GS02-7 **Anti-tumor effects of antisense oligonucleotide against *TUG1* in pancreatic cancers**

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Pancreatic cancer is one of the most malignant tumors with dismal prognosis. In spite of comprehensive therapies, such as tumor removal surgery and subsequent chemotherapy, such as 5-Fluorouracil, 5-year survival rate is less than 5%. Therefore, effective therapeutic strategies are urgently needed. Dysregulation of the long non-coding RNA (lncRNA) is known to play important roles in cancer formation via interactions with other molecules including, protein, DNA and RNA. Here, we investigated the roles of lncRNA, *TUG1,* in pancreatic cancer. A pancreatic cancer cell, BxPC3, was subcutaneously inoculated into nude mice. After inoculation, antisense oligonucleotides targeting *TUG1* (*TUG1*-ASO) coupled with drug delivery system (*TUG1*-DDS) was administrated intravenously every three days for 30 days. *TUG1*-DDS significantly reduced the tumor growth. Furthermore, we found that miRNA-X (miR-X) was profoundly increased upon inhibition of *TUG1*, together with downregulation of its candidate targets, dihydropyrimidine dehydrogenase (DPYD). DPYD is a pyrimidine catabolic enzyme and degrades 5-FU. Interestingly, combination therapy of *TUG1*-ASO and 5-FU significantly suppressed cell proliferation compared to 5-FU treatment alone. Our data indicate that oncogenic roles of *TUG1* in pancreatic cancer and provide a new paradigm whereby targeting *TUG1* might be an effective novel strategy for pancreatic cancer treatment.