



## Assoc Prof Yu Chun Kong, Victor



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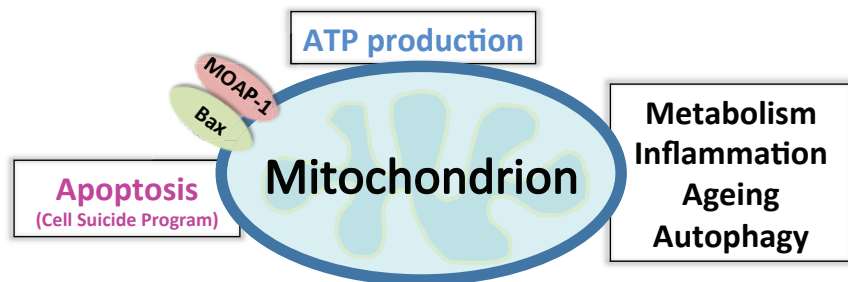
### Academic Profile

Born in Hong Kong, Dr. Yu obtained his B.Sc. Pharm. from University of Houston and Ph.D. in Pharmaceutical Chemistry from School of Pharmacy, University of California, San Francisco (UCSF) where he spent five years to study molecular pharmacology of morphine. He subsequently undertook post-doctoral training in molecular biology at the Howard Hughes Medical Institute (HHMI) at University of California, San Diego (UCSD). During this period, he made the discovery that the nuclear receptor protein RXR is the common co-regulator for retinoic acid (Vitamin A), vitamin D and thyroid hormone receptors. The discovery paved the way for rapid progress in understanding the molecular basis of the pleiotropic effect of nuclear hormones on gene transcription in multiple organs and cell types. He came to Singapore in 1993 to join the Institute of Molecular and Cell Biology (IMCB) as a Principal Investigator. In 2009, he joined department of Pharmacy at NUS as tenured Associate Professor.

### Research Interests

- Apoptosis mechanism in mitochondria
- Mitochondrial dynamics and cell death
- Liver and neurodegenerative diseases

Mitochondria are dynamic organelles that are constantly undergoing fusion and fission. In addition to being the powerhouse of cells by producing ATP as source of energy, increasing evidence suggests that mitochondria play central role in regulating a multitude of physiological processes, including apoptosis, aging and inflammation. Apoptosis is a physiological process by which unwanted, damage or infected cells are to be eliminated from multi-cellular organisms. Deregulation of mitochondrial apoptosis signaling has been linked to a plethora of major human ailments facing the ageing population worldwide, ranging from cancers to neurodegenerative diseases. His laboratory has been involved in identifying and characterizing several critical protein molecules in the mitochondrial apoptosis signaling pathway. Currently, they are focusing on characterizing the physiological and pathological roles of the Bax-binding protein MOAP-1 and its family of proteins in liver (hepatocytes) and brain (neurons) using combined genetic, biochemical and cell biology approaches.



### Selected Publications:

1. Yu V.C.; Delsert C.; Andersen B.; Holloway J.M.; Devary O.V.; Naar A.M.; Kim S.Y.; Boutin J.M.; Glass C.K.; Rosenfeld M.G. RXR $\beta$ : A co-regulator that enhances binding of retinoic acid, thyroid hormone, and vitamin D receptors to their cognate response elements. *Cell*, **1991**, 67, 1251-1266.
2. Tan K.O.; Fu N.Y.; Sukumaran S.K.; Chan S.L.; Poon K.L.; Hian K.J.; Chen B.S.; Yu V.C. MOAP-1 is a mitochondrial effector of Bax. *Proc. Natl. Acad. Sci. U.S.A.* **2005**, 102, 14623-14688. (Cited by Faculty 1000 Biology)
3. Fu N.Y.; Sukumaran S.K.; Yu V.C. Inhibition of ubiquitin-mediated degradation of MOAP-1 by apoptotic stimuli promotes Bax function in mitochondria. *Proc. Natl. Acad. Sci. U.S.A.* **2007**, 104, 10051-10056. (Cited by Faculty 1000 Biology)
4. Fu N.Y.; Sukumaran S.K.; Yu V.C. Bax $\beta$ : a constitutively active human Bax isoform that is under tight regulatory control by the proteasomal degradation mechanism. *Molecular Cell*, **2009**, 33, 15-29. (Selected as "Featured Article" by the Journal and Cited by Faculty 1000 Biology)
5. Sukumaran S.K.; Fu N.Y.; Chua B.T.; Wan K.F.; Lee S.S.; Yu V.C. A soluble form of the pilus protein FimA targets the VDAC-hexokinase complex at mitochondria to inhibit host cell apoptosis. *Molecular Cell*, **2010**, 37, 768-783.
6. Tan C.T.; Zhou Q.L.; Su Y.C.; Fu N.Y.; Chang H.C.; Tao R.T.; Sukumaran S.K.; Baksh S.; Tan Y.J.; Sabapathy K.; Yu C.D.; Yu V.C. MOAP-1 Mediates Fas-Induced Apoptosis in Liver by Facilitating tBid Recruitment to Mitochondria. *Cell Reports*, **2016**, 16, 174-185.