Electronic Supplementary Material

Influenza A virus-induced downregulation of miR-26a contributes to reduced IFNα/β production

Shijuan Gao¹#, Jiandong Li²#, Liping Song⁴, Jiaoxiang Wu², Wenlin Huang²,³#

¹. The Key Laboratory of Remodeling-Related Cardiovascular Diseases, Collaborative Innovation Center for Cardiovascular Disorders, Beijing Institute of Heart, Lung & Blood Vessel Diseases; Beijing Anzhen Hospital, Capital Medical University, Beijing 100029, China
². CAS Key Laboratory of Pathogenic Microbiology and Immunology, Institute of Microbiology, Chinese Academy of Sciences, Beijing 100101, China
³. Sun Yat-Sen University Cancer Center, State Key Laboratory of Oncology in South China, Collaborative Innovation Center of Cancer Medicine, Guangzhou 510060, China
⁴. Beijing Municipal Center for Food Safety Monitoring and Risk Assessment, Beijing, 100053, China

Supporting information to DOI: 10.1007/s12250-017-4004-9

Figure S1. miR-26a promotes the K63-linked ubiquitination of RIG-I. Lysates of 293T cells transfected with plasmids for Flag-RIG-I and HA-tagged K63-linked ubiquitin (HA-K63-Ub) or HA-tagged K48-linked ubiquitin (HA-K48-Ub), together with miR-26a or its negative control were immunoprecipitated with anti-Flag and immunoblotted with anti-HA.