UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): April 20, 2020

INMUNE BIO INC.
(Exact name of registrant as specified in charter)

Nevada	**001-38793**	**47-5205835**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

1200 Prospect Street, Suite 525, La Jolla, CA 92037
(Address of Principal Executive Offices) (Zip Code)

(858) 964 3720
(Registrant's Telephone Number, Including Area Code)

Not Applicable
(Former Name or Former Address, If Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mart if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.001 per share	INMB	The NASDAQ Stock Market LLC

Item 8.01. Other Events.

On April 20, 2020, INmune Bio Inc. (the "Company") held a conference call about its DN-TNF Clinical trial. A transcript from the conference call is attached as Exhibit 99.1 to this Current Report on Form 8-K.

Item 9.01 Financial statements and Exhibits

(d) Exhibits.

Exhibit Number	**Description**
99.1	Transcript of the INmune Bio Inc. Conference Call on April 20, 2020

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: April 21, 2020

INMUNE BIO INC.

By: */s/ David Moss*
David Moss
Chief Financial Officer

Exhibit 99.1

INmune Bio
DN-TNF Clinical Trial, Complications of Cytokine Storm Caused by COVID-19
April 20, 2020

Presenters
R.J. Tesi, CEO and Chief Medical Officer
David Moss, CFO

Q&A Participants
John Aschoff - Roth Capital
Michael Carmia - BTIG
Jason McCarthy - Maxim Group
Mikal Kaiser - BTIG
R.K. - H.C. Wainwright
Carl Byrnes - Northland Securities

Operator
Greetings, and welcome to INmune Bio's Conference Call to discuss the company's DN-TNF clinical trial to determine if it will successfully treat the complications of cytokine storm caused by COVID-19. At this time, all lines are placed on a listen-only mode, and the floor will be open for questions and comments following the presentation. If anyone should require operator assistance during the conference, please press star, zero on your telephone keypad. As a reminder, this conference call is being recorded. A transcript will follow within 24 hours of this conference call.

At this time, it is now my pleasure to introduce your host, R.J. Tesi, CEO and Chief Medical Officer, and David Moss, CFO. David, the floor is yours.

David Moss
Thank you, Devon, and good afternoon, everybody. We thank you for joining us on--for the INmune Bio conference call to discuss our strategy related to COVID-19. As we have stated before, we believe we have a unique drug in our DN-TNF platform that addresses complications of inflammation, and we know that in the case of COVID-19, rapid hyperinflammation can cause a series of events that can lead to death. With me on the call is CEO and Chief Medical Officer, R.J. Tesi, who will go into more detail about our clinical program related to COVID-19.

Before we begin, I remind everyone that except for statements of historical fact, the statements made by management and responses to questions on this conference call are forward-looking statements under the Safe Harbor provision of the Private Securities Litigation Reform Act of 1995.

These statements involve risk and uncertainties that can cause actual results to differ materially from those in such forward-looking statements. Please see the forward-looking statements disclaimer on the company's earnings press release as well as the risk factors in the company's SEC filing, including in our most recent quarterly filing with the SEC.

Our DN-TNF platform is beginning trials for the treatment of complications of cytokine storm, and there is no assurance of any specific outcome. Undue reliance should not be placed on forward-looking statements, which speak only as of the date they are made, as the facts and circumstances underlying these forward-looking statements may change. Except as required as law, INmune Bio disclaims any obligation to update these forward-looking statements to reflect future information (SP), events, or circumstances.

Now, I'd like to turn the call over to R.J. Tesi, the CEO of INmune Bio. R.J., please proceed.

R.J. Tesi
Thank you, and thank you, everyone, for joining us. And just on a personal note, if you see a health care worker who has been on the front lines, give them your hearty thanks. They deserve it.

COVID-19 has consumed us all for the last two months, particularly those of you in New York. You just can't get away from it. It's been a pretty remarkable period of time. The pathology is driven by the cytokine storm, which is an uncontrolled, disregulated, and sometimes lethal, inflammatory response, primarily driven by the innate immune system. The pro-inflammatory cytokines of IL-1, 6, and soluble TNF are elevated in patients who are hospitalized, and these are the main culprits in the cytokine storm of COVID-19. INmune Bio is the inflammation company focused on the innate immune system. We have a dog in this fight.

So, how did we get here? We signaled during our end-of-year conference call on March 11 that we were in the midst of a strategic analysis to determine if and how INmune Bio should get involved to help with the current pandemic. We did a careful scientific review. And I have to say any of you that have been following this pandemic know that the medical community has just been remarkable in the amount of data that has come out from places like China and Europe and beyond.

We assess the clinical need and consider the realities of financing and initiating a clinical trial in the midst of a pandemic. I will talk more about these in a moment. To be clear, our program is not a vaccine. We're not an antiviral program that kills virus. DN-TNF should decrease the severity of the inflammatory pathology associated with the viral infection. That is what we are planning to do.

So, what is the scientific evidence that says we should be going down this road? Soluble TNF is one of the cytokines that is elevated in patients with COVID-19, and it's part of the cytokine storm that is really the cause of much of the medical morbidity and mortality associated with the virus. I remind you that I'm talking only about soluble TNF. Any TNF that you hear about, read about, in the papers is soluble TNF.

They are not talking about transmembrane TNF, which, as you recall, is the good TNF. They are not talking about TNF receptors, which are really not that important in this disease at this point. We are the only ones that have a targeted agent that only affects--neutralizes soluble TNF that has advantages--may have advantages--in both biology, safety, and therapeutic breadth. We talk about this all the time. Please go to our website to learn more.

Soluble TNF plays actually two roles in the inflammatory response associated to COVID-19. The obvious role is that it activates immune cells, and the immune cells cause the inflammation and the tissue damage that promulgates the--the disease. The less obvious role is that soluble TNF activates endothelial cells. Endothelial cells express ICAM-1 and--and VCAM-1, which are proteins that signal the leukocytes in the blood to marginate then migrate through the blood vessel wall to the tissue, where they cause damage.

Put simply, soluble TNF brings the wood to the fire, and it supplies the match to light it. We think this double whammy is important. And endothelial activation is now being discussed more as the disease--as the threat of not enough ventilators has passed. And this hypercoagulable state, the problems with renal failure and the problems with increased cardiovascular risk, are probably all associated with this endothelial activation.

So, is DN-TNF the right drug? We believe the answer is yes for three reasons: the mechanism of action of the DN-TNF aligns well with the pathophysiology of cytokine storm; it is not immunosuppressive--and I'll come back to that; and it is very easy to use, something that's very important in the chaos of a surge.

We have been preaching for more than two years that DN-TNF neutralizes soluble TNF, and the soluble TNF levels in COVID-19 patients can reach 100 picograms per mL. That's more than 10 times the level that you and I have sitting here, assuming none of us have COVID-19. We know from the data from our oncology Phase I trial that stable trough levels of DN-TNF in the blood are achieved such that it will neutralize more than 99.9 percent of TNF in the patient's blood, even at those very high levels of 100 picograms per mL. The risk of overshooting is low because the drug is well tolerated. If someone only has 50 picograms per mL, nothing is lost.

Will--will DN-TNF therapy affect cytokine storm? We believe the answer is yes. IL-6, Il-1, and soluble TNF are elevated in the cytokine storm. We always preach that soluble TNF is the master cytokine that drives the expression of IL-6 and IL-1. They are located downstream of TNF translation. And data from our oncology Phase I confirm that when we neutralize soluble TNF in these patients, their IL-6 levels go down.

Finally, there are anecdotal points--reports--that if you treat one inflammatory cytokine of the cytokine storm--in this case IL-6--there are a number of anti-IL-6 trials in place--the patients with acute respiratory distress syndrome appear to get better. I will emphasize they are anecdotal reports at this point. But, safety is key. Any therapy that is used to treat patients with COVID-19 must not make the patient worse. Above all, do no harm.

I was a transplant surgeon in a previous life, and I spent years treating immunosuppressed patients with life-threatening viral infections. I have hard-won experience in this area. And anti-inflammatory strategies must cause no harm. And there aren't many that exist that are not immunosuppressive.

The clinical reports from Wuhan and the World Health Organization, WHO, are in agreement. Corticosteroids increase the mortality of patients with COVID-19. According to FDA labeling, the existing anti-IL-6, anti-IL-1, and anti-TNF therapies are contraindicated in patients with ongoing infection. DN-TNF is not immunosuppressive, and mirroring (SP) models of cancer and bacterial infection, DN-TNF improves the immune response. We have studied DN-TNF in models of viral infection. DN-TNF does not compromise the immune system to the virus.

Last--last year we worked with NIAID to study the effects of DN-TNF in animal models of EEE--that's eastern equine encephalitis virus--and coxsackie B3 virus. And in these infected animals, DN-TNF did not affect mortality, nor did it affect in the case of coxsackie B3 tissue invasion, the virus. These data are not yet published, but the official reports have been included in the document sent to the regulatory authorities and the investigators in preparation for this clinical trial.

Furthermore, speed and simplicity matter. I have been in close communication with a number of New York hospitals during their surge. And I tell you, it's--for someone who has lived in a war zone when I did my training in Brooklyn--my surgical training in Brooklyn--I mean, all of New York was really hard-pressed, and my hat is off to all of those on the front lines. But, it's got to be easy, or it's not going to happen. And this trial is designed to be easy.

Patients present with symptoms of COVID-19 to the--to a clinical facility. They can progress quite rapidly. So, time to therapeutic level is important. Based on data from the Phase I oncology trial, within six hours after the first dose, the patient will have blood levels that neutralize those very high levels of soluble TNF in the blood. It has to be easy. The trial is designed as a two-dose trial. That is, the first dose is given in the emergency room or the admitting clinic, and they don't need a second until seven days later. Patients as old as 86 years old have received this drug in the ongoing Alzheimer's disease trial.

And why did we choose two doses? Because two doses provides at least 14 days of protection from the cytokine storm. Since most patients present about 7 to 10 days after they become infected, that 14 days should be enough to cover them until they have developed their own immune response and the viral infection has run its course.

But, this is where not being immunosuppressive is quite important. I don't want to say that we will improve the immune system, but the data we have with viral infections is we do not get in the way of the immune response. And this is becoming increasingly important as you begin to see reports that patient are relapsing, and there's concern about patients shedding virus even after they leave the hospital.

So, our target population was carefully considered. Almost all the mortality, or shall we say most of the patients who die of COVID-19, at least have respiratory failure. In my mind, there are three points to intervene with respiratory failure. You either prevent the respiratory systems, you prevent the progression of the symptoms in patients that have them, or you treat ARDS, adult respiratory distress syndrome, in those patients in the ICU on ventilators. In my opinion, prevention is the purview of the antiviral therapies, and there will be viral therapies in the future.

ARDS has received a lot of attention by Biopharma. There--as of two days ago, there were 96 trials listed on clinicaltrials.gov treating ARDS. We decided to focus on what we think is an important underserved group. That is a group of patients that present to the hospital with hypoxia, so they can't be sent home. They are admitted, and they are at risk, particularly if they have one or more comorbidities, of progressing in their respiratory disease, potentially requiring ICU admission. It's difficult to predict exactly how many of these patients there are, but it's probably in excess of 20 percent of the patients that actually get admitted for COVID-19.

Our goal is to keep these patients out of the ICU, off mechanical ventilation, and get them out of the hospital quickly. This does not mean we don't believe that DN-TNF will be an effective therapy for ARDS. In fact, we think it would be a very good therapy, but we're going to wait for clinical teams to approach us with their own ideas. Our focus right now is to keep patients out of the ICU, and this becomes sort of a two-for, a two-for-one, that benefits the patient because they get better and go home. And it benefits the system because they don't require an ICU bed or a ventilator.

So, where do we stand with the clinical regulatory, and obviously the financial, logistics of this? We have a protocol, we have an investigatory brochure, the drug is ready to be labeled, we've identified the CROs, we have clinical sites. We are working closely with the FDA and the Australian regulatory authorities. We hope this all comes together in weeks, not months. We are ready to move. The breadth of our effort will depend on many factors, including the availability of non-dilutive financing, which we are applying to from various government sources. We have been invited to give a presentation to BARDA as part of the TechWatch program. I can't predict where that will go, but we made the first cut.

So in conclusion, the goal of this overview was to help you understand the why and the how that INmune Bio decided to get involved in COVID-19. Our decision to initiate the program is based on science, clinical need, and opportunity. The trial focuses on a patient population that plays an outsized role in determining the clinical--determining clinical, shall we say, outcomes and resource utilization, and we've been busy laying the groundwork for the program and hope to be in this clinic soon.

So with that, Devon, I'll open it up to questions.

Operator
Thank you. At this time, we will be conducting a question-and-answer session. If you would like to ask a question, please press star, one on your telephone keypad. A confirmation tone will indicate your line is in the question queue. You may press star, two if you would like to remove your question from the queue. For participants using speaker equipment, it may be necessary to pick up your handset before pressing the star key. One moment, please, as we poll for questions.

Our first question comes from the line of Jonathan Aschoff from Roth Capital. Please proceed with your question.

John Aschoff
Hi, guys. Thank you. So, I was wondering--I have two questions. Do you think that autopsies will show that, in addition to extensive lung damage that we keep hearing about, that there will be renal failure from, say, microangiopathy of the glomeruli as well as cardiac manifestations such as MI due to plaque rupture from endothelial cell rupture? And do you think that it will be discovered ultimately that lung damage maybe was not the actual cause of death in many instances?

R.J. Tesi
So, that's an interesting question. I have been anxious for--to be getting to see autopsy data for really three reasons. The first is there have been sporadic reports of neurologic complications. As you know, one of the, shall we say, defining symptoms is anosmia, which is inability to smell.
This suggests that olfactory nerves are being infected with the virus. I'm very curious if the virus is actually infecting the central nervous system because this may herald problems down the line. And as you know, we have a significant interest in the CNS.

As far as renal failure, I've done damn near 1,000 kidney transplants. I know a lot about renal failure. And I agree with you. I think that this is an important problem. And whether it's the chicken or the egg in death, I--I really think that the pulmonary complications are probably more often the cause of the death. But, the cause of the renal failure is unknown. Is it direct viral infection, or is it a--a coagulopathy, a microembolic disease to the glomeruli or something related to that, as the cause. And only autopsy data will show.

Plaque rupture is interesting. We know from the COVID--not from the COVID--from the CANTOS trial, that inflammatory cytokines do stimulate plaque rupture. We also know in a very wonderful paper that wasn't really well appreciated in the New England Journal of Medicine in 2018, that MIs in patients are--most commonly occur a week before and a week after they are diagnosed with influenza.

So, it is most likely that these plaques are rupturing because of the inflammatory cytokines in the blood associated with their viral infection. Mind you, this is an influenza infection. But, I suspect much of the same pathology is ongoing here with the--with the COVID-19. One of, we think, the strengths of our program is, as I mentioned early, the fact that TNF--soluble TNF--plays an important role in activating endothelial cells. So, if you eliminate and neutralize soluble TNF, you may have an outsized impact on some of these non-pulmonary complications of COVID-19. That's speculation, mind you, but it's things that we will study in our trial.

John Aschoff
Thanks for that. And the second question is, can you please briefly remind us of what agents are being tested in the clinic after your intended intervention point of immediately after hospital admission?

R.J. Tesi
So, I haven't heard of a lot of trials. Some--there are a number of trials that use corticosteroids. I think I've been clear that I don't think that's a--a wise choice. I think many--there are trials that many of the patients in New York City and a couple of the hospitals--they all get put on hydroxychloroquine.

I think the HCQ question is an interesting one and yet unsolved. In my opinion, the issue--I don't want to dive into the debate of whether it's antiviral, but it is clearly anti-inflammatory. That's why it's used in patients with autoimmune diseases. And one of the things that it does is actually--it affects endothelial cells and causes them to down-regulate these proteins, ICAM and VCAM.

So in my opinion, we have a long way to go. Obviously, the antiviral therapies and some of the IL-6 compounds are being used earlier. But I think the majority of the patients are getting primarily hydroxychloroquine, steroids, and then there's some interesting studies looking at things like Vitamin C and zinc. And I'm sure someone is doing a trial on using chicken soup out there also, so--.

John Aschoff
--Thank you very much, R.J.

Operator
Our next question comes from the line of Michael Carmia (SP) with BTIG. Please proceed with your question.

Michael Carmia
Hello, R.J., and thanks for taking the question. As you mentioned earlier, there are a couple different IL-6 approaches in the later stage clinical trials now for CRF. As (unintelligible) upstream of its pathway, can you just explain and elaborate why you believe this is a superior approach to the IL-6? Thanks.

R.J. Tesi
Yeah. So--so I think there's really three things. First of all, anti-IL-6 is immunosuppressive, full stop. Now, you may argue with me that a single dose of IL-6 will not have an effect on--from an immunosuppressive perspective--on a patient with COVID-19, particularly if the choice is in a patient who has ARDS and the choice is a little bit of immunosuppression and maybe a poor viral clearance and shedding a virus and potentially dying of their disease. And I'm not going to argue that in a moment. But (unintelligible) population, there's some things we don't know, and we don't know about how well--what kind of impact this will have on the immune response to the virus and viral clearance.

The other is what I just mentioned. I think that we are underestimating the importance of endothelial activation. And endothelial activation is, to my knowledge--and I could be wrong here--but to my knowledge is not as sensitive to IL-6 as--as it is to TNF. And I believe when you talk to the clinicians now, they're--I don't know whether they've grown comfortable with or they've gotten control of the pulmonary disease problems. And they are very focused on the coagulopathy and the renal failure. To me, those are unsolved answers. And I speculate, with an emphasis on speculate, that targeting soluble TNF is a superior approach.

Michael Carmia
Helpful. And thank you. I appreciate it.

Operator
Our next question comes from the line of Jason McCarthy with Maxim. Please state your question.

Jason McCarthy
Hey, R.J. Can you help me understand just a couple of things. On the--just to follow up on the last caller's theme on IL-6, is it--if you treat with toca (SP) or anything--hydroxychloroquine can go down the IL-6 road, too--do you end up suppressing the viral immune response with those drugs? And can that be an issue where DN-

TNF may be more ideal?

And my second question is, do you--it's more of an abstract question--do you look at DN-TNF in hypoxic patients that have not yet gone into the ICU as a means of, yes, keeping them out of the ICU but more of it like a triage therapy, where if you can get them coming into the ER, and you get their blood oxygen level somewhat stabilized, then you can get them out, whereas another TNF approach couldn't do that? Is that what you're looking to do here, to be that go-to drug to kind of triage the patient and get them out of the hospital before really they get in?

R.J. Tesi
So, yeah. Thank you, Jason. Those are both good questions. So once again, I want to reemphasize that to my knowledge no one has really started to study the immunosuppression issue because they've just been trying to save lives. Whether you're doing a better job of preventing viral shedding or preventing relapse, it becomes somewhat of a second order question.

I actually think that will start becoming an important question, particularly as we have our secondary surge this fall. It's going to happen again, guys. It won't be as bad, but it's going to happen again. And if these reports out of South Korea are true, that people are really relapsing--I'm suspicious of those reports, I'll be honest with you--but, the fact is there should be some real--there should be--there are theoretical advantages why an agent that is not immunosuppressive has advantages.

You can give it to anybody. You can give it to somebody who is obese, who is diabetic, who is elderly--remember, the elderly are already immunosuppressed, you don't want to double down on that--diabetics, you name--those are four of the probably six or seven comorbidities that are particularly high risk.

I like the simplicity. Our goal is the patient will be treated--basically they walk in, they have the diagnosis of COVID-19 to get a room air stat that's less than 93 percent, you're in, and, boom, they should be treated. If you--and that--it's a subcue injection. They can be treated while they're waiting for their other labs--this is theoretical now--be treated while their other labs are waiting to come back, while they're waiting for their test (unintelligible), etc.

The most important measure that the doc has in the ER is what that room--room air stat is--saturation. And that--they just put a little thing on the end of the finger and, boom, they get the results. The trial is also written that they get the second dose at seven days. My prediction is that many of the patients will actually leave the hospital before that seventh day, and we're going to have to chase them down and give it to them as an outpatient.

Jason McCarthy
Well--well--.

R.J. Tesi
--But the fact is I--yeah?

Jason McCarthy
I'm sorry.

R.J. Tesi
I strongly believe--.

Jason McCarthy
--What would you think--it's better to be--if--sorry. If you get the oxygen level--if they're hypoxic, and you get the oxygen level back to normal just from--just even from a single dose, as a physician, what would you consider that patient being stable in terms of--how long before you're comfortable--.

R.J. Tesi
--Yeah--.

Jason McCarthy
--Letting that--abstract. I'm holding you to it.

R.J. Tesi
Yeah, yeah. I--I can tell you that if--even if the patient got better in five or six hours, I doubt--and, remember, I haven't practiced medicine in a while--but I doubt a clinician would send them home that day because they came in with a life-threatening level of oxygenation. They would want to watch them overnight.

But, the fact is if you can convert a--an admission from a--to a 24 or 48, 72-hour admission from one that--instead of a 10-day admission, you're not going to be like Montefiore was just two days ago, sitting there with 1,600 COVID--or maybe it was only 1,200, what does it matter--but over 1,000 COVID-19 patients in their hospital, right, you'll be able to get patients out. And hopefully they won't infect anybody else while they're out of the hospital.

Jason McCarthy
And when the protocol parameters are finalized, will we know what the target change in oxygenation or whatever endpoints related to oxygen saturation are going to be required--for how long?

R.J. Tesi
Yeah. So--so good question. In general, the way the protocol is written, it allows the clinical teams to justify their criteria almost--when you read most of the protocols, they want patients--the need to be on room air with a standard respiratory rate, certainly less than 20, greater than 90 percent, 98 percent sat on room air before they'll go home.

And so, we leave that to the clinical teams because the last thing a protocol can do is dictate medical--that kind of medical care. The bottom line is, we think the difference will be that you're going to have fewer patients with what we call the catastrophic complications--admission to ICU, intubation, renal failure, cardiac problems, MI, congestive heart failure, neurologic problems, and death. That's--those are the primary endpoints. And we would expect to have shorter hospitalization. And--and that's really it in a nutshell.

This is--the way it's written, it's a very quick clinical trial. I mean, like I said, the second dose of drug, as it's written--and that may change as it goes through the final evolution with the clinical teams and the regulatory authorities--is they're just getting the second dose at day seven, and then they're basically home free, we hope.

Jason McCarthy
Great. Thank you, sir. Stay safe out there.

R.J. Tesi
You, too.

Operator
Our next question comes from the line of Mikal Kaiser (SP) with BTIG. Please proceed with your question.

Mikal Kaiser
R.J., thank you for taking my questions--just talking a little bit more about the protocol, just wondering the type of severity of these hypoxic patients that you guys are--are going to be looking at. Are these predominantly patients that are not going to be on any sort of medical mechanical ventilation or ECMO? Is that sort of the type of patients that you'd like to capture in this protocol?

And then, just to follow up on the--on the prior question, what are some of the more immediate sort of endpoints that you guys might be looking at in terms of cytokines or maybe sort of discharge timelines? What are--what are some of the things that you guys are considering from endpoints perspective? Thank you.

R.J. Tesi
Yeah. Yes. Yeah, so thanks. So clearly, the patient we don't want is one that is requiring mechanical ventilation. So in other words, we're--we're actually targeting the patient that's sitting there, and they're wondering if he's going to progress to needing more intensive care. And so, by intensive care we mean it could be CPAP, it could be intubation, it could be ICU care. I mean, that's the way the protocol is written.

Ideally, these patients get nasal prongs, they get just a face mask with oxygenation, and over two or three days they get better and go home. That's the ideal scenario. There are--as I mentioned in the lead-in, there's plenty of protocols for the more sick patients. And as you know, by the time they need ECMO, these are really sick puppies. And they are--are a handful.
But even the patients that have fully blown ARDS and on mechanical ventilation require very, very sophisticated care.

As I said, the primary endpoint are those catastrophic complications. The moment you end up in an ICU, the moment you end up intubated, the moment you develop renal failure, you are considered to have failed the protocol. Our goal is to keep patients well enough that they never reach that point. And we think we can have an impact on these patients, and that will make a very big difference in the number of patients that potentially die of the disease and certainly on the utilization of--of the health care resources.

Mikal Kaiser
Thank you.

Operator
Our next question comes from the line of Swayampakula Ramakanth from H.C. Wainwright. Please proceed with your question.

R.K.
Thank you. This is R.K. from H.C. Wainwright. Most of my questions have been answered. But, I have a couple of other (SP) things. R.J., as you know, CRS is not unique to COVID-19 infection. And do you see a larger indication where in DNF--DN-TNF could be used beyond the seemingly acute COVID-19 infection? I know--I know it doesn't look like that, but I would think and hope that within three to six months we are on the other side of this pandemic.

R.J. Tesi
So thank you. That's a good question, R.K. We--we--in the earnings call on, I think it was the 11th of March, one of the comments we made was that we would not get into the COVID-19 arena if we didn't see a commercial application beyond COVID-19. Like you, I worry that COVID-19 will be here today, gone tomorrow, like SARS, MERS, swine flu all were. Let's hope we have the same luck with COVID-19 because none of us want to have to go through this again.

But, you're exactly right. We--the reason we decided to move forward is we do see an opportunity for treating the cytokine release syndrome associated with either CAR-T cells or, even more interestingly, with the immune-mediated complications of check--immune checkpoint inhibitors. As you know, if patients fail steroids, which are immunosuppressive and make patients with cancer worse, they're given the nonselective TNF inhibitors which, by the way, are immunosuppressive, and they make patients worse.

Here we have a potent anti-inflammatory that is not immunosuppressive that in animal models of cancer actually improves the response to cancer and in animal models of infection actually improves the response to infection. So, we actually think there is a clinical opportunity that's important, based on today's use of immunotherapies, beyond the COVID-19 market. And so, in some ways this gave us an excuse to get into that market, although we're making a stop, taking care of patients with COVID-19 before we move on.

R.K.
So, thanks for that. In terms of evidence, is there any direct or indirect evidence that has shown that by lowering soluble TNF--TNF levels--leads to mitigation of CRS or any of the parameters of CRS?

R.J. Tesi
So, there are--are three bits of information. I've forgotten the drug, but remember about a decade ago when that normal volunteer study in the U.K. went sideways when they treated patients with an immunotherapy, and five or six of them ended up in the hospital. And these patients--it was the first time the term cytokine storm actually had been used in a setting of a pharmacological agent.

And in fact, in all those patients, the--the severity of the storm correlated with inflammatory cytokine levels, including IL-1, IL-6, and TNF. So as they got better, their levels went down. So, that's number one. Number two, in the COVID-19 patients, those that have elevated TNF levels, as they get better, their TNF levels come down spontaneously. So, that's in response to the viral--virus resolving, not a pharmacological intervention.

And the third piece is the indirect piece, that the current standard of care at many institutions after failure of steroids, or even after treatment of immune-mediated complications of you name (SP) the checkpoint inhibitor, is to use a TNF inhibitor. So, I think we have pretty good evidence that there's a role of soluble TNF in this disease and that treatment will have some benefit. But, I think it has never been proven to kind of a level one degree that is required for FDA labeling.

R.K.
Thank you. And the last question for me is, I know you talked a little bit--not a little--you talked quite a bit about the microcoagulation part, both in your lead-in and also in the Q&A session. But, I'm just trying to kind of piece it together in terms of how DN-TNF helps and lessens the microcoagulation. Is it--is it anything--is it systemic, is it tissue specific? How should we think about that?

R.J. Tesi
So, let's speculate here a little bit--and I've spent some time looking at this. And clearly what happens is the inflammatory cytokine soluble TNF activates endothelial cells to secrete tissue factor, and then tissue factor actually starts this coagulopathy going. So, it's not the direct effects on the coagulation parameters.

That's the role of tissue factor. But clearly they do stimulate endothelials to produce tissue factor.

And if you stop that endothelial activation, the hypothesis is--to be proven--that actually you will improve--improve that coagulopathy. D-dimer, which is a measure of fibrinolysis, is elevated in almost all patients with COVID-19. And the more severe their disease, the higher the D-dimer levels. So, we will be able to actually test this easily in our patients.

I mean, it's going to be a randomized trial, so we won't (SP) have a control arm, so we'll know--be able to tell if we get rid of--if D-dimer levels go down more rapidly in our patients that are treated with DN-TNF, I mean, that gives credence to my hypothesis--probably doesn't prove it, but it certainly supports it. I actually think--I actually think the coagulation issue is kind of the--the bogeyman of COVID-19. Everyone is focused on the--on the lungs, which are bad. But, I think--I think the coagulopathy is going to be a problem that we have to deal with for many years.

R.K.
Yeah, I agree. I believe hydraulic disease would be the long-term impact of (unintelligible).

R.J. Tesi
Yeah, yeah.

R.K.
All right. Thank you very much, and good luck with the program and hope to get more information on the study itself as time goes along.

R.J. Tesi
Thanks, R.K.

Operator
Ladies and gentlemen, once again, if you would like to ask a question, please press star, one on your telephone keypad. Once again, if you would like to ask a question, please press star, one on your telephone keypad.

Our next question comes from the line of Carl Byrnes with Northland Securities. Please proceed with your question.

Carl Byrnes
Thanks for the question, and congratulations on the progress. Assuming--assuming that everything goes smoothly, when might be the soonest you would expect to have data from the trial? And the bulk of my questions with respect to endpoints and secondary endpoints were addressed. Thanks so much.

R.J. Tesi
Yeah. So, that obviously depends on, one, how quickly we get started, and what the nature of the surges are. I mean, if you look at--in the U.S., I mean, even though Governor Cuomo is breathing a sigh of relief, I mean, you're still talking about extraordinarily high levels of admissions every day compared to many places in the country. I mean, many places in the country--some will never have a surge, and some are probably just starting their surge.

As you know, we're--we are--have particular interest in doing clinical trials in Australia and the U.K. Australia has done an extraordinary job of really blunting or squashing the curve. I mean, as of two days ago, they only had 7,000 cases. This is a country with as many patients or people as California--only 71 deaths total. That's 1 percent. Their ICU census was 49 for the whole country. I mean, that's like Lower Manhattan. That's like a 10-block area in Lower Manhattan.

And there were only 170 inpatients in the whole country with COVID-19. So, it has really been mild. That means either they've squashed it, or they haven't hit it. And we shall see. So, I think the best place to do the trial is the U.S., so we're talking to the FDA--I think the--the--as far as patient enrollment. And then, it should be reasonably quick.

From a financial point of view, we love Australia because of the rebate program. So, we're obviously working with the Australian authorities also on potentially setting up a trial there. I don't want to promise you which venue or if both will be involved, but those are the places where we're working with the regulatory authorities as we speak.

So, how quick? Hey, golly, I don't know. It's a beautiful thing from a clinical development point of view because it's a--really a 28-day study from time to enrollment to your last safety visit. But obviously it depends on how quickly the trial enrolls, and that depends on how long the virus sticks with it. So from a personal point of view, I hope it never enrolls because the virus has disappeared. From a business perspective, I suspect it will enroll faster than that because I think the virus is here to stay.

Carl Byrnes
Great. Thanks so much.

Operator
Ladies and gentlemen, this does conclude our question-and-answer session, and I would like to turn the floor back over to Mr. Tesi for any closing remarks.

R.J. Tesi
So, in my closing comments, before I turn it back to David, thank you for joining us. We appreciate your interest in learning about why we decided to really embark on the COVID-19 program. I think that we all have some responsibility to help with this pandemic, and so we are playing our role. David, anything you want to add?

David Moss
No, I really appreciate everybody's time. And that concludes our statements. Again, if you have any comments or questions, please reach out directly to us at any of the contact information you'll find on the website. Thank you.

Operator
This concludes today's--this concludes today's teleconference. You may now disconnect your lines at this time. Thank you for your participation. Have a wonderful day.