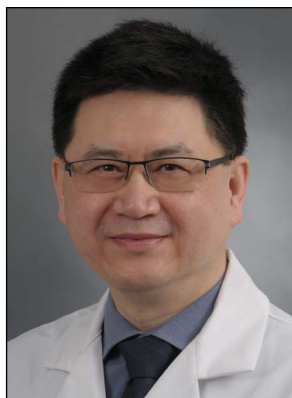


Cover legend: **Jingfang Ju**; a member of The Editorial Academy of
The International Journal of Oncology



Professor Jingfang Ju received his PhD degree in molecular biology and biochemistry with Dr Peter Danenberg at the Norris Comprehensive Cancer Center, University of Southern California in 1996, where his primary research focus was on the molecular mechanisms of thymidylate synthase and the tumor suppressor gene, p53, in the chemoresistance of colorectal cancer to 5-fluorouracil. In particular, he developed an antisense strategy to overcome chemoresistance by knocking down thymidylate synthase and the restoration of the tumor suppressor gene, p53 (Clin Cancer Res 4: 1315-1322, 1998; Clin Cancer Res 4: 2229-2236, 1998). Following the completion of his PhD training, Dr Ju joined Dr Edward Chu's group and completed his post-doctoral fellowship in molecular pharmacology at Yale University. During his post-doctoral fellowship from 1996 to 1999, he investigated the translational control of thymidylate synthase and p53 in colorectal cancer, where he discovered that as an RNA binding protein, thymidylate synthase binds to p53 mRNA to regulate its protein biosynthesis (Mol Cell Biol 19: 1582-1594, 1999; Proc Natl Acad Sci USA 96: 3769-3774, 1999). After completing his post-doctoral studies, he joined CuraGen Inc., a biopharmaceutical company as a senior scientist and team leader of genomics based high throughput drug target discovery and validation. He developed a high throughput approach to systematically identify mRNA targets that were regulated post-transcriptionally (Nucleic Acids Res 31: 5157-5166, 2003).

Dr Ju joined the faculty of the Mitchell Cancer Institute at University of South Alabama as the Head of cancer genomics

and as an Assistant Professor. He began to focus on his research on the roles of non-coding RNA, particularly microRNAs in chemoresistance. His group was the first to discover the regulatory relationship between p53 and microRNA in colorectal cancer [Clin Cancer Res 12 (7 Pt 1): 2014-2024, 2006]. As a transcription factor and key tumor suppressor, p53 directly binds to a number of promoters of microRNAs to regulate their expression and their function to regulate key downstream targets regulating cell cycle control and cell death. His group has identified a number of tumor suppressor miRNAs that play major roles in cell cycle control, apoptosis, autophagy, EMT and chemoresistance (Clin Cancer Res 4: 8080-8086, 2008; Oncogene 28: 4065-4074, 2009; Mol Cancer 9: 96, 2010; Mol Cancer 10: 99, 2011; Oncogene 32: 1570-1579, 2013). His group was the first to discover that miRNAs are highly stable in archival formalin fixed paraffin embedded specimens, which established the foundation for miRNA based biomarker discovery (RNA 13: 1668-1674, 2007).

In 2008, Dr Ju moved his laboratory to Stony Brook University, where he is currently Professor and Program Director of Oncogenic Drivers and Carcinogenesis Program of the Stony Brook Cancer Center, Renaissance School of Medicine. Dr Ju continues his main research on understanding the molecular mechanism of non-coding RNAs in cancer and cancer stem cells. The Ju laboratory studies the mechanisms of microRNAs (miRNAs) in cancer stem cell resistance, epithelial to mesenchymal transition (EMT), autophagy, and apoptosis (Cell Death Dis 4: 1-9, 2013; Cell Cycle 12: 246-250, 2013; Oncogene 35: 5501-5514, 2016). Dr Ju has a number of patents on miRNA-based therapeutics (Patent number: 11584932; Patent number: 11236337; Patent number: 10697020; Patent number: 8927207). The ultimate goal of his laboratory is to develop innovative miRNA based therapeutics for cancer (Mol Ther Nucleic Acids 19: 228-239, 2020; Mol Ther 30: 3450-3461, 2022; Mol Ther-Oncolytics 28: 277-292, 2023). In addition to having an NCI-funded laboratory, he was a recipient of the Glaxo Wellcome Oncology Clinical Research Scholar Award. Dr Ju is the scientific co-founder of Curamir Therapeutics Inc. to develop patented microRNA based cancer therapeutics.