Supplementary Material

Intramolecular interaction of NEP regulated by CRM1 ensures the unidirectional transport of M1 for the nuclear export of influenza viral ribonucleoprotein

Mikako Hirohama[1], Shun Yamashita[2], Masamitsu N. Asaka[1], Takahiro Kuroki[2], and Atsushi Kawaguchi[1,2,3,4]*

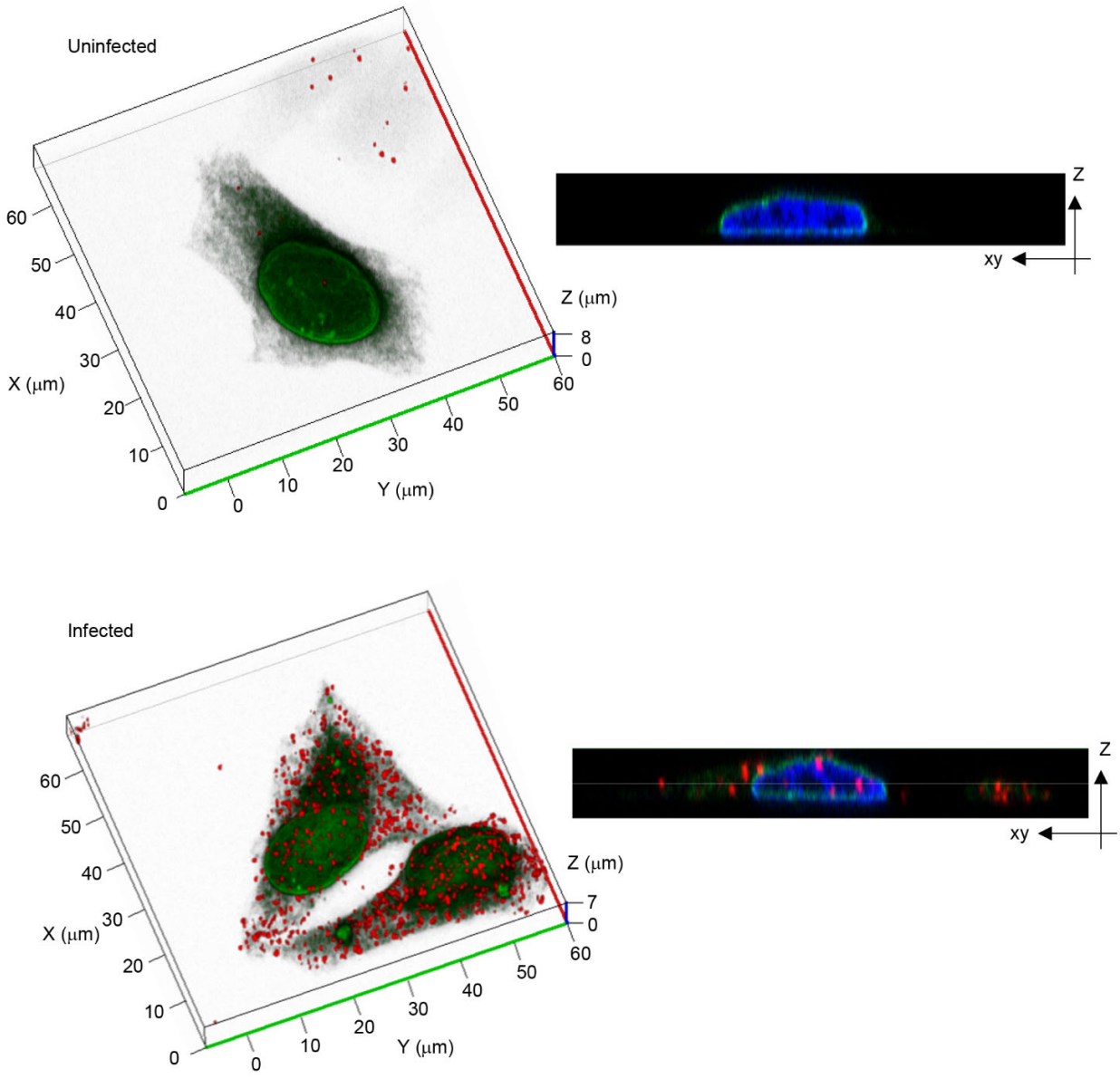
[1]Department of Infection Biology, Faculty of Medicine, University of Tsukuba, Tsukuba, Japan

[2]Graduate School of Comprehensive Human Sciences, University of Tsukuba, Tsukuba, Japan

[3]Transborder Medical Research Center, University of Tsukuba, Tsukuba, Japan

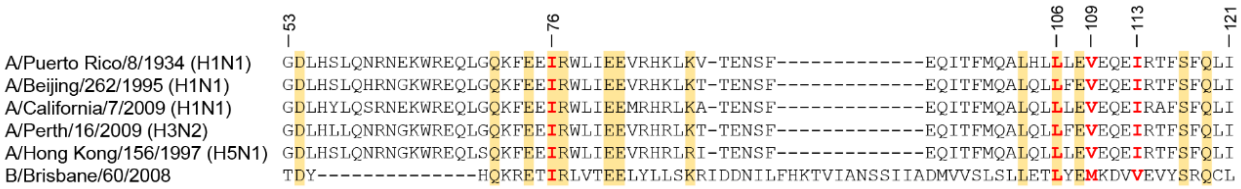
[4]Microbiology Research Center for Sustainability, University of Tsukuba, Tsukuba, Japan

*** Correspondence:**
Atsushi Kawaguchi
ats-kawaguchi@md.tsukuba.ac.jp



Supplementary Figure 1

The 3D-reconstruction images of Figure 1B. Samples shown in Figure 1B (0 and 9 h post-infectionFr) were analyzed with ZEN software for 3D reconstruction. The infected HeLa cells expressing GFP-emerin (green) were subjected to *in situ* PLA with rat anti-NEP and rabbit anti-M1 antibodies (red).



Supplementary Figure 2

The sequence alignment of NEP between H1N1, H3N2, H5N1 influenza A viruses and influenza B virus. Amino acid residues 53 to 121 of A/Puerto Rico/8/1934 (H1N1) NEP are aligned with that of A/Beijing/262/1995 (H1N1), A/California/7/2009 (H1N1), A/Perth/16/2009 (H3N2), A/Hong Kong/156/1997 (H5N1), and B/Brisbane/60/2008. Conserved residues are shown in yellow boxes. The amino acid positions 76, 106, 109, and 113 are shown in red.