



United States Department of State

Washington, D.C. 20520

July 31, 2024

Case No. FL-2022-00076

Mr. Sainath Suryanarayanan, PhD
U.S. Right to Know
4096 Piedmont Ave. #963
Oakland, CA 94611

Dear Mr. Suryanarayanan:

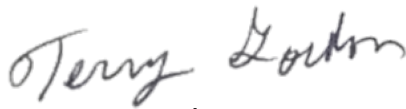
As we noted in our letter dated June 28, 2024, we are processing your request for material under the Freedom of Information Act ("FOIA"), 5 U.S.C. § 552. The Department of State ("Department") has identified an additional 19 responsive records subject to the FOIA. Upon review, we have determined that 13 records may be released in part and 6 records must be withheld in their entirety.

An enclosure explains the FOIA exemptions and other grounds for withholding material. Where we have made redactions, the applicable FOIA exemption are marked on each record. The records that were withheld in full are exempt from release pursuant to FOIA Exemption 5, 5 U.S.C. § 552(b)(5). The document identification numbers for the records withheld in full are: A-00000686366, A-00000686367, A-00000686368, A-00000686371, A-00000686369, and A-00000686372. Where applicable, the Department has considered the foreseeable harm standard when reviewing these records and applying FOIA exemptions. All non-exempt material that is

reasonably segregable from the exempt material has been released and is enclosed.

We will keep you informed as your case progresses. If you have any questions, your attorney may contact Savith Iyengar, Assistant U.S. Attorney, at savith.iyengar@usdoj.gov. Please refer to the case number, FL-2022-00076, and the civil action number, 22-cv-04359, in all correspondence about this case.

Sincerely,

A handwritten signature in dark ink, appearing to read "Terry Gordon". The signature is fluid and cursive, with the first name "Terry" being more prominent than the last name "Gordon".

Terry Gordon

Chief, Litigation and Appeals Branch
Office of Information Programs and Services

Enclosures: As stated.

The Freedom of Information Act (5 USC 552)

FOIA Exemptions

- (b)(1) Information specifically authorized by an executive order to be kept secret in the interest of national defense or foreign policy. Executive Order 13526 includes the following classification categories:
- 1.4(a) Military plans, systems, or operations
 - 1.4(b) Foreign government information
 - 1.4(c) Intelligence activities, sources or methods, or cryptology
 - 1.4(d) Foreign relations or foreign activities of the US, including confidential sources
 - 1.4(e) Scientific, technological, or economic matters relating to national security, including defense against transnational terrorism
 - 1.4(f) U.S. Government programs for safeguarding nuclear materials or facilities
 - 1.4(g) Vulnerabilities or capabilities of systems, installations, infrastructures, projects, plans, or protection services relating to US national security, including defense against transnational terrorism
 - 1.4(h) Weapons of mass destruction
- (b)(2) Related solely to the internal personnel rules and practices of an agency
- (b)(3) Specifically exempted from disclosure by statute (other than 5 USC 552), for example:
- | | |
|----------------|---|
| ARMSEXP | Arms Export Control Act, 50a USC 2411(c) |
| CIA PERS/ORG | Central Intelligence Agency Act of 1949, 50 USC 403(g) |
| EXPORT CONTROL | Export Administration Act of 1979, 50 USC App. Sec. 2411(c) |
| FS ACT | Foreign Service Act of 1980, 22 USC 4004 |
| INA | Immigration and Nationality Act, 8 USC 1202(f), Sec. 222(f) |
| IRAN | Iran Claims Settlement Act, Public Law 99-99, Sec. 505 |
- (b)(4) Trade secrets and confidential commercial or financial information
- (b)(5) Interagency or intra-agency communications forming part of the deliberative process, attorney-client privilege, or attorney work product
- (b)(6) Personal privacy information
- (b)(7) Law enforcement information whose disclosure would:
- (A) interfere with enforcement proceedings
 - (B) deprive a person of a fair trial
 - (C) constitute an unwarranted invasion of personal privacy
 - (D) disclose confidential sources
 - (E) disclose investigation techniques
 - (F) endanger life or physical safety of an individual
- (b)(8) Prepared by or for a government agency regulating or supervising financial institutions
- (b)(9) Geological and geophysical information and data, including maps, concerning wells

Other Grounds for Withholding

- NR Material not responsive to a FOIA request excised with the agreement of the requester

From: (b)(6)@state.gov
To: (b)(6)@state.gov
CC: EAP-Press <EAP-Press@state.gov>;
ISN-Press-DL <ISN-Press-DL@state.gov>;
Park, Christopher J (b)(6)@state.gov
Subject: RE: Vanity Fair Interview with SPOX edits
Date: Thu, 3 Jun 2021 12:27:48 +0000

Good morning,

Sorry if this is too late, but received additional edits/clearances.

EAP/P (b)(6) OK
EAP/CM: (b)(6) OK
EAP/CM: (b)(6) OK
EAP/EP: (b)(6) Info by Request
EAP/FO: Richard Buangan OK
EAP/FO: Jonathan Fritz OK

Thanks,

(b)(6)

From: (b)(6)@state.gov
Sent: Wednesday, June 2, 2021 10:32 AM
To: (b)(6)@state.gov; EAP-Press <EAP-Press@state.gov>
Cc: ISN-Press-DL <ISN-Press-DL@state.gov>; Park, Christopher J (b)(6)@state.gov
Subject: FW: Vanity Fair Interview with SPOX edits

(b)(6)

This just came back from the NSC. (b)(5)
(b)(5) Once you say go,
I push to my FO.

Thanks!

(b)(6)

From: (b)(6)@state.gov
Sent: Wednesday, June 2, 2021 10:18 AM
To: (b)(6) (b)(6)@state.gov; DL.NSC.Press <DL.NSC.Press@nsc.eop.gov>; Horne, Emily
J. EOP/NSC (b)(6)
Cc: ISN-Press-DL <ISN-Press-DL@state.gov>; Park, Christopher J (b)(6)@state.gov; Rowe, Courtney
M. EOP/WHO (b)(6); Munoz, Kevin EOP/WHO
(b)(6); SPOX2 <SPOX2@state.gov>; DiNardo, Nicholas A

<(b)(6)@state.gov>

Subject: RE: Vanity Fair Interview with SPOX edits

All:

See attached, with NSC cleared lines recommendations.

(b)(6)

From: (b)(6)@state.gov>

Sent: Wednesday, June 2, 2021 10:13 AM

To: (b)(6)@state.gov>; DL.NSC.Press <DL.NSC.Press@nsc.eop.gov>; Horne, Emily J. EOP/NSC <(b)(6)>

Cc: ISN-Press-DL <ISN-Press-DL@state.gov>; Park, Christopher J (b)(6)@state.gov>; Rowe, Courtney M. EOP/WHO (b)(6); Munoz, Kevin EOP/WHO

(b)(6); SPOX2 <SPOX2@state.gov>; DiNardo, Nicholas A

(b)(6)@state.gov>

Subject: Re: Vanity Fair Interview with SPOX edits

Adding a few others here (b)(6) (b)(5)

(b)(5)

From: (b)(6)@state.gov>

Sent: Wednesday, June 2, 2021 9:46 AM

To: DL.NSC.Press <DL.NSC.Press@nsc.eop.gov>; Horne, Emily J. EOP/NSC (b)(6)

(b)(6)@state.gov>

Cc: ISN-Press-DL <ISN-Press-DL@state.gov>; Park, Christopher J (b)(6)@state.gov>

Subject: Vanity Fair Interview with SPOX edits

Emily and NSC Press colleagues,

Yesterday, the Director of ISN's Biological Policy Staff received a call from Vanity Fair saying she is running a story on the origins of Covid-19. There are so many different versions of information out there, and our goal is to clarify and educate as best we can on what the Department knows and the particulars of State officials' meeting on this issue last December. (b)(5)

(b)(5) Note that Vanity Fair's deadline is at noon today. Once these lines are cleared, we will deploy off the record.

Thanks,

(b)(6)

Sender:	(b)(6)@state.gov>
Recipient:	(b)(6)@state.gov>; EAP-Press <EAP-Press@state.gov>;

ISN-Press-DL <ISN-Press-DL@state.gov>;
Park, Christopher J (b)(6)@state.gov>

**Vanity Fair Interview (on background) – State Department exploration of
COVID-19 Origins**

(b)(5)

(b)(5)



(b)(5)

Drafted by: CPark

ISN/PRESS: (b)(6) OK

OES/IHB: (b)(6) OK

AVC/PRESS: (b)(6) OK

AVC/CBW: (b)(6) OK

EAP

P: (b)(6) OK

S/P: (b)(6) Info by request

T

D

NSC/Press

From:	"DiNanno, Thomas G" (b)(6)@state.gov>
To:	(b)(6)@state.gov>
Subject:	RE: Schriever Wargame 2020 Coalition Council Information
Date:	Thu, 8 Oct 2020 20:42:24 +0000

From: (b)(6)@state.gov>
Sent: Thursday, October 8, 2020 4:40 PM
To: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>
Cc: DiNanno, Thomas G (b)(6)@state.gov>; (b)(6)@state.gov>
Subject: RE: Schriever Wargame 2020 Coalition Council Information

Hi (b)(6)

AA/S DiNanno won't be able to participate in this.

Thank you.

(b)(6)



(b)(6)

Chief of Staff
Bureau of Arms Control, Verification and Compliance
U.S. Department of State
HST Room 5950

Office: (b)(6)
Cell:

OpenNet: (b)(6)@state.gov
ClassNet: (b)(6)@state.sgov.gov
JWICS: (b)(6)@state.ic.gov

From: (b)(6)@state.gov>
Sent: Tuesday, October 6, 2020 1:03 PM
To: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)
(b)(6)@state.gov>
Cc: DiNanno, Thomas G (b)(6)@state.gov>; (b)(6)@state.gov>
Subject: FW: Schriever Wargame 2020 Coalition Council Information

(b)(6)

Can we block 2pm to 6pm on 3 and 4 November on AA/S DiNanno's calendar for the Schriever Wargame 2020 Coalition Council? I am aware that he may not be able to attend, but would like to pencil it in if he ends up being available. The 2pm start gives us plenty of time to get to the Pentagon (I can escort if he does not have access) and get him settled in the SVTC room with the rest of the D.C. based senior leaders.

Thank you!

Warm Regards,

(b)(6)

(b)(6)

AVC/ESC

U.S. Department of State

Tel: (b)(6)

Cell

Email: (b)(6)@state.gov

From: (b)(6) JSSF HQSF A2-3-6/S3/6TG (b)(6)
Sent: Tuesday, October 6, 2020 10:04 AM
To: (b)(6) HAF SAF/USSF (b)(6)
(b) USSPACECOM/CCE (b)(6) SAF-OS/Workflow (b)(6)
(b)(6)
(b)(6)
(b)(6)@bah.com>; (b)(6) (US)
(b)(6) (b)(6)@bah.com>;
(b)(6)
(b)(6)@state.gov>; (b)(6) HQSF HQSF/CDS
(b)(6) AFSPC HQ USSF/CDE2
(b)(6) JSSF HQSF AFSPC/S36E
(b)(6) HQSF AFSPC/S3/6
(b)(6) USSPACECOM JFSCC/J3
(b)(6) USSPACECOM JFSCC/J3
(b)(6) USSPACECOM/J5
(b)(6) HQSF AFSPC/SSDP
(b)(6) USSPACECOM

USSPACECOM/J006 (b)(6)
 (USA) (b)(6) (b)(6)@nsa.gov; (b)(6)@doc.gov;
 (b)(6)@noaa.gov; (b)(6); (b)(6)
 (b)(6)
 (b)(6) D Team <Dteam@nga.mil> (b)(6) CIV USSF SPOC CFSCC/CCS
 (b)(6) CFSCC/CCE
 (b)(6);
 (b)(6)
 (b)(6)@nasa.gov; (b)(6) AFELM
 Joint Staff AFELM JCS MCA JO/J-5/GPP/SMDP (b)(6)
 (b)(6) JSSF AFSPC JFSCC/NA (b)(6)
 Cc: (b)(6) A2-3-6/S3/6TG (b)(6)
 (b)(6) HQSF A2-3-6/S3/6TG (b)(6)
Subject: Schriever Wargame 2020 Coalition Council Information

Ladies and Gentlemen,

We completed the final vector check with General Raymond and here is some event information for your planning purposes:

1. We will send all SW 20 Coalition Council read-ahead materials by Tuesday, 13 October. The read-ahead materials will be sent to POCs for each by-name DV invite and the nation leads. The information will include:

- Overview and Schedule (Unclassified)
- Vignette (Secret)
- Threat Brief (Secret)
- Deep Dive Results (currently Unclassified but final version may end up Secret)
- Discussion Theme and Deliverables (Unclassified)
- Final Coordination Instructions (Unclassified)

2. The unclassified read-ahead materials will be sent via unclassified email and the secret read-ahead materials will be sent via the means we have to get classified products to each POC and nation.

3. The original plan was for the event to be a 4-hour discussion for both days. This has been reduced to a 3-hour discussion for both days. For your planning, below are the local times for each nation:

Locations	Times
Colorado Springs	1300-1600 (3-4 Nov)
Washington D.C.	1500-1800 (3-4 Nov)
Ottawa	1500-1800 (3-4 Nov)
London	2000-2300 (3-4 Nov)
Berlin	2100-2400 (3-4 Nov)
Paris	2100-2400 (3-4 Nov)
Tokyo	0500-0800 (4-5 Nov)
Canberra	0700-1000 (4-5 Nov)

Wellington

0900-1200 (4-5 Nov)

Respectfully,

(b)(6)

Contractor
Schriever Wargame Team
HQ USSF S3/6TG

(b)(6)

24 Talon Way
Schriever AFB, Colorado 80912

Sender: "DiNanno, Thomas G" (b)(6)@state.gov>**Recipient:** (b)(6)@state.gov>

Unusual Features of the SARS-CoV-2 Genome Suggesting Sophisticated Laboratory Modification Rather Than Natural Evolution and Delineation of Its Probable Synthetic Route

Li-Meng Yan (MD, PhD)¹, Shu Kang (PhD)¹, Jie Guan (PhD)¹, Shanchang Hu (PhD)¹

¹Rule of Law Society & Rule of Law Foundation, New York, NY, USA.

Correspondence: team.lmyan@gmail.com

Abstract

The COVID-19 pandemic caused by the novel coronavirus SARS-CoV-2 has led to over 910,000 deaths worldwide and unprecedented decimation of the global economy. Despite its tremendous impact, the origin of SARS-CoV-2 has remained mysterious and controversial. The natural origin theory, although widely accepted, lacks substantial support. The alternative theory that the virus may have come from a research laboratory is, however, strictly censored on peer-reviewed scientific journals. Nonetheless, SARS-CoV-2 shows biological characteristics that are inconsistent with a naturally occurring, zoonotic virus. In this report, we describe the genomic, structural, medical, and literature evidence, which, when considered together, strongly contradicts the natural origin theory. The evidence shows that SARS-CoV-2 should be a laboratory product created by using bat coronaviruses ZC45 and/or ZXC21 as a template and/or backbone. Building upon the evidence, we further postulate a synthetic route for SARS-CoV-2, demonstrating that the laboratory-creation of this coronavirus is convenient and can be accomplished in approximately six months. Our work emphasizes the need for an independent investigation into the relevant research laboratories. It also argues for a critical look into certain recently published data, which, albeit problematic, was used to support and claim a natural origin of SARS-CoV-2. From a public health perspective, these actions are necessary as knowledge of the origin of SARS-CoV-2 and of how the virus entered the human population are of pivotal importance in the fundamental control of the COVID-19 pandemic as well as in preventing similar, future pandemics.

Introduction

COVID-19 has caused a world-wide pandemic, the scale and severity of which are unprecedented. Despite the tremendous efforts taken by the global community, management and control of this pandemic remains difficult and challenging.

As a coronavirus, SARS-CoV-2 differs significantly from other respiratory and/or zoonotic viruses: it attacks multiple organs; it is capable of undergoing a long period of asymptomatic infection; it is highly transmissible and significantly lethal in high-risk populations; it is well-adapted to humans since the very start of its emergence¹; it is highly efficient in binding the human ACE2 receptor (hACE2), the affinity of which is greater than that associated with the ACE2 of any other potential host^{2,3}.

The origin of SARS-CoV-2 is still the subject of much debate. A widely cited *Nature Medicine* publication has claimed that SARS-CoV-2 most likely came from nature⁴. However, the article and its central conclusion are now being challenged by scientists from all over the world⁵⁻¹⁵. In addition, authors of this *Nature Medicine* article show signs of conflict of interests^{16,17}, raising further concerns on the credibility of this publication.

The existing scientific publications supporting a natural origin theory rely heavily on a single piece of evidence – a previously discovered bat coronavirus named RaTG13, which shares a 96% nucleotide sequence identity with SARS-CoV-2¹⁸. However, the existence of RaTG13 in nature and the truthfulness of its reported sequence are being widely questioned^{6-9,19-21}. It is noteworthy that scientific journals have clearly censored any dissenting opinions that suggest a non-natural origin of SARS-CoV-2^{8,22}. Because of this censorship, articles questioning either the natural origin of SARS-CoV-2 or the actual existence of RaTG13, although of high quality scientifically, can only exist as preprints^{5-9,19-21} or other non-peer-reviewed articles published on various online platforms^{10-13,23}. Nonetheless, analyses of these reports have repeatedly pointed to severe problems and a probable fraud associated with the reporting of RaTG13^{6,8,9,19-21}. Therefore, the theory that fabricated scientific data has been published to mislead the world's efforts in tracing the origin of SARS-CoV-2 has become substantially convincing and is interlocked with the notion that SARS-CoV-2 is of a non-natural origin.

Consistent with this notion, genomic, structural, and literature evidence also suggest a non-natural origin of SARS-CoV-2. In addition, abundant literature indicates that gain-of-function research has long advanced to the stage where viral genomes can be precisely engineered and manipulated to enable the creation of novel coronaviruses possessing unique properties. In this report, we present such evidence and the associated analyses. Part 1 of the report describes the genomic and structural features of SARS-CoV-2, the presence of which could be consistent with the theory that the virus is a product of laboratory modification beyond what could be afforded by simple serial viral passage. Part 2 of the report describes a highly probable pathway for the laboratory creation of SARS-CoV-2, key steps of which are supported by evidence present in the viral genome. Importantly, part 2 should be viewed as a demonstration of how SARS-CoV-2 could be conveniently created in a laboratory in a short period of time using available materials and well-documented techniques. This report is produced by a team of experienced scientists using our combined expertise in virology, molecular biology, structural biology, computational biology, vaccine development, and medicine.

1. Has SARS-CoV-2 been subjected to *in vitro* manipulation?

We present three lines of evidence to support our contention that laboratory manipulation is part of the history of SARS-CoV-2:

- i. The genomic sequence of SARS-CoV-2 is suspiciously similar to that of a bat coronavirus discovered by military laboratories in the Third Military Medical University (Chongqing, China) and the Research Institute for Medicine of Nanjing Command (Nanjing, China).
- ii. The receptor-binding motif (RBM) within the Spike protein of SARS-CoV-2, which determines the host specificity of the virus, resembles that of SARS-CoV from the 2003 epidemic in a suspicious manner. Genomic evidence suggests that the RBM has been genetically manipulated.
- iii. SARS-CoV-2 contains a unique furin-cleavage site in its Spike protein, which is known to greatly enhance viral infectivity and cell tropism. Yet, this cleavage site is completely absent in this particular class of coronaviruses found in nature. In addition, rare codons associated with this additional sequence suggest the strong possibility that this furin-cleavage site is not the product of natural evolution and could have been inserted into the SARS-CoV-2 genome artificially by techniques other than simple serial passage or multi-strain recombination events inside co-infected tissue cultures or animals.

1.1 Genomic sequence analysis reveals that ZC45, or a closely related bat coronavirus, should be the backbone used for the creation of SARS-CoV-2

The structure of the ~30,000 nucleotides-long SARS-CoV-2 genome is shown in Figure 1. Searching the NCBI sequence database reveals that, among all known coronaviruses, there were two related bat coronaviruses, ZC45 and ZXC21, that share the highest sequence identity with SARS-CoV-2 (each bat coronavirus is ~89% identical to SARS-CoV-2 on the nucleotide level). Similarity between the genome of SARS-CoV-2 and those of representative β coronaviruses is depicted in Figure 1. ZXC21, which is 97% identical to and shares a very similar profile with ZC45, is not shown. Note that the RaTG13 virus is excluded from this analysis given the strong evidence suggesting that its sequence may have been fabricated and the virus does not exist in nature^{2,6-9}. (A follow-up report, which summarizes the up-to-date evidence proving the spurious nature of RaTG13, will be submitted soon)

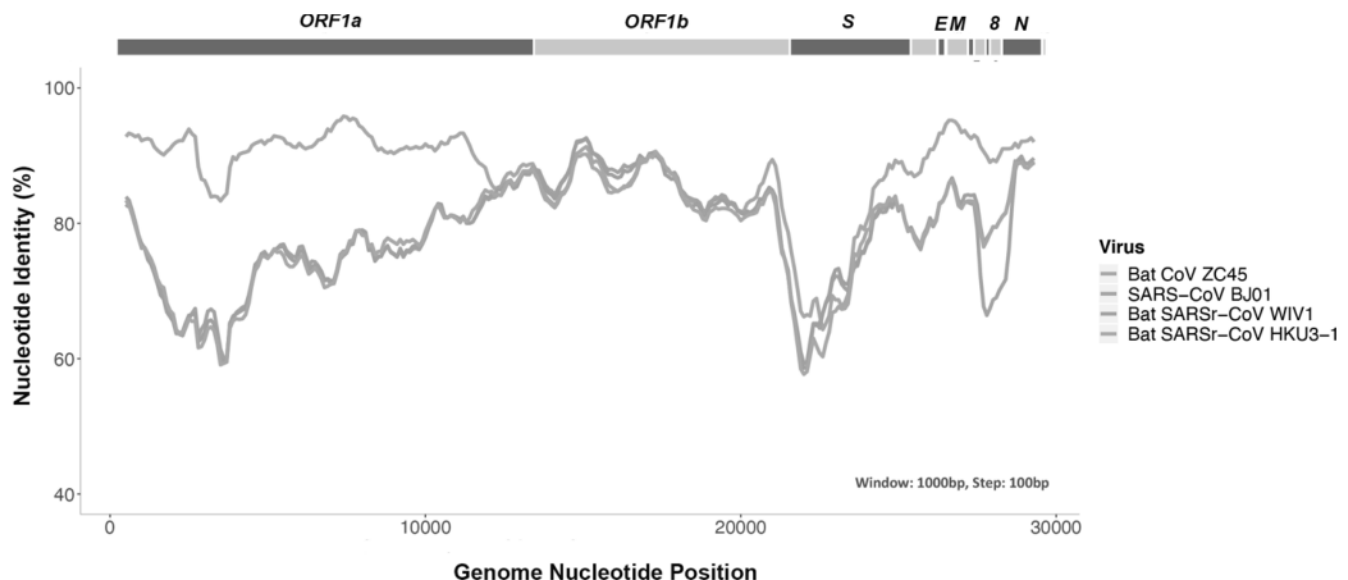


Figure 1. Genomic sequence analysis reveals that bat coronavirus ZC45 is the closest match to SARS-CoV-2. Top: genomic organization of SARS-CoV-2 (2019-nCoV WIV04). Bottom: similarity plot based on the full-length genome of 2019-nCoV WIV04. Full-length genomes of SARS-CoV BJ01, bat SARSr-CoV WIV1, bat SARSr-CoV HKU3-1, bat coronavirus ZC45 were used as reference sequences.

When SARS-CoV-2 and ZC45/ZXC21 are compared on the amino acid level, a high sequence identity is observed for most of the proteins. The Nucleocapsid protein is 94% identical. The Membrane protein is 98.6% identical. The S2 portion (2nd half) of the Spike protein is 95% identical. Importantly, the Orf8 protein is 94.2% identical and the E protein is 100% identical.

Orf8 is an accessory protein, the function of which is largely unknown in most coronaviruses, although recent data suggests that Orf8 of SARS-CoV-2 mediates the evasion of host adaptive immunity by downregulating MHC-I²⁴. Normally, Orf8 is poorly conserved in coronaviruses²⁵. Sequence blast indicates that, while the Orf8 proteins of ZC45/ZXC21 share a 94.2% identity with SARS-CoV-2 Orf8, no other coronaviruses share more than 58% identity with SARS-CoV-2 on this particular protein. The very high homology here on the normally poorly conserved Orf8 protein is highly unusual.

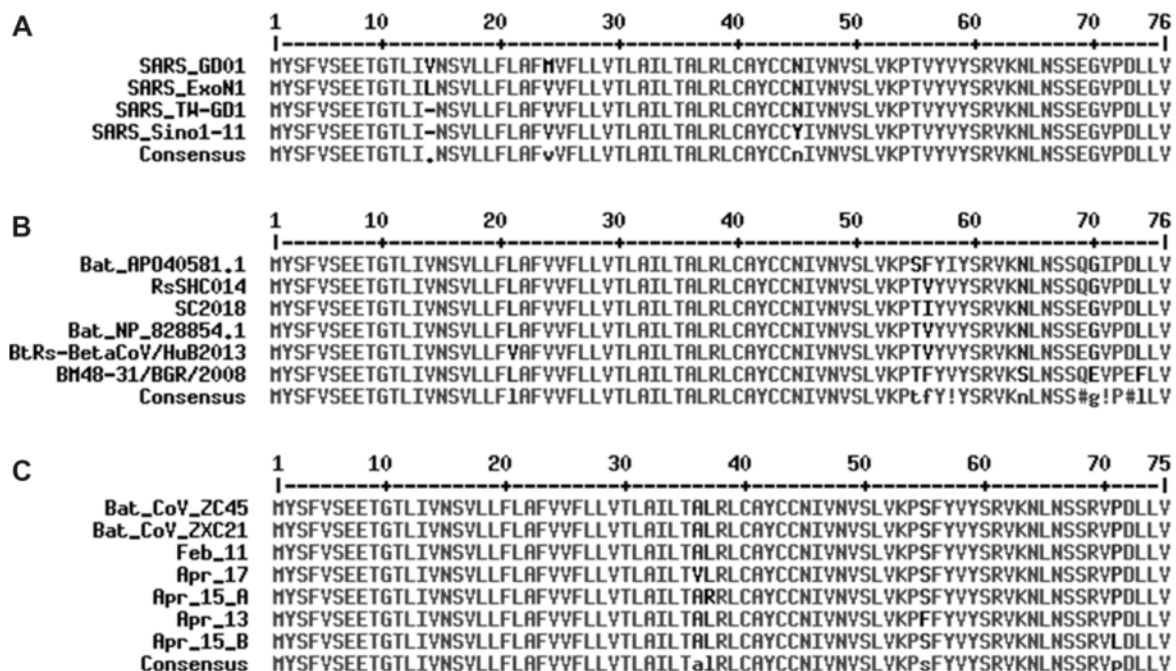


Figure 2. Sequence alignment of the E proteins from different β coronaviruses demonstrates the E protein's permissiveness and tendency toward amino acid mutations. A. Mutations have been observed in different strains of SARS-CoV. GenBank accession numbers: SARS_GD01: AY278489.2, SARS_ExoN1: ACB69908.1, SARS_TW_GD1: AY451881.1, SARS_Sino1_11: AY485277.1. B. Alignment of E proteins from related bat coronaviruses indicates its tolerance of mutations at multiple positions. GenBank accession numbers: Bat_AP040581.1: APO40581.1, RsSHC014: KC881005.1, SC2018: MK211374.1, Bat_NP_828854.1: NP_828854.1, BtRs-BetaCoV/HuB2013: AIA62312.1, BM48-31/BGR/2008: YP_003858586.1. C. While the early copies of SARS-CoV-2 share 100% identity on the E protein with ZC45 and ZXC21, sequencing data of SARS-CoV-2 from April 2020 indicates that mutation has occurred at multiple positions. Accession numbers of viruses: Feb_11: MN997409, ZC45: MG772933.1, ZXC21: MG772934, Apr_13: MT326139, Apr_15_A: MT263389, Apr_15_B: MT293206, Apr_17: MT350246. Alignments were done using the MultAlin webserver (<http://multalin.toulouse.inra.fr/multalin/>).

The coronavirus E protein is a structural protein, which is embedded in and lines the interior of the membrane envelope of the virion²⁶. The E protein is tolerant of mutations as evidenced in both SARS (Figure 2A) and related bat coronaviruses (Figure 2B). This tolerance to amino acid mutations of the E protein is further evidenced in the current SARS-CoV-2 pandemic. After only a short two-month spread of the virus since its outbreak in humans, the E proteins in SARS-CoV-2 have already undergone mutational changes. Sequence data obtained during the month of April reveals that mutations have occurred at four different locations in different strains (Figure 2C). Consistent with this finding, sequence blast analysis indicates that, with the exception of SARS-CoV-2, no known coronaviruses share 100% amino acid sequence identity on the E protein with ZC45/ZXC21 (*suspicious coronaviruses published after the start of the current pandemic are excluded*^{18,27-31}). Although 100% identity on the E protein has been observed between SARS-CoV and certain SARS-related bat coronaviruses, none of those pairs simultaneously share over 83% identity on the Orf8 protein³². Therefore, the 94.2% identity on the Orf8 protein, 100% identity on the E protein, and the overall genomic/amino acid-level resemblance between SARS-CoV-2 and ZC45/ZXC21 are highly unusual. Such evidence, when considered together, is consistent with a hypothesis that the SARS-CoV-2 genome has an origin based on the use of ZC45/ZXC21 as a backbone and/or template for genetic gain-of-function modifications.

Importantly, ZC45 and ZXC21 are bat coronaviruses that were discovered (between July 2015 and February 2017), isolated, and characterized by military research laboratories in the Third Military Medical University (Chongqing, China) and the Research Institute for Medicine of Nanjing Command (Nanjing, China). The data and associated work were published in 2018^{33,34}. Clearly, this backbone/template, which is essential for the creation of SARS-CoV-2, exists in these and other related research laboratories.

What strengthens our contention further is the published RaTG13 virus¹⁸, the genomic sequence of which is reportedly 96% identical to that of SARS-CoV-2. While suggesting a natural origin of SARS-CoV-2, the RaTG13 virus also diverted the attention of both the scientific field and the general public away from ZC45/ZXC21^{4,18}. In fact, a Chinese BSL-3 lab (the Shanghai Public Health Clinical Centre), which published a *Nature* article reporting a conflicting close phylogenetic relationship between SARS-CoV-2 and ZC45/ZXC21 rather than with RaTG13³⁵, was quickly shut down for “rectification”³⁶. It is believed that the researchers of that laboratory were being punished for having disclosed the SARS-CoV-2—ZC45/ZXC21 connection. On the other hand, substantial evidence has accumulated, pointing to severe problems associated with the reported sequence of RaTG13 as well as questioning the actual existence of this bat virus in nature^{6,7,19-21}. A very recent publication also indicated that the receptor-binding domain (RBD) of the RaTG13’s Spike protein could not bind ACE2 of two different types of horseshoe bats (they closely relate to the horseshoe bat *R. affinis*, RaTG13’s alleged natural host)², implicating the inability of RaTG13 to infect horseshoe bats. This finding further substantiates the suspicion that the reported sequence of RaTG13 could have been fabricated as the Spike protein encoded by this sequence does not seem to carry the claimed function. The fact that a virus has been fabricated to shift the attention away from ZC45/ZXC21 speaks for an actual role of ZC45/ZXC21 in the creation of SARS-CoV-2.

1.2 The receptor-binding motif of SARS-CoV-2 Spike cannot be born from nature and should have been created through genetic engineering

The Spike proteins decorate the exterior of the coronavirus particles. They play an important role in infection as they mediate the interaction with host cell receptors and thereby help determine the host range and tissue tropism of the virus. The Spike protein is split into two halves (Figure 3). The front or N-terminal half is named S1, which is fully responsible for binding the host receptor. In both SARS-CoV

and SARS-CoV-2 infections, the host cell receptor is hACE2. Within S1, a segment of around 70 amino acids makes direct contacts with hACE2 and is correspondingly named the receptor-binding motif (RBM) (Figure 3C). In SARS-CoV and SARS-CoV-2, the RBM fully determines the interaction with hACE2. The C-terminal half of the Spike protein is named S2. The main function of S2 includes maintaining trimer formation and, upon successive protease cleavages at the S1/S2 junction and a downstream S2' position, mediating membrane fusion to enable cellular entry of the virus.

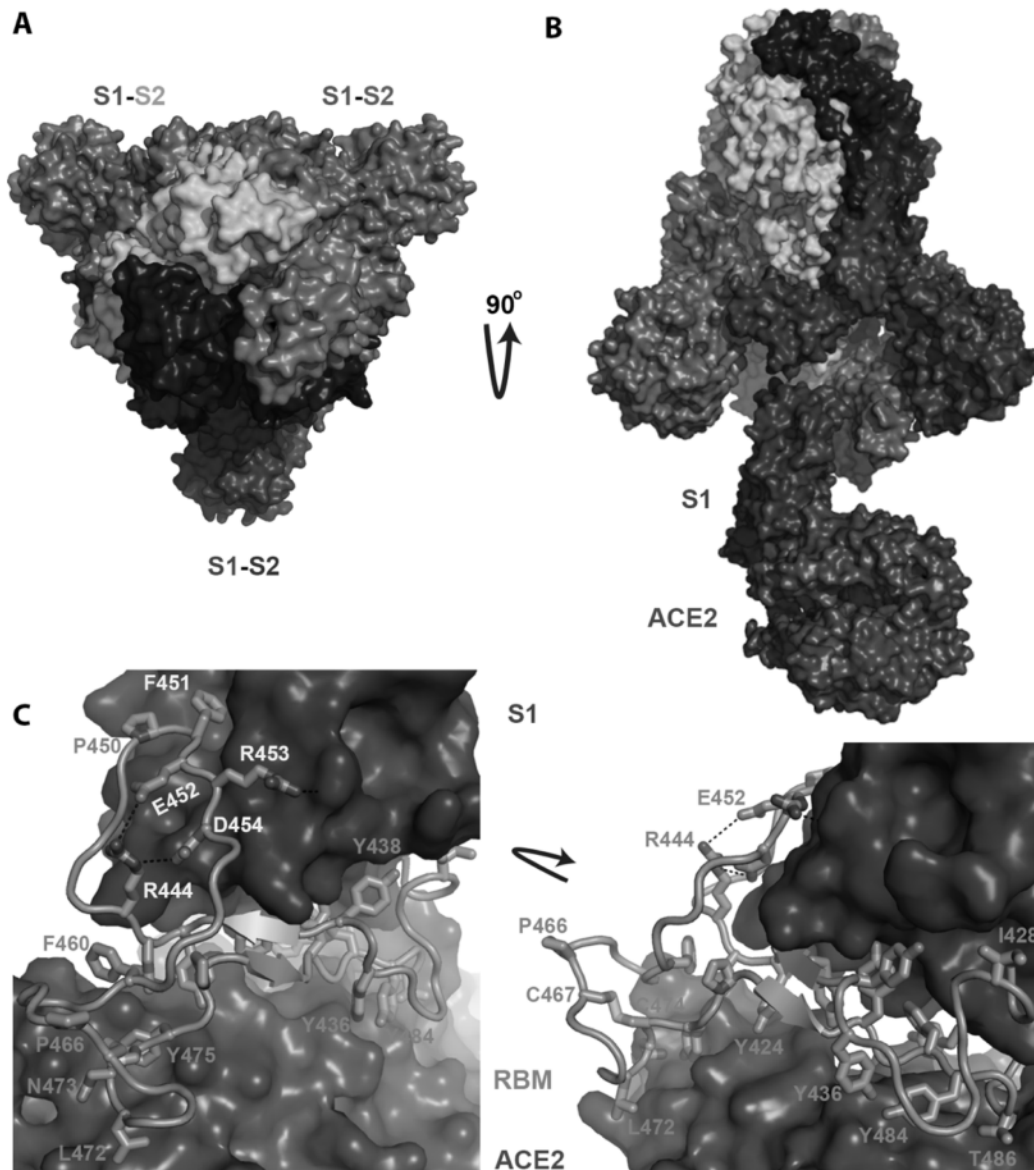


Figure 3. Structure of the SARS Spike protein and how it binds to the hACE2 receptor. Pictures were generated based on PDB ID: 6acj³⁷. A) Three spike proteins, each consisting of a S1 half and a S2 half, form a trimer. B) The S2 halves (shades of blue) are responsible for trimer formation, while the S1 portion (shades of red) is responsible for binding hACE2 (dark gray). C) Details of the binding between S1 and hACE2. The RBM of S1, which is important and sufficient for binding, is colored in orange. Residues within the RBM that are important for either hACE2 interaction or protein folding are shown as sticks (residue numbers follow the SARS Spike sequence).

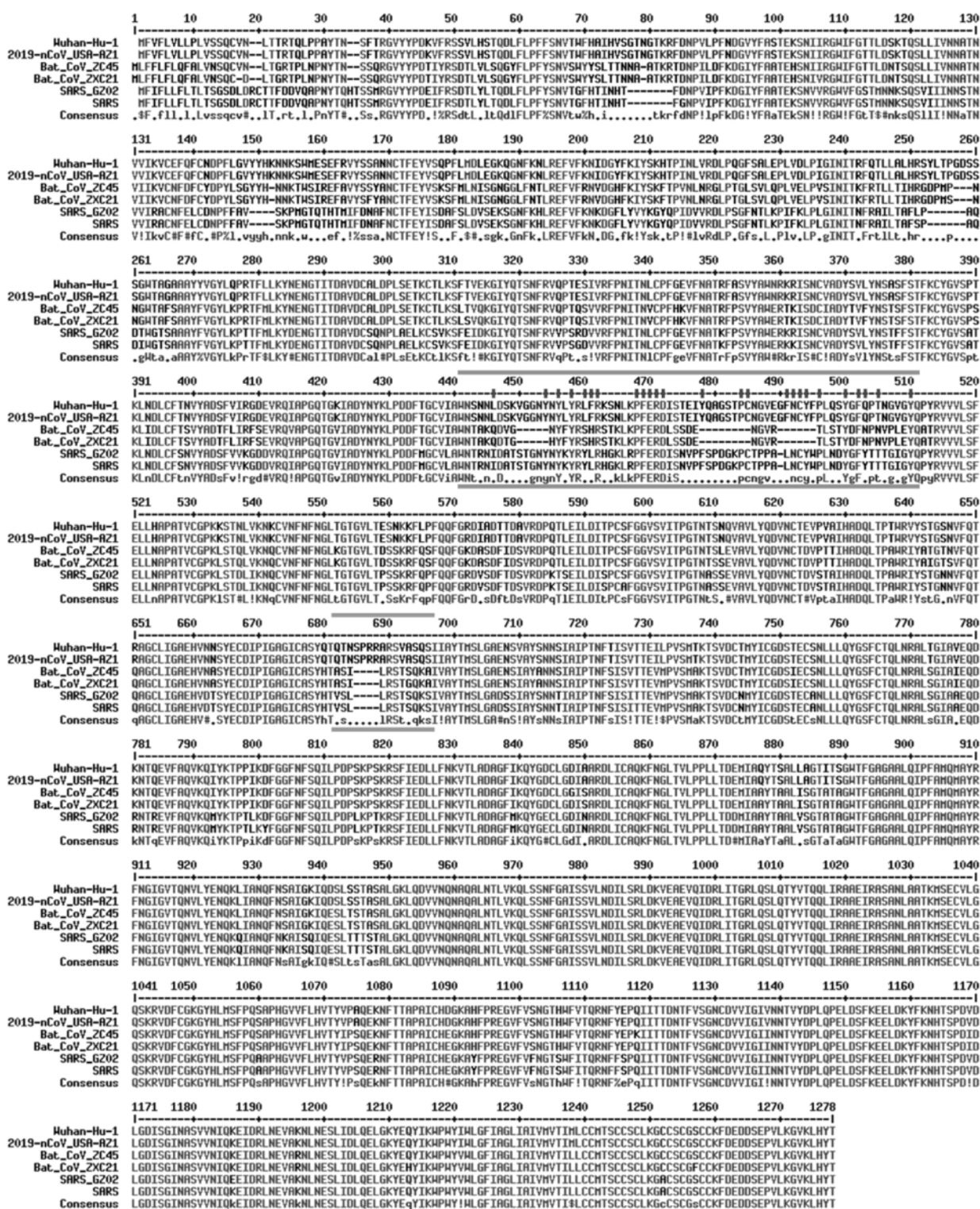


Figure 4. Sequence alignment of the spike proteins from relevant coronaviruses. Viruses being compared include SARS-CoV-2 (Wuhan-Hu-1: NC_045512, 2019-nCoV_USA-AZ1: MN997409), bat coronaviruses (Bat_CoV_ZC45: MG772933, Bat_CoV_ZXC21: MG772934), and SARS coronavirus (SARS_GZ02: AY390556, SARS: NC_004718.3). Region marked by two orange lines is the receptor-binding motif (RBM), which is important for interaction with the hACE2 receptor. Essential residues are additionally highlighted by red sticks on top. Region marked by two green lines is a furin-cleavage site that exists only in SARS-CoV-2 but not in any other lineage B β coronavirus.

Similar to what is observed for other viral proteins, S2 of SARS-CoV-2 shares a high sequence identity (95%) with S2 of ZC45/ZXC21. In stark contrast, between SARS-CoV-2 and ZC45/ZXC21, the S1 protein, which dictates which host (human or bat) the virus can infect, is much less conserved with the amino acid sequence identity being only 69%.

Figure 4 shows the sequence alignment of the Spike proteins from six β coronaviruses. Two are viruses isolated from the current pandemic (Wuhan-Hu-1, 2019-nCoV_USA-AZ1); two are the suspected template viruses (Bat_CoV_ZC45, Bat_CoV_ZXC21); two are SARS coronaviruses (SARS_GZ02, SARS). The RBM is highlighted in between two orange lines. Clearly, despite the high sequence identity for the overall genomes, the RBM of SARS-CoV-2 differs significantly from those of ZC45 and ZXC21. Intriguingly, the RBM of SARS-CoV-2 resembles, on a great deal, the RBM of SARS Spike. Although this is not an exact “copy and paste”, careful examination of the Spike-hACE2 structures^{37,38} reveals that all residues essential for either hACE2 binding or protein folding (orange sticks in Figure 3C and what is highlighted by red short lines in Figure 4) are “kept”. Most of these essential residues are precisely preserved, including those involved in disulfide bond formation (C467, C474) and electrostatic interactions (R444, E452, R453, D454), which are pivotal for the structural integrity of the RBM (Figure 3C and 4). The few changes within the group of essential residues are almost exclusively hydrophobic “substitutions” (I428→L, L443→F, F460→Y, L472→F, Y484→Q), which should not affect either protein folding or the hACE2-interaction. At the same time, majority of the amino acid residues that are non-essential have “mutated” (Figure 4, RBM residues not labeled with short red lines). Judging from this sequence analysis alone, we were convinced early on that not only would the SARS-CoV-2 Spike protein bind hACE2 but also the binding would resemble, precisely, that between the original SARS Spike protein and hACE2²³. Recent structural work has confirmed our prediction³⁹.

As elaborated below, the way that SARS-CoV-2 RBM resembles SARS-CoV RBM and the overall sequence conservation pattern between SARS-CoV-2 and ZC45/ZXC21 are highly unusual. Collectively, this suggests that portions of the SARS-CoV-2 genome have not been derived from natural quasi-species viral particle evolution.

If SARS-CoV-2 does indeed come from natural evolution, its RBM could have only been acquired in one of the two possible routes: 1) an ancient recombination event followed by convergent evolution or 2) a natural recombination event that occurred fairly recently.

In the first scenario, the ancestor of SARS-CoV-2, a ZC45/ZXC21-like bat coronavirus would have recombined and “swapped” its RBM with a coronavirus carrying a relatively “complete” RBM (in reference to SARS). This recombination would result in a novel ZC45/ZXC21-like coronavirus with all the gaps in its RBM “filled” (Figure 4). Subsequently, the virus would have to adapt extensively in its new host, where the ACE2 protein is highly homologous to hACE2. Random mutations across the genome would have to have occurred to eventually shape the RBM to its current form – resembling SARS-CoV RBM in a highly intelligent manner. However, this convergent evolution process would also result in the accumulation of a large amount of mutations in other parts of the genome, rendering the overall sequence identity relatively low. The high sequence identity between SARS-CoV-2 and ZC45/ZXC21 on various proteins (94-100% identity) do not support this scenario and, therefore, clearly indicates that SARS-CoV-2 carrying such an RBM cannot come from a ZC45/ZXC21-like bat coronavirus through this convergent evolutionary route.

In the second scenario, the ZC45/ZXC21-like coronavirus would have to have recently recombined and swapped its RBM with another coronavirus that had successfully adapted to bind an animal ACE2

highly homologous to hACE2. The likelihood of such an event depends, in part, on the general requirements of natural recombination: 1) that the two different viruses share significant sequence similarity; 2) that they must co-infect and be present in the same cell of the same animal; 3) that the recombinant virus would not be cleared by the host or make the host extinct; 4) that the recombinant virus eventually would have to become stable and transmissible within the host species.

In regard to this recent recombination scenario, the animal reservoir could not be bats because the ACE2 proteins in bats are not homologous enough to hACE2 and therefore the adaption would not be able to yield an RBM sequence as seen in SARS-CoV-2. This animal reservoir also could not be humans as the ZC45/ZXC21-like coronavirus would not be able to infect humans. In addition, there has been no evidence of any SARS-CoV-2 or SARS-CoV-2-like virus circulating in the human population prior to late 2019. Intriguingly, according to a recent bioinformatics study, SARS-CoV-2 was well-adapted for humans since the start of the outbreak¹.

Only one other possibility of natural evolution remains, which is that the ZC45/ZXC21-like virus and a coronavirus containing a SARS-like RBM could have recombined in an intermediate host where the ACE2 protein is homologous to hACE2. Several laboratories have reported that some of the Sunda pangolins smuggled into China from Malaysia carried coronaviruses, the receptor-binding domain (RBD) of which is almost identical to that of SARS-CoV-2^{27-29,31}. They then went on to suggest that pangolins are the likely intermediate host for SARS-CoV-2^{27-29,31}. However, recent independent reports have found significant flaws in this data⁴⁰⁻⁴². Furthermore, contrary to these reports^{27-29,31}, no coronaviruses have been detected in Sunda pangolin samples collected for over a decade in Malaysia and Sabah between 2009 and 2019⁴³. A recent study also showed that the RBD, which is shared between SARS-CoV-2 and the reported pangolin coronaviruses, binds to hACE2 ten times stronger than to the pangolin ACE2², further dismissing pangolins as the possible intermediate host. Finally, an *in silico* study, while echoing the notion that pangolins are not likely an intermediate host, also indicated that none of the animal ACE2 proteins examined in their study exhibited more favorable binding potential to the SARS-CoV-2 Spike protein than hACE2 did³. This last study virtually exempted all animals from their suspected roles as an intermediate host³, which is consistent with the observation that SARS-CoV-2 was well-adapted for humans from the start of the outbreak¹. This is significant because these findings collectively suggest that no intermediate host seems to exist for SARS-CoV-2, which at the very least diminishes the possibility of a recombinant event occurring in an intermediate host.

Even if we ignore the above evidence that no proper host exists for the recombination to take place and instead assume that such a host does exist, it is still highly unlikely that such a recombination event could occur in nature.

As we have described above, if natural recombination event is responsible for the appearance of SARS-CoV-2, then the ZC45/ZXC21-like virus and a coronavirus containing a SARS-like RBM would have to recombine in the same cell by swapping the S1/RBM, which is a rare form of recombination. Furthermore, since SARS has occurred only once in human history, it would be at least equally rare for nature to produce a virus that resembles SARS in such an intelligent manner – having an RBM that differs from the SARS RBM only at a few non-essential sites (Figure 4). The possibility that this unique SARS-like coronavirus would reside in the same cell with the ZC45/ZXC21-like ancestor virus and the two viruses would recombine in the “RBM-swapping” fashion is extremely low. Importantly, this, and the other recombination event described below in section 1.3 (even more impossible to occur in nature), would both have to happen to produce a Spike as seen in SARS-CoV-2.

While the above evidence and analyses together appear to disapprove a natural origin of SARS-CoV-2's RBM, abundant literature shows that gain-of-function research, where the Spike protein of a coronavirus was specifically engineered, has repeatedly led to the successful generation of human-infecting coronaviruses from coronaviruses of non-human origin⁴⁴⁻⁴⁷.

Record also shows that research laboratories, for example, the Wuhan Institute of Virology (WIV), have successfully carried out such studies working with US researchers⁴⁵ and also working alone⁴⁷. In addition, the WIV has engaged in decades-long coronavirus surveillance studies and therefore owns the world's largest collection of coronaviruses. Evidently, the technical barrier is non-existent for the WIV and other related laboratories to carry out and succeed in such Spike/RBM engineering and gain-of-function research.

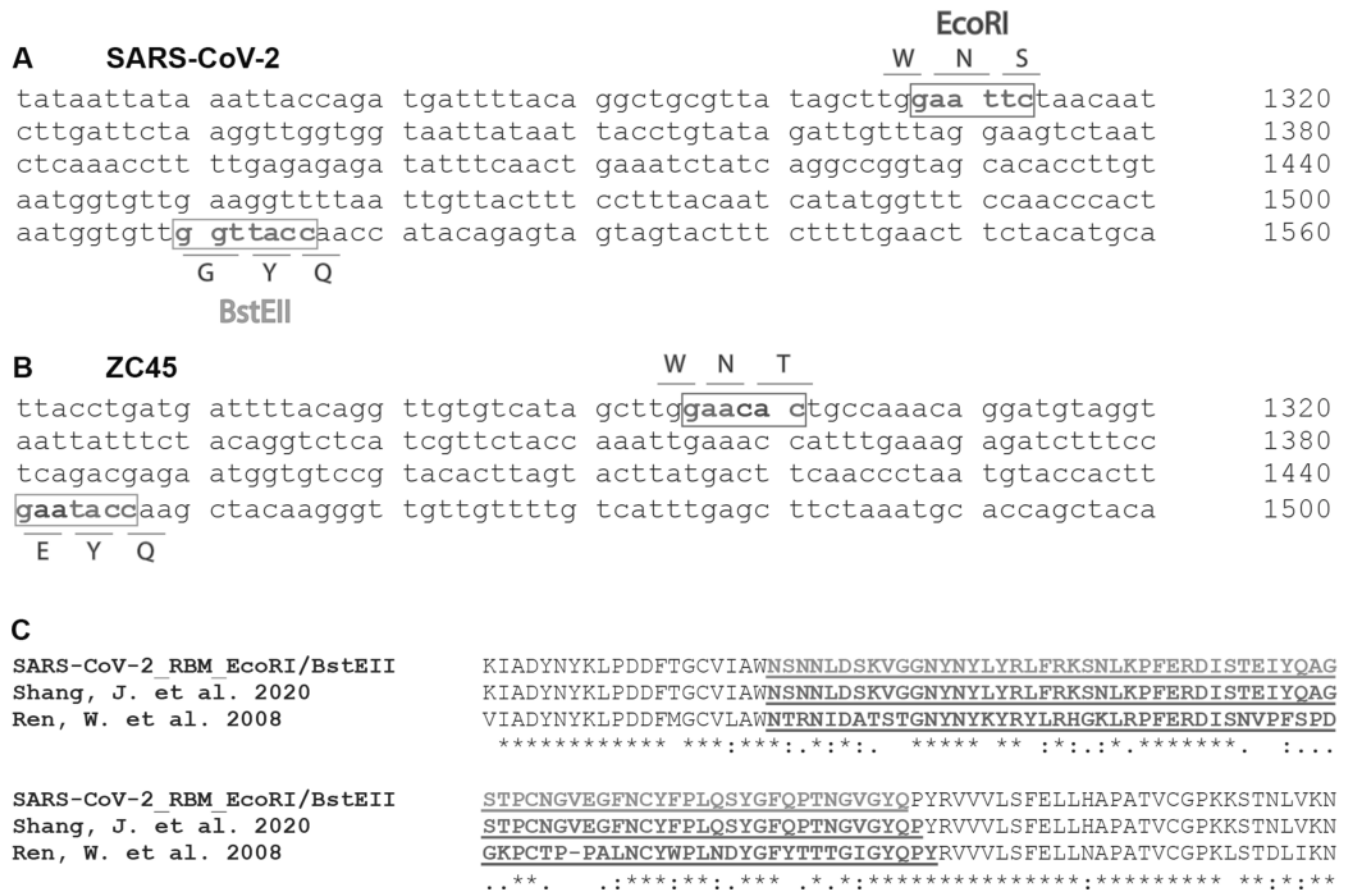


Figure 5. Two restriction sites are present at either end of the RBM of SARS-CoV-2, providing convenience for replacing the RBM within the spike gene. A. Nucleotide sequence of the RBM of SARS-CoV-2 (Wuhan-Hu-1). An EcoRI site is found at the 5'-end of the RBM and a BstEII site at the 3'-end. B. Although these two restriction sites do not exist in the original spike gene of ZC45, they can be conveniently introduced given that the sequence discrepancy is small (2 nucleotides) in either case. C. Amino acid sequence alignment with the RBM region highlighted (color and underscore). The RBM highlighted in orange (top) is what is defined by the EcoRI and BstEII sites in the SARS-CoV-2 (Wuhan-Hu-1) spike. The RBM highlighted in magenta (middle) is the region swapped by Dr. Fang Li and colleagues into a SARS Spike backbone³⁹. The RBM highlighted in blue (bottom) is from the Spike protein (RBM: 424-494) of SARS-BJ01 (AY278488.2), which was swapped by the Shi lab into the Spike proteins of different bat coronaviruses replacing the corresponding segments⁴⁷.

Strikingly, consistent with the RBM engineering theory, we have identified two unique restriction sites, EcoRI and BstEII, at either end of the *RBM* of the SARS-CoV-2 genome, respectively (Figure 5A). These two sites, which are popular choices of everyday molecular cloning, do not exist in the rest of this *spike* gene. This particular setting makes it extremely convenient to swap the *RBM* within *spike*, providing a quick way to test different RBMs and the corresponding Spike proteins.

Such EcoRI and BstEII sites do not exist in the *spike* genes of other β coronaviruses, which strongly indicates that they were unnatural and were specifically introduced into this *spike* gene of SARS-CoV-2 for the convenience of manipulating the critical RBM. Although ZC45 *spike* also does not have these two sites (Figure 5B), they can be introduced very easily as described in part 2 of this report.

It is noteworthy that introduction of the EcoRI site here would change the corresponding amino acids from -*WNT*- to -*WNS*- (Figure 5AB). As far as we know, all SARS and SARS-like bat coronaviruses exclusively carry a *T* (threonine) residue at this location. SARS-CoV-2 is the only exception in that this *T* has mutated to an *S* (serine), save the suspicious RaTG13 and pangolin coronaviruses published after the outbreak⁴⁸.

Once the restriction sites were successfully introduced, the *RBM* segment could be swapped conveniently using routine restriction enzyme digestion and ligation. Although alternative cloning techniques may leave no trace of genetic manipulation (Gibson assembly as one example), this old-fashioned approach could be chosen because it offers a great level of convenience in swapping this critical *RBM*.

Given that RBM fully dictates hACE2-binding and that the SARS RBM-hACE2 binding was fully characterized by high-resolution structures (Figure 3)^{37,38}, this RBM-only swap would not be any riskier than the full Spike swap. In fact, the feasibility of this RBM-swap strategy has been proven^{39,47}. In 2008, Dr. Zhengli Shi's group swapped a SARS RBM into the Spike proteins of several SARS-like bat coronaviruses after introducing a restriction site into a codon-optimized *spike* gene (Figure 5C)⁴⁷. They then validated the binding of the resulted chimeric Spike proteins with hACE2. Furthermore, in a recent publication, the RBM of SARS-CoV-2 was swapped into the receptor-binding domain (RBD) of SARS-CoV, resulting in a chimeric RBD fully functional in binding hACE2 (Figure 5C)³⁹. Strikingly, in both cases, the manipulated RBM segments resemble almost exactly the RBM defined by the positions of the EcoRI and BstEII sites (Figure 5C). Although cloning details are lacking in both publications^{39,47}, it is conceivable that the actual restriction sites may vary depending on the *spike* gene receiving the RBM insertion as well as the convenience in introducing unique restriction site(s) in regions of interest. It is noteworthy that the corresponding author of this recent publication³⁹, Dr. Fang Li, has been an active collaborator of Dr. Zhengli Shi since 2010⁴⁹⁻⁵³. Dr. Li was the first person in the world to have structurally elucidated the binding between SARS-CoV RBD and hACE2³⁸ and has been the leading expert in the structural understanding of Spike-ACE2 interactions^{38,39,53-56}. The striking finding of EcoRI and BstEII restriction sites at either end of the SARS-CoV-2 RBM, respectively, and the fact that the same RBM region has been swapped both by Dr. Shi and by her long-term collaborator, respectively, using restriction enzyme digestion methods are unlikely a coincidence. **Rather, it is the smoking gun proving that the RBM/Spike of SARS-CoV-2 is a product of genetic manipulation.**

Although it may be convenient to copy the exact sequence of SARS RBM, it would be too clear a sign of artificial design and manipulation. The more deceiving approach would be to change a few non-essential residues, while preserving the ones critical for binding. This design could be well-guided by the high-resolution structures (Figure 3)^{37,38}. This way, when the overall sequence of the RBM would appear

to be more distinct from that of the SARS RBM, the hACE2-binding ability would be well-preserved. We believe that all of the crucial residues (residues labeled with red sticks in Figure 4, which are the same residues shown in sticks in Figure 3C) should have been “kept”. As described earlier, while some should be direct preservation, some should have been switched to residues with similar properties, which would not disrupt hACE2-binding and may even strengthen the association further. Importantly, changes might have been made intentionally at non-essential sites, making it less like a “copy and paste” of the SARS RBM.

1.3 An unusual furin-cleavage site is present in the Spike protein of SARS-CoV-2 and is associated with the augmented virulence of the virus

Another unique motif in the Spike protein of SARS-CoV-2 is a polybasic furin-cleavage site located at the S1/S2 junction (Figure 4, segment in between two green lines). Such a site can be recognized and cleaved by the furin protease. Within the lineage B of β coronaviruses and with the exception of SARS-CoV-2, no viruses contain a furin-cleavage site at the S1/S2 junction (Figure 6)⁵⁷. In contrast, furin-cleavage site at this location has been observed in other groups of coronaviruses^{57,58}. Certain selective pressure seems to be in place that prevents the lineage B of β coronaviruses from acquiring or maintaining such a site in nature.

Human SARS-CoV BJ01	655 - GICASYHTVSSL----RSTS - 670
Human SARS-CoV CUHK-W1	655 - GICASYHTVSSL----RSTS - 670
Human SARS-CoV Tor2	655 - GICASYHTVSSL----RSTS - 670
Human SARS-CoV Frankfurt-1	655 - GICASYHTVSSL----RSTS - 670
Human SARS-CoV Urbani	655 - GICASYHTVSSL----RSTS - 670
Civet SARS-CoV civet020	655 - GICASYHTVSSL----RSTS - 670
Civet SARS-CoV sz16	655 - GICASYHTVSSL----RSTS - 670
Raccoon dog SARS-CoV A030	655 - GICASYHTVSSL----RSTS - 670
SARS-CoV-2	669 - GICASYQTQTNSPRRARSVA - 688
Fangolin CoV MP789	n/a - GICASYQTQTNS----RSVS - n/a
Bat SARSr-CoV RaTG13	669 - GICASYQTQTNS----RSVA - 684
Bat SARSr-CoV LYRa11	659 - GICASYHTASLL----RNTD - 674
Bat SARSr-CoV LYRa3	659 - GICASYHTASLL----RNTG - 674
Bat SARSr-CoV RsSHC014	656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs4084	656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV WIV1	656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs3367	656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs7327	656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs9401	656 - GICASYHTVSSL----RSTS - 671
Bat SARSr-CoV Rs4231	655 - GICASYHTVSSL----RSTS - 670
Bat SARSr-CoV WIV16	655 - GICASYHTVSSL----RSTS - 670
Bat SARSr-CoV Rs4874	655 - GICASYHTVSSL----RSTS - 670
Bat SARSr-CoV ZC45	646 - GICASYHTASLL----RSTS - 661
Bat SARSr-CoV ZXC21	645 - GICASYHTASLL----RSTG - 660
Bat SARSr-CoV Rf4092	634 - GICASYHTASLL----RGVG - 649
Bat SARSr-CoV Rf/JL2012	636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV JTM15	636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV 16B0133	636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV B15-21	636 - GICASYHTASLL----RSTG - 651
Bat SARSr-CoV YN2013	633 - GICASYHTASLL----RSIG - 648
Bat SARSr-CoV Anlong-103	633 - GICASYHTASLL----RSVG - 648
Bat SARSr-CoV Rp/Shaanxi2011	640 - GICASYHTASVL----RSTG - 655
Bat SARSr-CoV Rs/HuB2013	641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV YNLF/34C	641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV YNLF/31C	641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV Rf1	641 - GICASYHTASHL----RSTG - 656
Bat SARSr-CoV 273	641 - GICASYHTASHL----RSTG - 656
Bat SARSr-CoV Rf/SX2013	639 - GICASYHTASLL----RSTG - 654
Bat SARSr-CoV Rf/HeB2013	641 - GICASYHTASLL----RSTG - 656
Bat SARSr-CoV Cp/Yunnan2011	641 - GICASYHTASLL----RNTG - 656
Bat SARSr-CoV Rs672	641 - GICASYHTASLL----RSVG - 656
Bat SARSr-CoV Rs4255	641 - GICASYHTASLL----RSVG - 656
Bat SARSr-CoV 4081	641 - GICASYHTASLL----RSVG - 656
Bat SARSr-CoV Rm1	641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV 279	641 - GICASYHTASVL----RSTG - 656
Bat SARSr-CoV Rs/GX2013	642 - GICASYHTASVL----RSTG - 657
Bat SARSr-CoV Rs806	641 - GICASYHTASLL----RSTG - 656
Bat SARSr-CoV HKU3-1	642 - GICASYHTASVL----RSTG - 657
Bat SARSr-CoV Longquan-140	642 - GICASYHTASVL----RSTG - 657
Bat SARSr-CoV Rp3	641 - GICASYHTASLL----RSVG - 656
Bat SARSr-CoV Rs4247	642 - GICASYHTASLL----RSVG - 657
Bat SARSr-CoV Rs4237	641 - GICASYHTASLL----RSVG - 656
Bat SARSr-CoV As6526	641 - GICASYHTASLL----RSVG - 656

Figure 6. Furin-cleavage site found at the S1/S2 junction of Spike is unique to SARS-CoV-2 and absent in other lineage B β coronaviruses. Figure reproduced from Hoffmann, et al⁵⁷.

As previously described, during the cell entry process, the Spike protein is first cleaved at the S1/S2 junction. This step, and a subsequent cleavage downstream that exposes the fusion peptide, are both mediated by host proteases. The presence or absence of these proteases in different cell types greatly affects the cell tropism and presumably the pathogenicity of the viral infection. Unlike other proteases, furin protease is widely expressed in many types of cells and is present at multiple cellular and extracellular locations. Importantly, the introduction of a furin-cleavage site at the S1/S2 junction could significantly enhance the infectivity of a virus as well as greatly expand its cell tropism — a phenomenon well-documented in both influenza viruses and other coronaviruses⁵⁹⁻⁶⁵.

If we leave aside the fact that no furin-cleavage site is found in any lineage B β coronavirus in nature and instead assume that this site in SARS-CoV-2 is a result of natural evolution, then only one evolutionary pathway is possible, which is that the furin-cleavage site has to be derived from a homologous recombination event. Specifically, an ancestor β coronavirus containing no furin-cleavage site would have to recombine with a closely related coronavirus that does contain a furin-cleavage site.

However, two facts disfavor this possibility. First, although some coronaviruses from other groups or lineages do contain polybasic furin-cleavage sites, none of them contains the exact polybasic sequence present in SARS-CoV-2 (-PRRAR/SVA-). Second, between SARS-CoV-2 and any coronavirus containing a legitimate furin-cleavage site, the sequence identity on Spike is no more than 40%⁶⁶. Such a low level of sequence identity rules out the possibility of a successful homologous recombination ever occurring between the ancestors of these viruses. Therefore, the furin-cleavage site within the SARS-CoV-2 Spike protein is unlikely to be of natural origin and instead should be a result of laboratory modification.

Consistent with this claim, a close examination of the nucleotide sequence of the furin-cleavage site in SARS-CoV-2 *spike* has revealed that the two consecutive Arg residues within the inserted sequence (-PRRA-) are both coded by the rare codon CGG (least used codon for Arg in SARS-CoV-2) (Figure 7)⁸. In fact, this *CGGCGG* arrangement is the only instance found in the SARS-CoV-2 genome where this rare codon is used in tandem. This observation strongly suggests that this furin-cleavage site should be a result of genetic engineering. Adding to the suspicion, a *FauI* restriction site is formulated by the codon choices here, suggesting the possibility that the *restriction fragment length polymorphism*, a technique that a WIV lab is proficient at⁶⁷, could have been involved. There, the fragmentation pattern resulted from *FauI* digestion could be used to monitor the preservation of the furin-cleavage site in Spike as this furin-cleavage site is prone to deletions *in vitro*^{68,69}. Specifically, RT-PCR on the *spike* gene of the recovered viruses from cell cultures or laboratory animals could be carried out, the product of which would be subjected to *FauI* digestion. Viruses retaining or losing the furin-cleavage site would then yield distinct patterns, allowing convenient tracking of the virus(es) of interest.

FauI

tat	cag	act	cag	act	aat	tct	cct	cgg	cgg	gca	cgt	agt	gta	gct	agt	caa	tcc	atc	att
Y	Q	T	Q	T	N	S	P	R	R	A	R	S	V	A	S	Q	S	I	I

Figure 7. Two consecutive Arg residues in the -PRRA- insertion at the S1/S2 junction of SARS-CoV-2 Spike are both coded by a rare codon, CGG. A *FauI* restriction site, 5'-(N)₆GCGGG-3', is embedded in the coding sequence of the "inserted" PRRA segment, which may be used as a marker to monitor the preservation of the introduced furin-cleavage site.

In addition, although no known coronaviruses contain the exact sequence of -PRRAR/SVA- that is present in the SARS-CoV-2 Spike protein, a similar -RRAR/AR- sequence has been observed at the S1/S2 junction of the Spike protein in a rodent coronavirus, AcCoV-JC34, which was published by Dr. Zhengli

Shi in 2017⁷⁰. It is evident that the legitimacy of *-RRAR-* as a functional furin-cleavage site has been known to the WIV experts since 2017.

The evidence collectively suggests that the furin-cleavage site in the SARS-CoV-2 Spike protein may not have come from nature and could be the result of genetic manipulation. The purpose of this manipulation could have been to assess any potential enhancement of the infectivity and pathogenicity of the laboratory-made coronavirus⁵⁹⁻⁶⁴. Indeed, recent studies have confirmed that the furin-cleavage site does confer significant pathogenic advantages to SARS-CoV-2^{57,68}.

1.4 Summary

Evidence presented in this part reveals that certain aspects of the SARS-CoV-2 genome are extremely difficult to reconcile to being a result of natural evolution. The alternative theory we suggest is that the virus may have been created by using ZC45/ZXC21 bat coronavirus(es) as the backbone and/or template. The Spike protein, especially the RBM within it, should have been artificially manipulated, upon which the virus has acquired the ability to bind hACE2 and infect humans. This is supported by the finding of a unique restriction enzyme digestion site at either end of the RBM. An unusual furin-cleavage site may have been introduced and inserted at the S1/S2 junction of the Spike protein, which contributes to the increased virulence and pathogenicity of the virus. These transformations have then staged the SARS-CoV-2 virus to eventually become a highly-transmissible, onset-hidden, lethal, sequelae-unclear, and massively disruptive pathogen.

Evidently, the possibility that SARS-CoV-2 could have been created through gain-of-function manipulations at the WIV is significant and should be investigated thoroughly and independently.

2. Delineation of a synthetic route of SARS-CoV-2

In the second part of this report, we describe a synthetic route of creating SARS-CoV-2 in a laboratory setting. It is postulated based on substantial literature support as well as genetic evidence present in the SARS-CoV-2 genome. Although steps presented herein should not be viewed as exactly those taken, we believe that key processes should not be much different. Importantly, our work here should serve as a demonstration of how SARS-CoV-2 can be designed and created conveniently in research laboratories by following proven concepts and using well-established techniques.

Importantly, research labs, both in Hong Kong and in mainland China, are leading the world in coronavirus research, both in terms of resources and on the research outputs. The latter is evidenced not only by the large number of publications that they have produced over the past two decades but also by their milestone achievements in the field: they were the first to identify civets as the intermediate host for SARS-CoV and isolated the first strain of the virus⁷¹; they were the first to uncover that SARS-CoV originated from bats^{72,73}; they revealed for the first time the antibody-dependent enhancement (ADE) of SARS-CoV infections⁷⁴; they have contributed significantly in understanding MERS in all domains (zoonosis, virology, and clinical studies)⁷⁵⁻⁷⁹; they made several breakthroughs in SARS-CoV-2 research^{18,35,80}. Last but not least, they have the world's largest collection of coronaviruses (genomic sequences and live viruses). The knowledge, expertise, and resources are all readily available within the Hong Kong and mainland research laboratories (they collaborate extensively) to carry out and accomplish the work described below.

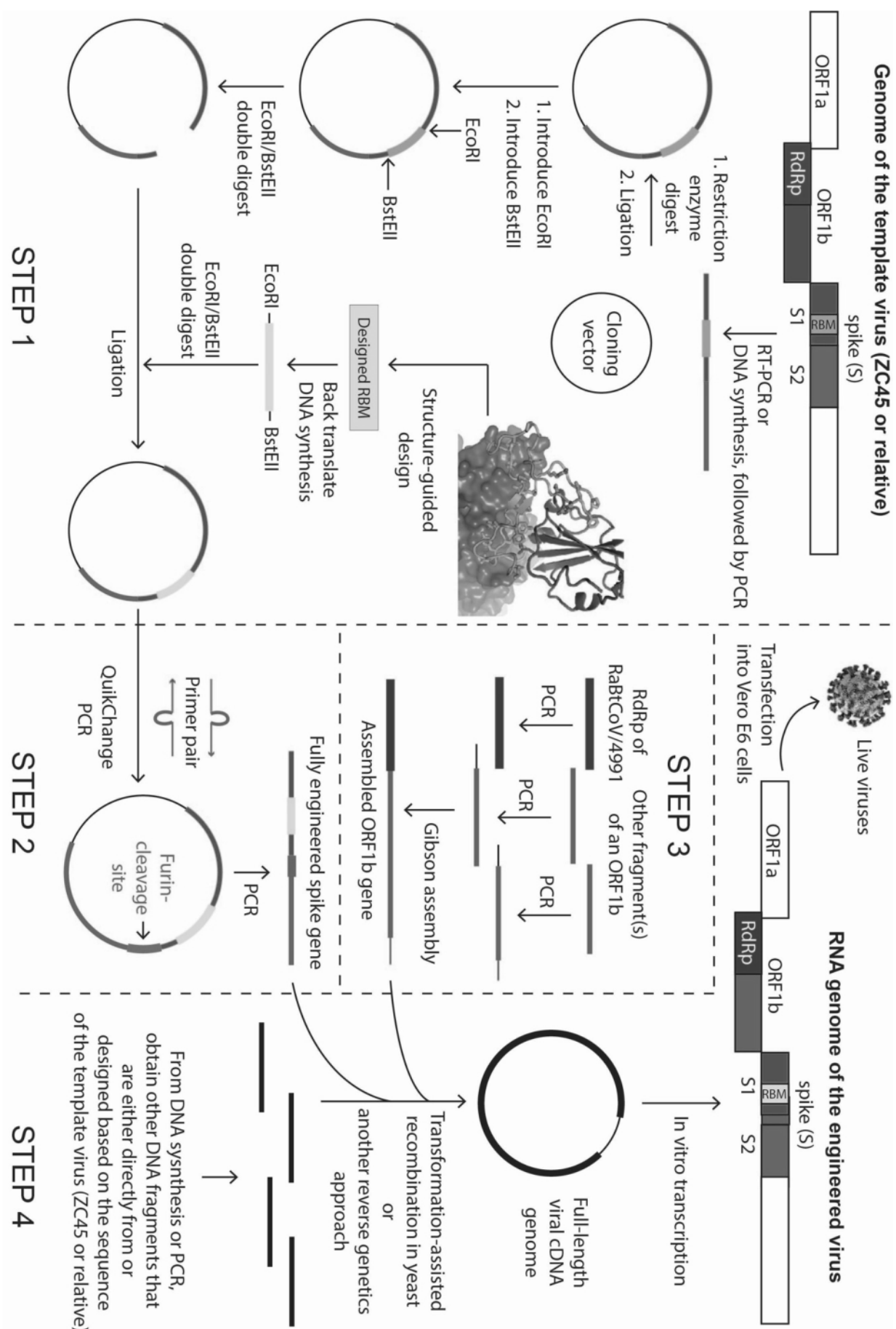


Figure 8. Diagram of a possible synthetic route of the laboratory-creation of SARS-CoV-2.

2.1 Possible scheme in designing the laboratory-creation of the novel coronavirus

In this sub-section, we outline the possible overall strategy and major considerations that may have been formulated at the designing stage of the project.

To engineer and create a human-targeting coronavirus, they would have to pick a bat coronavirus as the template/backbone. This can be conveniently done because many research labs have been actively collecting bat coronaviruses over the past two decades^{32,33,70,72,81-85}. However, this template virus ideally should not be one from Dr. Zhengli Shi's collections, considering that she is widely known to have been engaged in gain-of-function studies on coronaviruses. Therefore, ZC45 and/or ZXC21, novel bat coronaviruses discovered and owned by military laboratories³³, would be suitable as the template/backbone. It is also possible that these military laboratories had discovered other closely related viruses from the same location and kept some unpublished. Therefore, the actual template could be ZC45, or ZXC21, or a close relative of them. The postulated pathway described below would be the same regardless of which one of the three was the actual template.

Once they have chosen a template virus, they would first need to engineer, through molecular cloning, the Spike protein so that it can bind hACE2. The concept and cloning techniques involved in this manipulation have been well-documented in the literature^{44-46,84,86}. With almost no risk of failing, the template bat virus could then be converted to a coronavirus that can bind hACE2 and infect humans⁴⁴⁻⁴⁶.

Second, they would use molecular cloning to introduce a furin-cleavage site at the S1/S2 junction of Spike. This manipulation, based on known knowledge^{60,61,65}, would likely produce a strain of coronavirus that is a more infectious and pathogenic.

Third, they would produce an *ORF1b* gene construct. The *ORF1b* gene encodes the polyprotein Orf1b, which is processed post-translationally to produce individual viral proteins: RNA-dependent RNA polymerase (RdRp), helicase, guanidine-N7 methyltransferase, uridylylate-specific endoribonuclease, and 2'-O-methyltransferase. All of these proteins are parts of the replication machinery of the virus. Among them, the RdRp protein is the most crucial one and is highly conserved among coronaviruses. Importantly, Dr. Zhengli Shi's laboratory uses a PCR protocol, which amplifies a particular fragment of the *RdRp* gene, as their primary method to detect the presence of coronaviruses in raw samples (bat fecal swap, feces, etc). As a result of this practice, the Shi group has documented the sequence information of this short segment of *RdRp* for all coronaviruses that they have successfully detected and/or collected.

Here, the genetic manipulation is less demanding or complicated because Orf1b is conserved and likely Orf1b from any β coronavirus would be competent enough to do the work. However, we believe that they would want to introduce a particular Orf1b into the virus for one of the two possible reasons:

1. Since many phylogenetic analyses categorize coronaviruses based on the sequence similarity of the *RdRp* gene only^{18,31,35,83,87}, having a different *RdRp* in the genome therefore could ensure that SARS-CoV-2 and ZC45/ZXC21 are separated into different groups/sub-lineages in phylogenetic studies. Choosing an *RdRp* gene, however, is convenient because the short *RdRp* segment sequence has been recorded for all coronaviruses ever collected/detected. Their final choice was the *RdRp* sequence from bat coronavirus RaBtCoV/4991, which was discovered in 2013. For RaBtCoV/4991, the only information ever published was the sequence of its short *RdRp* segment⁸³, while neither its full genomic sequence nor virus isolation were ever reported. After amplifying the *RdRp* segment (or the whole *ORF1b* gene) of RaBtCoV/4991, they would have then used it for subsequent assembly and creation of the genome of SARS-CoV-2. Small changes in the *RdRp*

sequence could either be introduced at the beginning (through DNA synthesis) or be generated *via* passages later on. On a separate track, when they were engaged in the fabrication of the RaTG13 sequence, they could have started with the short *RdRp* segment of RaBtCoV/4991 without introducing any changes to its sequence, resulting in a 100% nucleotide sequence identity between the two viruses on this short *RdRp* segment⁸³. This RaTG13 virus could then be claimed to have been discovered back in 2013.

2. The RdRp protein from RaBtCoV/4991 is unique in that it is superior than RdRp from any other β coronavirus for developing antiviral drugs. RdRp has no homologs in human cells, which makes this essential viral enzyme a highly desirable target for antiviral development. As an example, *Remdesivir*, which is currently undergoing clinical trials, targets RdRp. When creating a novel and human-targeting virus, they would be interested in developing the antidote as well. Even though drug discovery like this may not be easily achieved, it is reasonable for them to intentionally incorporate a RdRp that is more amenable for antiviral drug development.

Fourth, they would use reverse genetics to assemble the gene fragments of *spike*, *ORF1b*, and the rest of the template ZC45 into a cDNA version of the viral genome. They would then carry out *in vitro* transcription to obtain the viral RNA genome. Transfection of the RNA genome into cells would allow the recovery of live and infectious viruses with the desired artificial genome.

Fifth, they would carry out characterization and optimization of the virus strain(s) to improve the fitness, infectivity, and overall adaptation using serial passage *in vivo*. One or several viral strains that meet certain criteria would then be obtained as the final product(s).

2.2 A postulated synthetic route for the creation of SARS-CoV-2

In this sub-section, we describe in more details how each step could be carried out in a laboratory setting using available materials and routine molecular, cellular, and virologic techniques. A diagram of this process is shown in Figure 8. We estimate that the whole process could be completed in approximately 6 months.

Step 1: Engineering the RBM of the Spike for hACE2-binding (1.5 months)

The Spike protein of a bat coronavirus is either incapable of or inefficient in binding hACE2 due to the missing of important residues within its RBM. This can be exemplified by the RBM of the template virus ZC45 (Figure 4). The first and most critical step in the creation of SARS-CoV-2 is to engineer the Spike so that it acquires the ability to bind hACE2. As evidenced in the literature, such manipulations have been carried out repeatedly in research laboratories since 2008⁴⁴, which successfully yielded engineered coronaviruses with the ability to infect human cells^{44-46,88,89}. Although there are many possible ways that one can engineer the Spike protein, we believe that what was actually undertaken was that they replaced the original RBM with a designed and possibly optimized RBM using SARS' RBM as a guide. As described in part 1, this theory is supported by our observation that two unique restriction sites, EcoRI and BstEII, exist at either end of the *RBM* in the SARS-CoV-2 genome (Figure 5A) and by the fact that such RBM-swap has been successfully carried out by Dr. Zhengli Shi and by her long-term collaborator and structure biology expert, Dr. Fang Li^{39,47}.

Although ZC45 *spike* does not contain these two restriction sites (Figure 5B), they can be introduced very easily. The original *spike* gene would be either amplified with RT-PCR or obtained through DNA synthesis (some changes could be safely introduced to certain variable regions of the sequence) followed by PCR. The gene would then be cloned into a plasmid using restriction sites other than EcoRI and BstEII.

Once in the plasmid, the *spike* gene can be modified easily. First, an EcoRI site can be introduced by converting the highlighted “gaacac” sequence (Figure 5B) to the desired “gaattc” (Figure 5A). The difference between them are two consecutive nucleotides. Using the commercially available QuikChange Site-Directed Mutagenesis kit, such a di-nucleotide mutation can be generated in no more than one week. Subsequently, the BstEII site could be similarly introduced at the other end of the *RBM*. Specifically, the “gaatacc” sequence (Figure 5B) would be converted to the desired “ggttacc” (Figure 5A), which would similarly require a week of time.

Once these restriction sites, which are unique within the *spike* gene of SARS-CoV-2, were successfully introduced, different *RBM* segments could be swapped in conveniently and the resulting Spike protein subsequently evaluated using established assays.

As described in part 1, the design of an *RBM* segment could be well-guided by the high-resolution structures (Figure 3)^{37,38}, yielding a sequence that resembles the SARS *RBM* in an intelligent manner. When carrying out the structure-guided design of the *RBM*, they would have followed the routine and generated a few (for example a dozen) such *RBM*s with the hope that some specific variant(s) may be superior than others in binding hACE2. Once the design was finished, they could have each of the designed *RBM* genes commercially synthesized (quick and very affordable) with an EcoRI site at the 5'-end and a BstEII site at the 3'-end. These novel *RBM* genes could then be cloned into the *spike* gene, respectively. The gene synthesis and subsequent cloning, which could be done in a batch mode for the small library of designed *RBM*s, would take approximately one month.

These engineered Spike proteins might then be tested for hACE2-binding using the established pseudotype virus infection assays^{45,49,50}. The engineered Spike with good to exceptional binding affinities would be selected. (Although not necessary, directed evolution could be involved here (error-prone PCR on the *RBM* gene), coupled with either an *in vitro* binding assay^{39,90} or a pseudotype virus infection assay^{45,49,50}, to obtain an *RBM* that binds hACE2 with exceptional affinity.)

Given the abundance of literature on Spike engineering^{44-46,84,86} and the available high-resolution structures of the Spike-hACE2 complex^{37,38}, the success of this step would be very much guaranteed. By the end of this step, as desired, a novel *spike* gene would be obtained, which encodes a novel Spike protein capable of binding hACE2 with high affinity.

Step 2: Engineering a furin-cleavage site at the S1/S2 junction (0.5 month)

The product from Step 1, a plasmid containing the engineered *spike*, would be further modified to include a furin-cleavage site (segment indicated by green lines in Figure 4) at the S1/S2 junction. This short stretch of gene sequence can be conveniently inserted using several routine cloning techniques, including QuikChange Site-Directed PCR⁶⁰, overlap PCR followed by restriction enzyme digestion and ligation⁹¹, or Gibson assembly. None of these techniques would leave any trace in the sequence. Whichever cloning method was the choice, the inserted gene piece would be included in the primers, which would be designed, synthesized, and used in the cloning. This step, leading to a further modified Spike with the furin-cleavage site added at the S1/S2 junction, could be completed in no more than two weeks.

Step 3: Obtain an *ORF1b* gene that contains the sequence of the short *RdRp* segment from RaBtCoV/4991 (1 month, yet can be carried out concurrently with Steps 1 and 2)

Unlike the engineering of Spike, no complicated design is needed here, except that the *RdRp* gene segment from RaBtCoV/4991 would need to be included. Gibson assembly could have been used here. In this technique, several fragments, each adjacent pair sharing 20-40 bp overlap, are combined together in one simple reaction to assemble a long DNA product. Two or three fragments, each covering a significant section of the *ORF1b* gene, would be selected based on known bat coronavirus sequences. One of these fragments would be the *RdRp* segment of RaBtCoV/4991⁸³. Each fragment would be PCR amplified with proper overlap regions introduced in the primers. Finally, all purified fragments would be pooled in equimolar concentrations and added to the Gibson reaction mixture, which, after a short incubation, would yield the desired *ORF1b* gene in whole.

Step 4: Produce the designed viral genome using reverse genetics and recover live viruses (0.5 month)

Reverse genetics have been frequently used in assembling whole viral genomes, including coronavirus genomes^{67,92-96}. The most recent example is the reconstruction of the SARS-CoV-2 genome using the *transformation-assisted recombination in yeast*⁹⁷. Using this method, the Swiss group assembled the entire viral genome and produced live viruses in just one week⁹⁷. This efficient technique, which would not leave any trace of artificial manipulation in the created viral genome, has been available since 2017^{98,99}. In addition to the engineered *spike* gene (from steps 1 and 2) and the *ORF1b* gene (from step 3), other fragments covering the rest of the genome would be obtained either through RT-PCR amplification from the template virus or through DNA synthesis by following a sequence slightly altered from that of the template virus. We believe that the latter approach was more likely as it would allow sequence changes introduced into the variable regions of less conserved proteins, the process of which could be easily guided by multiple sequence alignments. The amino acid sequences of more conserved functions, such as that of the E protein, might have been left unchanged. All DNA fragments would then be pooled together and transformed into yeast, where the cDNA version of the SARS-CoV-2 genome would be assembled *via* transformation-assisted recombination. Of course, an alternative method of reverse genetics, one of which the WIV has successfully used in the past⁶⁷, could also be employed^{67,92-96,100}. Although some earlier reverse genetics approaches may leave restriction sites at where different fragments would be joined, these traces would be hard to detect as the exact site of ligation can be anywhere in the ~30kb genome. Either way, a cDNA version of the viral genome would be obtained from the reverse genetics experiment. Subsequently, *in vitro* transcription using the cDNA as the template would yield the viral RNA genome, which upon transfection into Vero E6 cells would allow the production of live viruses bearing all of the designed properties.

Step 5: Optimize the virus for fitness and improve its hACE2-binding affinity *in vivo* (2.5-3 months)

Virus recovered from step 4 needs to be further adapted undergoing the classic experiment – serial passage in laboratory animals¹⁰¹. This final step would validate the virus' fitness and ensure its receptor-oriented adaptation toward its intended host, which, according to the analyses above, should be human. Importantly, the RBM and the furin-cleavage site, which were introduced into the Spike protein separately, would now be optimized together as one functional unit. Among various available animal models (e.g. mice, hamsters, ferrets, and monkeys) for coronaviruses, hACE2 transgenic mice (hACE2-mice) should be the most proper and convenient choice here. This animal model has been established during the study of SARS-CoV and has been available in the Jackson Laboratory for many years¹⁰²⁻¹⁰⁴.

The procedure of serial passage is straightforward. Briefly, the selected viral strain from step 4, a precursor of SARS-CoV-2, would be intranasally inoculated into a group of anaesthetized hACE2-mice. Around 2-3 days post infection, the virus in lungs would usually amplify to a peak titer. The mice would

then be sacrificed and the lungs homogenized. Usually, the mouse-lung supernatant, which carries the highest viral load, would be used to extract the candidate virus for the next round of passage. After approximately 10~15 rounds of passage, the hACE2-binding affinity, the infection efficiency, and the lethality of the viral strain would be sufficiently enhanced and the viral genome stabilized¹⁰¹. Finally, after a series of characterization experiments (e.g. viral kinetics assay, antibodies response assay, symptom observation and pathology examination), the final product, SARS-CoV-2, would be obtained, concluding the whole creation process. From this point on, this viral pathogen could be amplified (most probably using Vero E6 cells) and produced routinely.

It is noteworthy that, based on the work done on SARS-CoV, the hACE2-mice, although suitable for SARS-CoV-2 adaptation, is not a good model to reflect the virus' transmissibility and associated clinical symptoms in humans. We believe that those scientists might not have used a proper animal model (such as the golden Syrian hamster) for testing the transmissibility of SARS-CoV-2 before the outbreak of COVID-19. If they had done this experiment with a proper animal model, the highly contagious nature of SARS-CoV-2 would be extremely evident and consequently SARS-CoV-2 would not have been described as "not causing human-to-human transmission" at the start of the outbreak.

We also speculate that the extensive laboratory-adaptation, which is oriented toward enhanced transmissibility and lethality, may have driven the virus too far. As a result, SARS-CoV-2 might have lost the capacity to attenuate on both transmissibility and lethality during its current adaptation in the human population. This hypothesis is consistent with the lack of apparent attenuation of SARS-CoV-2 so far despite its great prevalence and with the observation that a recently emerged, predominant variant only shows improved transmissibility¹⁰⁵⁻¹⁰⁸.

Serial passage is a quick and intensive process, where the adaptation of the virus is accelerated. Although intended to mimic natural evolution, serial passage is much more limited in both time and scale. As a result, less random mutations would be expected in serial passage than in natural evolution. This is particularly true for conserved viral proteins, such as the E protein. Critical in viral replication, the E protein is a determinant of virulence and engineering of it may render SARS-CoV-2 attenuated¹⁰⁹⁻¹¹¹. Therefore, at the initial assembly stage, these scientists might have decided to keep the amino acid sequence of the E protein unchanged from that of ZC45/ZXC21. Due to the conserved nature of the E protein and the limitations of serial passage, no amino acid mutation actually occurred, resulting in a 100% sequence identity on the E protein between SARS-CoV-2 and ZC45/ZXC21. The same could have happened to the marks of molecular cloning (restriction sites flanking the RBM). Serial passage, which should have partially naturalized the SARS-CoV-2 genome, might not have removed all signs of artificial manipulation.

3. Final remarks

Many questions remain unanswered about the origin of SARS-CoV-2. Prominent virologists have implicated in a *Nature Medicine* letter that laboratory escape, while not being entirely ruled out, was unlikely and that no sign of genetic manipulation is present in the SARS-CoV-2 genome⁴. However, here we show that genetic evidence within the *spike* gene of SARS-CoV-2 genome (restriction sites flanking the RBM; tandem rare codons used at the inserted furin-cleavage site) does exist and suggests that the SARS-CoV-2 genome should be a product of genetic manipulation. Furthermore, the proven concepts, well-established techniques, and knowledge and expertise are all in place for the convenient creation of this novel coronavirus in a short period of time.

Motives aside, the following facts about SARS-CoV-2 are well-supported:

1. If it was a laboratory product, the most critical element in its creation, the backbone/template virus (ZC45/ZXC21), is owned by military research laboratories.
2. The genome sequence of SARS-CoV-2 has likely undergone genetic engineering, through which the virus has gained the ability to target humans with enhanced virulence and infectivity.
3. The characteristics and pathogenic effects of SARS-CoV-2 are unprecedented. The virus is highly transmissible, onset-hidden, multi-organ targeting, sequelae-unclear, lethal, and associated with various symptoms and complications.
4. SARS-CoV-2 caused a world-wide pandemic, taking hundreds of thousands of lives and shutting down the global economy. It has a destructive power like no other.

Judging from the evidence that we and others have gathered, we believe that finding the origin of SARS-CoV-2 should involve an independent audit of the WIV P4 laboratories and the laboratories of their close collaborators. Such an investigation should have taken place long ago and should not be delayed any further.

We also note that in the publication of the chimeric virus SHC015-MA15 in 2015, the attribution of funding of Zhengli Shi by the NIAID was initially left out. It was reinstated in the publication in 2016 in a corrigendum, perhaps after the meeting in January 2016 to reinstate NIH funding for gain-of-function research on viruses. This is an unusual scientific behavior, which needs an explanation for.

What is not thoroughly described in this report is the various evidence indicating that several coronaviruses recently published (RaTG13¹⁸, RmYN02³⁰, and several pangolin coronaviruses^{27-29,31}) are highly suspicious and likely fraudulent. These fabrications would serve no purpose other than to deceive the scientific community and the general public so that the true identity of SARS-CoV-2 is hidden. Although exclusion of details of such evidence does not alter the conclusion of the current report, we do believe that these details would provide additional support for our contention that SARS-CoV-2 is a laboratory-enhanced virus and a product of gain-of-function research. A follow-up report focusing on such additional evidence is now being prepared and will be submitted shortly.

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From:	(b)(6)@state.gov>
To:	Yu, Miles (b)(6)@state.gov>
Subject:	Re: query
Date:	Fri, 8 May 2020 20:59:12 +0000

Let me know if this worked.

From: (b)(6)@state.gov>
Sent: Friday, May 8, 2020 4:39 PM
To: Yu, Miles (b)(6)@state.gov>
Subject: Re: query

Hi Miles,

I made a few edits. It is very comprehensive.

Best,

(b)(6)

From: Yu, Miles (b)(6)@state.gov>
Sent: Friday, May 8, 2020 4:10 PM
To: (b)(6)@state.gov>
Subject: RE: query

(b)(6)

Could you go over it one more time? I (b)(5)

(b)(5) Make sure I gave you and E-PAI enough credit.

I plan to send this up in about an hour.

Much obliged.

Miles

Dr. M. Miles Yu
Policy Planning Staff (S/P)
Office of the Secretary
Department of State

(b)(6) (o)
(c)

~~SENSITIVE BUT UNCLASSIFIED~~

From: (b)(6)@state.gov>
Sent: Friday, May 8, 2020 3:08 PM
To: Yu, Miles (b)(6)@state.gov>
Subject: Re: query

Made a few edits to the doc and just responded.

From: Yu, Miles (b)(6)@state.gov>
Sent: Friday, May 8, 2020 2:41 PM
To: (b)(6)@state.gov>
Subject: query

(b)(6)

Have you taken a look at the draft and (b)(5)

Cheers!

Miles

Dr. M. Miles Yu
Policy Planning Staff (S/P)
Office of the Secretary
Department of State

(b)(6) (o)
(c)

Sender:	(b)(6)@state.gov>
Recipient:	Yu, Miles <(b)(6)@state.gov>

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

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Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

Withheld pursuant to exemption

(b)(5)

From:	(b)(6)@state.gov>
To:	Park, Christopher J (b)(6)@state.gov>
Subject:	RE: Press Guidance and Support to GPA? PLEASE REVIEW and CLEAR
Date:	Mon, 15 Mar 2021 16:19:09 +0000

Did you see these as well from us? Thoughts?

~~SENSITIVE BUT UNCLASSIFIED~~

From: Park, Christopher J <(b)(6)@state.gov>
Sent: Monday, March 15, 2021 12:17 PM
To: ISN-BPS-DL <ISN-BPS-DL@STATE.GOV>; T_SpecAssts <T_SpecAssts@state.gov>
Subject: FW: Press Guidance and Support to GPA? PLEASE REVIEW and CLEAR

I think this is actually pretty good with the (b)(5) as well. (b)(5)

(b)(5)

CJP

~~SENSITIVE BUT UNCLASSIFIED~~

From: (b)(6)@state.gov
Sent: Monday, March 15, 2021 12:09 PM
To: (b)(6)@state.gov; (b)(6)@state.gov; (b)(6)@state.gov; (h)(6)@state.gov; (b)(6)@state.gov; Park, Christopher J <(b)(6)@state.gov>; T_SpecAssts <T_SpecAssts@state.gov>; (b)(6)@state.gov; (b)(6)@state.gov; (b)(6)@state.gov; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>
Cc: (b)(6)@state.gov; (b)(6)@state.gov; (b)(6)@state.gov; ISN-Press-DL <ISN-Press-DL@state.gov>; AVC-Press-DL <AVC-Press-DL@state.gov>; (h)(6)@state.gov; (b)(6)@state.gov; (b)(6)@state.gov; (b)(6)@state.gov
Subject: RE: Press Guidance and Support to GPA? PLEASE REVIEW and CLEAR

Hi (b)(6)

Clear for (b)(5) with the edits from (b)(5)

Thanks,

(b)(6)

~~SENSITIVE BUT UNCLASSIFIED~~

From: (b)(6)@state.gov>

Sent: Monday, March 15, 2021 11:57 AM

To: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; (b)(6)@state.gov>; Park, Christopher J

(b)(6)@state.gov>; T_SpecAssts <T_SpecAssts@state.gov>; (b)(6)

(b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>; (b)(6)

(b)(6)@state.gov>

Cc: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; ISN-Press-DL <ISN-Press-DL@state.gov>; AVC-Press-DL <AVC-Press-

DL@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>;

(b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>

Subject: RE: Press Guidance and Support to GPA? PLEASE REVIEW and CLEAR

Thanks, (b)(6) a.

Attached are my edits (b)(5)

(b)(5)

(b)(6)

Office of Analysis for Strategic, Proliferation, and Military Issues
Bureau of Intelligence and Research (INR/SPM)
U.S. Department of State

~~SENSITIVE BUT UNCLASSIFIED~~

From: (b)(6)@state.gov>

Sent: Monday, March 15, 2021 11:56 AM

To: (b)(6)@state.gov>; (b)(6)@state.gov>;

(b)(6)@state.gov>; (b)(6)@state.gov>; Park, Christopher J

(b)(6)@state.gov>; T_SpecAssts <T_SpecAssts@state.gov>; (b)(6)

(b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>; (b)(6)

(b)(6)@state.gov>

Cc: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; ISN-Press-DL <ISN-Press-DL@state.gov>; AVC-Press-DL <AVC-Press-

DL@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>;

(b)(6)@state.gov>; (b)(6)@state.gov>; P@state.gov>; (b)(6)

(b)(6)@state.gov>

Subject: Re: Press Guidance and Support to GPA? PLEASE REVIEW and CLEAR

Thanks, (b)(6) (b)(5)

(b)(5)

Best,

(b)(6)

(b)(6)

Attorney-Adviser, L/OES

U.S. Department of State

Mobile: (b)(6)

From: (b)(6)@state.gov>

Sent: Monday, March 15, 2021 11:40 AM

To: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; Park, Christopher J <(b)(6)@state.gov>; T_SpecAssts

<T_SpecAssts@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; (b)(6)@state.gov>; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>

Cc: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; ISN-Press-DL <ISN-Press-DL@state.gov>; AVC-Press-DL <AVC-Press-

DL@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>;

(b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; (b)(6)@state.gov>

Subject: RE: Press Guidance and Support to GPA? PLEASE REVIEW and CLEAR

Thanks, (b)(6) (b)(5)

Best,

(b)(6)

~~SENSITIVE BUT UNCLASSIFIED~~

From: (b)(6)@state.gov>

Sent: Monday, March 15, 2021 11:28 AM

To: (b)(6)@state.gov>; (b)(6)@state.gov>; Park, Christopher J

(b)(6)@state.gov>; T_SpecAssts <T_SpecAssts@state.gov>; (b)(6)

(b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>

Cc: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>; ISN-Press-DL <ISN-Press-DL@state.gov>; AVC-Press-DL <AVC-Press-

DL@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>;

(b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)

(b)(6)@state.gov>

Subject: RE: Press Guidance and Support to GPA? PLEASE REVIEW and CLEAR

Colleagues—

Please review and clear on the attached draft points re (b)(5) (b)(6) story by noon or sooner.

Thanks,

(b)(6)

~~SENSITIVE BUT UNCLASSIFIED~~

From: (b)(6)@state.gov>

Sent: Monday, March 15, 2021 11:17 AM

To: (b)(6)@state.gov>; Park, Christopher J (b)(6)@state.gov>; T_SpecAssts <T_SpecAssts@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>; (b)(6)@state.gov>

Cc: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; ISN-Press-DL <ISN-Press-DL@state.gov>; AVC-Press-DL <AVC-Press-DL@state.gov>

Subject: Re: Press Guidance and Support to GPA?

(b)(5)

From: (b)(6)@state.gov>

Sent: Monday, March 15, 2021 11:16 AM

To: (b)(6)@state.gov>; Park, Christopher J (b)(6)@state.gov>; T_SpecAssts <T_SpecAssts@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>; (b)(6)@state.gov>

Cc: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>

Subject: Re: Press Guidance and Support to GPA?

Hi (b)(6) perfect timing.

(b)(5)

(b)(5)

Best - (b)(6)

From: (b)(6)@state.gov>

Sent: Monday, March 15, 2021 11:11 AM

To: Park, Christopher J (b)(6)@state.gov>; T_SpecAssts <T_SpecAssts@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; ISN-PC-East Asia Team-DL <ISN-PC-EastAsiaTeam-DL@state.gov>

Cc: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>

Subject: Press Guidance and Support to GPA?

Good Morning Everyone,

Last week, (b)(6) (b)(5)
(b)(5) (b)(5); (b)(6)
(b)(5)
(b)(5)

flagged the Fox story that I'm sure most of you have seen already:

<https://www.foxnews.com/world/top-state-official-coronavirus-bioweapon-accident> (appears to be the same as the NY Post article).

(b)(5)
(b)(5) (b)(5)
(b)(5) (b)(5)
(b)(5) (b)(5)
(b)(5) Is

there anything (b)(6) and I can do to help?

Thanks,

(b)(6)

Office of Analysis for Strategic, Proliferation, and Military Issues
Bureau of Intelligence and Research (INR/SPM)
U.S. Department of State

~~SENSITIVE BUT UNCLASSIFIED~~

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~~SENSITIVE BUT UNCLASSIFIED~~

~~SENSITIVE BUT UNCLASSIFIED~~

Sender:	(b)(6)	@state.gov>
Recipient:	Park, Christopher J (b)(6)	@state.gov>

From:	"DiNanno, Thomas G" (b)(6)@state.gov>
To:	(b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>
CC:	(b)(6)@state.gov>; Feith, David (b)(6)@state.gov>; (b)(6)@state.gov>
Subject:	Re: NIH discussion
Date:	Sat, 26 Dec 2020 16:03:58 +0000

Very compelling article

From: (b)(6)@state.gov>

Sent: Wednesday, December 23, 2020 10:26 PM

To: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>

Cc: DiNanno, Thomas G (b)(6)@state.gov>; (b)(6)@state.gov>; Feith, David (b)(6)@state.gov>; (b)(6)@state.gov>

Subject: Re: NIH discussion

(b)(6)

Reference your note below, attached are a few (b)(5)

(b)(5)

Note:

1. (b)(5)

2.

Cheers,

(b)(6)

From: (b)(6)@state.gov>

Sent: Wednesday, December 23, 2020 1:15 PM

To: (b)(6)@state.gov>; (b)(6)@state.gov>; (b)(6)@state.gov>

Cc: DiNanno, Thomas G (b)(6)@state.gov>; (b)(6)@state.gov>; Feith, David (b)(6)@state.gov>; (b)(6)@state.gov>

Subject: NIH discussion

(b)(6)

(b)(5)

(b)(5)

It is a terrible time to try to put together a call and it may be that this can't happen until after the New Year.

By then — assuming so things come out — it may be an easier time to have a serious discussion.

From: (b)(5); (b)(6)@state.gov>

Sent: Wednesday, December 23, 2020 1:05 PM

To: (b)(5); (b)(6) NIH/OD) [E] (b)(5); (b)(6)

Cc: DiNanno, Thomas G (b)(5);@state.gov>

Subject: Re: Scientific murder board

(b)(5)

(b)(5);

From: (b)(5); (b)(6) NIH/OD) [E] (b)(5); (b)(6)

Sent: Wednesday, December 23, 2020 9:37 AM

To: (b)(5); (b)(6)@state.gov>

Cc: DiNanno, Thomas G (b)(5);@state.gov>

Subject: Re: Scientific murder board

Hey (b)(6)

(b)(5)

(b)(5)

Take care,

(b)(6)

From: (b)(6)@state.gov>

Date: Tuesday, December 22, 2020 at 8:21 PM

To: (b)(6)

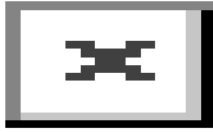
Cc: "DiNanno, Thomas G" (b)(6)@state.gov>

Subject: Re: Scientific murder board

(b)(6)

(b)(5)





Op-Ed: COVID-19 lesson:
Diseases can be ideal
biological weapons - Los
Angeles Times

The devastation COVID-19 has wrought on the U.S. population is staggering. Yet the risks it poses to our national security are also chilling: Diseases are, in many terrible ways, ideal weapons.

www.latimes.com

(b)(5)

(b)(6)

PS- I copy our senior official in AVC, Tom DiNanno who leads all WMD Treaty compliance. Tom

(b)(5)

(b)(6)

NSRI Strategic Advisor

Bureau of Arms Control, Verification and Compliance (AVC)

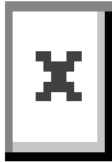
US Department of State

(b)(6)

NSTS: (b)(6)

JWICS: (b)(6)@state.ic.gov

SIPR: (b)(6)@state.sgov.gov



Biodefense in the Age of Synthetic Biology - The National Academies Press

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From: (b)(6) (NIH/OD) [E](b)(6) (b)(6)
Sent: Tuesday, December 22, 2020 6:44 PM
To: (b)(6)@state.gov>
Subject: Scientific murder board

Hey (b)(6)

I've now connected with a few different folks at NIH as a result of our conversation (b)(5)

(b)(5)

(b)(5)

Thanks!

(b)(6)

Sender: "DiNanno, Thomas G" (b)(6)@state.gov>

Recipient:

(b)(6)@state.gov>;
(b)(6)@state.gov>;
(b)(6)@state.gov>;
(b)(6)@state.gov>;
(b)(6)@state.gov>;
Feith, David <(b)(6)@state.gov>;
(b)(6)@state.gov>

From:	(b)(6)
To:	Park, Christopher J <(b)(6)@state.gov>
Subject:	RE: BTWC RevCon Geneva 2022
Date:	Wed, 28 Apr 2021 14:20:49 +0000

Thanks Chris – speak later

From: Park, Christopher J <(b)(6)@state.gov>
Sent: 28 April 2021 10:10
To: (b)(6)
Subject: RE: BTWC RevCon Geneva 2022

Sure.

From: (b)(6)
Sent: Wednesday, April 28, 2021 10:00 AM
To: Park, Christopher J <(b)(6)@state.gov>
Subject: RE: BTWC RevCon Geneva 2022

Thanks Chris

I can do 4pm today. Do you just want to call me on my cell (b)(6)

(b)(6)

(b)(6)

Strategic Threats, Non-Proliferation, Arms Control and International Security
British Embassy Washington,
3100 Massachusetts Avenue NW, Washington DC 20008-7870
Mobile (b)(6)

From: Park, Christopher J <(b)(6)@state.gov>
Sent: 28 April 2021 09:57
To: (b)(6) <(b)(6)@state.gov>
Subject: RE: BTWC RevCon Geneva 2022

Hi, (b)(6) Happy to discuss – I think a phone (or video, if you prefer) conversation would be the best option, since you can more easily ask follow-up questions. The best times for me this week are after 1 PM today or tomorrow between noon and 4 PM.

Chris

Christopher J. Park
Director, Biological Policy Staff
Bureau of International Security and Nonproliferation
Department of State
(b)(6)@state.gov
(b)(6)

From: (b)(6)
Sent: Wednesday, April 28, 2021 9:16 AM
To: (b)(6)@state.gov; Park, Christopher J <(b)(6)@state.gov>
Subject: RE: BTWC RevCon Geneva 2022

(b)(6) - thanks as ever - I thought I had it wrong!

Hi Chris,

I think we had a brief intro when you were in T's office ? Good to reconnect. Would you be kind enough to have a look at the questions below?

Many thanks

(b)(6)

From: (b)(6)@state.gov
Sent: 28 April 2021 09:04
To: (b)(6)
Cc: Park, Christopher J (b)(6)@state.gov
Subject: RE: BTWC RevCon Geneva 2022

Hi (b)(6)

Hope all is well with you! Our ISN colleagues in the Office of the Biological Policy Staff handle the BTWC. I've included the director, Chris Park, on this email so that you can be in touch directly.

I hope you are enjoying the warm weather and sunshine!

Best,

(b)(6)

(b)(6)
Deputy Director
Office of Multilateral Nuclear and Security Affairs
Bureau of International Security and Non-Proliferation
Office: (b)(6)
Cell: (b)(6)

(b)(6)@state.gov

From: (b)(6)
Sent: Tuesday, April 27, 2021 5:24 PM
To: (b)(6)@state.gov>
Subject: FW: BTWC RevCon Geneva 2022

Hi (b)(6)

Hope are you well? Who deals with BTWC in (b)(6) absence – it is your office in MNSA or have I walked off track?

Thanks as ever

(b)(6)

From: (b)(6)
Sent: 27 April 2021 17:14
To: (b)(6)@state.gov>
Subject: BTWC RevCon Geneva 2022

Hi (b)(6)

Hope all's well and that you are able to enjoy a bit of this lovely sunshine. I recall you lead on BTWC? Would you have time for a call this week? London are beginning to think about the BTWC with the MX and MSP meetings now scheduled. I have a few questions on the BTCW and verification policy and if that has changed under the new admin. You may still be in a review but I've listed the main questions if you'd prefer to e-mail back. They are:

- 1) What is the administration's policy on the BTWC, and specifically verification protocol?
- 2) Ambitions for the RevCon (Has the pandemic reignited ambition?)
- 3) Appetite for Quad co-ordination?
- 4) Appetite for broadening the likeminded group?
- 5) Thoughts on progressing less contentious measures – confidence building measures, lobbying the 14 remaining states to sign, increasing the capacity of the support group in Geneva..?

Many thanks in advance

(b)(6)

(b)(6)
Strategic Threats, Non-Proliferation, Arms Control and International Security
British Embassy Washington,
3100 Massachusetts Avenue NW, Washington DC 20008-7870
Mobile + (b)(6)

From: (b)(6)@state.gov>

Sent: 04 January 2021 10:58

To: (b)(6)

Cc: (b)(6)@state.gov>

Subject: RE: COVID/BW: US DNSA claims

Hi (b)(6)

Happy New Year to you and (b)(6)

We understand that the Secretary may be raising this issue with the UK and other key allies this week, possibly as soon as today. We'll let you know when we know more.

(b)(6)

From: (b)(6)

Sent: Monday, January 4, 2021 10:04 AM

To: (b)(6)@state.gov> (b)(6)@state.gov>

Subject: RE: COVID/BW: US DNSA claims

Hi (b)(6)

Ccing you in too. This is in UK media – is this something we may need to alert Ministers too?

(b)(6)

From: (b)(6)

Sent: 04 January 2021 10:02

To: (b)(6)@state.gov>

Subject: COVID/BW: US DNSA claims

Hi (b)(6)

Hope you had a great break. I see this press summary in the *Times* this morning. Nothing new on substance but noticeable it's come from DNSA Pottinger himself and attributed as such. Can I ask if this is now an official position ?

Biological weapons lab leaked coronavirus, claims US official

There is now strong evidence that the coronavirus was leaked from a Chinese biological weapons research laboratory, rather than developing in a livestock "wet market", a senior US official said.

Matthew Pottinger, a deputy national security adviser, claimed in a Zoom call to politicians around the world that there was "a growing body of evidence that the lab is likely the most credible source of the virus".

Mr Pottinger, a former US Marines officer, said: "Even establishment figures in Beijing have openly dismissed the wet market story." He said that the pathogen may have escaped through a "leak or an accident".

He said intelligence showed that the virus had been leaked from the heavily guarded Wuhan Institute of Virology, 11 miles from the market.

He was speaking on the eve of a trip to China by a group of experts from the World Health Organisation to investigate the source of the coronavirus, which critics claim will be a whitewash.

"MPs around the world have a moral role to play in exposing the WHO investigation as a Potemkin exercise," Mr Pottinger said. Sir Iain Duncan Smith, the former Conservative Party leader, who was on the Zoom call, told the Daily Mail that Mr Pottinger's comments showed the US was doubling down on the theory that the virus came from a leak at the laboratory. The US is said to

have been talking to a whistleblower from the Wuhan institute. Wuhan reported the first coronavirus cases in December 2019. Many of them were linked to a wet market but the existence of a bio laboratory that works on coronaviruses in the city has fuelled speculation over a possible leak.

"It is unfortunate that we have been targeted as a scapegoat for the origin of the virus," Wang Yanyi, director of the virology institute, said last August. "Any person would inevitably feel angry or misunderstood being subject to unwarranted or malicious accusations while carrying out research and related work in the fight against the virus."

Shi Zhengli, a virologist at the institute and known as the "bat woman" for her work related to coronaviruses, said that she would welcome WHO experts.

China has tightly controlled its own investigation into the origin of the virus.

Late last year foreign journalists were barred from visiting an abandoned copper mine in the southwest of the country, where the closest known relative of Sars-Cov-2 was discovered in 2013 by Chinese virologists and taken back to the Wuhan laboratory. A bat research team had samples taken on a recent visit to the mine confiscated.

Dr Shi discovered RaTG13 in the mine after six workers in Tongguan died from an unknown illness in 2012. She took the viral samples back to Wuhan for further analysis.

A majority of scientists believe that the Covid-19 virus originated in nature.

Many thanks

(b)(6)

(b)(6)

Strategic Threats, Non-Proliferation, Arms Control and International Security
British Embassy Washington,
3100 Massachusetts Avenue NW, Washington DC 20008-7870

Mobile (b)(6)

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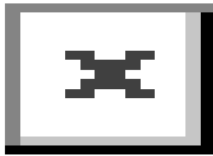
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Sender:	(b)(6)
Recipient:	Park, Christopher J <(b)(6)@state.gov>

From:	(b)(6)@state.gov> (b)(6)@state.gov>; DiNanno, Thomas G (b)(6)@state.gov>;
To:	(b)(6)@state.gov>; Feith, David (b)(6)@state.gov>; (b)(6)@state.gov>
CC:	(b)(6)@state.gov>; (b)(6)@state.gov>
Subject:	Re: Baric UNC FOIA Emails Update
Date:	Sun, 27 Dec 2020 00:02:10 +0000

(b)(6)(b)(5) via its support to EcoHealth Alliance, at least according to left leaning investigative journalists. The journalists provide supporting data, unlike so many scientists across USG and US academic labs funded by USG involved in GOF R&D cooperation with the WIV. (b)(5)

(b)(5) (b)(6)
<https://www.independentsciencenews.org/news/peter-daszaks-ecohealth-alliance-has-hidden-almost-40-million-in-pentagon-funding/>



Peter Daszak's EcoHealth Alliance Has Hidden Almost \$40 Million In Pentagon Funding And Militarized Pandemic Science - Independent Science News | Food, Health and Agriculture Bioscience News

A brief history of the PCR fiasco of the Pandemic that can't seem to end. It all started on Jan 10th 2020, when WHO reported that there is an outbreak in China caused by a novel coronavirus.

www.independentsciencenews.org

Peter Daszak's EcoHealth Alliance Has Hidden Almost

\$40 Million In Pentagon Funding And Militarized Pandemic Science

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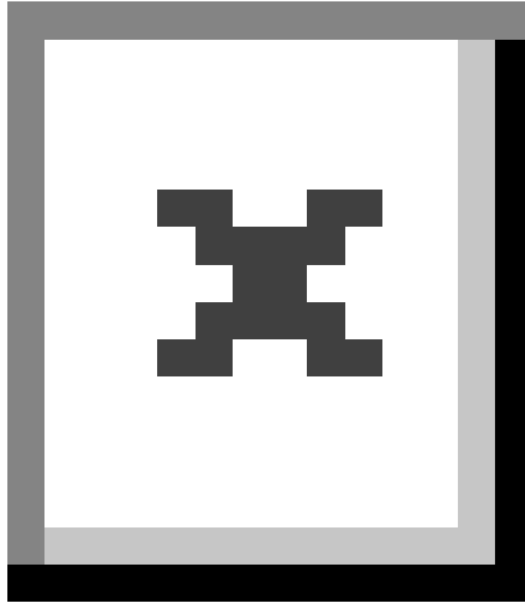
by Sam Hussein

“Pandemics are like terrorist attacks: We know roughly where they originate and what’s responsible for them, but we don’t know exactly when the next one will happen. They need to be handled the same way — by identifying all possible sources and dismantling those before the next pandemic strikes.”

This statement was written in the *New York Times* earlier this year by Peter Daszak. Daszak is the longtime president of the [EcoHealth Alliance](#), a New York-based non-profit whose claimed focus is pandemic prevention. But the EcoHealth Alliance, it turns out, is at the very centre of the COVID-19 pandemic in many ways.

To depict the pandemic in such militarized terms is, for Daszak, a commonplace. In an [Oct. 7 online talk organized by Columbia University’s School of International and Public Affairs](#), Daszak presented a slide titled “Donald Rumsfeld’s Prescient Speech.”:

“There are known knowns; there are things we know that we know. There are known unknowns; that is to say, there are things that we know we don’t know. But there are also unknown unknowns — there are things we don’t know we don’t know.” (This Rumsfeld quote is in fact from a news conference)



The Pentagon (Credit the Smithsonian)

In the subsequent online discussion, Daszak emphasized the parallels between his own crusade and Rumsfeld's, since, according to Daszak, the "potential for unknown attacks" is "the same for viruses".

Daszak then proceeded with a not terribly subtle pitch for over a billion dollars. This money would support a fledgling virus hunting and surveillance project of his, the Global Virome Project — a "doable project" he assured watchers — given the cost of the pandemic to governments and various industries.

Also on the video was Columbia University professor Jeffrey Sachs. Sachs is a former special advisor to the UN, the former head of the Millennium Villages Project, and was recently appointed Chair of the newly-formed EAT Lancet Commission on the pandemic. In September, Sachs' commission named Daszak to head up its committee on the pandemic's origins. Daszak is

also on the WHO's committee to investigate the pandemic's origin. He is the only individual on both committees.

These leadership positions are not the only reason why Peter Daszak is such a central figure in the COVID-19 pandemic, however. His appointment dismayed many of those who are aware that Daszak's EcoHealth Alliance funded bat coronavirus research, including virus collection, at the Wuhan Institute for Virology (WIV) and thus could themselves be directly implicated in the outbreak.

For his part, Daszak has repeatedly dismissed the notion that the pandemic could have a lab origin. In fact, a recent FOIA by the transparency group U.S. Right To Know revealed that Peter Daszak drafted an influential multi-author letter published on February 18 in the Lancet. That letter dismissed lab origin hypotheses as "conspiracy theory." Daszak was revealed to have orchestrated the letter such as to "avoid the appearance of a political statement."

Sachs for his part seemed surprised by Daszak's depiction of Rumsfeld but Daszak reassured him. "It's an awesome quote! And yes, it's Donald Rumsfeld, Jeff, and I know he's a Republican, but — what a genius!"

Following the EcoHealth Alliance's money trail to the Pentagon

Collecting dangerous viruses is typically justified as a preventive and defensive activity, getting ahead of what "Nature" or "The Terrorists" might throw at us. But by its nature, this work is "dual use". "Biodefense" is often just as easily biowarfare since biodefense and the products of biowarfare are identical. It's simply a matter of what the stated goals are.

This is openly acknowledged [See below] by scientists associated with EcoHealth Alliance when talking about alleged programs in other countries — like Iraq.

For much of this year, Daszak's EcoHealth Alliance garnered a great deal of sympathetic media coverage after its \$3.7 million five-year NIH grant was prematurely cut when the Trump administration learned that EcoHealth Alliance funded bat coronavirus research at the WIV.

The temporary cut was widely depicted in major media as Trump undermining the EcoHealth Alliance's noble fight against pandemics. The termination was reversed by NIH in late August, and even upped to \$7.5 million. But entirely overlooked amid the claims and counter-claims was that far more funding for the EcoHealth Alliance comes from the Pentagon than the NIH.

To be strictly fair to the media, Daszak's EcoHealth Alliance obscures its Pentagon funding. On its website EcoHealth Alliance states that "A copy of the EHA Grant Management Manual is available upon request to the EHA Chief Financial Officer at finance (at) ecohealthalliance.org". But an email to that address and numerous others, including Peter Daszak's, requesting that Manual, as well as other financial information, was not returned. Neither were repeated voicemails.

Only buried under their "Privacy Policy," under a section titled "EcoHealth Alliance Policy Regarding Conflict of Interest in Research," does the EcoHealth Alliance concede it is the "recipient of various grant awards from federal agencies including the National Institute of