no financial relationships to disclose