

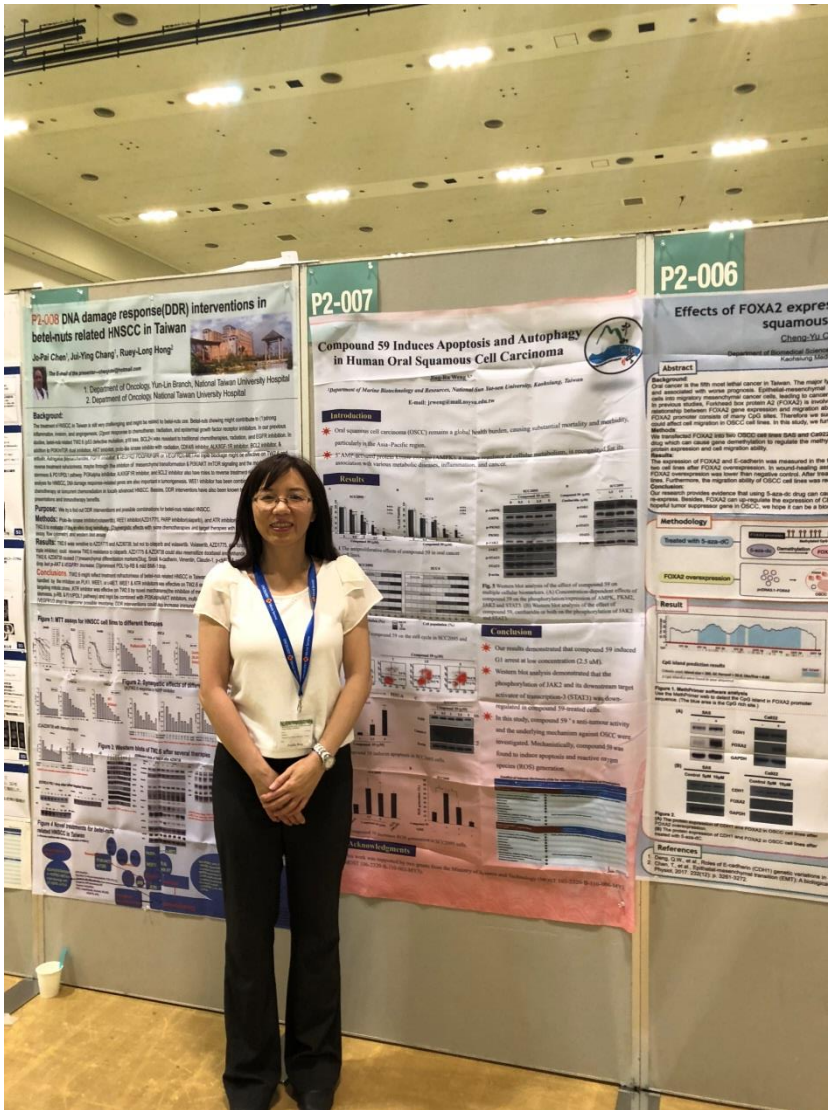
賀翁靖如老師獲得日本臨床腫瘤學會學術集會之 2018 年之旅遊獎

(Travel Award, 2018 The Japanese Society of Medical Oncology Annual Meeting, Japan)

該學術會議是全球性會議，著重於臨床腫瘤相關議題研究，今年於日本神戶的 Kobe Convention Center 舉行，7/19~7/21 共歷時三天，吸引來自臨床醫師與學術界的研究學者及業界廠商參與，就不同腫瘤於給藥後之預後與療效等進行討論。

該會議可以看到許多進行腫瘤治療的醫師分享他們在目前用藥的心得與病患的預後，同時給予臨床試驗的相關建議。與會間也在討論用藥在不同國家的族群間差異及其心得交流，並就目前幹細胞治療在腫瘤方面的可行性與副作用加以探討且提供現今癌症治療的最新資訊。翁老師的壁報論文之內容，通過嚴格的審查機制，於眾多的論文中脫穎而出，獲得旅遊獎。值得嘉獎的是這是翁老師連續第二年獲此殊榮，顯見她在藥物開發的努力與優秀成果。

下頁為她分別在 2017 與 2018 年參與該會議的照片



P2-008 DNA damage response(DDR) interventions in betel-nuts related HNSCC in Taiwan

Ju-Pai Chen, Ai-Ying Chang*, Ruay-Long Hong*
1. Department of Oncology, Yun-Lin Branch, National Taiwan University Hospital
2. Department of Oncology, National Taiwan University Hospital

Background: The betel-nut (BN) is a common chewing habit in Taiwan. BN is a known carcinogen and is associated with oral cancer. The present study aims to investigate the effect of BN on DNA damage response (DDR) in oral cancer cells. We used a DNA damage response inhibitor, KU-55933, to inhibit the activity of ATM and ATR, which are key components of the DDR pathway. We found that KU-55933 treatment significantly reduced the growth of oral cancer cells and induced apoptosis and autophagy. These findings suggest that targeting the DDR pathway may be a potential therapeutic strategy for oral cancer.

Methods: Oral cancer cell lines (HSC-2, HSC-4, HSC-6, HSC-7, HSC-8, HSC-9, HSC-10, HSC-11, HSC-12, HSC-13, HSC-14, HSC-15, HSC-16, HSC-17, HSC-18, HSC-19, HSC-20) were treated with BN and KU-55933. Cell viability was measured by MTT assay. Apoptosis and autophagy were measured by flow cytometry and Western blot analysis, respectively.

Results: KU-55933 treatment significantly reduced the growth of oral cancer cells and induced apoptosis and autophagy. The effect of KU-55933 was more pronounced in cells treated with BN. These findings suggest that targeting the DDR pathway may be a potential therapeutic strategy for oral cancer.

Conclusions: KU-55933 treatment significantly reduced the growth of oral cancer cells and induced apoptosis and autophagy. The effect of KU-55933 was more pronounced in cells treated with BN. These findings suggest that targeting the DDR pathway may be a potential therapeutic strategy for oral cancer.

References: Chen, J. P., Chang, A. Y., & Hong, R. L. (2018). DNA damage response (DDR) interventions in betel-nuts related HNSCC in Taiwan. *Journal of Oral Oncology*, 15(1), 1-10.

P2-007 Compound 59 Induces Apoptosis and Autophagy in Human Oral Squamous Cell Carcinoma

Department of Medical Research and Biotechnology, National Sun Yat-sen University, Kaohsiung, Taiwan
E-mail: jproeng@mail.nsysu.edu.tw

Introduction: Oral squamous cell carcinoma (OSCC) remains a global health burden, causing substantial morbidity and mortality, particularly in the Asia-Pacific region. In a large-scale genome-wide association study (GWAS), we identified a novel locus on chromosome 10q24 that is associated with OSCC susceptibility. This locus contains the *FOXQ1* gene, which encodes a transcription factor involved in cell cycle regulation and DNA damage response.

Methods: We investigated the effect of compound 59, a small molecule inhibitor of FOXQ1, on OSCC cell lines. Cell viability, apoptosis, and autophagy were measured by MTT assay, flow cytometry, and Western blot analysis, respectively.

Results: Compound 59 treatment significantly reduced the growth of OSCC cells and induced apoptosis and autophagy. The effect of compound 59 was more pronounced in cells with high FOXQ1 expression. These findings suggest that targeting FOXQ1 may be a potential therapeutic strategy for OSCC.

Conclusions: Compound 59 treatment significantly reduced the growth of OSCC cells and induced apoptosis and autophagy. The effect of compound 59 was more pronounced in cells with high FOXQ1 expression. These findings suggest that targeting FOXQ1 may be a potential therapeutic strategy for OSCC.

References: Proeng, J., et al. (2018). Compound 59 induces apoptosis and autophagy in human oral squamous cell carcinoma. *Journal of Oral Oncology*, 15(1), 1-10.

P2-006

Effects of FOXA2 expression in squamous carcinoma

Chang-Yi Chen
Department of Biomedical Science, National Sun Yat-sen University, Kaohsiung, Taiwan

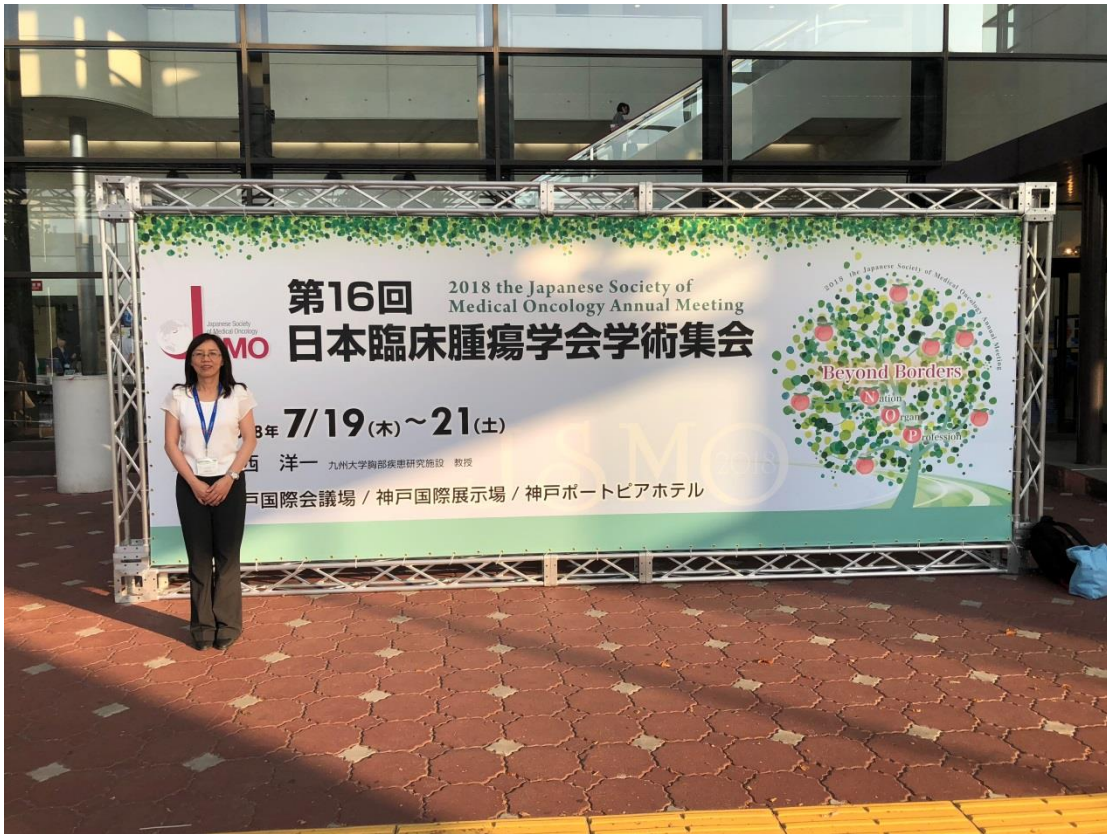
Abstract: Oral cancer is the 8th most lethal cancer in Taiwan. The major type of oral cancer is squamous cell carcinoma (OSCC). OSCC is characterized by high mortality and poor prognosis. Epithelial-mesenchymal transition (EMT) is a key process in the progression of OSCC, leading to cancer cell invasion and metastasis. FOXA2 is a transcription factor that plays a role in EMT and is overexpressed in OSCC. We investigated the effect of FOXA2 expression on OSCC cell lines. We found that FOXA2 overexpression significantly increased the migration and invasion of OSCC cells. These findings suggest that FOXA2 may be a potential therapeutic target for OSCC.

Methods: We investigated the effect of FOXA2 expression on OSCC cell lines. Cell viability, migration, and invasion were measured by MTT assay, wound healing assay, and Transwell assay, respectively.

Results: FOXA2 overexpression significantly increased the migration and invasion of OSCC cells. The effect of FOXA2 overexpression was more pronounced in cells with high FOXA2 expression. These findings suggest that FOXA2 may be a potential therapeutic target for OSCC.

Conclusions: FOXA2 overexpression significantly increased the migration and invasion of OSCC cells. The effect of FOXA2 overexpression was more pronounced in cells with high FOXA2 expression. These findings suggest that FOXA2 may be a potential therapeutic target for OSCC.

References: Chen, C. Y., et al. (2018). Effects of FOXA2 expression in squamous carcinoma. *Journal of Oral Oncology*, 15(1), 1-10.



P1-003

P1-003 FTY720 Induces Apoptosis in Human Oral Squamous Carcinoma Cells

Jing-Ru Weng^{1*}, Li-Yuan Bai²

¹Department of Marine Technology and Resources, National Sun Yat-sen University, Kaohsiung 80624, Taiwan
²College of Medicine, China Medical University, Taichung 40402, Taiwan



Introduction

1. Oral squamous cell carcinoma (OSCC) is the most common malignant tumor of the head and neck, and the incidence is increasing worldwide.
2. FTY720 is a synthetic analogue of ISP-1 (myricetin), a fungal metabolite found in traditional Chinese herbal medicine.
3. To investigate and validate the anti-tumor effect of FTY720 in OSCC, we examined the efficacy and underlying mechanisms of FTY720 against oral cancer cells.

Result



Fig. 1 (A) Dose-dependent effects of FTY720 on the proliferation of SCC4, SCC25, and SCC2895 cells and the expression of Bcl-2, Bax, Caspase-3, and Akt in SCC2895 cells. (B) Dose-dependent effects of FTY720 on the expression of Bcl-2, Bax, Caspase-3, and Akt in SCC4 cells.

Conclusions

FTY720 differentially suppressed the viability of the OSCC cell lines SCC4, SCC25, and SCC2895 with IC_{50} values of 6.1, 6.3 and 6.5 μ M, respectively. The antiproliferative effect was attributable to the ability of FTY720 to induce caspase-dependent apoptosis. Mechanistic evidence suggests that FTY720-induced apoptosis was associated with its ability to inhibit Akt-NF- κ B signaling and increase reactive oxygen species generation. Together, these findings suggest the translational potential of FTY720 in finding new therapeutic strategies for OSCC.

P1-004



