This Week in Virology

TWiV 1025 Clinical Update

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Vincent Racaniello: *This Week in Virology*, the podcast about viruses, the kind that make you sick. From MicrobeTV, this is *TWiV, This Week in Virology*, Episode 1,025, recorded on July 12, 2023. I'm Vincent Racaniello, and you're listening to the podcast all about viruses. Joining me today from New York, Daniel Griffin.

Daniel Griffin: Hello, everyone.

VR: This is not something that anyone cares about, but ever since *TWiV* 1,000 the clinical updates have gotten an even number, because if you do two a week, one is going to be even and one is going to be odd. But this week, I stuck in an extra midweek episode with Kostya Chumakov. Really good interview, you have to listen. Now, Daniel, you're going to be stuck on odd numbers for a while.

DG: From here on out, all my updates will be odd.

VR: That's right. Let's have an odd update, Daniel.

DG: Well, odd that - I'll start with my quotation. Now, unfortunately, people who are followers may have heard that Milan Kundera, the author of *The Unbearable Lightness of Being*, passed this week. I actually changed just this morning to a different quotation and let me go ahead with it. "For there is nothing heavier than compassion. Not even one's own pain weighs so heavy as the pain one feels with someone, for someone, a pain intensified by the imagination and prolonged by a hundred echoes." Huge fan of Milan Kundera. Those of you that haven't had a chance to read any of the works, I highly recommend them.

I remember one time I was talking to, I think it was the head of the Nobel Prize Committee, and his advice was to read broadly, not just stick in your little area, but try to bring some breadth to things. With that, I will talk about flu. The CDC recently published a new risk assessment for the H5N1 avian flu viruses that continue to circulate in wild birds and poultry, also impacting some cats. People are following that. For background, the Influenza Risk Assessment Tool, the IRAT, is an evaluation tool conceived by CDC, further developed with assistance from global animal and human health influenza experts. The IRAT used to assess the potential pandemic risk posed by Influenza A viruses that are not currently circulating in people.

A risk assessment for the potential emergence and public health impacts was conducted in March 2022. I'll leave a link in there, but using A/American wigeon/South Carolina/a whole bunch of letters. I won't go into that. This updated assessment includes new information available since that March 2022, including eight additional human cases. This updated assessment using the mink virus from an outbreak in Spain in 2022, they say, indicates that this virus has scored slightly higher in some risk elements compared with the previously assessed H5N1 clade 2.3.4.4b virus isolated from an American wigeon duck in 2022.

However, just to put this in context, the mean high and mean low acceptable score ranges for these viruses overlap, indicating that these viruses remain similar. Their overall risk scores remain moderate. I'm going to go out on a limb here, Vincent, and suggest that the question about whether we should be worried about an H5N1 flu pandemic should not be posed as a binary. It's national security and dare I say, a global security issue, that we should be prepared for such a possibility. The goal is not to stay up night worrying about this, that binary idea. What do you have nightmares about? But just to allocate appropriate resources, do the correct science, continue to educate, be prepared, so we do not have to come up with something at the last minute.

VR: Prepared? Did you say be prepared, Daniel?

DG: Prepared. Yes, I did say that.

VR: I agree. We should have been prepared for COVID and now we could make vaccines. We do have antivirals for flu. Maybe we should make enough of them so that everyone could have them.

DG: Maybe even better ones without resistance issues and better efficacy.

VR: Let's fund some science. What's the story here? Get with it. Our budget for science is not enough.

DG: It's interesting. If you look at the military budget, maybe we need to think about it that way. This is a security issue. What are we really more at threat from? I think we're more of a threat from pathogens than these foreign countries, but just my little two cents, and worth about as much.

VR: You're just the bleeding heart liberal, Daniel.

(laughter)

DG: I'm actually talking about funding the military but considering health to be part of the military. Malaria, two more cases of locally acquired malaria were reported in Sarasota County bringing this up to a number of six locally acquired cases of malaria in Florida. What is going on down there?

All right, moving into COVID. I'm going to jump right into testing. I like this article, “Development of Monoclonal Antibody-based Blocking ELISA for Detecting SARS-CoV-2 Exposure in Animals,” recently published in *Virology*. Our listeners are likely aware that

besides humans, SARS-CoV-2 can infect several animal species, so highly sensitive and specific diagnostic reagents might be worth developing.

Here we hear about a panel of monoclonal antibodies against the SARS-CoV-2 nucleocapsid protein. I like that. This is a serological test looking for antibodies against nucleocapsid. They use these monoclonal antibodies to develop a MAB-based, monoclonal antibody-based, blocking enzyme-linked immunosorbent assay, a b-ELISA. Test validation using a set of animal serum samples with known infection status, obtained a diagnostic sensitivity of 97.8% specificity of 98.9%. I wonder if our friends Paul Kelly and others at the Wildlife Conservation Society are listening but just remember, in addition to humans, SARS-CoV-2 can infect, we're going to go on with the list, cats, dogs, deer, mink, lions, snow leopards, tigers, et cetera, et cetera.

VR: You think some of the zoo animals are infected? Are they checking in the zoos? Do you know, Daniel?

DG: Well, as we know, several zoo animals got infected, got sick early on.

VR: I wonder if they're just doing routine checking now.

DG: It would make sense for us to keep track of this because once you start to see this circulating in the zoo - People go there. There can be back and forth.

VR: Last time I was at the zoo was for *TWiP*.

DG: Got to get you back there. All right, let's move right into, and we're moving fast this week, the COVID early viral upper respiratory done hypoxic phase. We've done that test. The person's positive. You've done the test, you're positive. It's not one of those expired tests that was sitting there for two years baking in the sun. It actually works. The article, “Repeated Antibiotic Exposure and Risk of Hospitalization and Death Following COVID-19 Infection (The OpenSAFELY Trial): A Matched Case-control Study,” was published in *eClinical Medicine*. Now, this is an interesting one because we know that age and comorbidities are associated with worse outcomes with COVID. You'd say, "I'm over the age of 50, I've got heart disease, hypertension."

We know that those are associated with worse outcomes. Here the investigators are asking whether prior antibiotic exposure is associated with severe COVID-19 outcomes. You're going to tell that, "I've been getting lot of antibiotics. I'm at high risk." Well, let's see. The investigators use this OpenSAFELY platform, which integrated primary and secondary care, COVID-19 test, and death registration data. This matched case-control data included 0.67 million patients aged 18 to, I like this, 110 years old, they had an older person in there, from an eligible 2.47 million patients with incident COVID-19 by matching with replacement.

We read that between February 1, 2020, and December 31, 2021, 98,420 patients were admitted to hospitals and 22,660 died. Fifty-five unique antibiotics were prescribed. A dose-response relationship between numbers of antibiotic prescriptions and risk of severe COVID-19 outcome was observed. Patients in the highest quintile with history of prior antibiotic exposure had almost two times, 1.80 times, greater odds of hospitalization compared to patients without antibiotic exposure. Similarly, the adjusted odds ratio for hospitalized

patients with death outcomes was increased 1.34. Larger number of prior antibiotic type was also associated with severe COVID-19-related hospital admissions.

The adjusted odds ratio of quintile 5 exposure, the most frequent, with more than three antibiotic types was around two times larger than the quintile 1, with the lowest about two times as likely. Lot, lots of questions here about causation versus, dare I say, correlation. This is, as the authors say, consistent with their prior Spanish study. Despite multiple ways of analyzing the data, it looked consistent and was even higher in certain age groups such as those and then, are you ready for this, the 40 to 59 age group odds ratio of 2.59. Lots of questions. This is related to disruption of the gut microbiome.

Could prior antibiotics impact what we refer to as the gut resistome, which comprises antibiotic-resistant genes and gut flora increasing potentially COVID-19 patient susceptibility to secondary bacterial infection, difficulty to treat. The obvious idea is that maybe people getting all these antibiotics are a bit different. They tried to do a sensitivity analysis adjusting for 17 individual diseases.

VR: Daniel, did these patients get antimicrobials before at a different hospitalization before they came in with COVID?

DG: Yes. That's it. You're looking over the period of time before they get COVID-19.

VR: I see.

DG: You're saying these people who get one antibiotic, people who've gotten multiple courses of antibiotics.

VR: Yes. Well, as you said, they may be just very sick people, right?

DG: That's my biggest concern. Even though they do all this matching, hard to match without a prospective cohort.

VR: All right, let's do a placebo-controlled trial, OK?

DG: All right, well, speaking of placebo-controlled trials, people might remember this article when it was a pre-print. It's now published, "The Coronavirus Disease 2019 Rebound Study: A Prospective Cohort Study to Evaluate Viral and Symptom Rebound Differences in Participants Treated with Nirmatrelvir Plus Ritonavir versus Untreated Controls," published in *CID*. Bit of a reminder, the uptake of Paxlovid in patients with COVID-19 has been limited, heard it again today, by concerns about the rebound phenomenon despite the scarcity of evidence around its epidemiology. The purpose of this study was to prospectively compare the epidemiology of rebound in Paxlovid treated and untreated patients with acute COVID-19 infection.

I could have a bit more here to say about all those pundits who jumped on to be interviewed to promote this idea of rebound despite a scarcity of evidence. But I will just move on to the literature. These are the results of a prospective observational study in which participants who tested positive for COVID-19 and were clinically eligible for Paxlovid were recruited to be evaluated for either viral or symptom clearance and rebound. Participants were assigned to

the treatment or control group based on their decision to take Paxlovid. Following initial diagnosis, both groups were provided 12 rapid antigen tests and asked to test on a regular schedule for 16 days and answer symptom surveys.

Viral rebound based on test results and COVID-19 symptom rebound based on patient-reported symptoms were evaluated. Not a lot of numbers here with 127 in the Paxlovid treatment group, 43 in the control, no treatment group. As we discussed this study at the pre-print stage, this preliminary report suggests that rebound after clearance of test positivity or symptom resolution is higher than previously reported, but was observed at a similar rate both between treatment and control groups.

VR: I think the pundits need to start reading, don't you, Daniel?

DG: It's really tough. I have to say people have died. You just got to put this right out there. People died because of this integrity issue. Science is powerful. Studies like this make it really clear that this is not a thing. You give a high-risk individual Paxlovid, you can reduce their risk of ending up in the ER, the hospital on a ventilator in the ICU or dying. When we're sitting with these 80,000 to 100,000 deaths a year, most of those are prevented, and a lot of reason these folks are not getting the right medicine is because folks were willing to get on there, sell their soul, not read the science.

VR: How many letters have we had where patients bemoaned their physicians said, "No, I'm not giving this to you. You'll get rebound, right?"

DG: I had it today. It was a woman in her 70s. Yes, her doc was telling her, "I'm just not comfortable. I hear a lot about this rebound. Now, what does that mean? I hear a lot about this rebound. What scientific journals are you reading where you're -"

VR: That doc goes to the local diner for coffee and the guy behind the counter, "Hey, doc, you hear all about this rebound?" You said, "Oh, really?"

DG: "Yes, I knew Jerry. Jerry got the Paxlovid and Jerry had the rebound." Oh my gosh. OK.

VR: It's not funny. We're not being joking about it, folks. It's just we're bemoaning how to get physicians on board, right?

DG: Yes. I am optimistic, and I hate to say this, and we have got to clean our own house. You can't wait till the pharmaceutical company comes around with a bunch of glossies. We have to keep up to date. This is a disease with mortality. When our patients come to us, there's an expectation that we're going to put in the time and the effort to give them evidence-based recommendation. Not just, yes, not just based on a bunch of anecdotes. All right. Number one, as we've been talking about for a while, Paxlovid, fully licensed, and beneficial in vaccinated, unvaccinated, under the age of 50, over the age of 50, really people who have risk factors.

Again, this isn't for everybody, and sometimes it's a little work looking at the kidney function and looking at the drug-drug interactions. Number two, remdesivir, we've been bemoaning issues with limited access, but that three-day early IV PINETREE-based approach with about an 87% reduction in progression. Number three, molnupiravir, only problem with

molnupiravir is the less-than-impressive efficacy, quite a problem. Number four, convalescent plasma really only recommended as a treatment option for immunosuppressed COVID-19 patients really early on. No other options, and as we keep saying, let's avoid doing harmful and useful things.

All right, now we move on. The early inflammatory person's progressed may become hypoxic. That's when we think about steroids, anticoagulation potentially ending up in the hospital with pulmonary support. Remdesivir, if early immunomodulation, and again, avoiding unnecessary therapies. Moving into the bulk of today's session, the late phase PASC and Long COVID. This is an area which actually I was glad that this article came out because I think there's a growing recognition of this as a problem. The article, "Exaggerated Blood Pressure Elevation in Response to Orthostatic Challenge: A Post-acute Sequelae of SARS-CoV-2 Infection (PASC) After Hospitalization," was published in *Autonomic Neuroscience*.

Maybe the first time we've referenced an Autonomic Neuroscience article. Another study where they investigated the effect of COVID-19 after recovery on blood pressure during orthostatic challenge. Thirty-one out of 45 patients hospitalized due to COVID-19-related pneumonia that developed PASC and did not have hypertension at discharge were studied. They underwent a head-up tilt test, it's called a HUTT, at about 10.8 plus or minus 1.9 months from discharge. All met the PASC clinical criteria, and an alternative diagnosis did not explain the symptoms. The population was compared with 32 historical healthy controls.

Exaggerated orthostatic blood pressure response was detected in 34.7% of the patients, representing a significantly increased prevalence, almost 7.67-fold increase, P evaluate to 0.009, compared to only seeing this in 6.4% of the healthy controls matched by age who underwent testing. This prospective evaluation in patients with PASC revealed abnormal blood pressure rise during the orthostatic challenge suggestive of autonomic dysfunction in about a third of the studied patients. I'd like to point out it may be a challenge to access the tilt table testing and the HUTT, the head-up tilt test.

For a lot of folks consider screening with the NASA Lean Test, as we do have therapies that can help address individuals with the orthostatic challenges.

All right, I also in the section like to cover what we recommend on hospital discharge, right? Patients surviving the hospitalization are getting ready to be discharged. The article, "Long-term Follow-up of a Multi-Center Cohort of COVID-19 Patients with Pulmonary Embolism: Anticoagulation Management and Outcomes," was published in *Thrombosis Research*. Again, not a time to just read the headlines. These are not just COVID-19 patients being discharged. They're COVID-19 patients with pulmonary emboli.

These are the results of a retrospective multi-center study in four Italian hospitals between March 1, 2020, and May 31, 2021, in patients who experienced a pulmonary embolism during hospitalization for COVID-19 pneumonia, excluding patients who died during hospitalization. Baseline characteristics were collected and patients were grouped according to duration of anticoagulation treatment. We're looking at folks that got three months or less, and we're going to look at folks that went on past greater than three months. The primary outcome was incidents of VTE recurrence, venous thromboembolism recurrence, while secondary outcomes were death, hemorrhages or recurrence during the follow-up.

One-hundred-six patients with pulmonary emboli were discharged. Of these, 89.6% had follow-up longer than three months. Seven folks were lost to follow-up. Four died in those first three months. Just to give you a sense, 106, right, four of those died the next three months. Not all that mortality is happening in the hospital. The median follow-up was 13 months. Overall, 23% of the patients were treated for the three months or less, 77% received anticoagulation for greater than three months. Of patients in the short treatment group, 4.5% died compared with 5.5% in the longer treatment group. No difference was shown in the risk of VTE recurrence.

Actually 0% in the less than three months versus 4% in the more than three months. Major bleeding, 4.5% versus 4.1%. When they looked at composite outcome less than three months 9.1%, more than three months 11%. No difference was found between the two treatment groups. Just to put this back together, in this retrospective multi-center cohort, prolongation of duration of anticoagulation beyond three months did not seem to affect any of these outcomes. Three months might just be enough.

Now another issue is a theme that we've hit on for a while. The risk of cardiovascular disease after COVID-19 diagnosis among adults with and without diabetes. This isn't getting diabetes, this is having diabetes, published in *Journal of the American Heart Association*. The authors comment that growing evidence suggests that incident cardiovascular disease may be a long-term outcome of COVID-19 infection and chronic diseases such as diabetes might influence the cardiovascular disease risk associated with COVID-19. The researchers evaluated the post-acute risk of cardiovascular disease greater than 30 days after diagnosis in 1,898,635 adults aged 20 or older with COVID-19 from March 1, 2020, through December 31, 2021. Think about the timing. We're pretty much looking at pre-vaccine.

A comparison was made with a contemporaneous control group comprising 11,180,192 adults without recorded diagnosis for COVID-19. Here they evaluated the post-acute risk of cardiovascular disease greater than 30 days after a COVID-19 diagnosis by diabetes status. They found that patients with a COVID-19 diagnosis had a significantly greater risk of all cardiovascular outcomes compared with patients without a diagnosis of COVID-19 hazard ratio 1.66 with diabetes, a little higher, 1.75, cardiovascular 1.66 with diabetes, 1.75 without diabetes.

Cardiovascular outcomes included cerebral vascular disorders, dysrhythmia, inflammatory heart disease, ischemic heart disease, thrombotic disorders, other cardiac disorders. Let us pull this back together. We're certainly seeing an increased risk of cardiovascular disease after COVID-19. Not clear to me that the diabetes is actually having a big impact here despite being in the title. Then again, as we talked about, think about the timeline here. We start looking in March 2020, and then we're going to follow through, think about when the vaccines come out, and we're going to follow out to December 31, 2021.

VR: Daniel, if you did this study with say, influenza patients, would you find cardiovascular events also higher in the flu patients?

DG: In the studies looking at that, the answer is clearly yes. When we looked at it later, United Health Group looked at patients to try to figure out what's the benefit of flu vaccine. The biggest benefit, particularly in younger individuals of getting your flu shot, is prevention of

cardiovascular outcomes, prevention of cardiovascular-related hospitalization. Certainly a connection here with viral diseases and cardiovascular outcomes. All right, I will wrap it up here before questions.

I see we have a few questions today, but no one is safe until everyone is safe. Only a couple, two, three weeks here left in our Foundation International Medical Relief of Children fundraiser. I was just checking on my cows today, my cattle today with Alice in Uganda. Because I guess now I've got cattle. I've got a cow and a bull. May, June, and July, donations made to Parasites Without Borders will be matched and doubled. We're trying to get up to a donation of $20,000 from PWB to Foundation International Medical Relief of Children.

VR: Time for your questions for Daniel. You can send them to daniel@microbe.tv. Larissa writes, "I'm a 55-year-old woman who got pericarditis with my first booster and similar symptoms after my second. I got the Moderna vaccine. My cardiologist told me I can't get any more boosters. It's been 18 months since my last shot. Do I have any protection against severe illness at this point, or am I essentially unvaccinated after all this time? Related question: Since I can't be boosted, treatment is important. I take clonazepam. The Liverpool drug interactions website says not to administer Paxlovid with clonazepam. My doctor said maybe I could reduce my dose, but she wasn't sure. The pharmacist said I could not take it. No way to get remdesivir in my area. Suggestions."

DG: This is a good challenge. I understand, and we've talked about this. It is rare, but there certainly are individuals who have issues with the vaccines and the cardiac inflammation that you're describing. I'd be on board with your cardiologist saying, "OK, maybe that's not the best way going forward, particularly with the mRNA vaccines." It would be nice for us to have a sense of maybe Novavax, is that an alternative that might be tolerated? I understand. That's the first question. The second is, do you have protection? We've gone through this, ideally you want to get three shots, you want to get that broadened or maybe even just need more of a gap between the first two.

You certainly have protection with two, maybe not what we would like with three. You're not walking around un-shielding. You're not walking around without some degree of protection. The next is, as you rightfully say, potentially, 80%, 90% reduction in progression if you can get early antiviral therapy. Shine a spotlight on the Liverpool COVID-19, drug interaction checker. I spent a little bit of time today, one individual, it was really easy, not on any medications, good kidney function, boom, easy lift. Second, we had a little bit of interactions. What about this? What about that?

Now clonazepam is metabolized through the cytochrome P-450, CYP3A. Everyone, make sure you memorize that. [laughs] If you take the Paxlovid, which has the ritonavir, that's going to really shut down the metabolism of clonazepam, that is not a safe thing to co-administer. You know what you can do? You could ask your doctor about temporarily stopping the clonazepam and replacing it with Lorazepam. Not all the benzodiazepines are metabolized the same way. There are ways around this and we went to medical school, we should be able to figure out those ways around it.

VR: This clonazepam is not one of these that persists a long time, right?

DG: It's a benzodiazepine. What you're going to want to do is stop it, make sure it's not going to start rising in levels. Stop it. The next day. Start your Paxlovid, but don't take it while you're on within 10 days. Because I would worry about it accumulating and actually it's a benzodiazepine. It can be associated with respiratory depression. It can be a dangerous thing. Actually, maybe talk to your doctor ahead of time about could you just switch from clonazepam to lorazepam, maybe use a different benzo because COVID is out there. It's nice to be said ahead of time.

VR: Now I got an idea for another episode we need to do. In multiple, we need to do one on what if your doctor wound won't give you Paxlovid and another one, what if you can't take Paxlovid. Five minutes you can tell them what to do. Then we'll get lots of views, right?

DG: Yes. You individually have to spend the time with each person saying, "Let's go through your medication list. Where are you on which medication?"

VR: Charmaine writes, "I just read that the national health system in the UK has stopped offering COVID vaccines to people who are not at high risk. This is nuts. They said it's to prioritize those most at risk and cut the backlogs and waiting lists. Good Lord. Don't they have enough vaccines and/or personnel to give people jabs? Is the demand still that high? Why would you tell people they can't have it? My sense is that we're swimming in the availability of vaccines." Charmaine. [laughs]

DG: OK, well, it's very entertaining. I'm a bit taken back by this. I can't imagine that right now in July in the UK, there's such demand for these vaccines and such a limited supply. As we've talked a little bit about the current bivalent boosters, the whole idea that you're going to get three to four months of reduction in infection and also some reduction in your risk of severe disease, I just don't think there's a lot of compelling data there. I think what we've got is once you've got those three shots, maybe there's a bivalent in there at some point, you're really in pretty good shape as far as, what do vaccines do? They prevent disease.

Maybe in the fall, when there's new, updated XBB-based vaccines, maybe when we see that they're actually producing neutralizing antibodies that are going to be effective against the circulating variants, they'll be more of an imperative. This sounds a little disturbing.

VR: I looked this up and here's a clarification. While the UK will stop providing vaccines to those under 50, anyone who has a clinical need, high risks of your disease, health care workers, caregivers, they can still get a shot. I'm sure there are a lot of people who are not at risk who would like to get shots, and I think they should be able to get it.

DG: Maybe there's folks who just decided, "I was going to wait," and now they waited. You really don't want to close the door if someone decides they want to get vaccinated.

VR: This is in line with the UK wanted to kill all the cats at the beginning because they were getting COVID.

DG: I think Boris was going to kill the old people too, until he realized he was old. OK, that was political. Sorry that I said that. [laughs]

VR: Paula writes, "Since you brought up shingles Shingrix, it reminded me to send a question about my friend who has repeated bouts of shingles in the roof of her mouth every few months. Nothing seems to stop it. Her doc gets her on antivirals immediately if she calls to say it's erupting again. She also seems to have it happen after any dental work. It's been going on for years. She had the shingles vaccine in between bouts of this, and it stopped for slightly longer, then came back. What causes this? What other options are there out there?"

DG: This virus is a problem in, let's just say, immunocompromised individuals, and we think of that broadly. Someone had a transplant, for instance, they're on immunosuppressive medications, maybe someone's on immunosuppressive medications for some autoimmune rheumatological disorder. This might be a case of someone with maybe even a particularly isolated immune issue controlling the virus. We do actually routinely put people on chronic suppressive antivirals. Standard would be maybe a Valtrex, which is an antiviral that you might take 500 milligrams once a day, maybe 1,000 milligrams once a day. There are ways of addressing this with chronic antivirals, so the person doesn't have to have the problem and then jump in with the treatment.

VR: It's actually shingles, Daniel, and not herpes?

DG: This can be shingles, yes, you can get the varicella zoster recurrent. That's also important to do. When you get a recurrence, swab it, do the viral PCR, make sure you know what you're treating, imagine that, and then once you know what you're treating it would be the same approach, but it's always good to know what you're treating.

VR: It's interesting because that's very frequent for shingles, isn't it?

DG: There are a few people that I've seen that have these recurrent, yes.

VR: Interesting. All right, our last one is from David. "Consistent with Dr. Griffin's comments on, if you are not thinking about Mpox in the differential, you will not test for it nor diagnose it. I wanted to share an article incorporating Mpox into the differential of genital skin lesions due to infectious causes. The idea was to have a table with multiple variables listed together for the clinician to work through a genital skin lesion differential. I thought this could be helpful. Of course, STIs may occur concurrently, thus, testing for coinfections is important to quickly identify all pathogens and appropriately treat individuals." David sends a link to this, which is a product of the U.S. military, Daniel, as you were mentioning at the top, Health.mil.

DG: OK.

VR: David is a lieutenant colonel in the Air Force Medical Readiness Agency.

DG: Oh, fantastic. We're talking a lot over this last weekend about the military, first, I'm going to say, because my college roommate's son is thinking of becoming a Navy SEAL. I don't know if you know this, Vincent, I tried to join the military several times. First I was thinking of going to the Naval Academy. I wasn't sure I could stay on the varsity sailing team, and they might make me work too hard but I couldn't do that. Then I tried to join the Air Force and tried to get them to agree that my first posting would be in Korea. Apparently, the needs of the government supersede the needs of the individual, so they wouldn't sign, but we could have almost been colleagues.

VR: Wow.

DG: But no. I think this is great. We need to have a broader view when we're looking at patients with sexually transmitted infections. Because we don't test for it, we don't think about it, we're not going to make the diagnosis. I applaud this. This is great. Hopefully we'll leave a link in our show notes.

VR: That's *TWiV* weekly clinical update with Dr. Daniel Griffin. Thank you, Daniel.

DG: Oh, thank you and, everyone, be safe.

[00:36:29] [END OF AUDIO]