

1 COMMITTEE ON OVERSIGHT AND ACCOUNTABILITY,
2 SELECT SUBCOMMITTEE ON THE CORONAVIRUS PANDEMIC,
3 U.S. HOUSE OF REPRESENTATIVES,
4 WASHINGTON, D.C.

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9 INTERVIEW OF: RALPH S. BARIC, Ph.D.

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13 MONDAY, JANUARY 22, 2024

14
15 The Interview Commenced at 10:07 a.m.

34 Appearances.

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54	Exhibits	
55	Minority Exhibit	Page No.
56	A - Nature Medicine December 2015 article,	
57	A SARS-like cluster of circulating bat	
58	coronaviruses shows potential for	
59	human emergence	57
60	B - Document, DARPA-PREEMPT-HR00111850017	89
61	Majority Exhibit No.	Page No.
62	1 - Email cover sheet, Bates	
63	UNC_SSCP00023674	105
64	2 - The National Academies of Sciences,	
65	Engineering, Medicine, Expert Meeting	
66	Agenda, Bates REV0000809	132
67	3 - 1R0AI110964 Year 4 Report 188	
68	4 - Letter dated May 28, 2016, with	
69	attachment	203
70	5 - Document, PREEMPT call (EHA,	
71	Ralph & Time of UNC) - 2 March	
72	2018	220
73	6 - Letter dated May 15, 2015, from	
74	Chernay Mason to Ms. Barbara	
75	Entwisle and Ralph Baric, Ph.D.,	
76	Bates commencing UNC_SSCP00002629	229

77 P R O C E E D I N G S

78 Mr. Benzine. We can go on the record.

79 This is the transcribed interview of Dr. Ralph Steven Baric
80 conducted by the House Select Subcommittee on the Coronavirus
81 Pandemic, the Committee on Oversight and Accountability, and
82 the Committee on Energy and Commerce under the authority
83 granted to them by House Resolution 5, House Rule 10, and the
84 Rules of the Committee on Oversight and Accountability and
85 Committee on Energy and Commerce.

86 This interview was requested by Chairman Brad Wenstrup,
87 Chairman James Comer, Chair Cathy McMorris Rodgers, Chairman
88 Morgan Griffith, and Chairman Brett Guthrie as part of the
89 Committee's oversight of the federal government's response to
90 the coronavirus pandemic.

91 Pursuant to House Resolution 5, the Select Subcommittee has
92 wide-ranging jurisdiction, but specifically to investigate
93 the origins of the coronavirus pandemic, including, but not
94 limited to, the federal government's funding of gain of
95 function research.

96 Pursuant to House Rule 10, the Committee on Oversight and
97 Accountability has jurisdiction to investigate any matter at
98 any time. And pursuant to House Rule 10 and 11, the
99 Committee on Energy and Commerce has jurisdiction for public
100 health service agencies, including the National Institutes of
101 Health and the entities it funds, as well as federal

102 biomedical research and development.

103 Can the witness please state his name and spell his last name
104 for the record?

105 The Witness. Ralph Steven Baric, B-A-R-I-C.

106 Mr. Benzine. Thank you. Dr. Baric, my name is Mitch

107 Benzine, and I am the staff director for the Majority staff
108 of the Select Subcommittee. I want to thank you for coming
109 in today for this interview. We recognize that you are here
110 voluntarily and appreciate that.

111 Under the Select Subcommittee and Committee on Oversight and
112 Accountabilities rules, you are allowed to have an attorney
113 present to advise you during this interview. Do you have an
114 attorney representing you in a personal capacity present with
115 you today?

116 The Witness. Yes.

117 Mr. Benzine. Will counsel identify themselves?

118 Mr. Ervin. I'm Clark Ervin at Squire Patton Boggs.

119 Mr. Benzine. For the record, beginning to my left, will the
120 rest of the Majority staff and the additional staff members
121 please introduce themselves with their name, title, and
122 affiliation?

123 Mr. Strom. John Strom, senior counsel, House Energy and
124 Commerce Subcommittee on Oversight Investigations, Majority.

125 Mr. Osterhues. Eric Osterhues, chief counsel, Select
126 Subcommittee, Majority.

127 Mr. Slobodin. Alan Slobodin, chief investigative counsel,
128 Majority staff, House Energy and Commerce Committee.
129 Ms. Brewer. Madeline Brewer, counsel for the Majority,
130 Select Subcommittee.
131 Mr. Spectre. Peter Spectre, professional staff member,
132 Select Subcommittee, Majority.
133 Ms Yass. Alicia Yass, senior counsel, Select Subcommittee,
134 Democratic staff.
135 Mr. Romero. Joseph Romero, Democratic counsel, Select
136 Subcommittee.
137 Mr. Lichtman. Miles Lichtman, Democratic staff director of
138 the Select Subcommittee.
139 Ms. O'Connor. Constance O'Connor, senior counsel, Committee
140 on Energy and Commerce Subcommittee on Oversight and
141 Investigations.
142 Mr. McAuliffe. Will McAuliffe, chief counsel for the
143 Minority, Energy and Commerce Committee, Subcommittee on
144 Oversight and Investigations.
145 Ms. Dockham. Kelly Dockham, director of federal affairs at
146 UNC Chapel Hill.
147 Mr. Lambeth. David Lambeth, counsel for UNC Chapel Hill.
148 Mr. Benzine. Thank you.
149 Mr. Chairman?
150 Mr. Wenstrup. Brad Wenstrup, Chairman.
151 BY MR. BENZINE.

152 Q Dr. Baric, before we begin, I would like to go
153 over the ground rules for this interview.
154 The way the interview will proceed is as follows: The
155 Majority and Minority staff will alternate asking you
156 questions, one hour per side per round until each side is
157 finished with their questioning.
158 The Majority staff will begin, and proceed for an hour, and
159 then the Minority staff will have an hour to ask questions.
160 We will then alternate back and forth in this manner until
161 both sides have no more questions.
162 If either side is in the middle of a specific line of
163 questions, they may choose to end a few minutes past an hour
164 to ensure completion of that specific line of questioning,
165 including any pertinent follow-ups.
166 In this interview, while one member of the staff for each
167 side may lead the questioning, additional staff may ask
168 questions.
169 There is a court reporter taking down everything I say and
170 everything you say to make a written record of the interview.
171 For the record to be clear, please wait until the staffer
172 questioning you finishes each question before you begin your
173 answer, and the staffer will wait until you finish your
174 response before proceeding to the next question.
175 To ensure the court reporter can properly record this
176 interview, please speak clearly, concisely, and slowly. The

177 court reporter cannot record non-verbal answers, such as
178 nodding or shaking your head, so it is important that you
179 answer each question with an audible, verbal answer.
180 Exhibits may be entered into the record. Majority exhibits
181 will be identified numerically. Minority exhibits will be
182 identified alphabetically.

183 Do you understand?

184 A I do.

185 Q We want you to answer our questions in the
186 most complete and truthful manner possible, so we will take
187 our time. If you have any questions or do not fully
188 understand the question, please let us know and we will
189 attempt to clarify, add context to, or rephrase our
190 questions. Do you understand?

191 A I do.

192 Q If we ask about specific conversations or
193 events in the past, and you are unable to recall the exact
194 words or details, you should testify to the substance of
195 those conversations or events to the best of your
196 recollection. If you recall only a part of a conversation or
197 event, you should give us your best recollection of those
198 events or parts of conversations that you do recall. Do you
199 understand?

200 A I do.

201 Q Although you are here voluntarily and we will

202 not swear you in, you are required, pursuant to Title 18,
203 Section 1001 of the United States Code to answer questions
204 from Congress truthfully. This also applies to questions
205 posed by congressional staff in this interview. Do you
206 understand?

207 A I do.

208 Q If, at any time, you knowingly make false
209 statements, you could be subject to criminal prosecution. Do
210 you understand?

211 A I do.

212 Q Is there any reason you are unable to provide
213 truthful testimony today?

214 A No.

215 Q The Select Subcommittee follows the rules of
216 the Committee on Oversight and Accountability. Please note
217 that if you wish to assert a privilege over any statement
218 today, that assertion must comply with the rules of the
219 Committee on Oversight and Accountability.

220 Pursuant to that, Committee Rule 16(c)(1) states, "for the
221 Chair to consider assertions of privilege over testimony or
222 statements, witnesses or entities must clearly state the
223 specific privilege being asserted and the reason for the
224 assertion on or before the scheduled date of testimony or
225 appearance." Do you understand?

226 A I haven't read the regulations, but I

227 understand what you're telling me.

228 Q All right, thank you. Ordinarily, we take a
229 five-minute break at the end of each hour of questioning, but
230 if you need a longer break or a break before that, please let
231 us know, and we will be happy to accommodate.

232 However, to the extent that there is a pending question, we
233 would ask that you finish answering the question before we
234 take the break. Do you understand?

235 A I do.

236 Q Do you have any questions before we begin?

237 A No.

238 Q Thank you. I want to start really briefly and
239 run through your education and experience.

240 Where did you attend undergraduate school and what degree did
241 you graduate with?

242 A I attended North Carolina State University,
243 actually on a swimming scholarship. I studied zoology and
244 received a bachelor of science degree there. I stayed on at
245 North Carolina State University in the Department of
246 Microbiology, where I received a Ph.D., studying emerging
247 alphaviruses.

248 From there, I went to University of Southern California,
249 working with a researcher who focused on coronaviruses,
250 specifically a virus called mouse hepatitis virus. And then
251 from there, I went to my faculty positions, which I assume

252 you're going to ask next.

253 Q Yes. More, I guess, who is your current
254 employer and current position?

255 A Currently, I am a William R. Kenan, Jr.
256 Distinguished Professor of Epidemiology and Microbiology and
257 Immunology in the Gillings School of Global Public Health at
258 the University of North Carolina, Chapel Hill.

259 Q And did you hold any academic positions prior
260 to joining UNC?

261 A I was hired at University of North Carolina as
262 an assistant professor in the department of parasitology in
263 laboratory practice. Ultimately, that department was merged
264 into the Department of Epidemiology in the School of Public
265 Health. And so I continued on as an assistant professor in
266 the Department of Epidemiology. Moved on to associate
267 professor, and then eventually full professor. And then a
268 few years later, distinguished professor.

269 Q And you currently run a lab at UNC?

270 A I do.

271 Q How many people report to you in the lab?

272 A Somewhere between 40 and 50. It depends on
273 how you count. There's undergraduates that come through and
274 do work, actually, more training to help move them forward,
275 either in graduate school or medical school. But they're not
276 really doing detailed scientific investigation.

277 Q And then what are kind of your normal duties
278 or roles and responsibilities?

279 A Review research, come up with ideas, try to be
280 innovative, problem solve. So if people are having
281 experiment problems with getting experiments to produce
282 results, I usually am a big help. I perform a lot of help
283 with problem solving. I write grants, I teach, perform
284 service for the university. I think basically all faculty do
285 research, service, and teaching, if that -- you're asking
286 more globally. I didn't know if you were asking more
287 specifically or not.

288 Q No, that answers the question.

289 A Okay.

290 Q Do you currently hold or have you previously
291 held any positions on boards of companies or nonprofits?

292 A Yes, I am on the scientific advisory board of
293 Vaxart, the scientific advisory board of a company called
294 Adagio, which changed their name to ILLiAD. I have been on
295 the scientific advisory board for Takeda Vaccines, and on the
296 scientific advisory board for Sanofi Pasteur with their
297 vaccines as well.

298 Q Do you currently hold or have you previously
299 held any honorariums or honorary positions?

300 A No.

301 Q Thank you. I am going to go through a list of

302 names, and just to the best of your recollection if you had
303 conversations with these folks, email, over the phone, in
304 person, regarding the origins of COVID-19, the Wuhan
305 Institute of Virology, or EcoHealth Alliance, beginning
306 January 1, 2020, until now.

307 A Okay.

308 Q Dr. Francis Collins.

309 A Yes, Dr. Collins, and Kizzmekia Corbett, and I
310 were honored by the governor of the State of North Carolina
311 for making contributions to humanity. That was the
312 Governor's Award. And Dr. Collins sent me an email in 2021
313 saying congratulations. I congratulated him back, so --

314 Q Any conversations with Dr. Collins specific to
315 the origins?

316 A No, not to my recollection.

317 Q Dr. Anthony Fauci?

318 A This is emails, or calls, or all of the above?

319 Q Any manner of communication.

320 A So -- and from this --

321 Q January 1st.

322 A I mention that, because the first time I
323 actually met him was at basically a conference on developing
324 strategies to move forward with MERS coronavirus, research
325 objectives, back in 2014. So that was the first time I met
326 him.

327 But after January 1st, 2020, I was on a phone conference with
328 him on February 1st of 2020 that had to do with the origins.
329 I met with him in his office with several staff, high level
330 staff, both including himself and other representatives from
331 both the extramural and intramural program for NIH on, I
332 think, February 12, 2020. And I believe that's it.
333 Oh, yes, I was also part of -- we were both part of an email
334 exchange that was associated with the Red Dawn group, which
335 was basically trying to help prepare the United States to
336 respond to -- to track and respond to the emerging COVID-19
337 pandemic.

338 Q Thank you.

339 BY MR. STROM.

340 Q On the Fauci meeting, you mentioned you
341 said -- I may have just misheard you -- intramural and
342 extramural NIAID staff?

343 A I believe so, yes.

344 Q Do you recall any names?

345 A Yeah. Auchinhue -- I've got to look at his
346 name.

347 Q Auchincloss?

348 A Yes, Auchincloss. Alan Embry. There's a
349 series of emails that included Maureen Beenan, and someone
350 else that I believe were also there. A few other names that
351 I can't recall.

352 Q David Morens?

353 A I can't recall whether he was there or not.

354 BY MR. BENZINE.

355 Q Emily Erbelding?

356 A We had email exchanges, and I actually talked

357 to her beforehand to try to find out what people wanted to

358 talk to me about. So I believe she was there, but I had

359 never met her personally, just talked to her on the phone.

360 So it wouldn't surprise me if she was there.

361 Q The same topics and timeframe. Dr. Lawrence

362 Tabak?

363 A No, I don't think so. Not to my recollection.

364 Q We touched on Dr. Auchincloss, but any

365 conversations with Dr. Auchincloss outside of the

366 mid-February meeting?

367 A I think there were some group emails, not

368 one-on-one emails like in May, but I can't recall the exact

369 nature of those emails. I'm sure you have my emails, so you

370 probably can figure it out.

371 Q Dr. Cliff Lane?

372 A I don't believe so, no.

373 Q Dr. David Morens?

374 A I don't believe so.

375 Q Dr. Ping Chen?

376 A Not to my recollection, no.

377 Q Dr. Victor Zhao?

378 A Not to my recollection.

379 Q Dr. Robert Redfield?

380 A He was part of the Red Dawn group emails as
381 well. So all of us -- none of us, I think ever, including
382 Fauci, ever made every single call, so we would have been on
383 some calls together.

384 Q But more of the group calls?

385 A It was all group calls, not a person.

386 Q Dr. Michael Lauer?

387 A Not to my recollection.

388 Q Dr. David Christian Hassell?

389 A Yes. He emailed me, I think on the 2nd of
390 February, sometime in February, but I can't recall actually
391 what the substance of that was.

392 Q But it was regarding one of these three topics
393 or COVID, kind of?

394 A It occurred after the origins call with Fauci,
395 so I imagine it was something along those lines, but I can't
396 recall the detail. I would have to see the email.

397 Q Dr. Jeremy Farrar?

398 A Indirectly. He had someone from his group
399 email me about a 4chan threat that had been made toward me.

400 Q Dr. Kristian Andersen?

401 A I met Kristian at a couple of meetings. He

402 emailed -- I think we were on the National Academy Origins
403 sort of committee together, so we would have interacted
404 there. He was on the call, on the February 1st call, so he
405 was there. I believe he emailed me the next day, and we were
406 going to have a call. But for the life of me, I can't
407 remember any details of that call, or whether it even
408 happened.

409 Q Dr. Michael Farzan?

410 A I've known Mike Farzan for a long time, all
411 the way back from the 2003 SARS epidemic, and so we have
412 communicated over the years. I believe he was on the May 1st
413 call, now that you mention his name, but I don't believe we
414 had any other direct emails with him.

415 Q May 1st or February 1st?

416 A Sorry, February 1st.

417 Q Dr. Eddie Holmes?

418 A I've known Eddie Holmes for a while as well.
419 He also emailed to pass on a 4chan threat. But otherwise,
420 no.

421 Q Dr. Ian Lipkin?

422 A I've known Ian Lipkin for a long time. We
423 were funded together on a grant that he was PI on for about
424 five years. Any time I go to New York, I visit him and talk
425 to him, sometimes stay at his house. We talk about science
426 off and on all the time, potential collaborative research

427 that we want to do, interesting results. He's a friend and a
428 colleague.

429 Q Any conversations regarding the origins of
430 EcoHealth?

431 A I think several months after, I don't exactly
432 remember when I was in New York City, but we did talk about
433 origins at that time. He told me about his trip in person,
434 in detail. We may have had a call on it as well, but he
435 talked about his trip to China early in the pandemic, when he
436 went to offer his assistance.

437 We talked about the diagnostic tests that were being run and
438 the lack of standardization among those tests, which was
439 probably his promoting, you know, resulting in some
440 inaccuracy in the reporting numbers, and offered to help with
441 that. He did mention George Gao's call to him, I think at
442 the end of December, so we've talked about that.

443 But I guess at some later date, after the Science paper that
444 I signed with others to say that the lab leak theory needed
445 to be looked at in more detail, he called me up to ask me
446 why. And I sent him a couple of papers that the Chinese had
447 published, where they were doing virus discovery work under
448 BSL-2 conditions, which is one of the main reasons why I felt
449 that the potential laboratory escape hypothesis shouldn't be,
450 in essence, put under the rug.

451 Q Do you recall what those papers were?

452 A I could provide them for you --

453 Q Okay.

454 A -- if you wanted.

455 Q That's fine.

456 A But they were basically Zhengli Shi's papers.

457 I can tell you her original paper on this, which was in

458 Nature around 2012, they were very vague about safety

459 conditions. They said they followed Chinese regulations.

460 But in a Journal of Virology paper, and I believe a PLOS

461 Pathogens paper are the two, I think, they actually stated

462 that they were doing the culturing work under BSL-2. And

463 then they continued that even into September of 2020, which I

464 thought was irresponsible.

465 Q Not the biosafety level that you would conduct

466 that work at?

467 A Well, I think you have to put it in

468 perspective. So biosafety regulations in the United States

469 are very clear, but they're heavily focused on known human

470 pathogens.

471 So when you move into animal pathogens, pathogens that are in

472 animals, where you don't really know the threat level, to

473 some extent, that becomes a decision between the investigator

474 and the local IBC, which may or may not talk to federal

475 authorities about whether this is appropriate or not.

476 So, for example, when we started working with zoonotic

477 coronaviruses, our underlying hypothesis was that there are
478 strains that exist in nature. They may be rare, but they
479 could -- they could potentially infect human cells. And if
480 that's your hypothesis, then you do it under BSL-3.

481 Q Yeah.

482 A The Chinese came to a different -- their
483 biosafety regulations are different. But, again, when you
484 ask me about specific regulations, as the Chinese would say
485 to me, Ralph Baric doesn't determine the biosafety levels in
486 this country, in China, right?

487 Q Yeah.

488 A So it's just different. So we were at a
489 higher level containment in the United States. And then
490 anyone who would ask me for these viruses, I would insist
491 that it be done at a higher level containment. So I kind of
492 set the standard in the United States.

493 Q Moving on with the communications questions.

494 Dr. Andrew Rambaut?

495 A Not to my recollection. Yeah, I don't even
496 know who he is, sorry.

497 Q Dr. Christian Drosten?

498 A I know Christian Drosten. We were members of
499 the Nidovirus Taxonomy Committee. So there was a large
500 number of emails between us and other members of the
501 committee about naming the novel coronavirus. Originally, it

502 was called -- what was it called, 2019 novel coronavirus, or
503 something like that, right?

504 And so that committee determined that we should name it SARS
505 Coronavirus 2, based on its viologenase, how closely related
506 it was to other sarbecoviruses, although it represented
507 completely different branches of the tree.

508 So the branch of the tree before SARS Coronavirus 2, there
509 were two branches. One were called clade 2 strains that
510 couldn't use human receptors or grow in human cells. And the
511 second was the SARS coronavirus 2003 related strains, like
512 WIV1 and SHC014 and a bunch of other viruses. So it's on
513 this branch of the tree. These have 6,000 nucleotide
514 differences than SARS2. So it was a new discovery.

515 So the taxonomy group basically says that it was closely
516 enough related to SARS1 and caused similar disease features,
517 that it should be named SARS2.

518 Q Do you recall receiving any pushback from the
519 Chinese?

520 A The Chinese were very unhappy about that. I
521 think several members of the committee received a lot of
522 pushback. I believe they ultimately wrote a paper that they
523 published saying that -- giving their reasons why they didn't
524 like that name.

525 Q Do you recall any of the reasons?

526 A I actually didn't read the paper, because I

527 didn't want to put up with the nonsense. But so you would be
528 asking me to speculate. I would guess that the SARS
529 coronavirus 2003 impact on Chinese society, and their view of
530 their nation was very -- was very extreme.
531 And so they're very sensitive. They're probably very
532 sensitive to any suggestion that they failed to put in
533 appropriate policies that would prevent another SARS-related
534 virus. That would be my guess, but I was not in the room,
535 right?

536 Q Thank you. Dr. Ron Fouchier?

537 A I've known Ron Fouchier for 15 years as well.
538 I'm part of a scientific advisory board for a CEIRR grant,
539 which is a center of excellence in virus research that is run
540 out of Mount Sinai. And Ron Fouchier is a member of that
541 group.

542 And so I'm familiar with his research. We talk about his
543 research when we had those meetings, I think they were by
544 Zoom, after COVID-19 occurred. He was one of the few
545 researchers that didn't shift his influenza virus program
546 into the COVID-19 at the time. So we didn't talk too much
547 about origins. He was on the February 1st call.

548 Q Do you recall any conversations with him
549 regarding kind of, like, genetic manipulation or being able
550 to manipulate viruses without leaving a trace?

551 A By -- from 2020 on?

552 Q Mm-hmm.

553 A Okay. So from 2020 on, there are a variety of
554 ways that you can make recombinant DNAs that are identical to
555 the sequence of a virus. One of the first ones was an
556 approach we developed using class IIS restriction enzymes
557 that you can orient either within the sequence of the virus
558 or on the outside of it.

559 So when they're on the outside, the way the enzyme is cut, it
560 cuts in the virus sequence, and it leaves actually the virus
561 sequence is the overhang. And they're different sequences,
562 so you end up with directional cloning.

563 So typically, with a restriction enzyme, if you cut and you
564 add an enzyme to make them come together, there's no
565 directionality to it, because the ends are all compatible.

566 So you get these large concatemers in a random fashion.

567 But some enzymes, especially the ones that were associated
568 with the approach that we developed, leave variable ends that
569 are unique, and can only link up with a complementary three
570 or four nucleotide. So that, then, allows you to assemble a
571 genome without leaving restriction sites that you engineered
572 into the genome.

573 Now, you might ask why. I mean, the reason you do this is
574 the primary sequence of the virus is virulence determinative.
575 So if you manipulate the primary sequence, you can attenuate
576 and get a different phenotype than you get from wild type.

577 So the way that we would deal with that is that we would then
578 engineer in signature sequences or mutations that would say
579 this was made in the Baric lab. So I guess to answer your
580 question more thoroughly, you don't have to do that, okay?

581 The other approach is now the synthetic DNA approaches allow
582 you to get much larger clones within the range of direct
583 synthesis.

584 And then there's another approach. There's a company that
585 does gateway cloning that allows you to assemble genomes
586 commercially that I believe that you can, or may or may not
587 decide you want to leave a trace. And then there's other
588 bacterial enzymes that they've used to make full length
589 genomes of bacteria species that the enzymes chew on one part
590 of the DNA. And so they leave an overhang that's specific
591 for the other fragments.

592 So, yeah, a variety of approaches that are available.

593 Q Any conversations with Marion Koopmans?

594 A I've known Marion Koopmans for years. She and
595 I both worked on noroviruses for years. And so if you look
596 historically through my emails, we talked off and on. I
597 don't believe when she took -- recently took the job to run
598 the sort of emerging infectious disease group in the
599 Netherlands in the beginning of the COVID-19 pandemic, I
600 can't recall any emails between us.

601 Q Dr. Michael Worobey?

602 A Let's see. I don't believe so, but I think he
603 was at the nidovirus meeting in Switzerland this year, and I
604 talked to him there. He may have been at -- either him or
605 Dr. Garry were also at the emerging infectious disease
606 meeting at the NIH, and I talked to him there as well.

607 Q Garry was my next one. Dr. Robert Garry.

608 A Okay. I don't think any direct emails. But
609 the nidovirus conference, I think so.

610 Q All right.

611 A But the nidovirus conference, I think so.

612 Q Dr. Jonathan Pekar?

613 A I don't believe so.

614 Q Dr. Florence Debarre?

615 A Oh, she emailed me, I don't remember when.

616 She's an evolutionary biologist in France, so she emailed me.

617 Q Dr. James LeDuc?

618 A I've known Jim LeDuc also for a long time. I
619 think he sent me -- I'd have to look at some notes. Yeah, he
620 invited me to be part of an origins group in, like, March
621 2020, but I couldn't -- I couldn't do it, because I was
622 swamped with other responsibilities, so I didn't participate.

623 Q Any conversations with him regarding biosafety
624 at the WIV?

625 A He was a member of the National Academy group.
626 This is prior to 2020, so National Academy of Sciences in the

627 United States and the National Academy of Sciences in China
628 held three joint meetings, one in Beijing, one in Harbin, and
629 one in Galveston Island, about biosafety and biosecurity.
630 So in the context of that, there were discussions about
631 biosafety and trying to harmonize -- in essence, trying to
632 harmonize and to teach each other's group about standard
633 practices and that kind of thing. But it wasn't more like
634 there was a small group sessions, where we talked about
635 biosafety. It was more of the science that we were doing and
636 the levels that it was done at.

637 Q Dr. Shi Zhengli?

638 A I've known her mostly by email. I think we
639 have met at a couple of meetings from about 2010 on. I have
640 emailed her, she has emailed me, and I have emailed her back
641 since January 2020.

642 Q Anything specific to origins or what was
643 happening at the Wuhan Institute?

644 A Most of our email exchanges, I think they
645 began -- they started initially with the naming of the virus.
646 She was one of the scientists that sent me an email
647 complaining about the name at some point. We had a couple of
648 email exchanges about some transgenic mice that I had sent
649 her under MTA that she was supposed to use at the Wuhan
650 Institute of Virology that somehow ended up at a commercial
651 group in China that they were trying to sell. There's emails

652 about a Cell paper that we were coauthors on.

653 I seem to recall there may have been an email after the paper
654 in Science saying about the potential for -- to open up the
655 investigation, almost -- if it did occur, almost assuredly
656 would be negative. But, again, you guys have my email, so
657 you may know better than I do.

658 Q The transgenic mice that you sent to the Wuhan
659 Institute under an MTA, you just said they ended up at a
660 Chinese commercial group. How did you learn that?

661 A I had a friend, a former post-doc from my lab
662 who works at the University of Maryland, Matt Freeman, sent
663 me an email or a phone text, I don't exactly remember which,
664 which had a product development plan on it saying how much
665 the mice were, which infuriated me because, to some extent,
666 NIH guidelines, should you receive a grant, and journals,
667 should you publish in journals, have a requirement that you
668 share reagents with other collaborative groups, and it's done
669 under MTA. And you don't try to make a profit off of
670 somebody else's discoveries.

671 And so the mice, again, I think it was around 2015, the
672 paperwork started. It probably took a couple years to get
673 through China, because it's really hard to get anything in or
674 out of China, but I think by 2017 or so, they might have the
675 mice. We would have it in our shipping records. So I don't
676 know the exact date, but I just remember it took a long time.

677 I'm sorry, what else is your question?

678 Q I guess, like, what is your presumption there,
679 that you provided the Wuhan Institute with these mice, they
680 had extra mice, and then sold them off, or do you think you
681 were kind of taken?

682 A I think in an expanding epidemic, there was a
683 desperate need for research groups to have access to mouse
684 models, so they could test countermeasures. It was a very
685 good reason to share reagents across nations, because
686 wherever an outbreak occurs, that's where countermeasure
687 development starts.
688 So it makes a lot of sense, just from a global health
689 perspective. What doesn't make sense is that it ends up at a
690 company, and the company is now trying to sell it back to the
691 United States with our emerging pandemic occurring here to
692 make a profit off. So that was infuriating.

693 Q Any conversations regarding the origins with
694 Dr. George Gao?

695 A I've met George off and on, a famous influenza
696 virus researcher, who ultimately became the head of their CDC
697 during the pandemic. George emailed me to share a paper that
698 he had published on one of the earliest variants of concern
699 called D614G. We had published on that, so he sent that.
700 More recently, he sent me an email inviting me to China to do
701 this kind of post-COVID thing that I decided not to go to.

702 Q And we're going to talk about this more, so
703 just briefly, conversations with Dr. Peter Daszak about the
704 origins?

705 A Just briefly about origins. So I think he, as
706 well as -- I don't know, several other people, as well as
707 seeing it on ProMED myself, sent me an email telling me that
708 there's an unknown respiratory disease in China, I think
709 around the 30th of December. So whenever that came out on
710 ProMED. And then on the 5th, he also emailed me to mention
711 that it was probably a coronavirus.

712 Q On January 5th?

713 A Around January 5th. I also had received
714 emails from other people that it was a coronavirus on January
715 5th. And by the 6th or so, I also knew it was a coronavirus,
716 because I was asked to review a paper.

717 Q Any conversations with Dr. Ben Hu?

718 A Not to my recollection.

719 Q What about Dr. Lanying Du?

720 A My capacity to link Chinese names to the
721 researchers is not good.

722 Q She was at the Blood Center of New York, and
723 is now at Georgia State.

724 A I don't think so, not to my recollection.

725 Q And Dr. Zhou Yusen or Yusen Zhou?

726 A I would have to do email research to know

727 that. No, nothing that comes to mind.

728 BY MR. SLOBODIN.

729 Q One more name. Dr. Lili Ren from the
730 Institute for Pathogen Biology in Beijing?

731 A If she did, it would not have been a
732 person-to-person email, I don't believe. It would have been
733 a group email.

734 So one of the things that was occurring in the early days of
735 the pandemic was that the National Academy set up some phone
736 conference calls between Chinese scientists and American
737 scientists. And they usually lasted an hour. And basically,
738 the goal of those calls was to discuss patient care,
739 diagnostics, public health control measures, those types of
740 issues, and basic science questions.

741 So it was very likely that there were several members from
742 China that would have been on that call. You had two pages,
743 two to three pages of pictures with names under them, and I
744 didn't take screenshots or anything. So I couldn't tell you.
745 The one person I know was on it was George Gao, and Zhengli
746 Shi was also on. Those are two people definitely I recall.

747 BY MR. STROM.

748 Q For the January 6th paper that you reviewed,
749 do you recall if that had the sequence of the virus?

750 A It did. When it was first sent, it did not.

751 All three reviewers immediately asked for the sequence.

752 BY MR. BENZINE.

753 Q Do you recall what the paper was?

754 A So review processes are normally confidential,

755 so if I tell you what journal it is and this comes out, then

756 I -- can we go off the record, so I can tell you that?

757 Q We can go off the record and talk about it,

758 and determine what to do. And I can talk to Clark about

759 redacting if we need to.

760 A Just the review process is supposed to be

761 confidential. So I would prefer that it remain confidential,

762 although I guess, to some extent, the paper got accepted,

763 so --

764 Mr. Benzine. We can go off the record.

765 (Discussion held.)

766 Mr. Benzine. We can go back on the record.

767 BY MR. STROM.

768 Q Dr. Baric, you referenced receiving a January

769 6th paper that was subsequently published?

770 A 6th or 7th.

771 Q It was subsequently published in Nature,

772 showing that the virus -- the unknown outbreak was caused by

773 a coronavirus.

774 A Yes.

775 Q And then you mentioned earlier that the

776 sequence of the virus was not initially provided. Do you

777 recall when you got access to the sequence?

778 A Within about 12 hours from requesting it from
779 the journal. And just for point of clarity, I knew it was a
780 coronavirus before I received the paper.

781 Q Do you recall if that version of the sequence
782 had the furin cleavage site in it?

783 A Are you asking me in the context of January
784 6th or 7th, or are you asking me in the context of --

785 Q You don't recall seeing a sequence that
786 omitted --

787 A No.

788 Q -- the furin cleavage site?

789 A No, it was not omitted.

790 BY MR. BENZINE.

791 Q Was this the first time that you saw the
792 sequence?

793 A Yes.

794 Q You also said, and ProMED did a notification
795 on December 30th, and you said that was around the same time
796 you were made aware. Were you made aware by the ProMED
797 notification or through other means?

798 A Well, the ProMED announcement came about the
799 same time I heard from other people that it was -- that there
800 was an unknown respiratory disease in Wuhan.

801 Q Who did you hear from?

802 A Peter Daszak, I believe Mark Denison sent me
803 an email. It wouldn't surprise me if Matt Freeman sent me an
804 email. Corona virologists, it's a small community, so
805 friends email all the time. And if there's an unknown
806 respiratory disease in China and you're a corona virologist,
807 you're thinking it could easily be a coronavirus.

808 Q And then you said January 5th was when you
809 knew it was a coronavirus. Am I remembering that right?

810 A Yes.

811 Q How did you know that?

812 A So I'm blanking on his name. Fred -- so Fred
813 Hayden is a clinician at the University of Virginia, who does
814 clinical trials for either vaccines or immunotherapeutics or
815 drugs against respiratory viruses, severe respiratory
816 viruses.

817 And he had -- Chinese scientists had contacted him around the
818 2nd or 3rd. And Fred was a member of the scientific advisory
819 board for our center for excellence in translational research
820 that was run by Rich Whitley out of the University of
821 Alabama.

822 So he knew we had a paper that was in press in Nature
823 Communication that compared remdesivir to what the Chinese
824 considered was the gold standard for the treatment of the
825 SARS-related infection, which was an HIV protease inhibitor
826 cocktail, lipinavir and ritonavir. So working with Gilead in

827 that paper, we had done a careful comparison of the efficacy
828 of those drugs compared to remdesivir in mouse models, both
829 MERS and SARS coronavirus in 2003.
830 So Fred called me to ask me if I would be willing to share
831 that paper with the Chinese, so that they could take a look
832 at it. So I said, yes, and two days later, he informed me
833 that -- by email, confidentially, as well as a couple other
834 people. So again, it's probably in my email. So if you look
835 for his name, you'll find him. But he told me that it was a
836 coronavirus and a SARS-related virus and was about 70, 80
837 percent identical to the original SARS strain. The sequence
838 confirmed that.

839 Q Thank you. My last kind of question in this
840 bucket, have you ever had any contracts, agreements, or other
841 binding paperwork with the Chinese Academy of Sciences or the
842 People's Liberation Army?

843 A I don't believe so. I've never had any
844 funding from China.

845 Q When we interviewed Dr. Daszak, he testified
846 that -- and there's emails to this effect of him putting your
847 gmail on emails, and dropping your UNC email, so it wouldn't
848 go through the state FOIA law. And I think a lot of it was
849 probably what you were referencing, the threats on 4chan and
850 various things, and trying to quell those a little bit while
851 the emails were getting FOIAed.

852 A He didn't do that email on my request.

853 Q Do you recall having any conversations with
854 him regarding putting your gmail on things?

855 A I told him it was irresponsible to do that,
856 and I was very unhappy with him, so, yeah.

857 Q I appreciate that. Do you recall, just for
858 our own kind of, like, document retention, do you recall
859 putting your UNC email back on or --

860 A What do you mean back on?

861 Q So Dr. Daszak would drop your UNC email, trade
862 it out with your gmail. Do you recall saying, no, I need
863 to -- this needs to go under my UNC email?

864 A At some point. I don't know how quickly I
865 did, but at some point, I did. I can't tell you exactly
866 when. I know that I would oftentimes answer, if he sent me
867 something by gmail, I would oftentimes send it back regular
868 mail. But I can't say that I did it every time.

869 Q I'm just trying to understand. Not a
870 substantial amount of communications over your gmail, most of
871 it over your UNC account?

872 A I don't think there's a substantial amount of
873 communication, but there would have been some because of
874 that, yes.

875 Q Prior to this interview, did you have
876 communications with anyone on that list regarding the

877 interview?

878 A No.

879 Q Have you had any conversations with Dr. Daszak
880 since his interview in November?

881 A Well, we're part of an emerging infectious
882 center disease grant that's run out of Southeast Asia that
883 includes a bunch of Southeast Asian countries except China.
884 So it's along the border. So if you want to know -- if you
885 really want to get to the questions of origins and whether or
886 not there are zoonotic strains very similar to SARS
887 coronavirus, you need to be along the Chinese border. You
888 need to be as close to China as you can.

889 So that's where he set up his emerging infectious disease
890 center. So we have quarterly reports and we have calls that
891 we share information and data. There is year-end progress
892 reports that we have to write up that we submit to the
893 grants.

894 And then, occasionally, I think there's a meeting each year
895 that the NIH puts on to have the different centers come
896 together, and share kind of what they're doing and be
897 reviewed by an outside review committee.

898 So, yeah, there's going to be emails back and forth about
899 that.

900 Q Nothing about his interview, though?

901 A No, I did not talk to him about that.

902 Q In the spirit of saving paper, I'm not going
903 to introduce Dr. Fauci's calendar from February 11th. But
904 that's when his calendar at least says that you met with him.

905 A Was it the 11th?

906 Q I'll introduce it.

907 A No, it's okay, I believe you.

908 Q Yeah, February 11, 2020.

909 A Okay. I was there for a reverse site visit,
910 so it sort of got blended in, so I don't exactly remember
911 which date it was.

912 Q And you already said it took place -- and I
913 just want to ask, Dr. Fauci was there at the meeting?

914 A He was there for a short period of time. I
915 already mentioned some of the names that were there. So he
916 was there for somewhere between five and ten minutes, at
917 most. And he got -- a secretary came in and said that he had
918 a call in the SCIF that he apparently had to go to, so he
919 apologized. So he wasn't there for the whole time.

920 Q Do you recall, specifically while he was
921 there, what you discussed?

922 A Well, these meetings, they always start off
923 with kind of pleasantries. But ultimately, the goal of the
924 meeting, to my recollection, was primarily focused on the
925 2015 paper that we published in Nature Medicine that
926 basically, in my opinion, warned the world that there were

927 viruses that existed in nature that could threaten human
928 health.

929 And so the first thing they wanted to do was talk about that
930 paper, and then they wanted to talk about the
931 regulatory -- the P3CO regulatory compliance that was
932 associated with that.

933 Q Do you recall the specific conversations
934 regarding the science of the paper?

935 A Yeah, sure. So I said that we had access to
936 the spike of proteins of this virus called SHC014 that was
937 provided by Zhengli Shi before she published it, which was
938 generous. Most scientists would not do that.
939 Later, she sent the plasmid on filter paper and coding the
940 spike sequence of that virus as well. But that's what we
941 had. And so -- and it's also cheaper, synthetic DNA costs at
942 the time, like the spike gene may cost \$3,000, a full length
943 genome may cost 17, 18,000. So we weren't a wealthy lab. So
944 it's a high-risk event to build a full-length virus,
945 especially if you don't have the sequence. So we synthesized
946 the spike gene and decided to place it into the context of
947 the SARS coronavirus 2003 mouse adapted strain.
948 So we talked about that. And then we talked about the
949 specific experiments that were done, the first of which we
950 compared the growth of this isolate to the parental virus
951 that we introduced the spike gene into. And it replicated

952 the same. So from our perspective, in terms of P3CO, that's
953 not called gain of function, that's called retention of
954 function, right?

955 We also looked at its ability to use different receptors,
956 ACE2 receptors from different animals, like the mouse, the
957 bat, the civet, and the human. And the chimera used those
958 receptors as well as the original SARS coronavirus strains.

959 So, again, no gain of function, it was retention of function.

960 So we looked at the growth in primary human cells and they
961 were the same. Ultimately, at some point -- and I should
962 probably put this in the perspective of a timeline.

963 So we were approved to do these experiments in early 2014
964 before the pause occurred from the Obama administration. So
965 by the time the pause occurred, we had already isolated the
966 chimeras and were in the process of isolating, if we hadn't
967 already isolated, the full length viruses as well.

968 So once we knew the spikes, could program infection, then you
969 could take a chance and spend \$17,000 and see if it works,
970 because there's a chance. There's a high error in
971 sequencing.

972 So that's the background. So then we -- ultimately, we
973 compared the chimeras to the full length SHC014 virus, in
974 which they grew about the same again as well, no real change
975 in any of those growth phenotypes. And then we went into
976 animals. The parental virus, in this case, it was the SARS

977 mouse who had the strains 100 percent lethal, the chimera was
978 not. It caused weight loss and the animals recovered.
979 Now, when you went into the older, vulnerable animals, again,
980 the wild type parent was 100 percent lethal. And the chimera
981 caused about 10 percent mortality, but most animals
982 recovered. So that is, again, a loss of function, it's not a
983 gain of function.
984 That information was all provided. So when the pause
985 occurred -- and then I explained this in the meeting. When
986 the pause occurred, we had that data. And so if you were
987 already doing experiments when the pause came out, you had a
988 choice, you could either pause or you could continue your
989 studies. The pause affected anything new that was funded.
990 So two things happened. In terms of new research that we
991 were doing, we were given a waiver to go forward with making
992 a MERS model, and you have that paperwork. In the case of
993 the 2015 paper, we paused and put in all the paperwork saying
994 these are the phenotypes that we see in the virus. As far as
995 we were concerned, the data is not consistent with a gain of
996 function phenotype. And ultimately, the NIH reviewed that
997 and came back and said that they didn't think it was gain of
998 function, either, and I could proceed. So then we proceeded
999 and eventually published the paper.
1000 So that kind of whole context, that's kind of -- and Fauci
1001 left in the early stages of that discussion, right, because

1002 that took about 25, 30 minutes. I don't know how long it
1003 took, probably too damn long probably.

1004 Q Less than 25 or 30 minutes. So was that the
1005 primary purpose of this meeting, was to review --

1006 A Yes.

1007 Q Like NIAID employees wanted to review that
1008 paper, and see if it had gone through the proper channels?

1009 A Yeah, I think I was also asked how closely
1010 related were these viruses to the SARS2 strain, which I
1011 already mentioned to the committee that they're on different
1012 branches of the phylogenetic tree, they differ by 6,000
1013 times. So one is not regenerative of the other, and that's
1014 been published by six or seven groups so far.

1015 Q In that meeting, did they ask you any
1016 questions about the Wuhan Institute, what research they were
1017 doing?

1018 A I don't recall that. I don't believe so, but
1019 I think you have to look at it from my perspective, which is
1020 I'm being called to talk about a paper I published on the
1021 gain of function regulation. And I'm freaked out that
1022 perhaps I didn't do the paperwork right. So I was focused on
1023 that.

1024 Q Okay.

1025 A And by the way, I did all the paperwork right.

1026 Q We appreciate good paperwork around here. At

1027 that meeting, and we're going to talk about this proposal in
1028 more detail, so we don't need to talk about the science. But
1029 at that meeting, did you bring up the DEFUSE proposal to
1030 DARPA?

1031 A No.

1032 Q Why not?

1033 A Mostly because I had forgotten about the
1034 DEFUSE proposal in DARPA, quite frankly. I read a lot of
1035 grants. And so the grant was not funded, so I moved on.

1036 Q I appreciate that.

1037 BY MR. WENSTRUP.

1038 Q When COVID hit, we were all in lockdown and
1039 started doing research. And I was looking for how do we
1040 treat people, what do we do? We don't have a test, we don't
1041 have a definitive treatment for this. It's called novel for
1042 a reason.

1043 And one of the things that I came across was your 2015
1044 article. And the first thing that occurred to me was gain of
1045 function, loss of function, regardless, to me, it was, like,
1046 wow, this can be done? And so for me, I was kind of like,
1047 this is kind of concerning here.

1048 And I'll talk about that again in just a minute, but in all
1049 of your research over the years, how close have you ever come
1050 to creating a virus similar to SARS-CoV-2, as far as
1051 structure, pathogenicity?

1052 A Before or after it emerged?

1053 Q Well, in retrospect, or after it emerged.

1054 A So before, I think what you need to think

1055 about is that no one had the sequence. So if you don't have

1056 the sequence of the pathogen, you don't have any guide to how

1057 to synthesize it or make it.

1058 Q But looking back?

1059 A Just to give you an example. Let's say I took

1060 SHC014 and I wanted to convert it to SARS-CoV-2. The first

1061 thing I have to know is the sequence of SARS-CoV-2, because

1062 if I don't know that, what I do know is that there are 6,000

1063 mutations -- let's say if I do it, there are 6,000 mutations

1064 that exist in SHC014 that don't exist in SARS.

1065 Q Let me clarify, because I'm not trying to get

1066 into that.

1067 A Well, statistically, you have to make four to

1068 the 6,000 mutants which can't be done.

1069 Q Okay.

1070 A Okay.

1071 Q My question really is maybe unrelated, maybe

1072 it's from a MERS virus, whatever. Anything close to the

1073 pathogenicity?

1074 A Never.

1075 Q Okay.

1076 A The only time that statement would be true

1077 would be with variants of concern that emerged after SARS
1078 emerged.

1079 So the first mutant that we made was a virus called D614G,
1080 which emerged in February, and then displaced the original
1081 Wuhan strain. So in that case, you have the sequence to
1082 guide your mutagenesis. The epidemiology indicated a new
1083 mutant had emerged in the population that was displacing
1084 everything else, and so it was a simple insertion of that
1085 nucleotide into the genome.

1086 Q When you were doing this type of work, what
1087 BSL level were you?

1088 A Always worked at BSL-3.

1089 Q What safety guards do you employ against that?
1090 You, personally, in your work?

1091 A So in our laboratory, we have a negative
1092 containment facility that is powered by backup fans, so
1093 there's two fans. So if one fan fails, there's a backup
1094 system that keeps the negative pressure. All of those backup
1095 fans are on the redundant power. And so emergency power. So
1096 if there's a failure in the system, it maintains. If
1097 everything fails, then the facility is designed to go
1098 neutral. So in other words, there's no air flow in or out.
1099 Within the facility, there are biological safety cabinets
1100 that are the primary containments for working with a
1101 pathogen. Those are also on emergency backup and also

1102 battery pack powered. The battery pack power gives you about
1103 30 minutes. So if there's a complete failure of all power
1104 and the facility goes negative, the hoods stay on, which
1105 gives the researcher and the facility about 30 minutes to
1106 decontaminate everything, clean it up, and put everything
1107 away.

1108 Now, our staff, the minimal regulations I think is lab
1109 jackets and goggles and an N95 mask. We take personal
1110 protective equipment at a much higher level. So we wear full
1111 Tyvek body suits with double gloves. People have an apron on
1112 top of the Tyvek suit, which is normally -- if there was any
1113 kind of aerosol or accidental spill, it would go on the
1114 apron.

1115 And then you have a hood and a shield that comes down to
1116 about here with a portable air breathing apparatus that pumps
1117 the air through Hepa filters and other chemical filters to
1118 pull out other toxins in the air.

1119 So if you think about protective barriers, it's basically a
1120 layered redundant system, where you have the negative
1121 containment facility, the hood. You have personal protective
1122 gear, and then you have SOPs that are in place, standard
1123 operating procedures, that are also designed to be redundant,
1124 so that if one thing fails, you have a backup.

1125 When I was setting up my BSL-3 lab, I was impressed by this
1126 television show called Seconds to Disaster. And in Seconds

1127 to Disaster, the common thread was always that there were
1128 redundant systems that had to fail before it occurred. So we
1129 put as many redundant systems as we could think of.

1130 Q So in that vein, what level lab was used when
1131 you were working with Dr. Shi Zhengli in 2015, the work that
1132 was maybe done in Wuhan, do you know?

1133 A There wasn't any work done in Wuhan. All the
1134 work was done at UNC, except for one experiment that was
1135 involving -- they had taken the SHC014 spike and placed it in
1136 a lentivirus, a pseudovirus.

1137 So, in other words, just the spike of SHC014 was placed into
1138 a virus particle. That's a single hit virus that can infect
1139 one cell, and then it can't spread. And it's used as a sort
1140 of bio-containment approach to ask questions about the
1141 functions of viral genes.

1142 And in this case, they did an experiment to ask whether the
1143 pseudotype virus they had could infect and use human ACE2
1144 cells. And it couldn't, and the reason for that is that a
1145 lot of the fundamental approaches that had been developed to
1146 make pseudotypes with coronaviruses weren't very efficient in
1147 2015.

1148 We subsequently did a lot of work with Barney Graham as we
1149 moved in to evaluating Moderna mRNA vaccines against MERS, to
1150 work out the technology, so that those pseudotype systems
1151 became much more efficient. So that you could do

1152 neutralization assays. Subsequently, they've been used all
1153 the over the United States and the world. So they didn't do
1154 any live virus work associated with that paper.

1155 Q Have you ever had a sense that research you
1156 did or some others in the field were doing could lead to a
1157 change of direction, where the outcome is different than
1158 expected?

1159 You talked about when you have a hypothesis, and so you think
1160 this will be okay to do, you don't expect it to be a pandemic
1161 pathogen. But have you ever had that concern, like, were you
1162 ever worried that the -- and also were you ever worried that
1163 the capabilities that you develop the expertise for could be
1164 used in some nefarious way or lead to a pandemic pathogen,
1165 not necessarily your work, but somebody else's?

1166 Like I always refer to when the Wright brothers invented the
1167 plane, they weren't thinking of flying into the buildings and
1168 killing 3,000 people, right, but somebody did.

1169 So when you have this type of technology, were you ever
1170 concerned that, hey, we've got to be careful who's doing this
1171 type of work because it's pretty dangerous, or can be?

1172 A Yeah, so we did -- I think a responsible
1173 scientist has to think about that. And I always call it the
1174 sort of unintended consequences, right? You're doing a
1175 series of experiments. But evolution follows its own path,
1176 not the path that you might necessarily think it's going to.

1177 So there's always a chance, some risk, for unintended
1178 consequences in any kind of virus evolution experiment.

1179 Q Evolution, I understand that. You can't
1180 really control that, except try and monitor it through
1181 surveillance, things like that. But I guess what I'm driving
1182 at is, one of the roles of this Committee is to have plans
1183 for the future. And so how do we protect ourselves?
1184 Because the technology exists, and so we have to come
1185 up -- or try to come up with ways as a country to make sure
1186 we have all the checks and balances in place, so an adverse
1187 reaction doesn't occur, either accidentally or intentionally
1188 by someone else.

1189 A So I can tell you what things we put in place
1190 in the 2015 paper. So for example, although we published the
1191 approaches for how to build molecular clones of
1192 coronaviruses, we never had anyone from Dr. Shi's lab or any
1193 of the Wuhan Institute of Virology come to our lab and train.
1194 We never taught them.

1195 In fact, if you look at their cloning technology, they use
1196 baculoviruses. They may assemble some of the full length
1197 molecule using some of the enzymes that we have, but they
1198 implant it directly into an insect virus to maintain it as a
1199 baculovirus, which was a technology developed in Europe, not
1200 my technology.

1201 We think our approach is safer because we've divided the

1202 genome into six pieces, so there's no way any of those can
1203 initiate an infection. And we don't assemble until we're in
1204 the BSL-3. So it's fundamentally safer than what was done by
1205 others.

1206 In terms of how we built the chimera, we didn't publish the
1207 sequence of the virus that we built, and we didn't share the
1208 sequence of that chimera with anyone at the Wuhan Institute
1209 of Virology. So we didn't give them the template on how to
1210 build the recombinant virus.

1211 Q Is that your own precaution?

1212 A Actually, that last precaution was done in
1213 collaboration with discussions with NIH, with our program
1214 officer, and the journal. And to some extent, it was a
1215 natural extension for -- in response to the transmissible flu
1216 studies, and whether or not the virus sequences should be
1217 made available.

1218 Ultimately, after the pandemic, we received a bunch of
1219 requests for the full-on sequence, and then we made it
1220 available just because there were conspiracy theories that
1221 were beginning to bounce around, that that virus was the
1222 cause of the pandemic in China. And people wanted to see the
1223 sequence. So for transparency, we really had no choice but
1224 to make it available.

1225 Mr. Wenstrup. Thank you.

1226 BY MR. STROM.

1227 Q One quick follow-up on the Chairman's
1228 question. But there isn't any sort of formal export review
1229 procedure for these kind of dual use technologies?

1230 A Yeah, export control regulations do -- they're
1231 complex.

1232 Q Yes.

1233 A And so the University of North Carolina has an
1234 export control group that regulates that. And so if we were
1235 going to have to -- if we were going to send anything to
1236 China directly, that at least it would be looked at in that
1237 context of export control, yeah. But those rules are kind of
1238 vague.

1239 Mr. Benzine. I think we're at time. We can go off the
1240 record.

1241 (Recess.)

1242 Ms. Yass. We can go back on the record.

1243 BY MS. YASS.

1244 Q Good morning, Dr. Baric. My name is Alicia
1245 Yass. I am senior counsel for the Democrats on the Select
1246 Subcommittee, and we want to express our thanks for you
1247 making the trip to come up here and for voluntarily agreeing
1248 to speak with us. We do have some questions for you today as
1249 well, and I will start by turning things over to my
1250 colleague, Joseph, for our first section.

1251 BY MR. ROMERO.

1252 Q Good morning, Dr. Baric.

1253 A Good morning.

1254 Q We would just like to ask you a few questions
1255 about the 2015 paper testing the SHC014 spike protein you
1256 coauthored in Nature Medicine. We discussed this paper some
1257 in the previous round.

1258 A Correct.

1259 Q I will introduce the paper now as Minority
1260 Exhibit A.

1261 (Minority Exhibit A was
1262 identified for the record.)

1263 BY MR. ROMERO.

1264 Q So in this paper, among other findings, you
1265 found that the SHC014 spike on a mouse-adapted backbone
1266 showed reduced pathogenicity compared to the full length
1267 mouse-adapted SARS backbone. Does that sound right?

1268 A That's correct.

1269 Q So the full length mouse-adapted SARS backbone
1270 has a name, MA15. And as you understand things, you helped
1271 to create that virus?

1272 A Yes, the virus was originally created in
1273 collaboration with Kanta Subbarao at the National Institutes
1274 of Health. She did the serial passage of the original SARS
1275 strain, which could replicate, but not cause disease in mice.
1276 And after about 15 passages, the virus became more

1277 pathogenic. There were six amino acid changes associated
1278 with the increase in virulence in the mouse, which we then
1279 engineered into the molecular clone that we had built to make
1280 a mouse-adapted strain that's been widely used in select
1281 agent labs across the U.S.

1282 Q Could you help us understand the scientific
1283 need to create this mouse pathogen virus, and what its uses
1284 ended up being?

1285 A Sure. One of the fundamental problems in the
1286 development of small molecule inhibitors and
1287 immunotherapeutics in drugs, as well as understanding the
1288 basic mechanism by which a virus causes disease, is that as
1289 viruses traffic from one species to the next, they oftentimes
1290 lose virulence.

1291 So the original SARS coronavirus virus strain, for example,
1292 caused 10 percent mortality rates in humans. But if you
1293 infected a mouse, it barely would grow to 10 to the 5th in
1294 the mouse. They didn't lose any weight, but the virus
1295 replicated primarily in a few cells in the mouse.

1296 So if you're developing drugs or antivirals or vaccines, it's
1297 actually very easy to make something work against a virus
1298 that's crippled in a model. It's not crippled in humans,
1299 right, so -- and standard practice is that you want to
1300 develop a model that closely phenocopies the human disease
1301 outcome.

1302 So this particular mouse-adapted strain, MA15, targeted
1303 epithelial cells in the airway, club cells at the transitions
1304 between the airways into the gas exchange, in essence, the
1305 little balloons that puff up and down, the alveoli. And
1306 targets AT2 cells in there, just like it does in the human.
1307 It results in an acute respiratory distress syndrome disease
1308 outcome, where there's a tremendous amount of fluid and a
1309 fibrin deposition in the lung. There's a breakdown of the
1310 alveoli/epithelial barrier that allows flooding. So, in
1311 essence, the mouse or the human patient infected with the
1312 original SARS strand is basically drowning in their own
1313 fluids.

1314 It also strips -- kills AT2 cells, which makes surfactant,
1315 which -- you know, when you get a balloon the first time out
1316 of a bag and you try to blow it up, it's really hard to cause
1317 it to inflate. Without surfactant, that's what your alveoli
1318 are like, it's hard to breathe.

1319 So the mouse model that we created mimicked the human disease
1320 phenotype as closely as we could, and it was lethal,
1321 especially in the older animals. So now you have a model
1322 that grows to higher titer, close to 10 to the 8th, it
1323 targets the right cells, the right organ, causes the right
1324 kind of disease. So now you have a rigorous model to develop
1325 small molecule inhibitors. And this was really important for
1326 us.

1327 One of the things that drove the 2015 paper was that SARS
1328 coronavirus emerged in 2003. It was controlled by public
1329 health intervention strategies because it didn't transmit
1330 until you got clinical disease. People thought it was a
1331 fluke, one-off, it's not going to happen again. Then MERS
1332 coronavirus emerged in 2012, again, highly pathogenic, 35
1333 percent mortality rate, but it didn't transmit very well.
1334 So that data made us ask the fundamental question: What is
1335 the risk level that exists in nature? This paper, in
1336 essence, said the risk in nature -- that risk existed in
1337 nature. And then the mouse models were then used to develop
1338 countermeasures.
1339 So almost immediately in parallel with this paper, we started
1340 working with Gilead Scientific to evaluate nucleoside
1341 inhibitors that might work against the coronavirus family.
1342 After testing a bunch of things, we eventually got down to
1343 remdesivir, demonstrating that it worked against the MERS
1344 coronavirus and the SARS coronavirus. That led to a
1345 companion paper that included these viruses in 2017 that said
1346 these are broad spectrum antivirals that work in robust
1347 animal models of disease. And the preclinical data was now
1348 available to move into the clinical trials. So that's why
1349 animal models are so important.
1350 Ultimately, remdesivir, molnupiravir, the Moderna vaccine, I
1351 don't know if we ever did the Janssen vaccine. But several

1352 therapeutic antibodies had all made it through the FDA and
1353 into the clinic, went through our lab, and many of them
1354 touched these viruses that were developed in the 2015 paper.
1355 These same viruses are being used for universal vaccine
1356 design for all sarbecoviruses and all betacoronaviruses.
1357 So if you want to really protect the public, you have to have
1358 the appropriate virologic reagents that challenge the
1359 effectiveness of either your drug or your antibody or your
1360 vaccine and prove performance.

1361 So ultimately, the goal of what resulted from this paper was
1362 the idea that we had to develop drugs, we had to develop
1363 immunotherapeutics that were broadly active. And we had to
1364 develop vaccines that were broadly active. And that paper,
1365 including the viruses, the human viruses that occurred, were
1366 included in studies that were used with the Moderna vaccine
1367 as well.

1368 So, again, animal model development is key to this. It's,
1369 again, very, very easy to make drugs that work against
1370 something that barely replicates, but then when they get into
1371 the humans, they fail. So that's the basis for it.

1372 That's probably a little longwinded. I apologize. Anyway,
1373 that's the thought process.

1374 Q So it sounds like this mouse-adapted virus was
1375 created to parallel the level of pathogenicity that I guess
1376 humans would experience?

1377 A Yes, with an important caveat. So a long
1378 history in virology is that serial passage of a pathogen
1379 that's adapted to one species, as it moves to another
1380 species, it rarely becomes a generalist. It usually loses
1381 its ability to cause severe disease in the original species.
1382 So serial passage has been used in virology for decades to
1383 make live virus vaccines, like the measles vaccine was
1384 passaged in subculture many times. The live polio virus was
1385 passaged in subculture to basically adapt it to the new
1386 environment where it loses its capacity to interact with host
1387 proteins that are specific to the natural host, and so it
1388 becomes attenuated.

1389 Q Is there a sense that because MA15 has
1390 enhanced replication and lethality, that it has been
1391 preadapted to be pathogenic in mice, that it is unsurprising
1392 that by removing its spike and replacing it with the spike
1393 from another virus, say SHC014, the resulting chimera would
1394 be less pathogenic than the full length original MA15?

1395 A That's a really good question. So it depends
1396 on the biochemistry and the receptor binding capabilities of
1397 the virus that you drop into the backbone of the strain that
1398 you chose.

1399 So in this case, the mouse-adapted strain, without question,
1400 had been selected for its ability to replicate and cause
1401 disease sufficiently in the mouse. It may be more difficult

1402 to make a virus more virulent than that. So if you dropped
1403 the SHC014 spike in there, the most likely phenotype is the
1404 mouse phenotype.

1405 Q You also coauthored another 2016 paper,
1406 "SARS-like WIV1-CoV poised for human emergence." Does what
1407 you just said also hold true for, like, creating a WIV1 MA15
1408 chimera and comparing that to full-length MA15?

1409 A Yes. So in the 2015 paper, we only compared
1410 pathogenesis in wild-type mice. In the PNAS paper in 2016,
1411 we compared pathogenesis in wild-type mice and also humanized
1412 mice that express the human ACE2 receptor. And if I remember
1413 correctly, the WIV1 virus was more attenuated than the
1414 wild-type virus. I would have to look at the paper to be 100
1415 percent sure.

1416 Q So back to the 2015 Nature Medicine paper, it
1417 also had two other things to say about the SHC014 spike
1418 protein vis-a-vis wild-type SARS Urbani.
1419 I would like to first just lay out those two things, and then
1420 ask you, at the time you wrote this paper, how you viewed
1421 those things together, and if there was any significance when
1422 juxtaposing them.

1423 The first was that full length SHC014 was less pathogenic in
1424 mice than full length SARS Urbani. Does that sound correct?

1425 A Both of them caused little, if any, weight
1426 loss, so I think they're pretty comparable. Comparable is

1427 the better word. Sorry, not "compare-able." I grew up in
1428 south Jersey, it happens, sorry.

1429 Q And the second was that the SHC014 spike on an
1430 MA15 backbone was more pathogenic in mice than the SARS
1431 Urbani spike on an MA15 backbone, correct?

1432 A Yeah, that was -- yeah. So in the discussion
1433 of this paper, we put in a statement saying that depending on
1434 how you compare gain of function and loss of function values
1435 in the system, the selection system that you're using, you
1436 can get different values. And that review panels need to be
1437 aware that when they review these things in the future, that
1438 they need to carefully consider the context of what kind of
1439 experiment is being done.

1440 So in this paper, we never did a head-to-head comparison of
1441 the mouse-adapted strain that was missing the single amino
1442 acid change in the spike that helped it to be mouse-adapted.
1443 So if you took the five mutations set where you had five of
1444 the six mutations without the spike-like protein, it was
1445 more -- it lost some of its virulence potential.

1446 Now, both of them are attenuated. And so you're asking me
1447 the question, in an attenuated backbone, which one is more
1448 attenuated. We never did a head-to-head comparison, right?
1449 So the experimental conditions like the age of the mouse,
1450 that's a little bit different. The mouse models and emerging
1451 coronaviruses all have this striking age-related phenotype.

1452 So after about 20 weeks, again, depending on the virus, the
1453 virus becomes more virulent as a function of age, just like
1454 in humans. So it recapitulates that phenotype.
1455 So to do this experiment properly, you actually need to set
1456 up the conditions where you have all three viruses with the
1457 same age mice that were housed under the same conditions, and
1458 then infected in the same dose.
1459 What we quoted on in this paper was that in the experiment
1460 where we removed -- in a different paper, where we removed
1461 the spike and you compare the clinical outcomes, the weight
1462 loss outcomes, there's a little more weight loss with the
1463 SHC014 as compared to the mouse-adapted virus, without the
1464 mouse-adapted spike mutation.
1465 So that's the problem with gain of function or loss of
1466 function. Depending on how you can compare it, you can end
1467 up with different phenotypes, and that's what we've tried to
1468 say at the end of the paper to future people doing this kind
1469 of work, that they needed to be aware that the conditions
1470 that you do these kind of experiments, and how you compare
1471 outcomes can have an effect on loss and gain of function
1472 phenotypes.
1473 Q So to the extent this question of comparing
1474 the different outcomes was on your mind, what were you
1475 thinking about whether this spike protein from SHC014 could
1476 be used to create something more pathogenic than SARS Urbani?

1477 A Well, there's no data. So the only data you
1478 have is that you can do a minimal tweak of pathogenesis in a
1479 mouse, not a human. We don't have any data on humans.

1480 Is that what you're asking, in the context of humans? Or are
1481 you asking me whether I can make a more virulent mouse virus?

1482 Q Well, in mice, and then also, I guess,
1483 transgenic mice later.

1484 A Yeah, ultimately, the -- so I believe the
1485 biochemistry on the SHC014 spike compared to the SARS 2003
1486 spike, the SARS 2003 spike binds the human ACE2 better than
1487 SHC014. But in the mouse, the SHC014 spike binds the mouse a
1488 little better than the human. So little tweaks in ortholog
1489 receptor usage that exists within the bat population can
1490 tweak it a little bit in directions, yes.

1491 Is that answering your question? I'm hoping I'm answering
1492 your question.

1493 Mr. Romero. I think so. I will turn it to Alicia.

1494 BY MS. YASS.

1495 Q I will say, we have a cursory understanding of
1496 all the science you are talking about, so we've done our best
1497 to get up to speed on it to have this conversation with you
1498 today. I want to talk to you about something a little more
1499 10,000-foot view, not in the weeds of the science, but about,
1500 in general, zoonotic origin of a human virus, and what that
1501 would look like.

1502 We've spent a lot of time in this Committee talking about lab
1503 leak versus zoonotic origin, and I think it's good to get a
1504 sense from somebody who is doing this work day-to-day on what
1505 that would be.

1506 So for a little bit of historical context, for zoonotic jumps
1507 with coronaviruses or even other viruses in general, could
1508 you just talk a little bit about how zoonotic jumps would
1509 happen or have happened?

1510 A In the context of coronaviruses?

1511 Q Or any other viruses, if that makes it easier
1512 for you to talk about.

1513 A Well, the first thing that has to happen is
1514 that human populations have to come into close contact with
1515 animals that encode these viruses. So that's obviously the
1516 first thing.

1517 So there are, like, people in the extractive industry who may
1518 be loggers or hunters or, you know, gathers or collects
1519 bushmeat, those kind of people are the most likely to come in
1520 contact with zoonotic viruses and become infected.

1521 Now, the vast majority of contacts where zoonotic viruses
1522 actually are introduced into a human being, most of those
1523 don't progress. The recent data with coronaviruses, for
1524 example, that was published in Southeast Asia argues that
1525 there's somewhere between 50 to 60,000 exposures where people
1526 working with bats come in contact with bat coronaviruses, and

1527 actually seroconvert. That means they get infected, probably
1528 had very mild disease and recovered. 50,000. So if you
1529 think about how many -- well, let's put it in the context of
1530 coronaviruses.

1531 So 2002, SARS emerged; 2019, SARS2 emerged. That's 17 years
1532 times 50,000 exposures a year, it's actually a little higher.
1533 So about a million exposures between human disease outbreaks.
1534 So the vast majority of exposures are self-contained and do
1535 not transmit to another person, and then do not establish or
1536 colonize the new population. But this is occurring all the
1537 time.

1538 And so when you get to origins, for example, and you ask the
1539 question, what's more likely, is it a lab leak or is it
1540 natural processes? You're looking at one in a million, a
1541 million exposures occurring over 17 years versus what happens
1542 in a laboratory setting. No chance it's even close. And the
1543 diversity in nature, hundreds of millions of times more
1544 diverse than what was in the Wuhan Institute of Virology.

1545 So that gradient is huge. And if you consider that, it's
1546 more likely to be a natural event than it is to come out of
1547 the laboratory. The data -- that's what the data screams.

1548 So that's the first event, is that most of those events don't
1549 actually spread and cause severe disease or transmit. So why
1550 is that? And I can tell you better for coronaviruses. I can
1551 tell you for other viruses. But for coronaviruses, for

1552 COVID-19, there are 49 what are called susceptibility loci in
1553 humans that regulate how bad the disease is going to be.
1554 There are 25 host proteins that interact with the virus to
1555 let it replicate well. So when an animal virus is coming
1556 from a bat into a human, there's a lot of variation in those
1557 25 genes that the virus has to be able to walk through and
1558 adapt to, and it takes time and it takes mutation.
1559 Now, the starting virus can make a difference. If it has a
1560 lot of intrinsic capability to use -- and these host proteins
1561 are all kind of conserved, if many of them are conserved,
1562 it's easier for them to make it through, but most of them
1563 can't.
1564 And then there's other barriers for pathogenesis. There's a
1565 whole set of genes for pathogenesis, which is important for
1566 producing symptoms and bringing the virus up to the right
1567 part of the upper respiratory tract, so it's sneezed and
1568 transmitted. And then there's other barriers for
1569 transmission to occur. So for a sarbecovirus to make that
1570 transit, it's hard, and the data in nature support that. So
1571 other viruses face the same fate.
1572 Now, some viruses use the same receptor across species, for
1573 example, like flu. But some of those receptors in an animal
1574 are expressed in the upper respiratory tract or the gut, and
1575 in the human, it's only in the lower respiratory tract. So
1576 when H5 infects an individual, it's a horrible lower tract.

1577 respiratory infection, but it doesn't replicate in the upper
1578 respiratory tract. So that's why I don't think it can
1579 transmit, so the virus has to figure that out.

1580 And so that's why most zoonotic transmission events in nature
1581 fail. And it's the same thing in the research laboratory.
1582 When you start, like, resurrecting bat viruses, and it sounds
1583 scary, but there are huge barriers. Even if you consider
1584 that, let's say that there was no protective barriers at all,
1585 humans have a huge number of protective barriers in terms of
1586 susceptibility loci that are in place to prevent that from
1587 occurring.

1588 In addition, humans have been exposed to four contemporary
1589 coronaviruses which provide some level of cross-immunity for
1590 new viruses to come in.

1591 So it's not a simple thing like there's a virus out there,
1592 you know, that looks like Pac-Man, it's got a big smile on
1593 its face and saying, give me a human, because I'm going to
1594 eat them, and then I'm going to keep eating. It's a
1595 difficult process for most of them.

1596 But, again, the important thing to consider when you think
1597 about biosafety is that some of them may have an easier route
1598 than others, and it's the ones with the easier route that you
1599 have to be concerned about.

1600 Q We've spoken about China. You've mentioned
1601 Southeast Asia is where currently a lot of research is being

1602 done on emerging viruses. What general characteristics or
1603 traits do China and Southeast Asia have that might be ripe
1604 for these zoonotic spillovers? We know several viruses have
1605 come out of that area in the past 20, 30 years.

1606 A Well, the scientific community has stated to
1607 the Chinese government several times that open markets are
1608 conduits for virus emergence. And that's because they stack
1609 animals on top of each other, including all kinds of wild
1610 animals.

1611 And also, there's an illegal trade. I don't know, what do
1612 you call people -- I guess they're smugglers, right? People
1613 who bring -- there's smuggling of animals into China as well
1614 that are brought into these markets as well that are sold.

1615 And so you have, in essence, mixing vesicles where a large
1616 number of different viruses in different mammals are brought
1617 in close proximity. And when you think about these
1618 susceptibility loci, they're going to vary for each animal.

1619 And so some animals are going to be -- if you take a bat
1620 virus, some bat viruses, sarbecoviruses can use a rabbit and
1621 a camel and bat receptors for entry. Others use 30 different
1622 mammalian receptors for entry.

1623 So some of those viruses may be able to slip -- they get
1624 through this, they go to another species, they're
1625 replicating, they're adapting. Some of those mutations allow
1626 more cross-jumping, and these mixing vesicles provide really

1627 efficient ways for viral disease emergence. And Chinese
1628 scientists, European scientists, and American scientists said
1629 that if you don't close these open markets down, you're going
1630 to have another sarbecovirus.

1631 So if you ask me -- one question could be, what was the cause
1632 of the pandemic? It's policy failure. There's plenty of
1633 science that said, close your markets, shut down the illegal
1634 trade and smuggling of animals. Otherwise, you're going to
1635 get another sarbecovirus. And they didn't do that.

1636 It's not only China that has open markets and traffic in
1637 bushmeat. It happens in Africa and South America, many
1638 different countries. And so also in the context of huge
1639 metropolitan areas. And so in essence, human beings are
1640 creating the appropriate environment for virus emergence.
1641 And so if you look at the 21st century, we've had somewhere
1642 between eight and 12 emerging pathogens that have occurred in
1643 20 years. This is not going to slow down.

1644 Q Thinking about some of the past zoonotic
1645 spillover viruses that we've had, SARS1 and MERS
1646 specifically, from our understanding, researchers didn't
1647 immediately know the path and what animal the virus had come
1648 from. Is that your understanding as well?

1649 A Well, the research in the flu field had always
1650 argued that open markets were a good conduit for virus
1651 emergence, for mixing of influenza virus strains. So the

1652 research community that's interested in emerging viruses know
1653 that anywhere where there's going to be the interaction
1654 between large number of animals and human populations is a
1655 potential way for virus emergence to occur.

1656 So you look as a civilization moves into and deforests areas,
1657 these are boundaries where emergence occurs. Open markets
1658 are boundaries where emergence events occur. Farming
1659 practices, anything that sort of changes the ecology or
1660 causes ecologic mixing is a way for this -- what was your
1661 question again?

1662 Q When we look at a virus and are trying to
1663 figure out the zoonotic point of origin, we don't always know
1664 right away which animal it came from. It may have passed
1665 through a couple animals before it got to humans, and that
1666 path is not always immediately clear.

1667 A Yeah, so in the case of SARS coronavirus, for
1668 example, because of what I just told you, one of the first
1669 places people start looking are animals in the area where the
1670 outbreak occurred. And so in the case of the SARS
1671 coronavirus 2003 outbreak, they found that people working in
1672 the open markets had a higher seropositive rate to these
1673 viruses, as compared to people outside of that work area.
1674 And they looked in the animals in those markets, and they
1675 found virus strains that were 99.8 percent identical to the
1676 SARS coronavirus 2003 that were transmitting in civets and

1677 raccoon dogs, and it was mostly happening in the metropolitan
1678 areas.

1679 I think Zhengli Shi went back to look at the farms that were
1680 producing the animals, and very few of those farms had virus.
1681 So it was somewhere in the transportation and the bringing
1682 large numbers of animals together that they become infected
1683 and they can potentially spread it to humans.

1684 Humans also in this case, in the case of 2003, could also
1685 reinfect the civets, setting up a transmission cycle. In the
1686 case of MERS, it was a change in practice associated with
1687 camels, where large numbers of camels were moving up from
1688 eastern Africa into the Middle East and being maintained as
1689 large herds.

1690 And they became seropositive and were transmitting MERS
1691 viruses probably as early as 1990 or so, unrecognized as
1692 causing -- either they didn't cause serious disease or they
1693 were causing some level of clinical disease that was going
1694 unrecognized.

1695 Now, that doesn't mean that you need an animal reservoir,
1696 right? I think that's really important. Because I just
1697 talked to you about viruses in nature that have different
1698 intrinsic levels, you know, of being positioned to emerge,
1699 like SARS coronavirus 2019 can use 30 to 40 mammalian
1700 receptors. One of the viruses that's close to it called
1701 pangolin GD can use all those same receptors and the mouse

1702 receptor.

1703 So there are strains in nature that have that intrinsic
1704 capacity as a generalist to bind ACE2 molecules of many
1705 species. Now, they don't necessarily need to set up a
1706 reservoir. We published a paper in 2023 on this, where a
1707 virus like that could infect a pangolin. And most
1708 people -- I could hold a pangolin and get it close to my face
1709 and not freak out. I would have trouble with a bat. I don't
1710 know about the rest of you, but I would have trouble holding
1711 a bat close.

1712 So a pass-through species is where a bat may infect another
1713 species, because the receptors in many of these barriers have
1714 been naturally circumvented. Then that virus is brought in
1715 close contact to a human. And if it's the right human, who
1716 has the right combination of susceptibility loci that make
1717 them more likely to be infected, or if they're elderly, or if
1718 they're partially immunosuppressed, all of these functions
1719 could allow the virus to infect that person and begin to
1720 replicate and adapt.

1721 And especially if they're immunosuppressed, because it
1722 doesn't clear, and that gives the virus plenty of time to
1723 make mutations and then transmit to another person.

1724 So in the case of SARS-CoV-2, large herds of pangolins don't
1725 exist. It's an endangered species. But the concept of one
1726 species acting, in essence, as a pass-through species is

1727 certainly possible. And I think it was one individual that
1728 infected some of the mink colonies in Europe, and exactly how
1729 the virus jumped from humans to deer is also open. And then
1730 deer back to humans is open.

1731 So again, this clade, which is called 1B that's
1732 SARS2-related, at least the viruses within the first 13 or 14
1733 of them that had ever been identified that are the closest
1734 thing to the SARS2, all from Southeast Asia. So if you hear,
1735 like, the virus came from somewhere else. No, it came from
1736 Southeast Asia. But all -- many of them have this feature of
1737 more of a generalist capacity. So the second possibility is
1738 pass-through.

1739 Q Sure. And just to be clear that I understand
1740 some of what you just said, it sounds like even though, for
1741 some of the example viruses, there's very clear evidence on
1742 pieces of the transmission of the virus, the entirety of the
1743 path is not always 100 percent settled?

1744 A That's correct.

1745 Q And when we're looking at the SARS-CoV-2 or
1746 COVID-19 pandemic, it sounds like you feel strongly that it
1747 was a zoonotic or natural origin. But would you say that
1748 it's not settled yet what the origin of the COVID-19 pandemic
1749 was?

1750 A Again, I have at different times speculated on
1751 three possibilities. The first is natural origin. The

1752 second is accidental escape from the laboratory setting,
1753 which can also include collection, which you can ask about if
1754 you'd like more details on that. And then the third would be
1755 the possibility of engineering.

1756 There is no hard evidence to support engineering. Initially,
1757 for example, the receptor binding domain was argued to be
1758 completely unique and perfectly positioned, perfectly
1759 designed to bind the human ACE2 receptor. Well, no, there
1760 are virtually identical strains in bat strains that are found
1761 in nature. So it's not been engineered.

1762 In addition, that spike gene has undergone successive sets
1763 of -- the RBD has gone successive adaptive changes that
1764 increases bind infinity for the ACE2 over a thousand fold.
1765 It is not perfectly designed. It's just like the origin
1766 SARS1, which underwent specific changes that enhanced its
1767 transmissibility as it was spreading. The exact same
1768 process. So the RBD is out.

1769 The second idea that it was engineered, there was a very bad
1770 bioinformatic paper, for example, that said -- it came from
1771 the HIV -- which was total nonsense.

1772 The better argument was that there might be a super antigen
1773 site, but there was a paper that was just published that
1774 said, no, there's no super antigen site. So, in essence, the
1775 scientific process says, okay, if this is the hypothesis,
1776 let's do experiments to see if we can disprove it. If we

1777 can't disprove it, then it's likely.

1778 So far there's no backbone genome that's close enough to have
1779 been engineered in the SARS2. Most of the components that
1780 were originally argued as being engineered failed. The only
1781 one that's left is the furin cleavage site, which has
1782 multiple explanations.

1783 So that leaves two possibilities. The first is escape from
1784 the laboratory. And you can't rule that out, because they do
1785 work at BSL-2. You just can't. But for the reasons I talked
1786 about earlier, just on the frequency and the exposure level
1787 in nature versus lab, it's massively -- what's that called,
1788 massive -- the scales are massively weighted to natural
1789 origins, yes, sorry.

1790 Q Sure. And taking out bioengineered, I think
1791 there's much consensus that that is not what we're looking at
1792 here. But with the lab leak and zoonotic, there would be
1793 possibilities for it to be somewhat more of a combination of
1794 the two. I'm thinking about, specifically, you said
1795 researchers go out and collect samples, they bring them back
1796 to the lab. Maybe they do no manipulation on it, so it's
1797 just whatever they collected out in nature. Something
1798 happens, there's a lab accident, and somebody is exposed to a
1799 virus and gets infected.

1800 While I understand this would be very rare, that would sort
1801 of be a combo of a lab accident with a natural virus,

1802 correct?

1803 A Yes, and still be a natural virus that

1804 inadvertently escaped the laboratory, because biosafety

1805 practices weren't sufficiently robust.

1806 Now, when you think about collection, at least the group at

1807 EcoHealth and the groups that they collaborate with, again, I

1808 haven't been in the cave with them, but the pictures that I

1809 have seen is they're fully dressed in Tyvek suits and with

1810 all the protective gear. So, in essence, they are

1811 collecting -- in essence, in laboratory appropriate

1812 conditions, and then bringing the samples back.

1813 Their weakness is trying to culture the viruses at BSL-2.

1814 It's just the chance of an accident is increased under BSL-2

1815 conditions, as compared to BSL-3.

1816 Q And I wasn't suggesting that this is what

1817 happened, just more that it's a possibility.

1818 One of the things that our Select Subcommittee is focused on

1819 is preventing the next pandemic, because, as you've said and

1820 as we're all aware, another pandemic does seem like a

1821 distinct possibility in the future. So we want to be

1822 learning lessons from this most recent pandemic to bring

1823 forward.

1824 You've talked about some policy ideas that were brought to

1825 China on ways to limit exposure to viruses, but are there

1826 other policy solutions that you think we should be

1827 considering to better prepare us for the next pandemic?

1828 A BSL-4 laboratory practices are well harmonized
1829 across the globe. BSL-3 practices are not well harmonized
1830 across the globe. And so there's quite an amount of
1831 variation that exists within BSL-3 laboratories from -- I
1832 don't know, from like conditions that I just described in our
1833 laboratory compared to the minimal conditions, which,
1834 depending on the pathogen, can actually be a lab coat and
1835 goggles, some sort of eye protective gear and gloves. And so
1836 that would be for a non-respiratory transmitted virus that
1837 may require bloodborne transmission or something like that.
1838 But different countries have different standards for how they
1839 work with pathogens. And it's not just China, for example.
1840 And so it would be good if, globally, there was a
1841 standardized set. There are other nations that also say they
1842 have BSL-3 facilities that do this work, where I would look
1843 at it and go, I don't want to do BSL-3 work in that facility,
1844 just because the standards aren't sufficiently high.
1845 I had another thought, too, that has now escaped me. Doggone
1846 it.

1847 Q Well, if I could just summarize that. I think
1848 we all know the virus doesn't know nations' borders, and can
1849 easily go across borders. And research is being done in
1850 these different countries, so it sounds like international
1851 cooperation and collaboration is key to preventing the next

1852 pandemic.

1853 A Yes, I would also, I guess, like to make the
1854 statement that regulation -- I actually have no problem with
1855 the current GOF or DURC regulations. I think they're
1856 appropriate, they're focused on pathogens of potential high
1857 consequence that we have a risk, that we know about risk.
1858 I have concerns about regulations that cover all of
1859 microbiology, for example. And my concerns are related to
1860 leadership. Leadership in terms of the scientific
1861 capabilities, leadership in terms of economic leadership.
1862 The bio-ag community, for example, is a multi-trillion dollar
1863 community, which may be the major economic driver of the end
1864 of the 21st century. And if we overregulate and put too much
1865 regulatory restrictions on that community, we will lose that
1866 economic battle.
1867 In addition, doing high containment research actually spurs
1868 the development of safer practices and safer facilities and
1869 safer equipment for biosafety work at a higher containment.
1870 So if you restrict it so much that very few people do it,
1871 those kind of advancements won't occur and will stagnate the
1872 system. And then I think there's biosecurity in terms of
1873 preparedness. What are the capabilities, what do you look
1874 for?
1875 So over-excessive regulatory restrictions on emerging
1876 pathogens or high containment research can be equally

1877 disastrous to the U.S. in the future. So there's a
1878 risk-benefit ratio. And if that risk-benefit ratio is wrong,
1879 the risk to the competitiveness of the United States could be
1880 impacted more than the benefit that would ever occur from the
1881 restrictions. And, unfortunately, you guys have to figure
1882 that out. I don't have to figure that out, but you guys have
1883 to figure it out.

1884 Q We appreciate your view on that. And one
1885 point of clarification. Early in that answer, you referenced
1886 the current GOF regulations. I assume you're referring to
1887 the current gain of function regulations, which are the P3C0
1888 framework; is that correct?

1889 A The P3C0 framework is designed around -- is
1890 specifically gain-of-function research related to viruses
1891 that are considered PPP. Those are viruses that either have
1892 the potential for high transmissibility in humans or high
1893 pathogenic outcomes in humans. And so it's a limited number
1894 of viruses that fall within that sphere. So for example,
1895 natural pathogens like zoonotic pathogens, at least my
1896 reading of the regulation, they don't fall within that
1897 category.

1898 If you're looking for -- if you're looking at -- if you're
1899 designing like mouse-adapted viruses, as was asked earlier,
1900 so that you can make better universal vaccines or test the
1901 breadth of drugs, those are exempt. If you're doing it to

1902 identify strains that are high risk, those are exempt under
1903 the current regulations.
1904 I'm talking about the harmonized regulations that are being
1905 discussed now, or the DURC regulations are mixed with the
1906 gain-of-function regulations, and currently, it's being
1907 considered that any animal, human, or plant pathogen or agent
1908 be under review.
1909 Now, the definition of agent is not defined, so the agent is
1910 someone or something that has an effect. AI has an effect,
1911 right? Biochemistry studies to identify what escape
1912 mutations can occur in a virus provides information that
1913 could be used as dual use. It has an effect. mRNA vaccines
1914 elicit an immune response, it has an effect. It can be used
1915 to deliver things to human hosts in a positive or negative
1916 manner. It has an effect.
1917 So you have these huge economic engines, CRISPR technology,
1918 and fixing genetic disorders that is coming head-on with
1919 these regulations. And the economic impact of that could be
1920 huge. Again, that's not my areas of expertise, it's your
1921 guys' area of expertise.
1922 I just hope you're aware that this is not insignificant, and
1923 in the harmonized regulations, they don't discuss the
1924 long-term impact of the regulatory structure. Like I said, I
1925 have abided by the regulatory structure to the best of my
1926 ability. I think the regulations are appropriate, especially

1927 early on with the coronaviruses. There were no drugs, there
1928 were no vaccines, there were no therapeutics. I mean, the
1929 human population was completely vulnerable, so we needed to
1930 have that in place.

1931 But remember how difficult it is for a zoonotic virus to move
1932 into a human. Most of the cases of laboratory escape that
1933 have led to transmission, these are human pathogens that were
1934 in the lab that already knew how to transmit. I don't know
1935 of any cases where a zoonotic virus immediately -- you know,
1936 they could infect somebody. But they're subclinical
1937 infections, they don't spread. At least to date.

1938 Again, it's not -- it's a balance. If you ask me whether
1939 that could never happen, well, of course it could happen.
1940 There's a risk there. And, again, governments around the
1941 world have to deal with that risk capability, and try to
1942 balance it as carefully as they can. And it could easily go
1943 in either direction in a disastrous way.

1944 Q Thank you for that context. I am going to
1945 change topics here, and I want to draw your attention to
1946 something that was briefly mentioned in the first hour, but
1947 the DEFUSE DARPA application.

1948 So on that grant proposal, you were not the leader of that
1949 team, correct, you were listed under other team members?

1950 A I was a coinvestigator, I was not the lead.

1951 Q Thank you. So there was a draft proposal that.

1952 was submitted amongst the team members, and you received that
1953 draft, correct?

1954 A Yes, I probably got a couple of drafts at
1955 various times.

1956 Q There is one draft that has been made public,
1957 so I'm just going to introduce that as Minority Exhibit B.

1958 (Minority Exhibit B was
1959 identified for the record.)

1960 BY MS. YASS.

1961 Q Does this look familiar to you?

1962 A Unfortunately, yes.

1963 Q Now, a lot of hay has been made out of this
1964 draft proposal. And specifically, there is a comment that
1965 you made, which, unfortunately, there are not page numbers.
1966 But if you count through one, two, three -- the fourth front
1967 page that is double-sided, there's a comment from you -- or
1968 that's been attributed to you. So I will make sure that is
1969 actually you. But on the very bottom, there's a comment that
1970 is identified as BRS17. Was that your comment?

1971 Mr. Ervin. You mean 7?

1972 The Witness. This comment 7 or 8?

1973 BY MS. YASS.

1974 Q It's identified "Commented," and then in
1975 brackets, "[BRS17]."

1976 A In the U.S.; is that correct?

1977 Q Yes, correct.

1978 A Yes.

1979 Q Is that your comment?

1980 A Yes.

1981 Q So I'm just going to read it.

1982 "In the US, these recombinant SARS CoV are studied under

1983 BSL3, not BSL2, especially important for those that are able

1984 to bind and replicate in primary human cells.

1985 "In China, might be growing these viruses under BSL-2. US

1986 researchers will likely freak out."

1987 Now, when I read that comment, I take it as advice against

1988 doing this work in a BSL-2, when it should be done in a BSL-3

1989 lab. Is that what you meant by the comment?

1990 A I think I'm responding to the comment above

1991 from Peter Daszak in two ways. First, I'm informing him,

1992 just in case he doesn't know, that a lot of the virus

1993 discovery work and culturing work that the Chinese do with

1994 zoonotic coronaviruses is done at BSL-2. The animal work

1995 they do is actually at their BSL-3, but the culturing is at

1996 BSL-2.

1997 And that while there aren't any actual U.S. regulations, but

1998 the Baric lab does this all under BSL-3. So anyone who had

1999 collaborated with us or had obtained the viruses from us

2000 always did it at BSL-3. And all of our paperwork said we're

2001 going to do it at BSL-3.

2002 So I'm letting him know there's a difference, and I say, "US
2003 researchers will likely freak out" to make sure he pays
2004 attention.

2005 Q Great. And this was not the final proposal
2006 that was submitted, correct?

2007 A I don't believe so, no.

2008 Q And that final proposal was finalized by
2009 EcoHealth Alliance, not you, correct?

2010 A I did not see the final proposal that went in.
2011 I made comments on it, but the final proposal, I didn't
2012 receive until after it had been submitted.

2013 Q And to be clear, that final proposal was not
2014 accepted by DARPA, correct, it was not funded?

2015 A That's correct.

2016 Q Dr. Daszak made a comment on the draft
2017 proposal as well, and suggests the one you mentioned,
2018 beginning with, "If we win this contract, I do not propose
2019 that all of this work will necessarily be conducted by
2020 Ralph." That was your point of concern?

2021 A Yes.

2022 Q But he was saying, "If we win this contract,"
2023 correct?

2024 A "If," yes.

2025 Q And the contract was not awarded?

2026 A That's correct.

2027 Q And as far as you know, the research that was
2028 outlined in this proposal has not been conducted through
2029 funding of other means?

2030 A Certainly not by my group. I don't know what
2031 China did, and I don't know what their grant funding was
2032 subsequent to this grant.

2033 So there was no evidence that they were doing this kind of
2034 work. Well, there was evidence that they were building
2035 chimeras using WIV1 as a backbone, so they were doing some
2036 discovery work about the functions of spike genes of zoonotic
2037 strains that they discovered later on, but I don't know if
2038 they did any of the engineering or anything.

2039 Q Because you had not been involved in any of
2040 that work?

2041 A I had not been involved, no.

2042 Q We've had heard others say that SARS-CoV-2 is
2043 the only virus in its subgenus with a furin cleavage site,
2044 although if you go one level above, there are other viruses
2045 with the furin cleavage in the genus. The DEFUSE proposal
2046 included inserting a furin cleavage site at the S1/S2
2047 juncture. So just a discrete question about that. Are S1/S2
2048 furin cleavage sites found in other coronaviruses in nature?

2049 A They're found in many betacoronaviruses and
2050 some alphacoronaviruses, yes.

2051 Ms. Yass. Thank you, Dr. Baric. We can go off the record.

2052 (Recess.)

2053 Mr. Benzine. We can go back on the record.

2054 BY MR. WENSTRUP.

2055 Q Dr. Baric, is it possible that SARS-CoV-2
2056 spent some of its life in the lab before the pandemic took
2057 off, even if it was brought into the lab from nature? Let me
2058 ask you this. Is there a way to find out? In other words,
2059 I'm thinking of, like, lab notebooks and documented
2060 sequences. Should that be possible?

2061 A If you had access to the laboratory notebooks,
2062 if you had access to the safety records of the Wuhan
2063 Institute of Virology, if you had access to the sequence
2064 databases, the level of assurance that you would have would
2065 be greater. No question.

2066 Q Which we didn't really have?

2067 A Which we don't really have, that's very true.

2068 Q And again, this is like going through a
2069 process, but -- so the sequences, they come from the lab,
2070 that's where the sequence is read, if you will, and maybe
2071 that's not be the right word.

2072 A Well, so many of them are collected in nature.
2073 They may collect it in inactivating chemicals so they
2074 maintain it as RNA. So I don't know how they actually break
2075 it down. So what they might do is half the samples may be
2076 nucleic acid, the other half may be a guano that would have

2077 live viruses.

2078 Q But there are data banks?

2079 A They would probably have --

2080 Q Whether it's found in nature, developed in a
2081 lab, they should be in the data bank, right?

2082 A It depends. Sorry to be -- but the problem is
2083 you have a certain level of depth that you can get at with
2084 sequencing that typically isn't going to capture everything.
2085 If they have 100 bats, it's not going to get everything in
2086 it.

2087 The second problem is, the way they normally culture viruses
2088 is they will pull samples, guano samples from 10 or 20 bats
2089 which they haven't gotten a full sequence on. And in the
2090 cell culture system, you could have what's -- a process
2091 called recombination, or it's kind of like the way viruses
2092 have sex with part of the genome, where one virus would
2093 joined to the other. And those wouldn't have been in the
2094 database, but you would have seen sequence signatures that
2095 something came -- was a recombinant that had information --

2096 Q Here's where I'm going. SARS-CoV-2, that was
2097 sequenced from human clinical samples in December of 2019,
2098 January of 2020. But if you later found in a previous data
2099 bank of sequences where there's maybe thousands, if you found
2100 that same sequence, it would imply that it was in the lab at
2101 some point?

2102 A That's correct. If it was in their sequence
2103 database and they sequenced it, it would have been in one of
2104 their samples. Now, whether they would have recognized it as
2105 being a thing of concern or not is a whole other question,
2106 because you're looking at potentially millions of sequences.

2107 Q I'm thinking you've got the sequence from the
2108 human. Can you do a Google search and see what's in the
2109 databank?

2110 A As soon as they had the sequence in humans,
2111 the Chinese had to have done a blast search to ask in the
2112 repository of sequences that the Wuhan Institute of Virology
2113 had, was it there or not.

2114 Q But we don't know that answer?

2115 A That's true, we do not.

2116 Q But normally, here, for example, you can track
2117 that, and when was it put in, who put it in?

2118 A That's correct.

2119 Q That answers my question. On to another
2120 topic. Do you now or did you have a security clearance at
2121 any time?

2122 A Let me ask a question. Is security
2123 clearances, is that kind of stuff -- is that --

2124 Q Top secret?

2125 A -- under security rules or not? If I have a
2126 security clearance, am I allowed to say that?

[REDACTED]

2193 BY MR. WENSTRUP.

2194 Q So I look at the advisory board -- and I'm not
2195 sure if that's the right name -- at NIH that reviews grants.
2196 And as Dr. Fauci said, once they're done reviewing it and
2197 they're okay, I just sign them. That's what he said. So I'm
2198 concerned, and if we're doing something in a foreign lab, are
2199 the people on the advisory board aware of the risks?

2200 A This is the NIH advisory board?

2201 Q Yes. And maybe you don't know, but I'm

2202 curious.

2203 A I've never been on those. They
2204 have -- basically, there's a review panel that will review
2205 them, and it will be scientists made up from across the
2206 country. Now, they may raise the issue that the expertise
2207 may or may not be available, especially if they feel that
2208 there's gain of function or DIRC related concerns. They may
2209 raise the issue, and then that would immediately go to the
2210 program officer.

2211 If they don't and the program officer, who is supposed to
2212 read the grant, reads the grant and sees an issue, they will
2213 flag it. And through either of those processes, I guess
2214 there's some kind of discussion that probably occurs in
2215 between.

2216 Q Yeah.

2217 A They will then notify the PI of the grant that
2218 there's some concerns related to -- and there's some concerns
2219 related to this grant that need to be addressed. So, for
2220 example, like on the grants where they may have looked at
2221 my -- they were concerned about gain-of-function research,
2222 they would then list what experimental protocols they were
2223 concerned about and may ask you to address it.

2224 Q My concern is, if they're the ones doing that,
2225 what they don't know, they don't know, the advisory board
2226 people. So they can't express concerns if they're not aware

2227 of what the concerns are about that lab. And I'm not just
2228 talking about China. It could be anywhere.

2229 A Yeah.

2230 Q So my concern -- I think my feeling is -- if
2231 we're going to do something in a foreign lab, there should be
2232 somebody on there that has that background.

2233 A To support what you just said, the
2234 transmissible flu work that was done by the Dutch, there was
2235 some concern about whether NIH should fund that lab. And
2236 they put in -- they then requested that they do all kinds of
2237 additional biosafety and stuff for the facility before they
2238 funded it. We're buddies with Europe.

2239 Q Yeah.

2240 A It's a fair question to ask whether, you know,
2241 if a nation state says it's going to accept U.S. money, there
2242 should probably be some kind of upfront agreement about being
2243 able to -- especially if it touches on any kind of sensitive
2244 subject.

2245 Q From the intelligence side, too. If you're
2246 getting a grant in an adversarial nation, does that grant
2247 come with some warnings before you go there? That's where
2248 I'm going.

2249 A But again, just to clarify, in this case, in
2250 the case of the EcoHealth grant, they were proposing to do
2251 work with zoonotic viruses that were not subject to the

2252 gain-of-function regulations. In other words, they weren't
2253 increasing -- they weren't working with PPPs. Those are
2254 strains that they knew were highly pathogenic or
2255 transmissible.
2256 They were working with zoonotic viruses that were not well
2257 characterized. So there's some inherent risk there, but it
2258 may not have triggered everything going up from the NIH,
2259 because it didn't make those regulations.
2260 Personally, I think it would have been in everyone's interest
2261 to look at that more carefully. But there are gray areas in
2262 regulatory science that things slip through, so, yeah.

2263 Q And that's my concern. That's where I'm
2264 going.

2265 A It's a fair concern.

2266 Q Thank you.

2267 A I don't disagree with it. I think it's a fair
2268 concern.

2269 Mr. Wenstrup. Thank you.

2270 BY MR. BENZINE.

2271 Q I want to talk about the Wuhan Institute, and
2272 any knowledge that you may have had. You made a comment, I
2273 think it was in the hour before lunch, that a lot of the work
2274 happens at BSL-2, but the animal work happens at BSL-3.

2275 A That's correct.

2276 Q How do you know that?

2277 A Their regulations state pretty clearly that
2278 they don't consider culturing bat viruses at BSL-2 as a
2279 biosafety concern. I also had that verbally confirmed by
2280 Zhengli Shi at a meeting in Harbin, when I was telling her
2281 she should move it all to BSL-3, and the reasons why. So I
2282 know that. And she also in that meeting said that all animal
2283 work is done at BSL-3.

2284 So I think the news reports also talk about -- and I don't
2285 know this, don't know the details again, but I thought the
2286 news reports said that there was big biosafety discussions
2287 sometime in October and November about whether they should
2288 change their regulations.

2289 I will note, you probably don't know this, we worked with a
2290 swine pathogen called severe acute diarrhea syndrome
2291 coronavirus, which was causing 99 percent lethal outbreaks in
2292 China. So we synthetically resurrected that virus and
2293 studied its biology, showed that it could grow in human
2294 cells, not very well, but it could grow in human cells,
2295 especially human enteric cells. And we wrote in that paper
2296 that all work on this should be done at BSL-3.

2297 The Chinese have been working on it at BSL-2 labs. And in
2298 2012, we had a virus called porcine epidemic diarrhea virus
2299 sweep through the country and kill millions of pigs.

2300 Ultimately, because of that paper, I have heard that they've
2301 moved all their SADS research to BSL-3.

2302 So in that particular instance, I think it's an example of
2303 where science done in one country can sometimes have a really
2304 positive impact on another country.

2305 Q I want to introduce what will be Majority
2306 Exhibit 1.

2307 (Majority Exhibit No. 1 was
2308 identified for the record.)

2309 BY MR. BENZINE.

[REDACTED]

[REDACTED] -- pursuant to a statute
2322 passed by the House, the Office of Director of National
2323 Intelligence had to release a report on specific intelligence
2324 they had on what the Wuhan Institute was doing, and what
2325 their capabilities were. I just want to read some passage
2326 from it, and ask if you have any personal knowledge of it.

2327 And for now, yes or no is good. And we can figure out, if
2328 yes, if we need to go any further.

2329 The ODNI assessed that WIV personnel have worked with
2330 scientists associated with the PLA. Do you have any
2331 knowledge of that?

2332 A I wouldn't know whether a Chinese scientist
2333 was a member of the PLA or whether they were -- unless they
2334 cleared -- unless they said it directly, and then, for
2335 whatever reason, I remembered.

2336 Most of the time, the times I've gone to China and seen a lot
2337 of Chinese scientists were a couple years apart, so there's
2338 no memory. Except for Zhengli Shi and George Gao, and more
2339 visible ones that traveled a lot. I can't remember them from
2340 one meeting to the next.

2341 Q ODNI also said -- and this kind of tracks what
2342 we've been talking about -- that the WIV first possessed
2343 SARS-CoV-2 in late December 2019. Is that kind of consistent
2344 with your understanding, that they at least had the sequence
2345 in late December?

2346 A It would be shocking to me if they did not
2347 have the sequence before January 1st. And I have seen -- I
2348 think it was Jerry Farrar's book, Jump, where I think there's
2349 a note between him and the evolutionary biologist out of
2350 Australia --

2351 Q Dr. Holmes?

2352 A Dr. Holmes, thank you. I have a problem with
2353 names -- noting that the Beijing -- I didn't see this until
2354 that thing came out, that the Beijing sequencing company had
2355 sequenced it on the 27th.

2356 But it makes sense to me. And it would also make sense to me
2357 that 23 days before that, they must have had PCR confirmation
2358 that it was a sarbecovirus. So I would say they had probably
2359 had enough sequence information to know it was a new
2360 coronavirus, maybe a sarbecovirus, before Christmas.

2361 Q So that goes to my next question. I was going
2362 to read that passage, so I'm glad that you've already seen
2363 Dr. Farrar's book.

2364 But you've told us, Dr. Daszak has told us, Dr. Farrar
2365 accounted in the book, ODNI said that China knew that this
2366 was a coronavirus by late December.

2367 A Yes.

2368 Q The dates can fluctuate, but they reported it
2369 as an undiagnosed pneumonia. Does that concern you, that
2370 they knew what it was, and didn't report it as such?

2371 A You just asked a political question. And so
2372 the political question is where countries around the world
2373 and the leadership in countries around the world, how
2374 transparent do they want to be and how quickly do they want
2375 to be transparent? And there are some scientific questions.
2376 The first question is, if they had one sequence, they might

2377 want to get a second one to confirm it before they announce
2378 it. That would be a logical thing to do.

2379 Number two, you have to think about it, you can't -- it's not
2380 appropriate to think about it in the scale of the pandemic
2381 that eventually happened. You have to think about it as
2382 where things were in December, late December. In which case,
2383 they -- well, at least they claimed they had no evidence that
2384 it was highly transmissible.

2385 And if you follow their literature, the first real case that
2386 they tracked for transmissibility, the exposure occurred on
2387 the 31st in one hospital, relatives flew in to see them, I
2388 think on the 1st, and then flew home on the 2nd. And then
2389 two or three of them became infected. And that ended up
2390 being the first report of transmissibility, which I think was
2391 published, I don't know, late January or somewhere in
2392 January.

2393 So in the interim of finding out the sequence, it would make
2394 sense for a government to want to confirm it at least within
2395 a second patient, because it could be that a second patient
2396 gives you a totally different sequence than which one's
2397 causing the pandemic. A fair question to ask.

2398 So I would expect some hesitation. I would also expect the
2399 Chinese government to be very sensitive about wanting to
2400 report that it was a SARS-related virus, especially if they
2401 didn't think it was transmissible.

2402 So it's unfortunate it was delayed. I'm not sure
2403 that -- it's harder for me to say what would happen in other
2404 governments around the world. In fact, you guys would
2405 probably know better than I would how quickly the CDC, if
2406 they found a new virus that looked like it was highly
2407 transmissible, would they report it immediately or would they
2408 call the State Department and warn and talk to Congress and
2409 the President first.

2410 You would think there would be almost some kind of -- you
2411 don't want the President or the leadership of the House or
2412 Senate to come out and say, what? You don't want to have
2413 them ask "what" to a reporter, I hadn't heard about it.
2414 So there's going to be some time there, but certainly by the
2415 beginning of January, they probably would have had the
2416 information.

2417 BY MR. WENSTRUP.

2418 Q So I was in Vietnam. Our CDC there did
2419 really, I think, good work in Vietnam to help Vietnam. We
2420 have a CDC representative in China. Any thoughts on whether
2421 that person was engaged or not early on?

2422 A I don't know whether the U.S. CDC
2423 representative -- are they in Beijing or Wuhan? Where are
2424 they?

2425 Q I think Beijing.

2426 A One of the problems with that sort of

2427 autocracy is the regional areas, if I understand correctly,
2428 the regional areas in China don't want to report they have
2429 got a problem to the higher levels. So I would guess that
2430 they were hesitant to pass it up the chain just because of
2431 the structure of their government.

2432 Q Or involve the U.S.?

2433 A Or definitely involve any other countries.

2434 Not just the U.S., but any other countries.

2435 BY MR. BENZINE.

2436 Q ODNI also reported that the WIV has created
2437 chimeras and SARS-like coronaviruses, and had the capability
2438 to use techniques that could make it difficult to detect.
2439 Intentional changes. We kind of talked about that.

2440 In your work with them, did you understand that they had that
2441 capability?

2442 A They use baculoviruses, and their molecular
2443 clone is a virus called WIV1, which I don't think they
2444 engineered with class IIS restriction enzymes that don't
2445 leave any sequence. So I think there's a sequence signature
2446 in that virus. I would have to go back and reread the paper.

2447 Q Okay.

2448 A But in general, yes, they had the technology
2449 to do it, but it would have -- they had -- they really
2450 struggled with trying to develop other molecular clones, like
2451 they were working on developing the SADS molecular clone from

2452 2016 on, and they failed. It's not easy technology. So we
2453 started three years later and beat them to press, just to
2454 show you. And I had no interest in teaching them how to do
2455 it faster, either.

2456 Q That was going to be my next question. Did
2457 you have any -- did you teach them any of the intentional or
2458 hard-to-track change techniques?

2459 A The only person that I ever really worked with
2460 on a molecular clone was George Gao, and this was prior to
2461 the 2020 SARS2 pandemic virus.

2462 If you remember, MERS coronavirus transmitted from the Middle
2463 East to Korea and infected a lot of Korean

2464 scientists -- sorry, citizens. One of those was a Chinese
2465 citizen who moved back to China and traveled back to Beijing
2466 and infected -- that they sequenced the virus from. And they
2467 couldn't culture it. So he asked me if I would be willing to
2468 help make a molecular clone for that virus.

2469 So we designed -- we worked with him -- actually, we reviewed
2470 their design, and so they tried to make a molecular clone.

2471 They failed. Ultimately, they never got it to work. They
2472 sent the clone to us. This was around 2016. We actually
2473 recovered the virus, it's still sitting in my lab. When I
2474 told them we have the virus, he never answered me, and so
2475 it's still sitting in my lab, and I've never used it.

2476 Q The last major point that ODNI states is that

2477 there were Wuhan Institute researchers that were ill in the
2478 fall of 2019. The illness doesn't necessarily support or
2479 refute either hypothesis or prove that it came from a lab.
2480 Did you have any awareness of any Wuhan Institute researchers
2481 being sick in the fall of 2019?

2482 A I've heard this report, but I'm not -- and
2483 I've heard that they've been named, but I haven't actually
2484 seen any of the data that supports that. So I don't know how
2485 authentic it is. I mean, there's, what, 5, 600 people who
2486 work in the Wuhan Institute of Virology. I don't know the
2487 full number, but -- and there was flu going on at the time,
2488 so it wouldn't surprise me if they got sick.
2489 And I believe they -- if they're just getting physicals, they
2490 go to the hospital. So that's their medical care system. So
2491 looking at it from that point of view, that doesn't tell me
2492 anything.

2493 Q Okay.

2494 A I will also note one other thing. If you look
2495 at the molecular clock of the virus, it emerged in the middle
2496 of October, late October, not the middle or end of November.
2497 So people who say that those were the first cases, no chance.
2498 There were five or six transmission cycles at least before
2499 they would have been infected.

2500 BY MR. STROM:

2501 Q Is there -- and I think everyone who has sat

2502 through one of these things is going to roll their eyes,
2503 because I ask this in about every single one of them.

2504 A I haven't sat through one of these, so I get
2505 to roll my eyes.

2506 Q You're welcome to do it. It won't be
2507 reflected in the transcript.

2508 A That's right.

2509 Q The 177 official WHO China corona reported
2510 cases, if you put the molecular clock to mid-October, then
2511 all of the activities around that -- the market in Wuhan is
2512 actually two months or so?

2513 A It's a major problem with that Wuhan
2514 study -- that market study, yes.

2515 Q Can you just elaborate on that a little bit?
2516 I don't have the expertise.

2517 A Okay, so keep it in context. The context is,
2518 what do you have data for?

2519 Q Sure.

2520 A And the only thing we have really solid data
2521 is that the market was the site of amplification in late
2522 December, January. That's still two months from the origin
2523 date, based on a molecular clock, which means it was
2524 circulating somewhere before it got there. And the question
2525 is, where was it?

2526 Q To that point, I guess without getting too far

2527 away from our next set of questions, how hard -- you're
2528 talking about several hundred, if not several thousand human
2529 cases by the time you're getting into January -- early
2530 January, late December?

2531 A Remember that 90 percent of those cases are
2532 asymptomatic.

2533 Q Right.

2534 A 85, 90 percent. So imagine trying to chase a
2535 transmission cycle.

2536 Q Yeah.

2537 A Early cases are almost impossible, because
2538 most -- many asymptomatics are in the middle of it. So now
2539 you have a case here and a case here, but they're actually
2540 truly linked by someone in the middle.

2541 Q Who just walked around with it.

2542 A Yeah. And you can't unravel that transmission
2543 cycle until you do deep sequencing on both of them. And then
2544 you look for SNPs, and you can say, this patient is linked to
2545 this patient. It had to go through somebody else because
2546 there's another marker.

2547 So all that -- so it's a fundamental problem with the papers
2548 that are reported to prove -- they write it too strong, I
2549 think, but they're very passionate about their data.
2550 And to be fair to them, it is the best data that's out there,
2551 that they can't -- they don't have the early cases. What

2552 they have, they have the cluster in the market and they have
2553 two SNPs, which they argue are indicative of two different
2554 zoonotic introductions, which other people argue with. It's
2555 one nucleotide that's making that call, so it's -- it
2556 actually claimed there were two independent introductions.

2557 Q And they had some --

2558 A It's a stretch. It's a stretch. There are a
2559 lot of virologists that look at that data and go, mmm.

2560 Q Because it looks like, as I understand those
2561 two differences between the two lineages, it's one looks
2562 marginally more like an ancestral bat virus?

2563 A Yes.

2564 Q And one looks a little more humanized?

2565 A At one nucleotide level. And they don't know
2566 what the ancestral bat virus really was.

2567 Q Sure.

2568 A So from my perspective, clearly, the open
2569 market was a conduit for expansion of the disease. Is that
2570 where it started? I don't think so.

2571 Q Keeping in mind the Chinese government's
2572 ability to cover things up, is it at all worrisome to you or
2573 notable to you that we don't have a second market or a third
2574 market or additional lineages coming out of nearby cities,
2575 like we saw with SARS1, where you had sort of a wave of
2576 spillover into the human population?

2577 A Remember that the Chinese Health Minister, I
2578 think on like the 24th of January, said community spread was
2579 rampant and asymptomatic spread was rampant. And they
2580 quarantined.

2581 Q A lot of people.

2582 A Within a few days of that, they quarantined 65
2583 million. They came in and cleaned the market in Wuhan on,
2584 like, the 30th of December. What I don't know is whether
2585 they went to every other market in Wuhan and other
2586 surrounding large metropolitan areas, or when they found
2587 them, they just wiped out -- they cleaned those out. I don't
2588 think -- I don't have any information on it. I don't know if
2589 you have any information on it.

2590 Q Not that we've seen.

2591 BY MR. BENZINE.

2592 Q The last kind WIV-specific question. The
2593 Chairman brought up about the importance of databases, and
2594 you concurred that if you did a blast search, that it would
2595 be kind of common practice for someone to do a blast search
2596 of the sequence to see if it was in there?

2597 A They had to have done a blast search.

2598 Q It was reported that the WIV database went
2599 offline in September of 2019, and was no longer public, at
2600 least publicly accessible?

2601 A That's what I've heard, yes.

2602 Q Do you have any other knowledge of that, or
2603 just based off the public report?

2604 A I think the rumors that I heard was that they
2605 were -- they shut it down because they were getting hacked.

2606 Q You just put the --

2607 BY MR. STROM.

2608 Q But you didn't talk to Zhengli Shi about it?

2609 A No, I didn't know until it was reported.

2610 Q You mentioned WIV1. Do you know if the WIV
2611 had access to additional backbones or unpublished full-length
2612 virus?

2613 A I'm sure they were working on other
2614 full-length molecular clones. But the ones that they
2615 published -- they were having trouble with it, because the
2616 ones that they published, they were taking the spike gene and
2617 dropping it into the backbone.

2618 One of the problems with sarbecoviruses, especially the
2619 full-length construct, is there are toxic regions. And in
2620 bacteria, when you try to maintain them, the toxic regions
2621 either kill the bacteria or the bacteria kicks them out. And
2622 so you end up with deletions in your construct.

2623 So we get around that by keeping the genome fragmented. It's
2624 another reason we would keep it fragmented. Besides
2625 biosafety issues, it's stable that way. Full-length
2626 constructs suffer from that.

2627 The group that actually developed the bat technology in
2628 Europe solved that problem in another coronavirus by
2629 carefully measuring where the region of toxicity was, and
2630 then inserting in a splice site. So they destroyed it and
2631 then allowed the splice site to rejoin the live virus. The
2632 Chinese bat clone doesn't have any of that kind of higher
2633 level.

2634 Q But I guess when you're saying that they only
2635 have WIV1, that is based on what they published. You don't
2636 have any insight?

2637 A That's based on what they published. I don't
2638 have any insights.

2639 Q Just that it's hard --

2640 A I guess I'm speculating, but I personally
2641 think I'm speculating near 100 percent certainty that they
2642 worked on that with a full-length clone. They would want to
2643 do that.

2644 Q It certainly seems plausible, based on
2645 certain --

2646 A That's the trajectory, so why wouldn't they
2647 have to be trying? They have to be trying.

2648 BY MR. BENZINE.

2649 Q I want to jump ahead and talk about the
2650 February 1st, 2020 conference call you referenced when I went
2651 through the names. In the email back-and-forths, and the

2652 notes and the invites, you're not listed anywhere, but you
2653 were on that conference call?

2654 A I wasn't listed on any of the invites?

2655 Q No.

2656 A I didn't know that. I'm kind of surprised.

2657 They clearly reached out to me. I don't know why they didn't
2658 reach out -- this must have been within the NIH staff?

2659 Q No, there was a conference call with Dr. Fauci
2660 and Dr. Andersen?

2661 A Wait, you're talking about the February 1st
2662 call.

2663 Q Yes, sir.

2664 A Not the February 11th call.

2665 Q Correct.

2666 A I'm sorry, I was confused. Can you restate
2667 the question?

2668 Q The February 1st call with Dr. Fauci,
2669 Dr. Andersen, and Dr. Farrar, and ten or so others, we have
2670 gotten emails from almost every American participant on the
2671 call, and haven't seen your name come up anywhere. So I was
2672 surprised to hear that you were on it. But I want to confirm
2673 that you were on the call?

2674 A I think I was. My recollection is this
2675 meeting was heavily dominated by the evolutionary biologists,
2676 who were split on the origin of the virus. Is that the

2677 meeting you're talking about?

2678 Q That sounds right.

2679 A So I must have been there.

2680 Q Do you recall how you got invited?

2681 A No, I thought I was on the email chain, to

2682 tell you the truth.

2683 Q I want to read a little bit from

2684 Dr. Andersen's interview.

2685 A Okay.

2686 Q We asked him these questions and asked him

2687 about the call.

2688 He said, "Ralph Baric, for example, is a name that came up.

2689 We all know Ralph, Ralph is a very important coronavirus

2690 biologist, but we also know that Ralph had very close

2691 associations and collaborations with the Wuhan Institute of

2692 Virology, for example. So if this did, in fact, originate

2693 from a lab, then, of course, he would not be a person to have

2694 on a call like this."

2695 A I must have been on that call. He may not

2696 have known it. It was -- again, right now, I have huge

2697 uncertainty about what call I was on, but he was there.

2698 Q I think we're talking about the same call.

2699 A I think we're talking about the same call.

2700 But I was on a phone, so it wasn't like a Zoom link for me.

2701 I didn't have anyone else's picture. So I was hearing mostly

2702 names, or I knew who they were, who was speaking.

2703 Q And you don't recall how you got on to the
2704 call?

2705 A I don't recall how I got invited.

2706 Q Okay.

2707 A No, I would have to look it up. I thought I
2708 knew, but apparently not.

2709 Q And you've discussed a little bit about the
2710 kind of back-and-forth of the people split on the origins
2711 question.

2712 A Yeah.

2713 Q Do you recall anything else from that
2714 conversation?

2715 A There was a fairly strong consensus, I think
2716 that was building toward the end of the call, that there
2717 wasn't data to support engineering, that there were other
2718 alternatives for the furin cleavage site.

2719 The receptor binding domain was still a little uncertain at
2720 that time, but if I remember correctly, one of the first
2721 pangolin strains had been sequenced and the sequence was
2722 available, which was very close to the SARS2 sequence, which
2723 argued that the RBD itself was natural origin.

2724 So that actually -- you know, in scientific method, you're
2725 trying to disprove a hypothesis. That actually was more
2726 against the current hypothesis, which was somebody tinkered

2727 with the residues in the RBD and made something totally
2728 unique. That couldn't have been the case, since it was
2729 already in nature.

2730 The furin cleavage site, the discussion was mostly around how
2731 furin cleavage sites can get in by natural
2732 replication-related processes. And so
2733 polymerase -- coronavirus polymerases can recombine. And
2734 there are group 1 coronaviruses that have snippets of group 2
2735 coronaviruses in the spike. The spike is like super plastic.
2736 It can tolerate all kinds of genetic change. And so it's
2737 possible it could have been inserted from another one.

2738 When polymerases are moving down template strands, they can
2739 slip back and then start again. You can duplicate sites.
2740 And then they evolve independently. They can stutter, where
2741 they're put in additional residues. And in the case of flu,
2742 the design of the sequence, right around that polyclonal
2743 cleavage site in flu is designed to confuse the polymerase
2744 and make it slip. And that's how it gets introduced in flu
2745 to make it pathogenic in birds.

2746 So those kind of things were possible. So there's other
2747 alternatives for the furin cleavage site, and so -- and there
2748 was no backbone, nothing.

2749 The other problem that they faced is that they only had a few
2750 genomes to look at. I think at that time, there were
2751 probably around 30, 40 genomes, maybe, max. Some of them,

2752 they couldn't use because the sequence quality was low read.

2753 And they needed more naturalized.

2754 So there was a lot of uncertainty from the evolutionary

2755 biologists, in terms of whether it could be lab escape or

2756 whether it could be natural processes, because both of them,

2757 it can pass between virus and culture, you'll get mutations.

2758 If you come from nature, it's got mutations.

2759 So it's hard to distinguish that, but what you could say is

2760 that it's normal evolutionary processes. It's not something

2761 unique.

2762 BY MR. WENSTRUP.

2763 Q One thing you might find interesting, which

2764 they didn't know at the time, but it's since been

2765 declassified or unclassified. ODNI has come out and said,

2766 well, they did have pangolin coronaviruses in the lab.

2767 A Hmm, okay. Actually, didn't they publish a

2768 paper like in September on the pangolin virus?

2769 Q I'm not sure the date.

2770 A It was very confusing, because different

2771 groups sequenced the same samples. And the first group had

2772 this low impact paper, nobody noticed. And then the next

2773 group was in Nature, and they came from the same place. It

2774 was all very confusing.

2775 BY MR. BENZINE.

2776 Q I want to ask about the furin site a little

2777 bit. Dr. Garry, after the call, in the notes, expressed
2778 concern over -- he called it a 13 nucleotide insertion that
2779 was created at the site, and said I just can't figure out how
2780 this gets accomplished in nature, but in a lab, it would be
2781 easy.

2782 How would you kind of refute Dr. Garry's points there?

2783 A The sequence, you only need to insert three
2784 amino acids to make a furin cleavage site. Four is a
2785 nucleotide. Four amino acids went in asymmetrically. Why
2786 would anybody engineer that and do it that way, putting in an
2787 extra residue which is a proline, which puts kinks in
2788 proteins, it usually screws things up. And ultimately, that
2789 proline changed within a few -- within one or two variants.
2790 So that didn't make a lot of sense to me. But if you were
2791 going to engineer it, I guess the question would be, you
2792 don't need to put four amino acids in, it's easier to put
2793 three amino acids in, in the frame. And also, you'd probably
2794 want to put one in that was efficient. The sequence in SARS2
2795 is not a very efficient cleavage site.

2796 Q So Dr. Garry was just kind of wrong?

2797 A You can make -- no, I'm not saying he's wrong.
2798 I'm just saying that means if it went in that way, then it
2799 was nefarious purposes to begin with, right? Because you're
2800 basically trying to cover up what you did.
2801 I don't think -- I mean, when I looked at it, when it went in

2802 asymmetrically, that was more akin to recombination for me.
2803 Because recombination is not always perfect. Sometimes you
2804 have perfect recombination, but oftentimes, you have its
2805 offset and it introduces additional residue. One nucleotide
2806 or two nucleotides, depending on how it goes in, it's sort of
2807 the random process of recombination.

2808 BY MR. WENSTRUP.

2809 Q Since we're on that sort of vein, referring to
2810 that DEFUSE proposal. And then this article of January 22nd,
2811 "Scientists say EcoHealth Alliance's DEFUSE proposal was a
2812 blueprint for SARS-CoV-2." And then from April of '23,
2813 "Endonuclease fingerprint indicates a synthetic origin of
2814 SARS-CoV-2." And that's by Bruttel.

2815 So I'm just reading from this, and I'm really seeking your
2816 opinion on some of the things. Have you read those, by any
2817 chance?

2818 A I have.

2819 Q So --

2820 A I have read this proposal.

2821 Q I know you've read that. So as they say in
2822 there, "and the EHA plan was to use six segments to assemble
2823 synthetic viruses to use unique endonuclease sites that do
2824 not disturb the coding sequence and to buy BsmBI" --

2825 A Can I answer those three questions? That's
2826 the standard way we've been doing genetics since 2003.

2827 Q Okay.

2828 A So none of that is novel.

2829 Q Okay. And the EHA proposal would create

2830 chimeric spikes, insert new receptor binding domains, and

2831 human furin cleavage sites.

2832 A Can we stop before the furin again?

2833 Q Sure.

2834 A Absolutely, the proposal talked about making

2835 chimeric spikes with WIV1 and SCH014 as the backbone. The

2836 sequence would come from the Chinese, depending on -- it

2837 would be some work with pseudotypes beforehand to make some

2838 kind of down selection about which ones you might want to

2839 work with.

2840 And then, primarily, because of cost, the first thing you do

2841 is you drop them into those backbones to see if they could

2842 program infection. So that's nothing new either in that

2843 proposal -- the DARPA proposal came out, what, 2020?

2844 Mr. Strom. Proposed in 2018.

2845 The Witness. But publicly, the group that released it --

2846 Mr. Benzine. 2021.

2847 The Witness. Okay.

2848 BY MR. WENSTRUP.

2849 Q After the FOIA?

2850 A No, it was done before the FOIA.

2851 Q And looking at the proposal, it appears there

2852 may have been a willingness, not necessarily by you, to do
2853 some of this work in the BSL-2 in China.

2854 A There was no willingness on my part to do any
2855 of this work.

2856 Q That's what I wanted to clarify.

2857 A Let me make that clear.

2858 Q That's fine. So in Bruttel, it says, "the
2859 restriction map of SARS-CoV-2 is consistent with many
2860 previously forwarded synthetic coronavirus genomes and meets
2861 all the criteria required for an efficient reverse genetic
2862 system." And then they discuss the rather improbable odds of
2863 a coronavirus having the patterns seen in SARS-CoV-2 without
2864 engineering. That's an opinion.

2865 A That is an opinion.

2866 Q And then they report a high likelihood that
2867 SARS-CoV-2 may have originated as an infectious clone in
2868 vitro.

2869 So what they're reporting is what EHA proposed to do is what
2870 is actually seen in the SARS-CoV-2 genome. I want to know if
2871 you agree. And if I give you this from the article, because
2872 at first blush, I have no idea, you may know, the top line.

2873 A Yeah.

2874 Q Does that makes sense to you? Do you see
2875 that?

2876 A So the first thing, what these are -- these

2877 lines describe naturally occurring BsmBI sites in the SARS
2878 coronavirus 2 genome. Now, one of the first things you
2879 notice is that those same sites are present in many of the
2880 bat strains that exist. So if they are engineered, if you
2881 use them to engineer SARS2, they wouldn't normally be in the
2882 same location in the bat strains.

2883 The second thing is, they do count six pieces, but one of the
2884 pieces is about 8 KB and the other is about 300 base pairs.
2885 If you look at any of the molecular clones that I've
2886 engineered, with SARS, they're usually 5 KB apart, so that
2887 you have five or six KB pieces that you can work.

2888 Having a tiny little piece like that, if I looked at it, that
2889 would irritate me, like, to no end, and we would silence it,
2890 one of those sites. And then separate this, so that the
2891 fragments are of equal size. The first size piece is also
2892 too small, and so it leaves larger pieces, and the larger
2893 clones are unstable with passage.

2894 Q Okay.

2895 A So you would want it more equally distributed,
2896 unless there was a region that was super toxic. If there was
2897 a toxic region, then you would have a little piece. There's
2898 no toxic site there.

2899 Q Thank you.

2900 A So this is biostatistical BS, in my opinion.

2901 And they come up and say that the pattern here is unique, and

2902 they do that by comparing most of the pattern to clade 2 and
2903 clade 1B coronaviruses.

2904 So the statistical number that they have for the ones that
2905 are far away is much more, and it gives them statistical
2906 power to make the claim that it was engineered.

2907 Q Thank you.

2908 A And it's a pathetic piece of work. By the
2909 way, you can see how I engineered the SARS-CoV-2 genome since
2910 it's published, and you will see that it's completely
2911 different than this.

2912 Mr. Benzine. I want to introduce Majority Exhibit 2. It's
2913 more to refresh your recollection on dates and people and
2914 stuff.

2915 (Majority Exhibit No. 2 was
2916 identified for the record.)

2917 BY MR. BENZINE.

2918 Q So this is the agenda for a National Academies
2919 of Sciences, Engineering, and Medicine meeting on Data Needs
2920 for COVID-19 from February 3rd, 2020.

2921 A He did send me an email. Did I say he sent me
2922 an email?

2923 Q This is a different meeting.

2924 A Okay. I always worry about names, about
2925 saying I didn't get an email.

2926 Q Absolutely. Do you recall attending this

2927 meeting?

2928 A This would have been by Zoom.

2929 Q Yes.

2930 A So I can't say with 100 percent certainty, but

2931 I can say that, most likely, yes. I would have to check my

2932 calendar, but I think I did. I was certainly part of that

2933 committee.

2934 Q Understanding you're not 100 percent sure, but

2935 do you have any recollection of what was said during this?

2936 A Well, I think the purpose of this meeting -- I

2937 think the purpose of this particular meeting was to outline

2938 an agenda for the group to write a report on origins. And so

2939 part of the meeting was to review the statement of work that

2940 had been given to the National Academies to try to come up

2941 with this plan.

2942 And then I don't recall what Fauci said at the meeting.

2943 Yeah, I don't recall what Fauci said at the meeting. And

2944 then there was discussion about writing objectives and things

2945 like that. That would have occurred. And what different

2946 groups need to get together to try to start formulating a

2947 response.

2948 Also, I think we had -- we may have had outside speakers come

2949 in and things like that, to try to inform the committee, but

2950 I would have to look. I would have to review the agenda.

2951 Part of the problem here is there's all kinds of things going

2952 on simultaneously, and so I could easily get things confused.

2953 Q Under a subpoena issued by this Committee,

2954 Dr. Andersen produced some Slack messages to us between him,

2955 Dr. Holmes, Dr. Garry, Dr. Rambaut, and then some were

2956 redacted, and we reviewed them in camera.

2957 Regarding this meeting, he said something about you, and I

2958 would like to get your side of the story on what he said. So

2959 this is --

2960 A Hopefully, he didn't say anything negative.

2961 Q This is a quote from Dr. Andersen's Slack

2962 messages. "I should mention that Ralph Baric pretty much

2963 attacked me on the call with NASEM, essentially calling

2964 anything related to potential lab escape ludicrous, crackpot

2965 theories. I wonder if he, himself, is worried about this,

2966 too."

2967 I'm just trying to get your side of this.

2968 A Can you read that again?

2969 Q "I should mention that Ralph Baric pretty much

2970 attacked me on the call with NASEM," National Academies,

2971 "essentially calling anything related to potential lab escape

2972 ludicrous, crackpot theories. I wonder if he, himself, is

2973 worried about this, too."

2974 A I don't recall this. So because of this, I'm

2975 going to at least say one thing that I gave in the BSEC

2976 meeting on January 25th or 26th. My summary of the origin of

2977 the pandemic was the following.

2978 There are three potential causes for that pandemic. First is
2979 natural origin, second was laboratory escape, and the third
2980 was genetically engineered.

2981 Q And what was the date of that again?

2982 A January 25th or 26th of 2020. So I don't know
2983 where he's coming from. That may have been his
2984 interpretation, but I'm surprised. I'm really surprised.

2985 Q When we saw it, I wanted to make sure we got
2986 your perspective on the record.

2987 A Can you read it one more time?

2988 Q Yes. "I should mention that Ralph Baric
2989 pretty much attacked me on the call with NASEM, essentially
2990 calling anything related to potential lab escape ludicrous,
2991 crackpot theories. I wonder if he, himself, is worried about
2992 this, too."

2993 A I'm really surprised about this, because I
2994 wrote a piece on his origin paper in Immunology, and said
2995 that laboratory escape was possible because of safety
2996 procedures in their laboratories. So it's not consistent
2997 with what I also reported to other groups publicly on when
2998 interviewed. So I don't believe he's attributing that to the
2999 right person.

3000 Q That's fair. And I wish I could show you the
3001 message, but like I said, it's redacted, so I don't have it.

3002 A What do you mean, it's redacted?

3003 Q When Dr. Andersen's counsel produced the Slack
3004 messages to us, they redacted some. So there's a big black
3005 box over them, and we requested to review them in camera.

3006 A So he's talking to somebody else, then.

3007 Q Yes.

3008 A Okay. No, I would just say that's
3009 inconsistent with what I've said publicly and privately that
3010 can be verified.

3011 Q Dr. Andersen was then the lead drafter of "The
3012 proximal origin of SARS-CoV-2" that came out in Virological
3013 in February, and then Nature Medicine in March. I know
3014 you're aware of the paper. Have you had an opportunity to
3015 review the paper in the last four years?

3016 A I looked at it before this meeting. I figured
3017 you guys might ask.

3018 Q So it came to two kind of conclusions. The
3019 first in the summary, and we've heard different stories from
3020 different authors, of the reviewers kind of ramped up the
3021 language to, we -- when we said laboratory construct, we
3022 meant like bioweapon, all kinds of things.
3023 But the first conclusion was, "our analysis clearly show that
3024 SARS-CoV-2 is not a laboratory construct or a purposefully
3025 manipulated virus."

3026 Do you agree?

3027 A I would agree with that statement, in terms of
3028 the data that was available at the time. That's absolutely
3029 true. It's still true today.

3030 Q Laboratory construct, how do you define
3031 laboratory construct?

3032 A It doesn't matter how I define it. What
3033 matters is how they define it. I would -- laboratory
3034 construction, to me, personally, would be an engineered
3035 virus.

3036 Mr. Strom. One that does not have --
3037 The Witness. You have a molecular clone, and you reconstruct
3038 it somehow in the laboratory.

3039 BY MR. BENZINE.

3040 Q Like serial passage wouldn't fall under
3041 laboratory construct?

3042 A No, I don't think so.

3043 Q Okay.

3044 A But they may have interpreted it that way.

3045 You would have to ask him.

3046 Q We did.

3047 A Did he answer?

3048 Q I would have to go back and look. I
3049 think -- what I recall from that, both from their hearing and
3050 the interviews, is that they meant bioweapon or --

3051 Mr. Strom. A de novo --

3052 BY MR. BENZINE.

3053 Q A de novo, built virus.

3054 A What they would have had is no true actionable
3055 intelligence, and said it was engineered. Because if you
3056 don't have a backbone sequence that's close enough, you don't
3057 have any substrate on which to build anything that could have
3058 been close enough to SARS that people would have said it was
3059 novel. So we still don't have a backbone sequence that's
3060 close enough.

3061 Q The second conclusion was, "we do not believe
3062 that any type of laboratory-based scenario is plausible."
3063 Do you agree with that?

3064 A I signed a paper that said that that
3065 was -- that a laboratory scenario needed to be carefully
3066 evaluated. I think that says it all as well.

3067 Q And then after the fact --

3068 A Which is also inconsistent with the statement
3069 he just made.

3070 Q It is. I'm not a scientist, but even reading
3071 that confuses me beyond just the science.

3072 A It's the first I've ever heard it, so I'm very
3073 confused about it myself, yes.

3074 Q After the fact -- and then there's a reporter
3075 at Science Magazine named John Cohen.

3076 A I know him.

3077 Q He put out some emails after the fact of an
3078 anonymous person that claimed that the "proximal origin"
3079 authors plagiarized some ideas and went a little bit too far.
3080 Are you aware of those emails?

3081 A John contacted me.

3082 Q Were you the --

3083 A No, I was not. I was not. I was building
3084 suspense.

3085 Q So Dr. --

3086 A And it worked.

3087 Q It did. Part of it is because Dr. Holmes
3088 thinks you were the one that contacted John Cohen.

3089 A Well, that's why he may say it. He and -- I'm
3090 forgetting his name, sorry -- Andersen. If that's what they
3091 thought, he may have been really irritated with me if he felt
3092 that it was me, but it was not.

3093 Q What did Mr. Cohen contact you about?

3094 A He was asking me the same question you asked
3095 me, was I the author of that statement? And I said, no, I
3096 was not.

3097 Q Do you know who is?

3098 A No, I don't.

3099 Q Shifting to another publication, going a
3100 little bit back in time, but the Lancet correspondence from
3101 February 19th, 2020.

3102 A This is the Daszak request for support of
3103 Chinese science?

3104 Q Yes.

3105 A Okay.

3106 Q You're obviously aware of it. Dr. Daszak
3107 testified, and I'm quoting, that you didn't want to be on the
3108 letter, and that you were very hesitant. Do you recall
3109 Dr. Daszak asking you to join the letter?

3110 A Yeah, there is an email chain, but I can tell
3111 you what preceded the email chain was a phone call, where he
3112 asked me to be on that correspondence. And I said, no, that
3113 I felt that we both had a conflict of interest because we
3114 work with Wuhan Institute of Virology. That if we were on
3115 it, and that could be construed as, in
3116 essence -- what's -- sorry, I must be getting tired, because
3117 I'm forgetting the terminology.

3118 Mr. Strom. Competing interest or a conflict.

3119 The Witness. Like we were doing it for our own benefit,
3120 right? So I didn't think it was appropriate to sign it. The
3121 next day, he emailed me and said that he talked to Linfa
3122 Wang, and he agreed that we shouldn't be authors.

3123 And I did something I normally don't do, which is say more
3124 words than "great," which is what I usually said. But I
3125 said, great, it's better this way, or something along -- the
3126 summation was it's better this way. So that's the genesis of

3127 that.

3128 Q But Dr. Daszak did end up signing it?

3129 A He did end up signing it.

3130 Q Did you have any conversations regarding his
3131 change of heart?

3132 A No. I think it was a mistake on his part, and
3133 later, I think when he went -- when he was part of the WHO
3134 committee that went to China to review it, he also had a
3135 conflict of interest. And that it would have been better for
3136 the scientific community if he hadn't attended.

3137 Q You've kind of already answered this, but I'm
3138 going to ask it very directly. In the letter, it said, "we
3139 stand together to strongly condemn conspiracy theories
3140 suggesting that COVID-19 does not have a natural origin,"
3141 that was widely construed as any kind of lab leak hypothesis
3142 is a conspiracy theory.

3143 A I think you might want to put that in context,
3144 because the context of that letter came out shortly after a
3145 report went up on a reprint server saying that the SARS2
3146 genome had pieces of HIV. And what that researcher had done
3147 is he had done sequence comparisons under the most relaxed
3148 conditions possible, and so he allowed big deletions and
3149 things to occur.

3150 So you could allow those deletions to occur and say, okay, is
3151 there a sequence of HIV in SARS2, and, boom, it occurred.

3152 What he didn't tell you is if you did the search on all the
3153 biota in nature, you would have found it like in a pine tree,
3154 and all kinds of other stuff.

3155 So the scientific community was really upset about that
3156 paper, because it was -- my wife told me not to describe it
3157 that way, so I'm not going to describe it that way, but it
3158 was really poor quality science, and ultimately, the group
3159 retracted the paper.

3160 There were several groups that immediately showed what they
3161 did, and why it was inappropriate. That letter came out
3162 shortly -- I believe came out shortly after that report. And
3163 so that was the first big conspiracy report, which would have
3164 dominated that letter. So keep that in context.

3165 Q That makes sense. And like John said about
3166 rolling eyes, everyone in here is going to roll their eyes
3167 when I say this, but we have kind of had this recurring theme
3168 of people getting out in front of their skis and maybe
3169 writing a little bit more than they know or mean, to combat
3170 things. So, completely understand the HIV sequence was a
3171 conspiracy theory. They could have written that,
3172 understanding that you didn't sign it, but they could have
3173 said that was a conspiracy theory, not any theory suggesting
3174 COVID-19 does not have a natural origin.

3175 A They said there was no chance, what?

3176 Q We stand together to strongly condemn

3177 conspiracy theories suggesting that COVID-19 does not have a
3178 natural origin.

3179 A Yeah, I would say, that date, I would probably
3180 have been more comfortable not signing it, in any event, even
3181 if I didn't have a conflict of interest.

3182 Mr. Benzine. Thank you. We are at our time, so we will take
3183 a break and go off the record.

3184 (Recess.)

3185 Ms. Yass. Back on the record.

3186 BY MR. ROMERO.

3187 Q So, Dr. Baric, in the previous round of
3188 questioning, you were asked about your attendance on a
3189 February 1st conference call, and you mentioned that on that
3190 call, there was some talk about the pangolin virus, its
3191 receptor binding domain, and its similarity to the RBD of
3192 SARS-CoV-2. Does that sound correct?

3193 A That's correct.

3194 Q So as far as the highly scrutinized February 1
3195 call that we've come to understand was organized by
3196 Dr. Jeremy Farrar, we have talked to other scientists, other
3197 virologists who attended that call, and we were told that, at
3198 that time, they didn't actually know about the pangolin
3199 virus.

3200 So hearing that, and knowing that you were on a lot of calls
3201 around this time in early February 2020, is it possible that

3202 you weren't on the February 1 conference call organized by

3203 Jeremy Farrar?

3204 A Since I apparently wasn't on the email invite,

3205 there's uncertainty in what call I was on. But certainly

3206 Dr. Fauci was there, certainly there were four evolutionary

3207 biologists there, certainly there were people like Ron

3208 Fouchier, who I think was also on the call, and several other

3209 corona virologists, so I'm pretty sure I was on that call.

3210 And I believe that the statement was from one of the

3211 evolutionary biologists that the sequence of the pangolin

3212 virus either was out, or it might have been coming out. I

3213 may have misspoke and said it was out, but it was out very

3214 shortly thereafter. If it wasn't out at the time of the

3215 meeting, it was within a couple of days, and I may have

3216 pooled them together. But within a few days, those sequences

3217 became available.

3218 So that might be a memory lapse. There's already a potential

3219 memory lapse about whether I was even on the call, so -- but

3220 I'm pretty sure I was on the call.

3221 Q Okay. So last hour, I think around that

3222 time -- it ended with a discussion about the "proximal

3223 origin" paper.

3224 A Yeah.

3225 Q So we would like to ask a few more questions

3226 about that paper, and some of the conclusions reached.

3227 A Sure.

3228 Q Again, related to its conclusion that

3229 SARS-CoV-2 is not a "purposefully manipulated virus."

3230 So again, we have interviewed the authors, and our

3231 understanding through those conversations is that

3232 "purposefully manipulated virus" refers specifically to the

3233 idea of deliberate engineering. So that would mean combining

3234 bits and pieces of genetic material in order to create a

3235 virus. And there are other techniques that are encompassed

3236 here, but constructing a chimera, I believe, would fall under

3237 this concept.

3238 A Sure.

3239 Q So the paper rules out purposeful manipulation

3240 on two grounds. Premise 1 is that the virus, SARS-CoV-2's

3241 receptor binding domain, which is housed on the spike

3242 protein, is imperfect. And you have kind of gone into this

3243 discussion in our first hour of questioning, that no

3244 scientist would intentionally construct a virus whose

3245 receptor binding domain would not perfectly bind to human

3246 ACE2?

3247 A No, I don't think I -- you need to say that

3248 again. I'm not sure I would have said it the way you said

3249 it. Can you say it again?

3250 Q Okay. So our understanding is that the

3251 receptor binding domain of SARS-CoV-2 is an imperfect

3252 receptor binding domain that does not bind perfectly to
3253 SARS-CoV-2. Does that sound correct?

3254 A It binds well to human ACE, but it is not
3255 perfectly designed to bind to human ACE.

3256 Q So I guess the question is, what does that say
3257 about the possibility that this receptor binding domain was
3258 constructed by a scientist?

3259 A I think the more telling information that's
3260 also in that paper is that there's a pangolin sequence that I
3261 think has four amino acid changes in it over several hundred
3262 amino acids in the RBD, which indicates that it's more likely
3263 a natural origin derivative.

3264 I think this was then later substantiated by sequences from
3265 Thailand isolates, like BANAL-52 that only had one amino acid
3266 change in that region and not in a receptor binder, which
3267 argued again that it was natural, it's related to natural
3268 isolates.

3269 So what's your question again? I'm trying to understand the
3270 context of it.

3271 Q So I guess, on the one hand, we have a
3272 receptor binding domain that can bind to a human ACE2, but
3273 does not perfectly bind to human ACE2. And on the other, we
3274 have a pangolin virus found in nature that has a very
3275 similar, if not identical, receptor binding domain.

3276 A Except it binds much better to human ACE2.

3277 Q Okay. So taking those two things together,
3278 what does that say about the likelihood that this receptor
3279 binding domain in SARS-CoV-2 is not natural and was created
3280 in a lab?

3281 A It says it wasn't created in a lab.

3282 Q Okay. So that's kind of the conclusion that
3283 the "proximal origins" authors possibly reached in their
3284 paper?

3285 A I think I said that I was in agreement with
3286 their interpretation of the data as it sat at the time, that
3287 there wasn't any evidence, scientific evidence that it was
3288 engineered. It doesn't mean that that kind of data won't
3289 emerge in the future. It just means that, at that moment in
3290 time, there was no data to support it.

3291 Q I guess that kind of flows into a criticism of
3292 that conclusion of the "proximal origin" paper that, in the
3293 abstract -- and correct me if you disagree. But is it
3294 possible that SARS-CoV-2 is a chimera that was constructed by
3295 taking a receptor binding domain from a virus similar to the
3296 pangolin virus and attaching it to the backbone of a virus
3297 that is similar to RaTG13?

3298 A If you took the separate binding domain of
3299 SARS2 and put it into RaTG13, every evolutionary biologist in
3300 the world would say, hey, somebody took the SARS2 or some
3301 other RBD and stuck it into RaTG13, which has about 1100 or

3302 1200 nucleotide changes, a fingerprint all across that genome
3303 that says, I'm RaTG13. And if you put a SARS RBD in it, it
3304 still says, I'm RaTG13 and somebody stuck an RBD in me. So
3305 the footprint would have been there.
3306 There's no genome close enough that is engineerable using
3307 current standards that could have resulted in SARS2.
3308 Q Okay.
3309 A Now, that may happen in the future, but at
3310 this time -- and at this time, it was not going to be
3311 possible. And it was even worse because, let's say if you're
3312 going to engineer it, if you're going to engineer it, that
3313 means you don't know what the sequence is.
3314 So with RaTG13 -- and I tried to point this out before,
3315 there's like -- I'm going to do it 1200, it's actually 1100
3316 and, I don't know, 47, or something like that, but the math
3317 is too hard. So there's about 1200 changes, so it's four to
3318 the 1200th power of combinations of mutations that you have
3319 to try to get SARS2. That's a huge number.
3320 Now, I'm going to tell you why it can't be done. The
3321 transfection efficiency of a molecular clone for
3322 coronaviruses was, at best, 5,000 cells. So that means you
3323 can quarry 5,000 genomes at a time. Four to the 1200th power
3324 is a whole lot of zeroes. I calculated it out. One
3325 researcher would require something like 500,000 years. So if
3326 you've got 100 researchers doing it, you could get it down to

3327 54 years. Then you have the problem of figuring out which
3328 one was going to be pathogenic in humans. So that's just the
3329 start. So it's not possible to actually do that with the
3330 current technology.

3331 Now, people will say, well, you can do shotgun mutagenesis
3332 across the genome, but you still have all those genomes that
3333 you have to filter through to the one that would be
3334 pathogenic in humans.

3335 How would you select them? I know how I would select them.
3336 I'm not going to tell you how I'm going to select them, but I
3337 would, because you don't want me to answer the question on
3338 the table unless you press me.

3339 Mr. Romero. I think that's good for the "proximal origin"
3340 questions, so I am going to turn it over to Alicia.

3341 Ms. Yass. Great.

3342 BY MS. YASS.

3343 Q So I am going to ask you, Dr. Baric, some
3344 questions about what's been termed the one log growth rule.
3345 This Committee previously spoke to Dr. Daszak, and during his
3346 interview, he said that the idea for his one log growth rule
3347 that EcoHealth Alliance worked on and used in its grants with
3348 NIAID in their year 3 award conditions for their study of bat
3349 coronavirus, and he said that he got the idea for this rule
3350 from you, and work that you had previously done. Are you
3351 aware of this?

3352 A Absolutely.

3353 Q So Dr. Daszak said, as he was responding to
3354 questions that he got from NIAID about his work and the gain
3355 of function pause in effect at the time, and he said, "I got
3356 advice on what a good proper response to this should be from
3357 Ralph Baric, who responded to other requests for that."
3358 Did you speak to Dr. Daszak about your use of the one log
3359 growth rule?

3360 A Yes. So this goes back to the review of the
3361 chimeric viruses with SHC014 and WIV1.
3362 Despite all the data that argued that it was attenuated, one
3363 of the things that NIH wanted us to do or think about was to
3364 come up with some criteria that you would use as a benchmark
3365 that if it happened in your lab, let's say we put those
3366 viruses in some other system and suddenly they're growing
3367 like bandits, or they grew tenfold higher in a humanized
3368 mouse for some reason. We needed a benchmark. They wanted a
3369 benchmark.
3370 They didn't want to give you approval to move forward without
3371 some other regulatory -- not a restriction, but a regulatory
3372 benchmark that if you saw this benchmark, you would
3373 immediately pause, you would immediately tell your local
3374 environmental health and science committee to say, listen, I
3375 found this growth phenotype that's tenfold above what we
3376 would have normally seen with this virus in this system.

3377 They would have looked at it, and communicated with NIH. And
3378 then we would have had a call about what to do. And the
3379 outcomes could be destroy the virus, which is fine. Alter
3380 the containment conditions, maybe move it up to BSL-4, which
3381 would mean we wouldn't work on it anymore, or -- I can't
3382 think of a reason, like right now, I would be alarmed if we
3383 continue with it, so I would probably destroy it. But I
3384 can't think of a reason why they would say, don't worry about
3385 it, and go forward, right?

3386 But from their perspective, they're developing new
3387 regulations for things that had never been regulated before,
3388 and our application was one of the first ones that went
3389 through. And so in the discussions, the back and forth
3390 discussions, we decided that there needed to be some kind of
3391 additional benchmark that you could use as a way that would
3392 tell the research community and the university and the NIH
3393 that you've got an unexpected result and you need to stop.
3394 And you need to then debate and discuss and make an informed
3395 decision on how to move forward.

3396 Q Thank you.

3397 A So he called me and asked me what we did, and
3398 I told him that's what we did.

3399 Q In your use of this one log growth rule, in
3400 your research, we would just like to hear a little bit about
3401 that. But specifically thinking about the measurement for

3402 the one log growth, we have heard some witnesses talk to us
3403 about using a PCR measurement, others talk about using viral
3404 titers. So can you please explain the difference between
3405 those measurements and how you utilize them in your
3406 experiments.

3407 A Sure. So viruses, RNA viruses when they
3408 replicate, they have an error rate. They also make mistakes
3409 when they package viral genomes into the virions which are
3410 released from the cells. So sometimes they're not
3411 infectious.

3412 In addition, some of the errors that occur during replication
3413 can be lethal, so those viruses are not infectious.

3414 So in virology, for RNA viruses, there's a function called
3415 particle to PFE ratio, where you count the number of virus
3416 particles and you ask, can they form plaques in monolayers,
3417 or what's the titer, what's the -- it's usually plaques and
3418 monolayers.

3419 You can also do it in animals, too, and you have to titer
3420 down to -- it depends on how well a virus -- if a virus is
3421 lethal, one PFE, you can use a mouse. So you could put the
3422 virus in a mouse and figure out exactly what the lethal dose
3423 is or the number of plaques.

3424 So if you have a monolayer of cells, so you've got holes in
3425 them, so you count those plaques and those are viable viruses
3426 that can infect cells. So we use viable viruses to infect

3427 cells, because that tells us exactly what number of cells in
3428 that tube can infect a cell.

3429 PCR will detect anywhere from 100 to 1,000 fold higher titer
3430 than is seen with plaque assays for RNA viruses because of
3431 this particle to PFE ratio, and the numbers of particles that
3432 are noninfectious. So we always focus on particle PFE.

3433 I wouldn't do it with -- I wouldn't use the standard with PCR
3434 genome equivalents, because the particle to PFU -- there's a
3435 genetic term called epistasis, and that's where mutations at
3436 one location affect the viability and the function of
3437 sequences in another location. So when you make a chimera,
3438 you break apart epistatic interaction, so the particle to PFE
3439 ratio can shift.

3440 So you could think you had a high titer by PCR, but by
3441 plaques, there wouldn't be a tenfold increase.

3442 Q So --

3443 A So I would prefer -- I mean, we preferentially
3444 do plaques. I don't know what NIH regulations are, what
3445 other people may ask.

3446 Q But just in the most simple terms, you're
3447 using that because it's more accurate and more reliable?

3448 A Yes. In simple terms, I think it's a more
3449 reliable metric of the potential hazards to the experiment.

3450 Q Does it also give you realtime results as the
3451 experiment is happening?

3452 A Within a week or two, yeah, sure.

3453 Q And we would just be interested in hearing
3454 your perspective on how virus growth relates to a virus's
3455 pathogenicity or transmissibility, particularly in the
3456 context of this rule.

3457 Is it as simple as if a virus's growth is enhanced by more
3458 than one log, then that virus has been made more pathogenic
3459 or transmissible, or are they not necessarily correlated?

3460 A It's complex.

3461 Q Okay.

3462 A In humans, there is a general correlation
3463 between titer and disease severity. In individuals, that
3464 relationship may not hold. And I can describe it best in the
3465 context of mouse experiments with a genetic -- what's called
3466 a genetic reference population called a collaborative cross.
3467 You can infect collaborative cross mice with the same dose of
3468 virus, and the virus grows to identical titers at day 2 and
3469 4. And it clears at the same rate. One animal doesn't lose
3470 a drop of weight, the lungs are clean, completely subclinical
3471 infection. The next animal, lose 25 to 30 percent of its
3472 weight loss, it can die, the lungs look like a liver, and
3473 that's because of all those host susceptible loci that occur
3474 after the virus gets in and replicates. So it's complex.

3475 Q Sure.

3476 A So when we do a correlation analysis in

3477 outbred rodent populations, there is no correlation between
3478 titer and disease severity, but there are individuals where
3479 it correlates, okay? So it's a function of genetics and
3480 individual variation.

3481 Now, the second part of your question had to do with
3482 transmissibility. Prior to COVID-19, there were no
3483 transmission levels for any coronavirus, so we had no
3484 information on that. And it wasn't until -- because SARS1
3485 doesn't grow very well in the hamster and nobody tried
3486 transmission studies.

3487 So in general, with COVID-19, there seems to be a correlation
3488 between titer and transmission. But transmission is
3489 contrived. There's about two inches apart in two cages for
3490 airborne transmission and air blows from one to the other.
3491 It doesn't happen in nature, like in humans.

3492 Q Sure.

3493 A So in that scenario, it's kind of a contrived
3494 model. In real life, it's probably multigenic, it's
3495 stability of the virus, it's where it grows and how easily it
3496 aerosols. Different people clearly make different size
3497 particles when they breathe and talk, some make very small
3498 particles, they're more likely to aerosol; others don't, make
3499 large droplets. So it's very complex in terms of
3500 transmissibility.

3501 So I don't think that's been studied sufficiently to give you

3502 a clear answer except, in general, it's thought that higher
3503 titer in the right compartment correlates with more efficient
3504 transmission.

3505 Q And just from your use of this one log growth
3506 rule, what has your experience been in it being a good
3507 guardrail or benchmark, as you said?

3508 A Well, we haven't done anything that's
3509 triggered it yet, so we're happy with that. Again,
3510 generally -- well, we haven't made chimeras in quite a while.
3511 But in general, when you make a chimera, you're breaking
3512 apart some epistatic interactions, so in general, it's a
3513 little more debilitated, so the virus has to pass it a few
3514 times to figure out how to fix itself.

3515 Q I appreciate that science lesson. I'm going
3516 to change topics a bit. We have heard from multiple
3517 witnesses that the creation of a vaccine for COVID-19
3518 happened almost miraculously fast, and they credit this speed
3519 to the fact that coronavirus research and mRNA research had
3520 been going on for years prior to the COVID-19 pandemic.
3521 You were a part of this process, both with ongoing research
3522 and active involvement in the COVID-19 vaccine testing,
3523 correct?

3524 A That's correct.

3525 Q In terms of the development and testing of a
3526 COVID-19 vaccine, in 2020, your involvement was running

3527 safety and efficacy trials for Moderna's vaccine using your
3528 lab's chimeric coronavirus strains, human respiratory cell
3529 cultures, and lab mice. Is that accurate?

3530 A For the COVID-19 vaccine, I don't think we
3531 tried any -- we used any chimeras. The only thing we really
3532 used was the mouse-adapted SARS2 coronavirus, the MA10, which
3533 was called MA10 in this case. It was ten passages in mice
3534 that produced a lethal infection.

3535 But I can tell you that our involvement with mRNA technology
3536 started in 2016 in collaboration -- 2016, early 2017, in
3537 collaboration with Barney Graham and Kizzmekia Corbett at the
3538 NIH VRC, where they had just worked. Well, Jason McLellan
3539 and Barney had really worked out the technology to freeze the
3540 coronavirus spike glycoprotein in what was called the
3541 prefusion state, which had all the big, juicy neutralization
3542 epitopes in the right context.

3543 So they wanted to evaluate mRNA vaccine performance, and so
3544 they contacted us and we worked with them on mRNA vaccines
3545 for MERS coronavirus mostly, but also SARS coronavirus in
3546 2003, and were actually writing the paper in December 2019
3547 when COVID hit. And so we stopped writing the paper.

3548 When they received the sequence, they ordered the constructs.
3549 I was told that I had to have a mouse model available by the
3550 end of April, so my job was to make a robust mouse model in
3551 sufficient time to test that vaccine in April and May, so

3552 that the final reports could be compiled, including some
3553 studies that were designed to look for what are called
3554 variant phenotype vaccine associated -- oh, crap, I forget
3555 the name. Do you have to type everything that I say? Great.

3556 Q We're all allowed to have those moments.

3557 A I'm having a moment. But they're probably
3558 going to become more frequent over the next hour, I have to
3559 admit. But it's vaccine associated deleterious outcome. In
3560 this case, there's something, either the vaccine enhances the
3561 availability of the virus to grow or it causes some kind of
3562 pathology. And it needed to be tested for that, because,
3563 earlier, it had been shown with earlier vaccines with the
3564 SARS strain that you've got those phenotypes. My job was to
3565 make the mouse model and design those experiments and have
3566 them all done by April.

3567 Q And we've heard from multiple people that this
3568 was all on a timeline that was way faster than any other
3569 vaccine.

3570 A It was very stressful.

3571 Q I'm sure.

3572 A It was very stressful.

3573 Q You mentioned that you had been working on
3574 this, on vaccines, prior to 2016. I know, reading articles
3575 and research that you've done, it seems like you've been
3576 working on a pan-coronavirus vaccine for many years, and

3577 that's been one of your research focuses; is that right?

3578 A Well, again, the discovery work we did said
3579 that there was a zoonotic virus. There are animal viruses
3580 out there that are high risk. You don't know which one will
3581 evolve. So the only kind of countermeasure you can make is
3582 broad spectrum. It either has to be a broad spectrum drug,
3583 or you have to have a vaccine that provides like an umbrella
3584 of breadth to many strains.

3585 And so what you try to do with your discovery work is to find
3586 the strains that are the most different, and then some in the
3587 middle. So then you can say, well, it works on the bookends,
3588 it works in the middle, I hope it works against the new
3589 thing, right?

3590 Q Sure.

3591 A That's the only way to do it.

3592 Q You mentioned a little bit throughout today
3593 some therapeutics that you were testing before and other
3594 research that was sort of useful for the pandemic. Can you
3595 elaborate on what pieces or findings from research prior to
3596 the pandemic were useful in determining and finding vaccines
3597 and therapeutics once the pandemic was widespread?

3598 A Well, certainly having isolates and robust
3599 mouse models of human disease, using the human strain of MERS
3600 and the SARS strain that caused human disease were really
3601 important. But that captured this much of the variation,

3602 like a paper thin sliver of the variation that exists in the
3603 family.

3604 So you need to have natural, other zoonotic isolates with
3605 robust mouse models, so you'll be able to really evaluate the
3606 performance of the vaccine when it's not a perfect match,
3607 because when the vaccine's not a perfect match is when all
3608 these adverse reactions can occur, or you have this because
3609 you have a breakthrough.

3610 So we did discovery work. That discovery work is important
3611 because it gave us breadth both with MERS and with SARS. In
3612 addition, at the same time, we were part of a grant that was
3613 funded to try to develop drugs against coronaviruses, with
3614 Mark Denison at Vanderbilt and Gilead were collaborators.

3615 And so Gilead was gracious enough to provide a fairly robust
3616 panel of nucleoside inhibitors that we screened working down
3617 to remdesivir, that we then moved from -- the classic
3618 approach was, you know, cells, continuous cells and culture,
3619 to primary human cells, to the animal models, and
3620 demonstrated that it not only worked against SARS and MERS,
3621 but it worked against all these other bat coronaviruses,
3622 other human coronaviruses, other animal coronaviruses, 12
3623 different viruses.

3624 So we knew it had broad spectrum. So now the hypothesis is,
3625 you have a broad spectrum drug. Any new virus comes along,
3626 you immediately test the hypothesis and evaluate remdesivir,

3627 molnupiravir, Paxlovid, therapeutic antibodies, vaccines, to
3628 see if they provide breadth. And simultaneously, you use
3629 that information in a reiterative fashion now to develop
3630 broader-based vaccine platforms.

3631 So one of the innovations that we did was to take spike
3632 glycoproteins across the phylogenetic tree, blend them
3633 together as a chimera, delivered on mRNA vaccine that would
3634 provide neutralizing breadth against a greater percentage of
3635 the strains.

3636 Q So would it be accurate to say that research
3637 on a pathogen that's not yet infecting people gives
3638 scientists a basis to make their hypotheses for how a
3639 pathogen that is infecting people may react to therapeutics
3640 or a vaccine?

3641 A It's more than that. It's absolutely
3642 essential. You have no idea of the breadth of performance of
3643 your product if you don't have natural isolates available in
3644 the virus family.

3645 So, for example, calls to shut down discovery work in the
3646 natural world will basically mean that the U.S. is at greater
3647 risk for future emerging diseases because we don't know
3648 what's there, and we can't test products against it.

3649 Q Agreed.

3650 Ms. Yass. And I think that leads into some questions my
3651 colleague will have for you.

3652 BY MR. McAULIFFE.

3653 Q Good afternoon. Will McAuliffe from the

3654 Energy and Commerce Committee.

3655 You mentioned a lot about, I think, things that are sort of

3656 fairly out of our control, both the American scientific

3657 enterprise and then certainly the U.S. government, in terms

3658 of what other countries do, wildlife trade, markets in urban

3659 centers that may be engaging in things that are risky from a

3660 natural spillover and viral evolution context, right? I

3661 mean, as you said earlier, some of that is like a political

3662 question, it's not really somebody in the government here can

3663 push a button and change what everybody else is doing.

3664 A That's absolutely correct.

3665 Q Despite what we would like to do sometimes,

3666 often, maybe. So thinking of the things that are in our

3667 control, and following up on some of the things that Alicia

3668 was talking about, it seems like leading up to the COVID-19

3669 pandemic, there was already an anticipation, as a result of

3670 SARS and MERS, that this is a type of virus that is going to

3671 continue to present a threat to people that we need to be

3672 looking closely at. Is that fair?

3673 A Yes, with the caveat that many scientists and

3674 many public health officials felt that the risk was very low,

3675 and that's because the original SARS strain was controlled by

3676 public health intervention strategies, completely because you

3677 didn't transmit that various until you got really sick, and
3678 asymptomatic spread was zilch.

3679 With MERS, it didn't transmit efficiently except for a few
3680 super spreaders, like, transmitted it really efficiently,
3681 which actually tells you a little bit about the potential,
3682 right?

3683 Asymptomatic infections occurred and they could transmit,
3684 which is a little bit different, but it wasn't very
3685 efficient. It could be controlled by public health
3686 interventions.

3687 So the -- I'm forgetting the word. Standard is not the word
3688 that I want, but the standard in the field was that if a
3689 coronavirus emerged, it would be subject to control by
3690 classic public health intervention strategies. And that was
3691 lunacy to me, because human coronavirus OC43, HKU1, 229E, and
3692 NL63 transmitted efficiently and have been transmitting
3693 efficiently for anywhere from 100 to 800 years in human
3694 populations. And in the animal world, efficient transmission
3695 and pandemics were occurring. That means they have the
3696 rudimentary intrinsic capacity to do that.

3697 We just got warned. That's how I viewed it. We were warned
3698 that nature had some things in store for us and we weren't
3699 paying attention to it.

3700 Now, in NIH's defense, they funded research specifically to
3701 do work on developing drugs against coronaviruses. They

3702 funded work with Barney Graham and our group to develop mRNA
3703 vaccine technology. We were eventually going to get to
3704 nanoparticle-based technology, but the pandemic hit before it
3705 was there.

3706 So NIH had it on their threat list and were supporting
3707 fundamental research, which in the end, saved millions of
3708 lives across the globe, but there was resistance to that
3709 idea, and many health officials thought that it wasn't going
3710 to be an issue.

3711 Q Is it fair to say that that kind of resistance
3712 can result less from a desire to potentially downplay a
3713 threat altogether versus choosing among competing priorities
3714 of threats to people with limited resources?

3715 A Absolutely. I think -- I can only speak
3716 for -- I can't even speak for NIH. I can speak for what my
3717 opinion is, right?

3718 Q Yes.

3719 A So my understanding is NIH uses data to
3720 determine policy. The experiments with transmissible
3721 flu -- I need something to drink, excuse me.
3722 The experiments with transmissible flu were to address a
3723 question about policy. And the virus had emerged in '99, it
3724 was still around in 2009, half the scientific community was
3725 saying there's some risk or some fraction. Some fraction of
3726 the community was saying it couldn't get through fitness

3727 trials to be able to cause -- to be transmissible. Never was
3728 going to happen.

3729 The other part of the community said, yes, that it could.

3730 And NIH is spending a lot of money on surveillance, vaccines,

3731 developing drugs, spending a lot of time and resources on

3732 this. They wanted to know the answer. So they had meetings

3733 with the WHO, and the FDA, and the USDA, and the CDC to

3734 determine priorities. And the priority was, we need to ask

3735 the question, is transmissibility possible.

3736 The answer was yes. And that continued to result in drugs,

3737 surveillance. You can go to the CDC site and get a whole

3738 list of mutations that are associated with pathogenesis or

3739 transmission.

3740 So these types of questions provide information for policy.

3741 Policy then implements it in terms of some kind of strategy

3742 to try for preparedness.

3743 Did I answer your question? I get off on a tangent. I'm

3744 losing focus.

3745 Q This is all very interesting. Don't worry

3746 about it. I think one of the questions I have, then, is

3747 investments like the ones that NIH made prior to the COVID-19

3748 pandemic, there were folks during the time of those

3749 investments who thought maybe those weren't as wise as other

3750 investments that could be made.

3751 A Absolutely.

3752 Q Now, we're sitting here with the benefit of
3753 hindsight.

3754 A Yes.

3755 Q And again, I'm sure those people had other
3756 very good, pressing concerns. But is one of the lessons, as
3757 we sit here trying to figure out what should we bring back,
3758 what does Congress do, is one of the lessons to make sure
3759 that there are adequate resources for NIH and other research
3760 institutions, such that even within prioritizing, you're not
3761 having to wholesale exclude a category of threats because you
3762 think it is less at a time. And there can still be
3763 background work that is happening at all times that may
3764 suddenly, over the course of weeks, become incredibly
3765 relevant to the entire world?

3766 A That's correct. And a potentially risky
3767 experiment may be in the pipeline in making that decision.

3768 Q So that's what I want to talk about as well.
3769 I think you gave a very helpful background on how we should
3770 sort of think about risk, and that it seems like some of the
3771 folks who are thinking about risk the most are those who are
3772 physically entering into a lab and interacting with different
3773 things that pose different kinds of risks under different
3774 kinds of circumstances.

3775 But I think, with all the understandable discussion that
3776 we've had about risk at top of mind, the potential or actual

3777 reward, I think, can sometimes get pushed to the side, or the
3778 reason for why it is being done.

3779 And folks who aren't familiar, who haven't sat in a room and
3780 listened to this and been educated numerous times by

3781 scientists about why this work is done, could sort of walk
3782 away from reading an article or seeing a headline and

3783 thinking, why would we touch viruses? Why would we think

3784 about it? This seems dangerous, these are dangerous things.

3785 Why can't we just sort of, like, leave it alone and just

3786 treat whatever we have that we know exists and people are

3787 getting sick with.

3788 But it seems like one of the reasons for this work, and I'm

3789 curious -- correct me on this. One of the reasons for this

3790 work is, as you said, viruses are constantly evolving on

3791 their own. It's not like they only evolve in a lab.

3792 Frankly, that is a tiny sliver of where anything with a virus

3793 is changed. It is evolving and changing many, many, many

3794 times over all across the globe.

3795 A And looking for new niches to colonize, yes.

3796 Q And some of them may pose a very distant

3797 threat, and then there may be some currently in animals that

3798 are on the cusp of becoming an actual threat to the human

3799 population.

3800 A That's correct.

3801 Q So one of the things I've come to understand

3802 from all these conversations is some of the work that is
3803 happening in a lab where you are examining and altering a
3804 virus to something that at least we don't know yet has
3805 happened in nature, we haven't collected it from nature, but
3806 it may well exist, is to be able to sort of see around the
3807 corner and say, this is where nature may be heading next.
3808 And what would that mean for the human population and what
3809 defenses do we currently potentially have against it? Do
3810 they work? Do we need something new?
3811 Is that a fair assessment of why you do viral alteration in a
3812 lab?

3813 A Well, that's the fundamental reason that we
3814 built the chimeras in the 2015 and 2016 paper, was to assess
3815 the threat level that existed in nature. And it was either
3816 going to be a very rare event, or it was going to be more
3817 frequent. And our data said that there was a large reservoir
3818 of viruses that could potentially be threats, and that we
3819 needed to develop countermeasures of some kind.
3820 That was not done through policy of the NIH. Those
3821 particular experiments were done at the individual level.

3822 Q So again, thinking of folks who hear about the
3823 term gain of function or hear about viral work in labs, it
3824 can sound scary. I mean, it is scary if you're not doing it
3825 right.

3826 A Yes, it could be. It could be very scary,

3827 yes.

3828 Q But the goal is not to come up with something
3829 that nature wouldn't, just out of curiosity and your
3830 fascination and to just spend grant money and see what
3831 happens. The purpose is more to anticipate where nature may
3832 be heading next on its own, and be a step or two steps ahead
3833 in terms of being able to either develop new practices,
3834 whether it's public health policy, whether it's therapeutics,
3835 vaccines, other countermeasures. The point is to be ahead of
3836 nature, not to do something that nature otherwise may not,
3837 and create some new kind of risk?

3838 A Well, again, just to make sure we're all on
3839 the same page, in the '90s, I participated in a large number
3840 of studies that actually demonstrated that coronaviruses
3841 could undergo RNA recombination at high frequency.
3842 So that means if you took two coronaviruses that were
3843 somewhat closely related and put them in cells at the same
3844 time, 30 percent of the progeny are recombinants. That's the
3845 highest among any of the RNA viruses. So this is a normal
3846 mechanism that coronaviruses use to cause diversity.
3847 So I think there was a question earlier, could you take parts
3848 of different viral genomes and sort of build the SARS-CoV-2.
3849 Actually, the recombination analysis using natural isolates
3850 says SARS2 is a creation from three or four recombination
3851 events with animal strains.

3852 Now, keep in mind that that kind of analysis is only as good
3853 as the sequence of the number of genomes you have, right? So
3854 if you get double the number of genomes, you may find, well,
3855 this region wasn't really a recombinant, it was evolving by
3856 natural -- by genetic descent from an ancestor.

3857 But in general, recombination processes are fundamental to
3858 how coronaviruses replicate. So for a corona virologist,
3859 building a chimeric spike in the laboratory isn't doing
3860 anything different than nature does all the time.

3861 Q That's very helpful. In terms of being able
3862 to monitor viruses in wildlife, understanding that we will
3863 never have perfect information as much as we wish we could,
3864 there's simply too many animals, too many things going on.
3865 Is it fair to say that one of the lessons from the pandemic
3866 is that wildlife monitoring is an essential part of our
3867 pandemic preparedness and potential response? Should we be
3868 doing as much or more of it, I guess, as we were prior to the
3869 pandemic?

3870 A I think so, because there's pretty clear
3871 networks in terms of how natural products flow from the wild
3872 into small cities to large cities. It's like airline
3873 networks, you know, they can say these three cities in the
3874 world are the most likely cities to experience a pandemic
3875 first, just because of flights.
3876 We can do the same thing with how products travel from very

3877 rural areas to urban areas. And that's one of the goals of
3878 the Southeastern -- the center grant that we are on emerging
3879 infectious diseases, is to try to track those conduits, so
3880 that you know where to place a surveillance network that
3881 would capture these emerging coronavirus or pathogen events
3882 that occur from nature and animals.

3883 Q And having advanced notice of viruses that are
3884 either prime to jump into humans or maybe prime to jump into
3885 an intermediate host, and then into humans, that's the ideal,
3886 right, if we could actually spot it before it made the jump
3887 into the humans, and say, this will infect humans inevitably,
3888 and we can take steps now in terms of medicinal
3889 countermeasures, but also maybe isolating populations,
3890 changing animal populations, changing practices, being able
3891 to take steps before it jumps, or maybe just immediately
3892 after. It may happen in a more rural area.

3893 A I can build a really nice example of this, is
3894 public health intervention strategies. So SARS 2003 emerges
3895 as an R0 and transmits to about three people. SARS2 emerges,
3896 transmits to about 2.8 people. They have the same
3897 transmission rate.

3898 When you apply public health intervention on that, the
3899 original 2003 strain now went below 1 to 0.7. SARS2 went to
3900 1.4. What that means is the doubling time went from three
3901 days to 15 days. What happens in that interval? You have

3902 more time to develop countermeasures. It's not perfect,
3903 masking and social distancing was not perfect, but it was
3904 slowing the spread.

3905 And one of the things you do not want to be in the beginning
3906 of the pandemic is one of the first patients in the hospital
3907 with a new disease, because physicians don't know how to
3908 treat it, and they are using historic references of this
3909 organ disease to try to figure out how to treat the clinical
3910 symptoms. That means they're, to some extent, making
3911 intelligent guesses, and they don't always work out. So
3912 people die. And the physicians communicate and they say,
3913 this didn't work or that didn't work, but this is working.

3914 And the clinical medicine gets better within about a month or
3915 two.

3916 At that point, they stop -- you know, two or three months in,
3917 they stopped using respirators. Why? Because the
3918 respirators were causing all kind of sheer stress in the
3919 alveolar region of the lung that were killing people who had
3920 COVID because there was so much damage in that region anyway.

3921 And they rolled them over and they gave them different
3922 breathing apparatuses and the survival rate went up.

3923 Those kind of things occur in the beginning of a pandemic.

3924 So it doesn't matter -- if you don't like social distancing,
3925 after six months or after eight months, the importance of
3926 those actually falls, but in the beginning, it's so

3927 dramatically important. And any kind of early surveillance
3928 has this big impact on the survivability of the population
3929 and individuals' health.

3930 And so rapid diagnosis, rapid intervention with public
3931 health, doing whatever you can to slow that spread to give
3932 physicians time to learn with less patients than having the
3933 hospital filled with them, and the clinical medicine gets
3934 better and more people survive. So all of that is
3935 intricately linked.

3936 Q Thank you.

3937 A Later on, it's probably of less value, but in
3938 the beginning, absolutely critical.

3939 Mr. McAuliffe. Understood. We can go off the record.

3940 (Recess.)

3941 Mr. Benzine. We can go back on the record.

3942 BY MR. BENZINE.

3943 Q I want to discuss the NIAID grant processes a
3944 little bit.

3945 A Sure.

3946 Q And you can sense some of the confusion from
3947 the Chairman on how steps in the process, especially for
3948 foreign labs and foreign collaborators including biosafety.
3949 But I want to talk about the scoring process really quick.
3950 If a grant receives a fundable score, the lower the better,
3951 does it guarantee that it will be funded?

3952 A Usually if it's within the pay line, it will
3953 be funded, unless there's some flag that comes up during the
3954 post review process.
3955 So in essence, the review committee will rank order the
3956 grants based on scientific merit. That information then goes
3957 to council, where typically program officers do short
3958 presentations on each of the programs, each of the projects
3959 that are sort of in the fundable category, and there will be
3960 discussion there.
3961 If there are concerns, there will be another round of review.
3962 I don't know whether it occurs before it or after, quite
3963 frankly, but there will be another -- like, if there's GOF or
3964 DIRC considerations, those will have to be satisfied before
3965 the money is released.
3966 I don't know if there's instances where grants that receive
3967 really fundable scores were then not funded at council. What
3968 typically happens at council is that the National Institutes,
3969 all the different institutes, have priority areas. And so
3970 grants that come close to those, close to fundable scores
3971 that would make the percentiles, but are in high priority
3972 areas, they're usually pulled into council and then presented
3973 for special consideration for funding.

3974 Q Okay.

3975 A And that usually -- it usually, as I said,
3976 requires that it meets one of these criteria of special

3977 emphasis areas within one of the institutes.

3978 Q And then during the course of the grant, is it
3979 the principal investigator's responsibility to monitor
3980 sub-grantee compliance with the terms and conditions?

3981 A The PI of the grant is responsible for all of
3982 those issues, yes. Typically, those are all set up before
3983 the grant of money is released to any of the subs.

3984 So you have to show your animals, you know, your animal use
3985 forms are in compliance. If you are doing DIRC or GOF, that
3986 has to have been reviewed, and there has to be some
3987 resolution to whatever was presented. Biosafety of the
3988 facility has to be validated by the university, and the
3989 university will then review and sign off on all that stuff.

3990 Q So that touches on one of the questions. From
3991 all the people we talked to at NIH and NIAID, it's been
3992 unclear how the U.S. government vets foreign labs' biosafety.

3993 A I think the best answer you can get to that is
3994 to talk to them about what they did with Fouchier's
3995 laboratory with the transmissible flu, because I think there
3996 was some vetting of that facility before he was allowed to
3997 proceed.

3998 I'm also 99 percent sure that was not done in China, for
3999 example, right? They receive some certification and
4000 accreditation for their BSL-3/BSL-4 facility based on Chinese
4001 regulatory, but I don't -- I have not run PI foreign grants,

4002 so I don't know exactly how NIH deals with that, or whether
4003 they do deal with it.

4004 Q Another question we've had is obviously
4005 there's biosafety and security regulations that govern how
4006 you do things. You've taken it a little bit of a step
4007 further of erring on the side of caution.

4008 A We try to.

4009 Q And if you don't know, you don't know. But
4010 for U.S. money going abroad, do the foreign labs have to
4011 follow U.S. standards or is it the standard in the country
4012 that they reside?

4013 A I don't know the answer to that. For BSL-4,
4014 it would be straightforward. Yes, the standards are pretty
4015 much uniform across countries just because of the cost of
4016 building those facilities.

4017 BSL-3 is much more difficult. BSL-2, probably more similar
4018 across countries except for certain pathogens. And I told
4019 you one gray area. Animal zoonotic viruses is a gray area
4020 because nobody really knows the threat level associated with
4021 them if there hasn't been a human infection.

4022 So you would have to ask NIH administrators how they deal
4023 with that. My guess is they or no one else probably deals
4024 with it all that well.

4025 Q So we have heard the CDC does it, the State
4026 Department does it, DOJ does it, NIH does it, the principal

4027 investigator does it. And to us in Congress, when you hear
4028 five people are doing it, it means nobody is doing it.

4029 A Well, and basically it's a sign that the
4030 regulatory framework around that particular set of pathogens
4031 is gray. And so people are -- there's individual initiative
4032 that's occurring.

4033 Q I want to shift gears and talk about EcoHealth
4034 and Dr. Daszak a little more, in specific, the grant work
4035 with the WIV.

4036 When I asked about your gmail earlier, you expressed some
4037 frustration or upsetness that that happened, that Dr. Daszak
4038 would put your gmail on things. What's your current
4039 relationship with Dr. Daszak?

4040 A I generally don't harbor a lot of ill will
4041 toward people. Peter is a good man who is trying to make a
4042 difference in the world, and he firmly believes that there
4043 are questions that need to be answered. Sometimes he's
4044 overexuberant in how he does things, and he doesn't think it
4045 through very clearly.

4046 In the case of my gmail, sending that out to everyone and
4047 saying use his gmail, don't use his regular email because he
4048 gets FOIAed all the time, ensures that I get FOIAed in all my
4049 email. And he apologized for that.

4050 Q I want to talk about -- you touched on the one
4051 log growth and there might be a couple follow-up questions.

4052 But talk about more 2020 to present, and just if you had
4053 conversations with him regarding some of the enforcement
4054 actions that NIH was taking.

4055 So in April 24, 2020, NIH sent a letter to EcoHealth
4056 terminating that grant. Did you have any conversations with
4057 Dr. Daszak regarding the termination?

4058 A I hadn't received any of the money to do
4059 anything on that grant yet when the termination notice hit.
4060 So he called me and told me that the grant had been
4061 terminated and that the EcoHealth lawyers were looking into
4062 it. So I knew about it. But in terms of how that would
4063 impact my program, that was a very small component on that
4064 grant.

4065 Q When did you get added to the grant?

4066 A After the first round. So it would have been
4067 the second round, I don't know exactly. I can't remember.

4068 Q So going into year 6?

4069 A It would have been going in -- if year 6 was
4070 around 2019 or 2020, that's when I would have been a part of
4071 it. And my role was to study a couple of the viruses that
4072 the Wuhan Institute of Virology found that they were willing
4073 to share with me. So I always viewed that as not number one
4074 or number two on the list, maybe number five or number six on
4075 the list.

4076 Q I understand.

4077 BY MR. STROM.

4078 Q I think I understand what you're saying. But
4079 when you say not one or two on the list, but number five on
4080 the list, is that as far as they are giving you the fifth
4081 most interesting virus that they had found?

4082 A Well, to be fair to them, they did the
4083 discovery work and they're going to choose the priority of
4084 what they want to work on first. And so I'm not going to get
4085 the dregs, that would be an unfair characterization, but I'm
4086 not going to get number one. I'm going to get somewhere down
4087 the list, which is okay, and I understand that process.
4088 Hopefully, it would be something that they felt would be
4089 interesting as well.

4090 BY MR. BENZINE.

4091 Q In July of 2021, Dr. Lauer informed EcoHealth
4092 that at this point -- at that point, they were 22 months late
4093 on their year 5 progress report. Did you have any
4094 conversations with Dr. Daszak regarding that?

4095 A No, that was the first set of -- that was the
4096 first grant that I was not part of.

4097 Q We've asked almost everybody this, and our
4098 understanding now is that it's common to be a little late on
4099 progress reports, but maybe not 22 months late. Is that
4100 fair?

4101 A NIH really tightened down on that timing.

4102 They used to be pretty lax, actually more lax than you might
4103 imagine, but not 22 months. You know, some people might
4104 delay -- well, there's a couple reasons to delay. One reason
4105 you can delay is, you don't have to write a final report. If
4106 you have unspent funds and you roll it over to a one-year
4107 extension, that means by definition the final report goes in
4108 at the end of that extension.

4109 So I don't know if they rolled money over and they did a
4110 one-year extension, in which case, it wouldn't be 22 months
4111 late, it would be eight or nine months late.

4112 So I would look into that and see what the scenario was. I
4113 don't know the scenario. So if they didn't -- if they didn't
4114 do a one-year extension, then 22 months is -- it's not in the
4115 middle of the bell shaped curve, it's on that side.

4116 Q Absolutely. We've also been going through
4117 this, and you touched on it a little bit, but the difference
4118 between -- we have to operate with what we know, what's been
4119 published versus what we don't know, the always kind of known
4120 unknowns.

4121 Do researchers in your field publish every experiment that
4122 they conduct?

4123 A No.

4124 Q Do they publish every sequence that they
4125 collect?

4126 A I don't believe so. Sometimes you get

4127 distracted. You can be working on an area -- we were doing
4128 several research questions on a SARS-related virus when MERS
4129 came along, and we immediately pivoted to MERS-related
4130 research, as you might expect. And then post-docs may leave
4131 and take jobs, and then you end up with a dataset which the
4132 PI has to write the paper, which is almost like death for the
4133 paper.

4134 Q That makes sense.

4135 A There are other PIs that are better than me,
4136 but I can tell you that if I have to write the paper and
4137 it's -- I'm constantly getting pulled away to do other
4138 things, and so it's just -- time passes.

4139 Q In the year 5 report, obviously before your
4140 time on the grant, EcoHealth reported an experiment that
4141 exhibited a greater than one log growth, and that experiment,
4142 or at least that data was not reported in year 4. Dr. Daszak
4143 says the year 4 experiment and the year 5 experiment are the
4144 same ones.

4145 A Can you -- was the data presented in year 4,
4146 or was it presented in year 5, or was it presented in both?

4147 Q Both, but different.

4148 A Oh. What does different mean?

4149 Q Year 5 had the actual greater than one log
4150 growth data.

4151 A Okay.

4152 Q Year 4 didn't have that. Under Daszak's
4153 grant, which we talked about, he had to immediately stop and
4154 report anything that showed a greater than one log growth.
4155 A That's correct.
4156 Q He didn't after year 4.
4157 A Or if there was an increase in pathogenesis.
4158 So did he show an increase in pathogenesis with those
4159 studies?
4160 Mr. Slobodin. It might be helpful -- I have an exhibit here.
4161 I think this would be helpful to you, Doctor.
4162 Mr. Benzine. This will be Majority Exhibit 3.
4163 (Majority Exhibit No. 3 was
4164 identified for the record.)
4165 BY MR. SLOBODIN.
4166 Q So we have a two-page excerpt from the year 4
4167 RPPR, and then a two-page excerpt -- this is all on the
4168 humanized mice experiments or experiment and the results that
4169 were reported, you know, what parts of it. If I could have
4170 you take a moment to review.
4171 A The year 4 report is on the MERS coronavirus.
4172 Q I don't know what you're looking at, on the --
4173 A The first page.
4174 Q You have page 25?
4175 A This is --
4176 Q So at the bottom, In Vivo Infection of Human

4177 ACE2 Expressing Mice with SARS-related CoV S Protein.

4178 A Okay.

4179 Q And then if you could, look at the next page

4180 at the top of the two charts.

4181 A Okay. 35B. That's here, okay. Looking at

4182 genome equivalents.

4183 Okay, what's the question?

4184 Q I will give you a little more prep here to

4185 give you the full picture.

4186 If you go to the third page of this, the excerpt for year 5,

4187 and you'll see Specific Aim 3: Testing Predictions of CoV

4188 Inter-Species Transmission.

4189 A Which?

4190 Q It's the narrative section, again at the

4191 bottom of the page. It starts off, "In Year 5, we continued

4192 with in vivo infection experiments," and then there are

4193 charts on the following page.

4194 A Mm-hmm.

4195 Q So if you go to the last page.

4196 A I need to read this whole paragraph, I'm

4197 sorry.

4198 Q Take your time.

4199 A Okay, what's the next thing?

4200 Q If you could take a moment there just to see

4201 those two charts -- I'm sorry, three.

4202 Mr. Ervin. On the last page?

4203 BY MR. SLOBODIN.

4204 Q So you have got a survival chart, you have got
4205 one with the brain tissue, and then two slides --

4206 A Pathology.

4207 Q -- with the lung tissue.

4208 A Yeah.

4209 Q So now, if you look to both excerpts, so if we
4210 can go back to year 4.

4211 A Yeah.

4212 Q There is a statement in there, and it's
4213 supported by the figure 35 on the left-hand chart about mice
4214 challenged with the WIV1 SHC014 spike have experienced about
4215 a 20 percent body weight loss by sixth day post infection,
4216 while two other chimeras produced less body weight loss.
4217 Does that body weight loss have any significance?

4218 A So for example, on figure 34 on the first
4219 page, you can see those error bars with significant markers.

4220 Q Right.

4221 A So they did statistics, right? So on the
4222 weight loss, the percentage of stark body weight on figure
4223 35, they go through day 6 and there's no statistics, right?

4224 There's no error bars. So I don't know how many -- to
4225 know -- how do you want me to answer this question?

4226 Q Well, just honestly.

4227 A I'm going to answer it honestly.

4228 Q I'm just trying to figure out what this means.

4229 A I guess I'm trying to ask the question, for

4230 you to, in essence, say they were noncompliant, you need

4231 statistical values here that show that the weight loss of the

4232 chimera was greater than the weight loss of WIV1. And they

4233 don't tell you the number of animals and they don't have

4234 error bars.

4235 Q Right.

4236 A So the data looks like they lost more weight.

4237 I would personally believe they lost more weight. But if you

4238 were thinking about it as regulatory or some sort of action

4239 against the grant, you probably need to know statistics here,

4240 because the argument you may get back, let's say people were

4241 arguing as -- if I were a lawyer, I would say, well, they had

4242 insufficient animals for statistics, so there's no

4243 statistical difference between the two, so there is no

4244 difference.

4245 That's why I was trying to answer. I wasn't trying to be

4246 circumventive. I am just trying to tell you that that's

4247 where you're going to end up with this argument.

4248 Q We're trying to get back to the oversight --

4249 A Yeah.

4250 Q -- which you were raising the opinion about

4251 cautioning policymakers about not overregulating --

4252 A Sure.

4253 Q -- important virus research. So one of the
4254 things we're trying to look at is to see, how are things
4255 being overseen? And there are obviously current discussions
4256 going on, on how that oversight process can be tweaked.

4257 A Yeah.

4258 Q And NIH took compliance actions and took
4259 certain positions on this, but we would like to get your
4260 professional judgment on a couple of questions about what's
4261 in these reports.

4262 A Okay. To add on to this.

4263 Q Yes, please.

4264 A The titer that's next in 35 has error bars.
4265 So they -- if they had sufficient animals numbers, there
4266 would be a statistical difference between -- all of their
4267 data is arguing that the WIV1 backbone that they have,
4268 especially with SHC014 spike, is more pathogenic than WIV1,
4269 which would be a gain of function in which they would then be
4270 required to have paused the experiment and told NIH that
4271 here's the data, we need to discuss it.
4272 At this point, they don't mention statistics anywhere here,
4273 and they don't talk about animal numbers, so there's
4274 uncertainty in what I just told you.

4275 Q Right. Now --

4276 A However, the biology would argue -- the

4277 biology would argue, since SHC014 likes the mouse receptor
4278 better than WIV1, WIV1 is -- we talked about it one time.
4279 The gradient of phenotypes that you're measuring, WIV1 is
4280 down here at the bottom and SHC014 is down here, you've
4281 really set your experiment up for a gain.

4282 Q Okay.

4283 A So it's probably a gain, but sort of the more
4284 compliant thing that you're thinking about is there are no
4285 statistics.

4286 Q There are no numbers. You don't know the
4287 samples.

4288 A You don't know numbers.

4289 Q Right.

4290 A So that kind of information would be really
4291 important.

4292 BY MR. STROM.

4293 Q Is there a reason that they would run an
4294 experiment like this, where they're not trying to make it
4295 statistically --

4296 A They have the statistics. They just didn't
4297 put it in.

4298 Q We were wondering if it's a pilot program?

4299 A It probably wasn't nefarious. It probably was
4300 just they were writing a report at the last minute and
4301 somebody gave them figures without error bars, and they just

4302 stuck it in. But at the same time, it leaves some
4303 uncertainty about the gain of function.

4304 BY MR. SLOBODIN.

4305 Q What about the NIH program officers? Do they
4306 just not really critically review this stuff? I mean, you're
4307 looking at this. I mean, there's some pretty basic issues as
4308 far as error bars and basic numbers, like a sample size.

4309 A Yeah.

4310 Q You tell me, because I don't live in this
4311 world. Are they that lax that they wouldn't even raise the
4312 question? I'll take that they rushed this to meet a deadline
4313 and they included this in the report, but is there no quality
4314 control at all on what's in these RPPRs on the NIH side?

4315 A There is quality control, because I've had
4316 program officers --

4317 Q Okay.

4318 A -- look at reports that we put in and ask
4319 questions.

4320 Q Okay.

4321 A The broader question is, I think what NIH
4322 should probably do is there should be some sort of specific
4323 flag on any grant that has DIRC or GOF -- that touches on
4324 DIRC or GOF with a list of things that have to be in the
4325 grant. And that's not there.

4326 So then the program officer is not just dealing with one

4327 grant, they're dealing with probably a pile of -- they may
4328 get two grants funded, two to three grants funded a year,
4329 they last five years. They may have 15, 20 grants because
4330 they also usually have several different virus families that
4331 they're studying. So they may just get lost in the workload.
4332 That's not an excuse. There's a way to deal with that
4333 probably from a regulatory standpoint that would be more
4334 efficient, and it would specifically say you need to know the
4335 answer to these questions on this particular application, and
4336 it's flagged at a higher level, it's ranked higher in terms
4337 of oversight.

4338 Q Okay.

4339 A I don't believe they do that, but they might.

4340 You should ask NIH.

4341 Q Sure. And then just on this right-hand chart,

4342 this is on the viral load in the lung tissues.

4343 A Yes.

4344 Q If you look at the bar graph, two days post

4345 infection. If I'm reading it right, and you tell me, I'm

4346 looking at the bar for WIV1, and it looks like it's 4.7 or

4347 maybe, I don't know, something like that, and the bar right

4348 next to it SHC014 is close to --

4349 A I think the bar graph on day 2 is SHC014.

4350 Q Yeah, I'm saying there's more than one line.

4351 A Oh, yeah, there's no titer in the other one.

4352 So basically, that's saying that SHC014 is going to the brain
4353 faster than WIV1.

4354 Q This is one, year 5?

4355 A This is brain.

4356 Q Oh, I'm still on year 4.

4357 A Sorry.

4358 Q So on year 4, the bar graph shows two days
4359 post infection.

4360 A Yeah, there's two logs difference in genome
4361 copy number.

4362 Q So my question is --

4363 A Almost certainly is statistically significant
4364 if they had more than three animals in each group.

4365 Q So my question is, when are these measurements
4366 taken? When would the WIV/EcoHealth have known about this
4367 result? Because I'm hearing two different things. One is --

4368 A From me?

4369 Q No, from the virology community.

4370 A Okay.

4371 Q From your colleagues. So one way, a two-week
4372 experiment with these humanized mice, testing these chimeras.
4373 They would take these whatever specimens at these intervals
4374 and then do all the testing on them or measurements all at
4375 the same time, so there's no variation on the -- in other
4376 words, you wouldn't know until the end of the experiment,

4377 until you did all the measurements. Or do you do them pretty
4378 close to realtime while -- during these intervals? When do
4379 you do the measurements?

4380 A If you're doing realtime measurements, in this
4381 case, you probably would wait until the end of the
4382 experiment. At least I would. Then you have a single
4383 standard curve, and everything is done at the same time, so
4384 you can put it on that standard curve.

4385 Q But here's the problem.

4386 A I probably wouldn't do it at day 2 and day 4,
4387 day 6. It's just the workload to set up the experiment and
4388 the time it takes to do it means you're doing it four times,
4389 versus if you did it all at once, it would be one-and-a-half
4390 to two times.

4391 Q So let's go back to this one log viral growth.

4392 A Yeah, two logs.

4393 Q Well, this is two logs here.

4394 A Yeah.

4395 Q But in terms of there was language, I think
4396 you know at this point, because it has been pretty publicly
4397 reported. But EcoHealth Alliance required it.

4398 A Tenfold.

4399 Q So my question, though, is this. The language
4400 says if you see it, you're supposed to stop the experiment
4401 and then notify the IBC and the NIH.

4402 A In their case, the WIV should have notified
4403 the PI.

4404 Q Right.

4405 A And the PI should have immediately notified
4406 the NIH.

4407 Q But when?

4408 A As soon as the PI found out within some short
4409 period of time of doing the experiment.

4410 Q So say, hypothetically -- we don't know the
4411 date of this experiment.

4412 A I do not.

4413 Q No, we don't, either. Nobody knows because we
4414 didn't get the lab notes. But it would appear maybe it was
4415 the early part of 2018, because they submitted this RPPR in
4416 April of 2018.

4417 So let's say it was conducted in January 2018, just for the
4418 sake of the hypothetical. So this experiment, first, I don't
4419 understand, if the experiment's already done by the time
4420 you're taking your measurements, then what's the point of
4421 even having that policy? It's already done. There's nothing
4422 to be stopped. It's all done. The stoppage requirement
4423 doesn't make any sense.

4424 A How would you stop something before you didn't
4425 know it occurred?

4426 Q Well, that's what I'm trying to get at.

4427 A Okay.

4428 Q You don't know when one log virus growth
4429 occurred -- in excess of one log virus growth occurred until
4430 the end of the experiment. And yet NIH is saying, well, stop
4431 the experiment if you see it. But Dr. Daszak says there's a
4432 single experiment, this was it, they split up the reporting
4433 of the results.

4434 And so -- and NIH is saying, well, there's no violation here
4435 because, yeah, there was a difference of day 2, but we only
4436 count it at the end of the experiment and then they converged
4437 again.

4438 Do you agree with that?

4439 Mr. Strom. The transient nature of the viral growth doesn't
4440 cause it to trigger the policy?

4441 The Witness. Yeah, I can't comment on what NIH or Daszak
4442 said about this. I can only give you my opinion.

4443 BY MR. SLOBODIN.

4444 Q I just want your opinion.

4445 A So there was a tenfold difference in titer
4446 early on, so that would alarm me. It was still present in
4447 day 4, and eventually by day 6 or 8 in the brain, it
4448 would -- I'm not sure -- lung tissue. At some point, those
4449 titers merged. But the other phenotype that's going on is
4450 that the chimera is causing much more weight loss, so it's
4451 more virulent. So what I would have done is stopped the

4452 experiment at that time and notified NIH.

4453 Q But the experiment is already done. That's my
4454 point.

4455 A I am going to talk about that, because what
4456 you just said alarmed me a lot.

4457 Q Yeah.

4458 A And you're suggesting that you do one
4459 experiment, you're done, you're never going to do any work
4460 with that virus again. That's not the case. There are all
4461 kinds of things you can do here, evaluating vaccines, they
4462 may want to look at host expression patterns in the animal,
4463 they may want to do all kinds of systems biology analysis.
4464 So this basic experiment here, the whole beginning to ask the
4465 fundamental question, why is the chimera more virulent?
4466 So if that regulation was in place, you're talking about
4467 another dozen set of experiments that occurred that could
4468 potentially occur along this research pipeline. And you
4469 don't want to do that.

4470 The risk of one experiment versus a dozen experiments or 20
4471 experiments is very different, okay? But the way that you
4472 just said, what's the use of it, because the experiment's
4473 over, what you've really said is you should never do any
4474 experiments at all on the potential of enhanced disease. On
4475 the potential of enhanced disease.

4476 And so if the U.S. government wants to do that regulation,

4477 they certainly have every right to put it in place and the
4478 U.S. scientific community needs to follow it, but we're going
4479 to be behind.

4480 Q I'm not implying that. What I'm implying is
4481 whether this system of oversight is adequate.

4482 A That's a very fair question.

4483 Q For public confidence.

4484 A That's fair.

4485 Q To go forward with the virus research. That's
4486 what I'm trying to explore with you, because it looks to me
4487 like there's some serious questions about this. I mean, as
4488 an outsider, it doesn't make sense. They don't talk about
4489 that this is -- like you providing a fuller context, but if
4490 you want, I can go to the letters, and maybe we'll do that so
4491 you can see the exact --

4492 A Are these comments from the PI to the NIH?

4493 Q I am going to try to shorten these up.

4494 Mr. Strom. This will be Exhibit 4.

4495 (Majority Exhibit No. 4 was

4496 identified for the record.)

4497 Mr. Benzine. One question.

4498 BY MR. BENZINE.

4499 Q Dr. Baric, you've read the year 5 paragraph
4500 now, the in vivo infection where five of the seven mice
4501 infected with just the WIV1 backbone survived, but only two

4502 of the eight mice infected with the WIV1 SHC014.

4503 A You should be able to do the statistics on
4504 that, and it should show that there's a statistical
4505 difference, which means there was an increase in virulence
4506 and the entire review process would have been triggered.

4507 Q So that's --

4508 A I think, if you did the statistics on those
4509 numbers.

4510 Q That's my question, is that this wouldn't have
4511 triggered P3 because it's not a human virus.

4512 A It doesn't matter whether it triggered P3 or
4513 not. It triggered the regulation that they agreed to in the
4514 document to follow. So if that statistics -- your problem
4515 right now is you have no statistical significance on here.
4516 So I'm just saying from kind of a legal position, you're in a
4517 gray area if you want to be successful.

4518 Mr. Slobodin. But what he just read to you had numbers, the
4519 year 5 had numbers.

4520 The Witness. That's right. But they weren't put into the
4521 figure, but they are in the text. So the data is there for
4522 you to determine statistics if you want to, if you can link
4523 it. Well, you have mortality statistics, so you can probably
4524 do that.

4525 BY MR. BENZINE.

4526 Q So my question is, and we've gotten different

4527 answers on everything, and it depends on if you're using the
4528 P3 definition or whatever definition. This reads like a gain
4529 of function to me.

4530 A Okay. So what year was this? I just want to
4531 make sure I'm in the right gain of function regulation.

4532 Q 2019.

4533 A So it's the NSABB regulation. So the NSABB
4534 regulations say a potential pathogen, a potential pandemic
4535 pathogen is a pathogen that shows increased
4536 replication -- I'm sorry, increased pathogenesis or
4537 transmissibility in humans. Humans. This gets to the DARPA
4538 grant, by the way.

4539 Natural isolates that exist in nature are not considered
4540 PPEs -- PPPs. So the backbone virus that they're working
4541 with is a natural isolate. The virus that they're moving the
4542 spike from is a natural isolate. Neither of those are
4543 potential PPPs, because they've never been documented to
4544 infect a human and they've never been documented to transmit.
4545 It's a gray area because we do know they can use human
4546 receptors.

4547 So your alarm level should go up a little bit, but it doesn't
4548 trigger the regulation because of that. Now, the chimera is
4549 a gray area because you're putting one from the other, and
4550 so -- but the regulation, I don't believe, is specific on
4551 that.

4552 The second part, the next part is that if they're doing these
4553 experiments for surveillance purposes or for vaccine
4554 purposes, even if they've engineered them and they're not
4555 PPPs, they're exempt.

4556 So the regulatory framework from 2017 actually argues that
4557 these are exempt. Now, the gray area is that -- and you have
4558 to go back to the Obama administration. They said they were
4559 concerned about SARS and MERS coronavirus. The NSABB and the
4560 National Academy of Science, I believe, said that was SARS
4561 and MERS coronavirus that were in the definition. Bat
4562 sarbecoviruses or bat merbecoviruses were not included in the
4563 definition.

4564 Other people outside of that review funnel that were not part
4565 of Obama's administration or part of the NSABB review say
4566 that that was a bureaucratic switch of the regulations that
4567 were supposed to cover all merbecoviruses and all
4568 sarbecoviruses. It never says that in the regulation. It
4569 says SARS and MERS coronavirus.

4570 So based on those regulations, yes, this is -- as my
4571 interpretation, is that, yes, these would be exempt. But is
4572 it a gain of function phenotype? Absolutely. You can't
4573 argue with that.

4574 BY MR. STROM.

4575 Q Do you think it's two experiments, the year 4
4576 and the year 5?

4577 A Almost certainly. The second one -- let's
4578 see. The first one stopped at day 6 and the second one stops
4579 at day 14. So they probably set up a repeat. Normally, you
4580 want to repeat experiments.

4581 Q To prove that they're replicable?

4582 A To make sure that they're correct. So again,
4583 that's -- the reason why one experiment triggers, because you
4584 would want to review that before you proceeded.

4585 BY MR. BENZINE.

4586 Q Should the year 4 have triggered?

4587 A I'm sorry, I keep forgetting.

4588 Q That one.

4589 A I think it should have. There's no statistics
4590 here, but I think it should have triggered a review.

4591 Q Thank you.

4592 A If you're going to put in a metric that you're
4593 supposed to respond to, you don't want it to be sloppy,
4594 right? You don't want it to be variable. You want to say if
4595 it crosses the line, you call NIH and you let them know.

4596 That's my feeling.

4597 BY MR. STROM.

4598 Q So going back to DEFUSE, which I believe is
4599 Minority Exhibit B, the proposal.

4600 A Yeah.

4601 Q That same page, and again, unfortunately, it's

4602 not numbered, but I believe it is page 4. It's got comments
4603 16 and 17 on it.

4604 A Right.

4605 Q So I would like to focus on comment 16. I
4606 realize it's coming from Dr. Daszak and not from yourself,
4607 but what is your recollection of what he's trying to convey
4608 there?

4609 A I think -- I mean, it's pretty
4610 straightforward, right? He's saying that he's going to
4611 revisit this topic if, after potential review, the
4612 grant -- and that he's going to focus it more in terms of
4613 U.S. research for work at BSL-3 than in China. And my
4614 response to that is this is a bad idea.

4615 Q So the part is -- so that DARPA is comfortable
4616 with our team. So is that to minimize the appearance of the
4617 WIV portion in the grant?

4618 A You're going to have to ask him exactly what
4619 he was thinking. I think there's a variety of ways you can
4620 interpret it, but I think my response indicated that I was
4621 concerned about his statement.

4622 Q And then but you don't recall the time, and it
4623 looks like you guys had either standing fairly periodic calls
4624 as drafts were going through iterations. I'm not sure how
4625 involved you were with those, but you don't recall that
4626 coming up in any conversations?

4627 A I recall this being a very last minute
4628 production to put the grant together. And so I don't recall
4629 many calls beyond the first one, which was to establish the
4630 team that was going to go after the question and what the
4631 question was going to be.

4632 Q Sure?

4633 A And then different groups were writing
4634 different parts that were being assembled and sent around.
4635 So some parts of the grant, I may not have seen until the
4636 last time I read it, and I never saw the final copy until
4637 after it was submitted.

4638 BY MR. BENZINE.

4639 Q Is there sort of post-award wiggle room on who
4640 does what? The way I read it, and in fairness, you're not
4641 Dr. Daszak, so we can't get into his mind, and we got these
4642 documents after we interviewed Dr. Daszak, so we're in a
4643 tough spot, too. But, once we get the funds, we can then
4644 allocate who does what exact work. Is that kind of standard
4645 that you can shift the grant after it's been awarded?

4646 A The PI has control of the budget, so they can
4647 move money any way they want. They can take people off the
4648 grants. I have removed people from grants before who weren't
4649 being productive.

4650 In essence, the PI is responsible to be a steward of the
4651 federal money and the public's money. And if people aren't

4652 doing their job, it's their responsibility to remove them
4653 from the grant. If they don't, sadly enough, they're not
4654 doing their job. I hope I've done my best over the years.

4655 Q This just seems like intentionally hiding the
4656 ball.

4657 A Yeah, the optics don't look great. I agree.

4658 Q I want to --

4659 Mr. Benzine. I'm sorry for cutting you off.

4660 Mr. Strom. You're fine.

4661 BY MR. BENZINE.

4662 Q I wish there were page numbers, but it has
4663 comment 24 on the page.

4664 Mr. Strom. Third to last.

4665 BY MR. BENZINE.

4666 Q It's in the resume section, and the comment
4667 from Dr. Daszak on this one. "I'm planning to use my resume
4668 and Ralph's. Linfa, Zhengli, I realize your resumes are also
4669 very impressive, but I'm trying to downplay the non-U.S.
4670 focus of the proposal, so that DARPA doesn't see this as a
4671 negative."

4672 This comment, taken in conjunction with the last one, seems
4673 like an intentional effort to hide the Chinese portion of the
4674 grant in order to get funding.

4675 A That's a fair question to ask him.

4676 Q Did you have any conversations with him about

4677 this while this was being written?

4678 A You saw my comment, which was again designed
4679 to stimulate, let him know that there's sort of a fundamental
4680 difference, and that this is a concern.

4681 Q All right.

4682 BY MR. STROM.

4683 Q You mentioned that in the first hour, but
4684 essentially, that you kind of forgot about the DEFUSE
4685 proposal?

4686 A Yes, I did. People probably say no chance.

4687 Q And I'm trying to battle hindsight here.

4688 A Yeah.

4689 Q But it would be helpful for context, I think,
4690 if you could share just how many SARS-related coronavirus
4691 proposals you were sort of working on in a given year,
4692 because there's about an 18-month gap between this proposal
4693 being put forward and then the pandemic.

4694 A I believe I have the record at University of
4695 North Carolina for submitting grants and getting grants
4696 rejected.

4697 Q Okay. A rough approximation in sort of a
4698 year-and-a-half period?

4699 A In one year, I know that I submitted at least
4700 20 grants.

4701 Q Okay.

4702 A Some years, it may actually be higher, because
4703 of the few times I -- so you can write grants a couple of
4704 different ways. One way is where you're a PI, where you're
4705 responsible for really putting it together.
4706 The second is co-investigator, where you're writing like a
4707 section, but you're not responsible for completely doing the
4708 entire grant. You read it and make comments but you usually
4709 don't -- you're not refining it, refining it to the very end,
4710 but you build a section.
4711 And then a third level is where you're kind of an
4712 investigator, where you're not actually leading a lot of the
4713 work, you're providing some support and you're providing a CV
4714 that says, I can do this set of experiments that they need,
4715 and I will be there to do it. But you're not actually
4716 working.
4717 So if you use that strategy appropriately, you can write a
4718 lot of grants.

4719 Q Okay. And then do you have a moment where
4720 your memory was sort of jogged about DEFUSE?

4721 A After it was released by -- I forgot the name
4722 of that group that -- the computer sleuths that found it and
4723 released it, and it popped up on the news. And I was
4724 thinking, what's this? And I read it. Yeah, I wrote the
4725 grant, part of it, yeah.
4726 I can also tell you one of the drivers that sort of stopped

4727 me thinking about that line of research was we were
4728 interested in protease cleavage sites, for example, because
4729 it was a second barrier for virus emergence. And we were
4730 having -- there were several MERS-related strains and SARS
4731 strains that we couldn't culture. We knew the clone was
4732 infectious and the virus could replicate, but it couldn't
4733 spread.

4734 So what we realized is that if we add exogenous trypsin,
4735 another protease, if you put it in the media, some of those
4736 viruses will grow. It's a simple solution to the problem.

4737 So you didn't exactly have to engineer anything to make it
4738 grow. So we published a paper on that, and we used that on a
4739 variety of viruses. It's kind of a simple solution to a more
4740 technologically different approach.

4741 Q So within this DEFUSE team, whose idea was it
4742 to sort of target the cleavage site for that S1/S2 junction?
4743 As I understand it, they occur randomly in a series of
4744 different viruses, but the location itself, the location
4745 within the genome is important for it to work.

4746 A Yeah, so it's -- there's a lot of redundancy
4747 in proteases that cleave the coronavirus spike. So to start
4748 off, the idea of manipulating the protease was clearly mine.
4749 No question.

4750 I want to take you back to what the -- I have to look at my
4751 notes here. But I want to take you back to what the proposal

4752 requested. This was in response to the National Biodefense
4753 Strategy. They wanted to improve U.S. biosecurity by
4754 detecting and containing bio threats adopted for active
4755 posture, stem ID outbreaks at the source.

4756 They wanted to understand both pathogen interactions, and
4757 they wanted to develop models that you could look at how
4758 those viruses jumped between species. And they wanted to
4759 know down to the nucleotide level, down to the nucleotide
4760 level how the viruses jumped.

4761 Now, there's two ways to do that. You can do loss of
4762 function which tells you a potential mechanism, it's not
4763 causal. And the reason it doesn't tell you that is if you
4764 knock out one of those protease sites, and the best example
4765 is with furin and SARS2 that was done later, you knock out
4766 that furin site, you knock out cleavage by two or three, at
4767 least one other restriction enzyme, which is TMPRSS2,
4768 nobody's ever measured cathepsin L, and nobody measured the
4769 other proteases that chew at that S1 boundary. But that
4770 deletion wasn't furin specific, it was a generalized
4771 processing defect, because it was a loss of function
4772 mutation.

4773 So the true interpretation of the furin cleavage site in
4774 SARS2 is that if you disrupt cleavage of spike, it's going to
4775 be attenuated because none of those proteases can chew. All
4776 right? So it's not specific. Gain of function experiments

4777 allow you to say this site --

4778 Q This is it?

4779 A -- is it, right? Now, the way the furin

4780 cleavage site was built in that grant, at least in the

4781 earlier versions, some of that may have been lost as they

4782 tried to condense it to get it to fit, was that the first

4783 part was that we were fundamentally interested in why didn't

4784 sarbecoviruses have a furin cleavage site.

4785 There had been studies done in 2010, 2011, 2012 using

4786 pseudotypes. Catherine Holmes published one in JB, there was

4787 a Chinese group that published it, where they dropped the

4788 furin cleavage site into the SARS1 from 2003. There was no

4789 increased infectivity, there was just a little bit more

4790 fusion between the cells. So no really big phenotype.

4791 Another example of furin cleavage sites with coronaviruses, a

4792 researcher at University of Pennsylvania knocks out the furin

4793 cleavage sites in mouse hepatitis. No change in pathogenesis

4794 for the ability of the virus to replicate.

4795 Feline infectious peritonitis virus, it's an enteric form,

4796 it's got a furin cleavage site, it replicates, and it got

4797 very mild infection. When the furin cleavage site is lost,

4798 it kills the cat. So it's a flip, right? Furin cleavage

4799 site is the loss of -- it's protecting from virulent disease.

4800 So the data going into that proposal, the exact role of furin

4801 cleavage site was not clear. We were interested in it

4802 because most other coronaviruses in family had those sites.

4803 Why didn't sarbecovirus?

4804 So the way the grant was designed was that the discovery
4805 group would look, as they did discovery, if they found one
4806 with the furin cleavage site, we would first study the
4807 pseudotypes.

4808 The second thing we would do is move it into the chimeras to
4809 see what the effect on applicants was. The third thing was
4810 we would probably build virulent viruses and study
4811 pathogenesis, and then we would knock out the furin cleavage
4812 site.

4813 Q As I understand, to see what you've got?

4814 A To see what would happen. If you knocked it
4815 out and you lost all the virulence, then you're going to
4816 think twice before you start dropping it into things, right?
4817 So it's a step-wise process. It's not like it's portrayed in
4818 the news where researchers were going to take furin cleavage
4819 sites and just shotgun them into every coronavirus they could
4820 find until they found something happened. It was a
4821 systematic process that was initially designed.

4822 And it wasn't just the furin site. It was also TMPRSS2
4823 sites, it was also HAT, and the cathepsin L protease. So
4824 there were four proteases we were interested in.

4825 Q Was there also an effort to identify, and it's
4826 maybe RMYN02, if that's the one I'm thinking of that has a

4827 partial?

4828 A That was published after, I guess, SARS2
4829 emerged.

4830 Q Would that have been one that if this project
4831 had been done, that you -- the team would have been
4832 interested in to see what additional -- I guess I'm
4833 wondering, you talked about --

4834 A It didn't have a full furin cleavage site,
4835 just two or three of the residues. It was close, right?

4836 Q Right.

4837 A And so some people argue it was on the way to
4838 get a furin cleavage site, but I personally don't believe
4839 that. It just had additional residues in there, so --

4840 Q And then on the other aspect of looking -- and
4841 this may relate to sort of the search for a broad spectrum
4842 coronavirus vaccine. What was the rationale between looking
4843 for a SARS-related coronavirus that sort of a 10 to 20
4844 percent divergent in the spike from SARS1?

4845 A Sure. So SARS 2003 is the bookend, right?
4846 You know how much variation. WIV1 and SHC014 have about 8 to
4847 12 percent variation in the spike or the RBD. The clade 2
4848 strains like HKU3 have 30 to 35 percent variation in the
4849 spike, they've got deletions in the RBD, they can't use human
4850 ACE2 receptors.

4851 If you take those two numbers, subtract 10 or 12 from 35,

4852 divided by 2, added to 12, you get a number between 20 and
4853 25. And that was our prediction, that there would be strains
4854 with that much variation that could still use human ACE2
4855 receptors.

4856 It turns out SARS2 had 22 percent variation, so we were
4857 within the range, but we were really not completely right.
4858 In MERS, there are strains with 35 percent variation in the
4859 RBD that could still use the human. So in reality, it's
4860 probably much greater than 20, 25 percent.

4861 Q Really?

4862 A That was our estimate. And the reason we're
4863 interested in that, the strains with the most variation
4864 become important for developing countermeasures in vaccines.
4865 So if you have a strain that's really different than
4866 therapeutic antibodies, you can look for broadly neutralizing
4867 antibodies. They may not work. Your vaccine, if you have an
4868 animal model, you can ask, does it cover this much variation?
4869 And if it doesn't, it gives you the starting material to
4870 develop a second generation vaccine that can capture it.
4871 So again, that variation -- I have no interest in simply
4872 resurrecting every single coronavirus.

4873 Q Sure.

4874 A I'm interested in the bookends and a couple
4875 intermediate ones because that's what's best for
4876 countermeasure development.

4877 Q And this came out in the recent FOIA release.
4878 I can make it an exhibit if it's helpful. But there was a
4879 call about PREEMPT EcoHealth and Ralph is the title, March 2,
4880 2018.
4881 There's a bullet here that says, "another idea is...if you
4882 build chimera that broadly reduces heterogeneous population
4883 of SARS-related coronaviruses in bat caves, this might be
4884 something you'd want to develop for humans.
4885 "RB has already generated SARS-like chimeras with RBD from
4886 group of bat viruses called 293, which is 20 percent
4887 different" -- sorry, "(for S1), which is 20% different than
4888 the epidemic strains."
4889 Mr. Ervin. Could we look at that?
4890 (Majority Exhibit No. 5 was
4891 identified for the record.)
4892 The Witness. So in 2008 or 2009, we had a PNAS paper where a
4893 clade 2 SARS-related virus called HK3, which is about 30, 35
4894 percent different than SARS, we made a molecular clone for
4895 that, and it could infect cells and it could replicate but it
4896 couldn't spread to the next cell.
4897 So we did an experiment with Vanderbilt University where we
4898 took the receptor binding domain of the 2003 SARS strain and
4899 swapped it into the HK3 backbone. So we built a chimera.
4900 That virus could grow, but it was highly attenuated in mice.
4901 I can't remember the growth curve comparisons.

4902 BY MR. STROM.

4903 Q HKU3 is one of the standard cold causing
4904 viruses?

4905 A No, HKU3 is a bat coronavirus that is very
4906 different. So the coronavirus tree with three branch -- I
4907 can't use these. No, I can't do that.

4908 Q Anyway.

4909 A So the three branches --

4910 Q It's not videotaped, so you're all right.

4911 A That's good.

4912 Q But so the same three group of viruses.

4913 A It's called -- there's a clade 1A, which is
4914 SARS 2003; a clade 1B, which is SARS2; and a clade 2, which
4915 is bat strains that don't grow on human cells, don't use
4916 human ACE2 receptors. They have deletions in their receptor
4917 binding domains, so they don't even engage human receptors.
4918 Those could replicate, but they couldn't cause disease. So
4919 we wanted -- we were asking a fundamental question about
4920 recombination. Are the RBDs interchangeable between
4921 coronaviruses by recombinatory practices. And so we inserted
4922 the SARS RBD into the HKU3 backbone and it replicated. It
4923 was attenuated in mice. We ultimately passed it in mice and
4924 made a more mouse-adapted strain.

4925 Why would we want to do that? Well, variation in the
4926 polymerase is important for testing drugs without breadth.

4927 Was it 293, is that what it says?

4928 Q The group of bat viruses, generates SARS-like
4929 chimeras with RBD from a group of bat viruses called 293.

4930 A So the experiment I just told you about was
4931 2008 or 2009. We took that backbone around 2012 and
4932 introduced a triple chimera. In essence, it had, if I
4933 remember correctly, the HKU3 NTD, the SARS1 RBD, and the S2
4934 domain from this other bat virus. I actually don't think
4935 it's 293, I think 3 is a typo. It might be 96, but I would
4936 have to look at the recombinant DNA thing that I submitted to
4937 UNC, which I have, by the way.

4938 So in 2012, in the fall of 2012, we made that virus and had
4939 recovered it. And then MERS kind of hit and then we didn't
4940 do very much on it besides showing that it was replication
4941 competent.

4942 Q Okay.

4943 A So this is a clade 2, clade 1A chimera. It's
4944 got mostly the HKU3 backbone, but what it showed is that all
4945 three major components of the spike glycoprotein are
4946 interchangeable.

4947 Q And then my last question relating back to
4948 something that Dr. Wenstrup asked, I guess --

4949 A And that was before any GOF regulations were
4950 in place, so it was IBC approved at UNC.

4951 Q As of like December 2019, what was, I guess,

4952 the SARS-related coronavirus you had at UNC that would be
4953 most similar -- we'll start with sort of the whole genome
4954 level to SARS-CoV-2. Even if it's just a percentage, if you
4955 can't remember the specifics or in-house designation for it.

4956 A All the clade 1A strains, like SARS, SCH014,
4957 WIV1, are anywhere from 22 to 25 percent different than
4958 COVID-19. The HKU3 virus, I don't remember how similar it is
4959 to -- I would have to go back and look at the data. I would
4960 be surprised if it was less than 1A, because it has so much
4961 more variation to begin with.

4962 Q I guess my question is, Shi Zhengli went back
4963 to her holdings and found RaTG13. I don't know if you did a
4964 similar one just to see if you had something similar from a
4965 previous --

4966 A I don't do surveillance.

4967 Q Well, that would be --

4968 A So I don't go out and collect bat samples. I
4969 had a research assistant professor that did some bat
4970 discovery work in Maryland, and he found mostly group 1
4971 coronaviruses at the time. So we didn't -- I don't do bat
4972 discovery, so I don't have large repositories of bat samples
4973 to look for coronaviruses.

4974 Q Okay.

4975 A I usually look for sequences, and if I find
4976 something interesting, then I'll go after it.

4977 Mr. Benzine. I have one final question.

4978 BY MR. BENZINE.

4979 Q Notwithstanding what we talked about earlier

4980 and discussed, at any point during the intelligence

4981 community's review of the origins, were you contacted by any

4982 agencies?

4983 A FBI, CIA, and many other three-letter

4984 agencies.

4985 Q Okay, to help with their review?

4986 A Yes.

4987 Q And did you tell them substantially what you

4988 told us today?

4989 A I did. I said there were three potentialities

4990 for the origin.

4991 Mr. Benzine. Thank you. We can go off the record.

4992 (Discussion held.)

4993 Mr. Benzine. We can go back on the record.

4994 BY MR. SLOBODIN.

4995 Q So why did -- when we're reading the grant

4996 documents -- we're going back to the humanized mice

4997 experiments.

4998 A This is the EcoHealth ROI in the first five

4999 years of the grant.

5000 Q Right.

5001 A Okay.

5002 Q And the mice -- as I understand, the mice for
5003 that experiment were obtained from your lab?

5004 A I don't believe so, but I don't know for sure.

5005 Q Well, you were telling us before that you had
5006 the mice, that you were curious about them commercializing --

5007 A That's correct.

5008 Q -- the mice you shared through an MTA?

5009 A Yes. And the discussions to send those mice
5010 to them started in 2015, and I think I told you I was unsure
5011 of whether they got them in '16 or '17, and when they had
5012 sufficient numbers to do it.

5013 Q Why would they want your mice? There's plenty
5014 of mice in China. In the grant documents here, they said
5015 they got them from Wuhan University. So what was it that's
5016 special about your lab's mice that they wanted them?

5017 A I knew that researchers in China developed
5018 humanized mice in 2004 at Peking University. And actually, I
5019 tried to get those mice and they tried to send them to me,
5020 and the Chinese government sort of shut it down. That
5021 researcher got out of coronavirus research, so I assume he
5022 left the colony. And I didn't know that they had access to
5023 humanized mice. I got a request and I responded to it.
5024 So I don't know if these were my mice that came from our lab
5025 or not. It's a good question to ask. I don't know.

5026 Q But you didn't get any details from them in

5027 the request about why they were coming to you?

5028 A No, I think the MTA agreed that the first
5029 paper they published with it, they would include me as an
5030 author, and that was the 2020 paper.

5031 Q Did --

5032 A On SARS2.

5033 Q Did they include any specifications, like age,
5034 gender, type of mice?

5035 A In the Cell paper?

5036 Q No. When they wanted to -- when they were
5037 trying to get --

5038 A No, they just request mice. So you send the
5039 breeding pairs, and then they breed them.

5040 Q Okay. What is the scientific basis for the
5041 one log difference in virus growth being used as sort of a
5042 marker, a benchmark as you called it? Where does that come
5043 from?

5044 A Plaque assays have some level of variability
5045 in the ability to distinguish between differences. So
5046 there's about 15 to 20 percent variation in plaque assays.
5047 So if you take a virus ten to the sixth, and you do a series
5048 of plates with the same stock and titers, you'll see titers
5049 ranging from like -- I have to do the math -- eight times ten
5050 to the fifth. That's not the right number, I'm getting
5051 tired.

5052 But you're going to get a range between like eight times ten
5053 to the fifth, and two times ten to the sixth, so you get some
5054 variability in the response just because of the distribution
5055 of viruses in the 200 microliters that you take out of the
5056 sample and place on the plate.

5057 Q Is there a study on that? How did it become a
5058 standard? Is that something you've always done through your
5059 career as a virologist?

5060 A For virus titer? Yeah, I started in graduate
5061 school.

5062 Q So it had nothing to do with a gain of
5063 function regulation?

5064 A It had nothing to do. The tenfold value
5065 was -- I think was -- well, we were asked to come up with a
5066 metric. A tenfold value, you can be pretty sure is
5067 statistically significant.

5068 In general, in humans, there's a correlation between
5069 increased titer and disease, so that means there's some level
5070 of potential risk even though we know that host genetics can
5071 make a big difference in that, so -- but that's not really
5072 what the purpose is.

5073 The purpose is to have some kind of metric that provides a
5074 meaningful bar that you use to initiate additional review
5075 processes. There are other ones that you could use. You can
5076 use the degree of fusion, but that's really hard to measure,

5077 especially in 2014, 2015, 2016. You know, how big the fused
5078 areas are, how many nuclei are in the fusion area.
5079 There are other metrics you can use. But this was a very
5080 straightforward, very definable, quantifiable measure that is
5081 meaningful. And we felt that was -- that if you saw that
5082 difference, then you should at least pause and discuss it.

5083 Q Okay.

5084 A Some others may disagree.

5085 (Majority Exhibit No. 6 was
5086 identified for the record.)

5087 BY MR. SLOBODIN.

5088 Q So this is a letter from the NIAID vice
5089 chancellor to you. I'm only interested actually in one
5090 sentence on the second page.

5091 A All right.

5092 Q And it's at the bottom. And it's the last
5093 paragraph, the first sentence that says, "NIAID acknowledges
5094 that if any unanticipated outcomes are observed, including
5095 enhanced virus growth greater than one log in any mammalian
5096 cells, enhanced virus titers by greater than one log in any
5097 mammalian cells, or enhanced clinical disease or death in
5098 mice as defined by significantly increased weight loss,
5099 percent mortality, or decreased mean day to death, you will
5100 immediately stop all experiments and notify NIAID and the
5101 UNC-Chapel Hill IBC of the results."

5102 So where did that formulation come from? Because that's not
5103 just on virus. This seems to be a little more -- how would
5104 you describe it?

5105 A It's absolutely to the letter of the State
5106 Department's gain of function pause in 2014. So the way the
5107 pause of 2014 read was any increase in pathogenesis or
5108 transmissibility in any mammal, okay, any mammal. All 6400
5109 of them that exist on Planet Earth, there's only one BSL-3
5110 facility that handles aquatic species, and the whales can't
5111 fit in them. There's no whale cell lines that I know of.

5112 So this was an impossible metric for any scientist to follow.

5113 NIH recognized that after they -- this came down from the
5114 State Department, it didn't come from the NIH.

5115 In the NSABB, the revived regulations of 2017, they dropped
5116 the mammal requirement because it was experimentally not
5117 doable.

5118 So the way that regulation really should have meant is anyone
5119 doing a gain of function experiment needs to stop now because
5120 you cannot measure it in every single mammal, either as a
5121 cell line or whatever, because they don't exist.

5122 Also, who wants to do it? You know, you have to test it in
5123 6400 cell lines. Really? I'm not going to do that
5124 experiment. I'm not going to do the experiment at all,
5125 because it's crazy.

5126 And so in the revised revision, they dropped any mammal and

5127 focused on humans, which was reasonable, at least in my
5128 opinion. But you see the dichotomy, how can you do it? And
5129 if you want to see animal in vivo studies, there's one BSL-3
5130 facility with water in it in the United States, and it's for
5131 little things, not for whales.

5132 Q So the question to take away on this lesson,
5133 on overseeing these types of research proposals where there
5134 are risk issues, should there be one consistent standard that
5135 every researcher has to meet? And two, should it specify
5136 certain data elements that should be included with a certain
5137 level of detail?

5138 A Statistics should be there.

5139 Q Okay.

5140 A Statistics definitely should be there. I like
5141 the 2017 regulations, quite frankly. I've lived by them, I
5142 think they're appropriate. They're focused on pathogens that
5143 are risky. The DIRC regulations don't include any
5144 coronaviruses, but they cover 15 pathogens and six or seven
5145 experiments of concern which are well articulated. So it's
5146 very well articulated. Things get added to that list as the
5147 scientific community says, hey, there's a pathogen here that
5148 needs to be included on this list.

5149 The harmonized regulations that recently the federal
5150 government asked for public comment on had three pieces in
5151 it. One piece was to use -- apply the regulations, the DIRC

5152 regulations and the GOF regulations pulled together on any
5153 human animal or plant pathogen and agent. And agent was not
5154 defined. So you look it up in the dictionary and it says
5155 it's something or someone that mediates an effect. mRNA
5156 vaccines mediate effect. AI predictions mediate effect.
5157 All of the products that are being developed in
5158 microorganisms where you're dropping -- you're basically
5159 farming the genetic information on Planet Earth to build
5160 synthetic biosynthetic pathways to make two carbon molecules,
5161 which is the basis of the petrochemical industry and perfumes
5162 and drugs, that is all now subject to those regulations as
5163 written.

5164 I personally think we're going to crush the bio-economy with
5165 that regulation. So I wrote that and said this regulation is
5166 too extreme, because it doesn't distinguish between any
5167 pathogen, and it closes down potential
5168 commercial -- economically commercial and viable research
5169 pathways that are going to drive the U.S. economy in the
5170 future.

5171 And so I'm concerned about that because overregulation is
5172 going to be -- it's sort of the risk-benefit. The
5173 risk-benefit of a flu experiment is if it gets out and it's
5174 truly transmissible, it can kill a million to a billion
5175 people. That's pretty quantifiable, right? That's high
5176 risk. But working with a virus that's mildly pathogenic,

5177 that most of us get exposed to when we're two years of age
5178 and get repeated exposures the rest of our life, that's not a
5179 big risk. Even if you engineered it, it would have a huge
5180 problem getting past the immunity that's in the population.
5181 So you can't do these regulations with a sledge hammer. You
5182 have to use a scalpel. And that means there has to be some
5183 refinement and consideration for the long-term impact of
5184 those regulations on scientific leadership, our economy, the
5185 biosecurity field, the biosafety fields, and
5186 entrepreneurship, innovation, discovery. And if you close
5187 all that down, microbiology is gone to China, and they have a
5188 ten-year plan to be number one, and we're helping them.
5189 That's my interpretation.

5190 Q So my question to you --

5191 Mr. Ervin. Can we make this the last one?

5192 Mr. Slobodin. Yeah.

5193 BY MR. SLOBODIN.

5194 Q -- is in trying to figure out the sweet spot
5195 on this policy.

5196 A It's very difficult.

5197 Q As part of the implementation to address
5198 public confidence in the safety of this research, we have
5199 this policy, sort of this backup system talking about the one
5200 virus log growth. Maybe there are other things, but right
5201 now, you said that's the best?

5202 A To be frank on that, if you get a bunch of
5203 virologists and bacteriologists together, they may come up
5204 with a better metric. This is what I came up with.

5205 Q Sure.

5206 A It shouldn't be the standard.

5207 Q So my question is, whatever it is, if you
5208 implement a policy to make sure the research is being done
5209 safely and to be prepared in case of an unexpected outcome,
5210 shouldn't that policy be consistent with every grant research
5211 proposal that's being reviewed, the same rule for everybody?
5212 Or is there such a thing as different versions of this?
5213 Should there be certain standards or certain template and
5214 pieces of information, like how it's to be measured, when
5215 it's to be measured, certain statistics, you've got to
5216 include certain information? Because Daszak is saying, oh,
5217 well, there was nothing here anyway, we weren't statistically
5218 powered. This doesn't make any sense. Why were you even
5219 doing research if it wasn't statistically powered.

5220 A It should have been statistically powered.

5221 Q So my point is, what should that regime look
5222 like? Shouldn't there be -- to me as an outsider, I do not
5223 understand. I think we're going to see as we're doing this
5224 oversight, variations in how this virus log growth is
5225 articulated and how it is applied by the NIH. And that
5226 raises concerns about whether that's really a good way to go

5227 to address this public confidence issue.

5228 So what should that look like? To what extent should there
5229 be some standardization for that kind of rule?

5230 A Let me address your first comment, which was
5231 more focused across all of virology or microbiology.

5232 There are things in this world that you're not too concerned
5233 about if you get infected with. The common cold is certainly
5234 one. But I bet your concern level would go way up if it was
5235 Ebola. And so there are pathogens that are at much higher
5236 threat level than others.

5237 So because of that, and because of their biology and how they
5238 transmit and where they cause disease and how severe the
5239 disease is, there is a gradient. It is not one standard fits
5240 all. There has to be some level of flexibility in
5241 interpreting those regulations that you develop that make
5242 intelligent and informed predictions about what should be
5243 regulated and what should the standards be.

5244 And there's going to be some variation in that. And there's
5245 some things that probably shouldn't be regulated, unless the
5246 technology or the capabilities in the scientific community
5247 occur that would allow for DIRC related research to occur.

5248 So if you figured out -- let's say if you had an AI program
5249 that could look at the common cold, look at all the common
5250 cold viruses, like 170 of them, and you run AI programs and
5251 say, okay, I want to make a new rhinovirus that escapes all

5252 the immunity that could have been made if you got infected
5253 with all of them, let's say if AI ever got there.
5254 Number one, as a nation, if this was -- you might want to
5255 know if that capability existed. You would want to know when
5256 that technology emerged. You might want to think about how
5257 you would apply those standards to things that are low risk
5258 or high risk.

5259 So depending on the technology and the capabilities, those
5260 are just things that, you know, you might find smarter people
5261 than me that can come up with a better standard for
5262 regulatory control. But I just think there's a lot of
5263 variation in pathogenesis and pathogens, and how they cause
5264 disease and how they transmit.

5265 And we should stay focused on those pathogens that are the
5266 highest risk level that we need to develop countermeasures
5267 for, so that we have things in our box that we can rapidly
5268 implement in the population to protect them, should either
5269 one emerge from nature or by some sort of nefarious purpose:
5270 Mr. Benzine. We can go off the record.

5271 [Whereupon, at 4:32 p.m., the taking of the instant interview
5272 ceased.]