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## WHY CHINA MAY HAVE Manufactured COVID-19

04/19/2020 – FROM THE DESK OF: <mark>Edward C. Noonan, Former 2012 & 2016 Presidential Candidate</mark>

## Why China may have manufactured COVID-19

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In order for COVID-19 to infect humans, the virus must attach to the target human cell, fuse with the cell membrane in order to inject the virus' genetic material and then use the cell's own biochemical mechanisms to replicate the virus.

Understanding those processes through scientific experimentation is key to the development of vaccines against and treatments for viral infections.

When dealing with highly contagious and deadly viruses and given the widespread use of bioengineering, extra precautions need to be taken not to inadvertently create dangerous new viruses or release viruses from containment facilities, whether they be naturally-occurring or man-made.

SARS-CoV, the coronavirus responsible for the 2002-2003 pandemic, infects human cells first by attaching to the human angiotensin-converting enzyme-2 receptor (ACE2), then, via SARS-CoV's S-protein virus-to-cell membrane fusion is initiated.

In 2008, scientists at the Wuhan Institute of Virology, led by Zhengli Shi, <u>demonstrated</u> the importance of the initial human ACE2 binding step by using bioengineering techniques to "splice" the receptor-binding domain of SARS-CoV onto a non-human-infecting bat coronavirus, essentially creating an entirely new viral entity capability of infecting humans.

After the 2012 Middle East respiratory syndrome coronavirus (MERS-CoV) pandemic, similar scientific studies were undertaken to understand its human infectivity and pathogenesis.

Although MERS-CoV uses a different receptor to bind to human cells, dipeptidyl peptidase 4 or DPP4, the S-protein, which may be similar to SARS-CoV, is <u>important</u> for virus-to-cell membrane fusion.

After binding to the human cell, the MERS-CoV S-protein undergoes two cleavages. The first cleavage splits the S-protein into two subunits, S1 and S2, which allows an unfolding process proceeding virus-to-cell membrane fusion. The second cleavage occurs within the S2 segment, called S2-prime (S2'), and is <u>thought to trigger</u> membrane fusion.

The importance of the S2' cleavage site in pathogenicity has long been recognized in <u>bird influenza</u> and <u>cat peritonitis</u> coronavirus infections.

COVID-19 has just such a S2' furin polybasic cleavage site, which does not exist in any of the naturally-occurring bat coronaviruses <u>identified as</u> close relatives of COVID-19.

That "close relative" article has been widely cited as evidence to support the Chinese government's contention that COVID-19 "jumped" from animals to humans inside the Wuhan Seafood Market.

The bird infectious bronchitis Beaudette coronavirus strain, which has a furin polybasic cleavage site amino acid sequence of Arginine-Arginine-Lysine-Arginine, closely matches COVID-19's furin polybasic cleavage site of Arginine-Arginine-Alanine-Arginine.

It is important to note that furin cleavage sites have been <u>"introduced"</u> into coronaviruses using widely-known genetic engineering techniques since, at least, 2006 in order to study the effect of cleavage to mediate cell-to-cell fusion and affect viral infectivity.

It is, therefore, not inconceivable to conclude that in the course of scientific investigation, a Chinese laboratory may have bioengineered a furin polybasic cleavage site into a bat coronavirus in order to study its effects on pathogenicity.

In the absence of conclusive evidence that COVID-19 is naturally-occurring, the burden of proof as to the origin of COVID-19 is now on China.

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