IMSUT Distinguished Professor Unit

Division of Virology
ウイルス感染部門

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Viruses can cause devastating diseases. The long-term goal of our research is to understand the molecular pathogenesis of viral diseases by using influenza virus, Ebola virus, and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections as models. Interactions between viral and host gene products during viral replication determine the consequences of infection (i.e., the characteristics of disease manifestation, whether limited or widespread); hence, our research has centered on such interactions during these viral infections.

1. Characterization of SARS-CoV-2 Omicron BA.2.75 clinical isolates.

Uraki R, Iida S[1], Halfmann PJ[2], Yamayoshi S, Hirata Y[1], Iwatsuki-Horimoto K, Kiso M, Ito M, Furusawa Y, Ueki H, Sakai-Tagawa Y, Kuroda M[2], Maemura T[2], Kim T[2], Mine S[1], Iwamoto N[3], Li R[4], Liu Y[4], Larson D[4], Fukushi S[5], Watanabe S[6], Maeda K[6], Wang Z[4], Ohmagari N[3], Theiler J[7], Fischer W[8,9], Korber B[8,9], Imai M, Suzuki T[1], Kawaoka Y. [1]Department of Pathology, National Institute of Infectious Diseases, Tokyo, Japan. [2]Influenza Research Institute, Department of Pathobiological Sciences, School of Veterinary Medicine, University of Wisconsin-Madison, Madison, WI, USA. [3]Disease Control and Prevention Center, National Center for Global Health and Medicine Hospital, Tokyo, Japan. [4]Department of Animal, Dairy, and Veterinary Sciences, College of Agriculture and Applied Sciences, Utah State University, Logan, UT, USA. [5]Department of Virology 1, National Institute of Infectious Diseases, Tokyo, Japan. [6]Center for Influenza and Respiratory Virus Research, National Institute of Infectious Diseases, Tokyo, Japan. [7]Space Data Science and Systems, Los Alamos National Laboratory, Los Alamos, USA. [8]Theoretical Biology and Biophysics, Los Alamos National Laboratory, Los Alamos, USA. [9]New Mexico Consortium, Los Alamos, USA.

The prevalence of the Omicron subvariant BA.2.75 rapidly increased in India and Nepal during the summer of 2022, and spread globally. However, the virological features of BA.2.75 are largely unknown. Here, we evaluated the replicative ability and pathogenicity of BA.2.75 clinical isolates in Syrian hamsters. Although we found no substantial differences in weight change among hamsters infected with BA.2, BA.5, or BA.2.75, the replicative ability of BA.2.75 in the lungs is higher than that of BA.2 and BA.5. Of note, BA.2.75 causes focal viral pneumonia in hamsters, characterized by patchy inflammation interspersed in alveolar regions, which is not observed in BA.5-infected hamsters. Moreover, in competition assays, BA.2.75 replicates better than BA.5 in the lungs of hamsters. These results suggest that BA.2.75 can cause more severe respiratory disease than BA.5 and BA.2 in a hamster model and should be closely monitored.

2. In vitro and in vivo characterization of SARS-CoV-2 strains resistant to nirmatrelvir

Kiso M, Furusawa Y, Uraki R, Imai M, Yamayoshi S, Kawaoka Y.

Nirmatrelvir, an oral antiviral agent that targets a SARS-CoV-2 main protease (3CLpro), is clinically useful against infection with SARS-CoV-2 including its omicron variants. Since most omicron subvariants

have reduced sensitivity to many monoclonal antibody therapies, potential SARS-CoV-2 resistance to nirmatrelvir is a major public health concern. Several amino acid substitutions have been identified as being responsible for reduced susceptibility to nirmatrelvir. Among them, we selected L50F/E166V and L50F/E166A/L167F in the 3CLpro because these combinations of substitutions are unlikely to affect virus fitness. We prepared and characterized delta variants possessing Nsp5-L50F/E166V and Nsp5-L50F/E166A/L167F. Both mutant viruses showed decreased susceptibility to nirmatrelvir and their growth in VeroE6/TMPRSS2 cells was delayed. Both mutant viruses showed attenuated phenotypes in a male hamster infection model, maintained airborne transmissibility, and were outcompeted by wild-type virus in co-infection experiments in the absence of nirmatrelvir, but less so in the presence of the drug. These results suggest that viruses possessing Nsp5-L50F/E166V and Nsp5-L50F/E166A/L167F do not become dominant in nature. However, it is important to closely monitor the emergence of nirmatrelvir-resistant SARS-CoV-2 variants because resistant viruses with additional compensatory mutations could emerge, outcompete the wild-type virus, and become dominant.

3. In vitro and in vivo characterization of SARS-CoV-2 resistance to ensitrelvir.

Kiso M, Yamayoshi S, Iida S[1], Furusawa Y, Hirata Y, Uraki R, Imai M, Suzuki T[1], Kawaoka Y.

Ensitrelvir, an oral antiviral agent that targets a SARS-CoV-2 main protease (3CLpro or Nsp5), is clinically useful against SARS-CoV-2 including its omicron variants. Since most omicron subvariants have reduced sensitivity to most monoclonal antibody therapies, SARS-CoV-2 resistance to other antivirals including main protease inhibitors such as ensitrelvir is a major public health concern. Here, repeating passages of SARS-CoV-2 in the presence of ensitrelvir revealed that the M49L and E166A substitutions in Nsp5 are responsible for reduced sensitivity to ensitrelvir. Both substitutions reduced in vitro virus growth in the absence of ensitrelvir. The combination of the M49L and E166A substitutions allowed the virus to largely evade the suppressive effect of ensitrelvir in vitro. The virus possessing Nsp5-M49L showed similar pathogenicity to wild-type virus, whereas the virus possessing Nsp5-E166A or Nsp5-M49L/E166A slightly attenuated. Ensitrelvir treatment of hamsters infected with the virus possessing Nsp5-M49L/E166A was ineffective; however, nirmatrelvir or molnupiravir treatment was effective. Therefore, it is important to closely monitor the emergence of ensitrelvir-resistant SARS-CoV-2 variants to guide antiviral treatment selection.

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