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The focus of my research is on the interaction between viruses and their hosts in molecular level. The most recognized work of mine until now was the identification of a key molecule in response to virus infection, MITA/STING, as the host substrate of dengue viral protease. Cleavage of this protein dramatically impairs interferon pathway and benefits dengue virus replication. Currently, I am studying the biological functions of host and viral proteins involved in innate immunity, metabolism, and mitochondrial biology.

Publications:

1. Hom-Ming Yeh, **Chia-Yi Yu**, Ho-Chun Yang, Shih-Han Ko, Ching-Len Liao, and Yi-Ling Lin. *Ubiquitin-specific protease 13 regulates interferon signaling by stabilizing STAT1*. **Journal of Immunology** (2013) Sep 191(6):3328-36.
2. Yu-Chun Tu, **Chia-Yi Yu**, Jian-Jong Liang, Elong Lin, Ching-Len Liao, and Yi-Ling Lin. *Blocking dsRNA-Activated Protein Kinase PKR by Japanese Encephalitis Virus Nonstructural Protein 2A.* **Journal of Virology** (2012) Oct 86(19):10347-58.
3. Tsung-Hsien Chang, Siang-Ru Chen, **Chia-Yi Yu**, You-Sheng Lin, Yao-Shen Chen, Toru Kubota, Mayumi Matsuoka and Yi-Ling Lin. *Dengue virus serotype 2 blocks extracellular signal-regulated kinase and nuclear factor-κB activation to downregulate cytokine production.* **PLoS One** (2012) Aug 7(8): e41635.
4. **Chia-Yi Yu**, Tsung-Hsien Chang, Jian-Jong Liang, Ruei-Lin Chiang, Yi-Ling Lee, Ching-Len Liao and Yi-Ling Lin. *Dengue virus targets the adaptor protein MITA to subvert host innate immunity.* **PLoS Pathogens** (2012) Jun 8(6): e1002780. [Postdoctoral Fellow Outstanding Publication Awards, National Science Council of Taiwan]
5. Jian-Jong Liang, **Chia-Yi Yu**, Ching-Len Liao, and Yi-Ling Lin. *Vimentin binding is critical for infection by the virulent strain of Japanese encephalitis virus.* **Cellular Microbiology** (2011) Sep 13(9):1358-70.
6. **Chia-Yi Yu**, Ruei-Lin Chiang, Tsung-Hsien Chang, Ching-Len Liao, and Yi-Ling Lin. *The interferon stimulator mitochondrial antiviral signaling protein facilitates cell death by disrupting the mitochondrial membrane potential and by activating caspases.* **Journal of Virology** (2010) Mar 84(5):2421-31.
7. **Chia-Yi Yu**, Yun-Wei Hsu, Ching-Len Liao, and Yi-Ling Lin. *Flavivirus infection activates the XBP1 pathway of the unfolded protein response to cope with endoplasmic reticulum stress*. **Journal of Virology** (2006) Dec 80(23):11868-80.