「The Novel Coronavirus Is Mutating. More Dangerous or less harmful?」

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The novel coronavirus (SARS-CoV-2) is spreading and infected more than 5 million people in just a few months.[1] Recently, researchers from University College London (UCL) identified 198 mutations that appear to have independently occurred more than once, which may hold clues to how the virus is adapting.[2]

The mutation number looks scary, but it is actually very normal in all RNA viruses such as SARS-CoV-2. "Viruses mutate naturally as part of their life cycle," says Ewan Harrison, scientific project manager for the Covid-19 Genomics UK Consortium. What scientists want to know is: may these mutations change the severity or infectiousness of the disease?

The first known infections from the SARS-CoV-2 strain were discovered in Wuhan, China. SARS-CoV-2 is believed to have zoonotic origins and has clos genetic similarity to bat coronaviruses. However, there is no evidence yet to link an intermediate animal reservoir to its introduction to humans. So far, many subtypes of SARS-CoV-2 have been identified but the origin is still a puzzle. [3, 4]

The current global virus database shows that the repeated mutations displayed in different strains are mainly induced and edited by the host RNA. Among the mutant strains, the non-synonymous mutation rate of ORF8, ORF1a and ORF9 of virus is the highest, and other genes including spike protein are relatively conservative.[5]

Notably, A recent study from the Los Alamos National Laboratory tracked mutations throughout the outbreak using a database called the Global Initiative on Sharing All Influenza Data (GISAID). The researchers found 14 such mutations and suggested one particular mutation, D614G, was "of urgent concern" because it may make the disease more infectious.[6] It has a non-silent (Aspartate to Glycine) mutation at 614th position of the spike protein and generates an additional serine protease (Elastase) cleavage site near the S1-S2 junction of the spike protein.[6] D614G began spreading in Europe in early February, and when introduced to new regions it rapidly becomes the dominant form.

It is known that the new coronavirus attaches to human cells through a "spike protein." After the spike protein binds to the ACE2 receptor on a human cell, the viral membrane fuses with the human cell membrane, allowing the genome of the virus to enter human cells.[7, 8] The RBD (receptor binding domain) mutation of the virus is the reason for the higher affinity of SARS-CoV-2 and ACE2. Researchers

found that a few genetic mutations led the SARS-CoV-2's spike protein to develop a more compact molecular "ridge" and allows SARS-CoV-2 to attach more strongly to the human ACE2 receptor.[9]

Genetics and biology experts say it's still too early to know whether any of the mutations are meaningful. Lawrence Young, a professor of molecular oncology at Britain's University of Warwick, said that while there is "much speculation about the possible emergence of more aggressive strains". This virus still has many mysteries. It evolves while spreading and we must find a way to deal with it as well as disclose its origin mystery.

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