NATIONAL CITIZENS INQUIRY

Quebec, QC **Day 1**

May 11, 2023

EVIDENCE

(Translated from the French)

Witness 4: Dr. Jean-Marc Sabatier
Full Day 1 Timestamp: 06:05:46–07:00:07
Source URL: https://rumble.com/v2sjzn2-quebec-jour-1-commission-denqute-nationale-citoyenne-franais.html

[00:00:00]

Konstantinos Merakos
So hello again everyone. We had a little dinner break. So thank you for being here. We have our next witness with us, but before that, I'll start by introducing myself. It's good to know the lawyers are here today to help the situation. So my name is Konstantinos Merakos. I am a lawyer in Canada for the firm Bergman & Associés. A brief word about our experience. In 2020, our firm represented members of the public service in a legal action against the federal government on the basis of violations against the Constitution of the Charter of Rights and Freedoms and Human Rights. This was after the federal government expelled public servants over their right to privacy, bodily integrity and their medical choices. So I would quickly like to thank the forum for its professionalism and respectful exchanges, and I want to emphasize that it is not only important but crucial in a free and democratic society to have forums like this.

So thank you and congratulations. Without further ado, we'll move on to the next witness, Monsieur Sabatier, who is on Zoom with us right now. Monsieur Sabatier, can you hear us?

Dr. Jean-Marc Sabatier
Yes, hello.

Konstantinos Merakos
Are you doing well?

Dr. Jean-Marc Sabatier
Yes, very well, thank you.

Konstantinos Merakos
Thank you for being with us. So I'm going to start by having you sworn in. So do you swear or solemnly affirm to tell the truth, the whole truth and nothing but the truth? Say "yes" or "I solemnly affirm it."

Dr. Jean-Marc Sabatier
Yes, I solemnly affirm it.

Konstantinos Merakos
Good. Your full name, please?

Dr. Jean-Marc Sabatier
Jean-Marc Sabatier.

Konstantinos Merakos
Ok, and where are you currently located?

Dr. Jean-Marc Sabatier
Pardon?

Konstantinos Merakos
Where do you currently live?

Dr. Jean-Marc Sabatier
I live in Rousset, so in the south of France.

Konstantinos Merakos
Okay.

Dr. Jean-Marc Sabatier
It's near Marseille.

Konstantinos Merakos
And are you alone in the room or is there someone else?

Dr. Jean-Marc Sabatier
Yes, yes, yes, yes, I am alone.

Konstantinos Merakos
Okay. So Monsieur Sabatier, today, we will first of all speak about you, your CV and— I have here the message that you sent and essentially, it will be before the committee here, with

whom you spoke. So I'd like to start by discussing your CV, your background and your expertise. So briefly, in a few sentences, your expertise, please.

Dr. Jean-Marc Sabatier
Yes. In fact, I am a research director at the CNRS: the National Center for Scientific Research. It is the French research body. My educational background is a doctorate in cell biology and microbiology, and I have a Habilitation to Direct Research, therefore an HDR in biochemistry. And so I've been working in a research lab since 1985. I've worked in different fields, but my specialty is toxins, microbes, and protein engineering. And in particular, I have worked on vaccines since I joined the CNRS in 1989 on the topic of vaccines. At the time, they were HIV vaccines. On that occasion, I worked on the subject with Professor Montagnier, since we had a partnership with the Institut Pasteur in Paris.

Konstantinos Merakos
Perfect, thank you very much. And currently, you are still working in the field. What is your present employment?

Dr. Jean-Marc Sabatier
Yes, so I currently work at the Institute of Neurophysiopathology in Marseille, and I research COVID. Among other things, I am editor-in-chief of infectious disease journals, in particular a journal called *Coronaviruses*, which is really specialized in coronaviruses, and another journal that is more specialized in germs, let's say, and then diseases associated with germs. It's a journal called *IDDT*. Both are peer-reviewed international journals.

Konstantinos Merakos
Excellent. Thanks. In a few words, I see your résumé here, which is very extraordinary. Can you say a few words about patents? There are quite a few pages on the subject here. Can you say a word or two? Are these patents that you participated in creating? Is it something that is under your name? We will perhaps identify one or two patents which would be important for today.

Dr. Jean-Marc Sabatier
Yes. I was the co-author of 55 patents, there are joint patents with the Institut Pasteur—moreover, old patents signed by Professor Montagnier, so in virology on HIV.

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And then, more recently, there are also patents on toxins and on microbes, on antibacterials, for example; and in particular, we filed a patent on a molecule that I had designed and produced chemically, which has been tested in an FDA protocol against HIV, the human immunodeficiency virus. I've worked quite a bit on microbes. I'm also editor-in-chief of another journal which specializes in antibiotics, in other words, molecules that are active against bacteria.

Konstantinos Merakos
Excellent. Thank you. I know that here, if possible, we will talk about the virus's mode of operation and the pathological problems associated with vaccine injections, but I will leave

the floor to our commissioners to ask you questions. So I thank you and I will leave you to it.

Dr. Jean-Marc Sabatier
Thank you.

Konstantinos Merakos
Thank you.

Commissioner Massie
Hello, Dr. Sabatier, my name is Bernard Massie. I am also a researcher, but I finished my career a few years ago. I was in biotechnology so I know the whole history of patents. I know that during this pandemic, there was a lot of work that was done around the axis of the ACE2, I don't know how to say it in French. . .

Dr. Jean-Marc Sabatier
Yes, the ECA2 [in French].

Commissioner Massie
. . .which regulates an extremely important function, and that you have particularly focused on trying to perhaps explain both the pathology that could be detected with the infection—with SARS-CoV-2—but also with pathologies that arise from, or rather, the undesirable effects that result from the injection and the abundant expression of the spike protein following the gene injections. Can you briefly describe to us the problems that can be detected, and perhaps draw a parallel between being in a condition of infection versus injecting these coding sequences to produce, or overproduce, the spike protein?

Dr. Jean-Marc Sabatier
All right. So first of all, to describe the virus's mode of infection, I must remind you of how the renin-angiotensin system works. In fact, it is a system that I must explain in some detail for you beforehand in order to understand precisely how the virus works on this system, and how current vaccines—which are essentially based on the spike protein—can act. More specifically, messenger RNA vaccines. First of all, this renin-angiotensin system is extremely important because it is the number one system for the functioning of the human body. It really allows the functioning of all our organs and tissues; and it is in this capacity, therefore, that it has a truly essential role for our body to function. In particular, it is responsible for renal, pulmonary, and cardiovascular functions. It also controls innate immunity, and it controls the different microbiota, therefore: the intestinal microbiota, which is the second brain, and also the vaginal, cutaneous, and oral microbiota. So you see that it is a very important system.

So to outline in a few steps how this system is affected by the virus, as well as the vaccine spike protein works. First of all, in some cases, you have a substance in the liver that will be produced which is called angiotensinogen, and then you also have the kidney which will produce an enzyme called renin. And in fact, this renin will degrade angiotensinogen to give angiotensin 1, which is a hormone. This angiotensin 1, in turn, will be degraded by another receptor called ACE1, which is the angiotensin-converting enzyme 1. And when this molecule is degraded, it will produce angiotensin 2, which is another hormone. This

angiotensin 2 is the key to COVID diseases. If you will, this angiotensin 2 normally recognizes a receptor called ACE2, which is the angiotensin-converting enzyme 2. Now, this ACE2 receptor is the target of the spike protein, either of the virus during a natural infection or, in certain cases, of the vaccine spike protein—in other words, the one which will be produced by the vaccines, in particular messenger RNA ones. Since messenger RNA vaccines are vaccines in which RNA is injected into the deltoid muscle, and is coupled to lipid nanoparticles which allow penetration into the cell, these RNAs will be translated into spike proteins, which are actually the vaccine spike proteins.

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So what happens, if you will, is that this spike protein— whether viral or vaccine-induced—will be able to recognize the ACE2 receptor, in other words, the angiotensin-converting enzyme 2. And in fact, by binding to this ACE2 receptor, they will interfere with the degradation of angiotensin 2 because normally angiotensin 2 is degraded by the ACE2 receptor to give another hormone called angiotensin 1-7. And so when you have a natural infection or when you receive a vaccine injection, at least in certain cases, you can hinder the degradation of angiotensin 2, which will then end up in excess and which will over-activate its own receptor. Its own receptor is called AT1R, and it is a receptor that can be extremely harmful. That means it is a receptor that is completely essential for the human organism to function because it just so happens that it pilots all these renal, pulmonary and cardiovascular functions. It controls innate immunity and it controls the different microbial flora, so it has a very, very important function. But on the other hand, when it is overactivated—and that is precisely the case when there is an infection of the SARS-CoV-2 virus, like when we have COVID or when we receive a vaccine injection or a vaccine booster injection—at that time, this receptor can be overactivated, which can be very harmful because it is capable of launching a host of cellular signalling pathways since it is an extremely complex receptor. It's one of the most complex receptors that we know of—one of the seven-transmembrane segment G protein-coupled receptors—and it can do a lot of things because it activates pathways, or cellular signaling cascades.

So I won't go into details, but the best known are JAK/STAT, p38 MAP kinases, NF-kB. There are many more. And in fact, what this receptor does when it is overactivated—Because you have to know that we find the renin-angiotensin system on which this receptor depends in all the organs of the human body: in the heart, in the lungs, in the liver, in the spleen, in the intestines, in the adrenal glands, in the thyroid. We even find it in the brain, we find it in the gonads, in the reproductive organs. So it really is absolutely everywhere. And so this AT1R receptor, when it is overactivated—which just happens to be a consequence of the attachment of the spike protein to the ACE2 receptor, and therefore, of the overactivation of the AT1R receptor—can cause vasoconstriction. In other words, it will be pro-hypertensive. It will also be pro-inflammatory, which means it will launch a storm of pro-inflammatory cytokines, for example, a production of interleukin-1, interleukin-1 beta, interleukin-6, TNF alpha, interferon gamma, so it's very harmful because it can start a lot of inflammation. At the same time, it is pro-oxidant, which means it will generate oxidative stress at the cellular level. And this is very harmful since, in fact, oxidative stress can kill cells because it can put them into apoptosis—in other words, into programmed cell death—and then that can also put them into autophagic dysfunction. In any case, it is very harmful.

So the AT1R receptor has this pro-oxidant effect because it activates an enzyme called NADPH oxidase, whose nickname is NOX. This enzyme will release reactive oxygen particles which are very harmful because they can kill mitochondria, which are the energy centers of the cell; and so when they kill the mitochondria, they also kill the cells. So this

AT1R receptor is also pro-angiogenic; that means it will promote the growth of blood vessels, and so, among other things, it will be able to grow tumors, even launch tumors. It has a pro-cancer effect too, which is also problematic. The overactivated AT1R is a receptor as well, which is prothrombotic; in other words, it can initiate thrombosis. We know how serious this is since the majority of people who die from severe COVID die from thrombosis. It is also pro-hypoxemic; that is to say, it will reduce the oxygen load of red blood cells—the red blood cells that carry oxygen to our cells, tissues, and organs so that they can work. So it decreases this dioxygen load since it, in fact, hinders the incorporation of dioxygen on the iron, which is present at the level of the hemoglobin of the red blood cells. At the same time, you also have this receptor which is also pro-hypoxic. In other words, being pro-hypoxemic by causing the blood saturation to drop suddenly, it causes a deficit in the supply of oxygen to our tissues and organs. We consider it hypoxia when we are at a saturation level of less than 95 per cent oxygen in the blood.

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You also have a pro-fibrotic receptor, which means it will be able to induce fibrosis of organs, which is also very harmful because it is often completely irreversible. It could be fibrosis of the heart; it could be fibrosis of the lungs. And it's a receptor that's also pro-hypertrophic, meaning it causes organs like the heart and lungs to swell, simply because the renin-angiotensin system is actually involved in cell differentiation and multiplication, and that's why it can make organs grow and enlarge. And that's also why it can have a pro-cancerous effect since cancers are in fact an anarchic proliferation of cells. So alongside all that, this AT1R receptor can also lower the production of nitric oxide, which is a very important substance because it is involved in all the inflammatory, immune and memory phenomena, all the cognitive problems. That's why people who have long COVID often have memory problems or cognitive problems. So it's due to this drop in nitric oxide or NO. You see, therefore, that this overactivated AT1R receptor, either by the viral spike protein, or by the vaccine spike protein, can be very harmful. Because it is, to sum up: pro-hypertensive, pro-inflammatory, pro-thrombotic, pro-hypoxic, pro-hypoxemic, pro-fibrotic, pro-hypertrophic, and lowers nitric oxide.

And besides this, the essential problem with current vaccines—for the messenger RNA vaccines—is the toxicity of lipid nanoparticles. So just as a reminder, lipid nanoparticles are what allow these messenger RNAs to enter the cells. In fact, there are four types in vaccines: so Spikevax from Moderna, and the Pfizer vaccines, Pfizer BioNTech and Comirnaty. Actually, these lipid particles are cholesterol and phospholipids, so they are not a problem. And the ones that are problematic are the other two types of lipids because they are pegylated lipids and cationic lipids, which are not natural. And so these smaller-sized substances can be picked up by the different organs, and then, what is even more concerning, they can cross barriers: in particular the placental, blood-brain barrier, et cetera. And so these messenger RNAs which cause the spike protein to be produced are simultaneously harmful precisely because this spike protein was badly chosen from the start; that is to say, it was slightly modified. You know, actually, the spike protein is like a string of pearls made up of 1,273 pearls, with the pearls being amino acids. And you have twenty different types of pearls. In fact, the designers, that is, the designers of these messenger RNA vaccines, have modified two pearls: one bead at position 986 and one bead at position 987. They actually replaced them with two proline residues. However, prolines are amino acids, which are somewhat special because they can make a connection with the amino acid that is upstream, in either cis or in trans [configuration]. In fact, that means that at the level of these two modified prolines in the messenger RNA vaccines, we can have several types of configurations, so a trans/trans, cis/cis, cis/trans, or trans/cis configuration.

So what does this actually mean for our listeners? This means that at the level where the beads were modified, the peptic chain can have four different orientations. In other words, at that level, the pearl necklace's spike protein can be oriented in four directions. And in fact, these four orientations enhance or increase the probability that the S protein—or the spike protein, which is actually produced by the translation of these vaccine messenger RNAs, or RNA vaccines— This means that it can, in fact, adopt different shapes in space, and that enhances the possibility that these S proteins, or spike proteins, combine into a trimer. And when it associates in threes—in other words, when it is an association of three spike proteins—at that moment, it looks like the spike protein of the virus: it looks like the spicule, which is, in fact, an association of three S proteins. And when it looks like the spicule, these vaccine proteins in trimeric form have the ability to recognize the ACE2 receptor. And once attached to the ACE2 receiver, what do they do? They do exactly what the virus does, which is to interfere with the breakdown of angiotensin 2.

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This angiotensin 2 will therefore be in excess. It will over-activate the AT1R receptor, which will produce all these harmful effects—which I spoke to you about five minutes ago—and it will trigger a lot of more or less severe diseases, and will be able to affect many organs. This is why the COVID diseases that we find in long COVID are very varied. Because this renin-angiotensin system is pervasive and is, in fact, connected to all the organs since it is found on the surface of many cell types—in particular all the endothelial and epithelial cells, as well as all of the nerve cells. On nerve cells, you have neurons and oligodendrocytes. In the immune system in the brain, you have astrocytes and microglial cells. All of these cells have ACE2 receptors.

Also at the level of the reproductive organs, you find these ACE2 receptors in the prostate, the penis, and the testicles; and in women, in the endometrium and the ovaries. So it really is present everywhere. And it also lines the entire vascular system, and that is precisely why we can have cardiovascular problems since it covers the entire internal lining of the blood vessels. And it's really pervasive because we find it even at the level of mitochondrial membranes, as well as inside cells. So it's not only on the exterior of cells. There's also a renin-angiotensin system which is intracrine and which, in fact, controls all the functioning of the cell. And we find it particularly in all the cell membranes: on the internal membrane of the mitochondria, which are the energy centers of the cell and allows the cells to live. But we also find it in the membranes of cell organelles such as the endoplasmic reticulum and the sarcoplasmic reticulum, even the nuclear membrane, so they are found in the endosomes, exosomes, and lysosomes.

Well, they are really present everywhere, and that means, in fact, that this renin-angiotensin system that controls our organs can be extremely harmful precisely because it is present everywhere. And the problem with the current vaccines is that they are all based on the spike protein, and this spike protein is, in fact, to a certain extent able to recognize this ACE2 receptor and to make the system malfunction. And by causing this system to malfunction, well, these vaccine spike proteins effectively do the same thing as the virus, which is to say they disrupt the renin-angiotensin system, they over-activate the AT1R receptor, and they cause all the pathologies that we know today.

Commissioner Massie
If I can take the liberty of summarizing what you are saying— If I correctly understand what you are saying, it is that the spike protein found on the coronavirus will engage this

system and can potentially cause any series of dysfunctions at the cellular level, and even at the organ level. And similarly, the spike protein, which is expressed as a result of gene injections, can do the same thing. So can I ask you a question regarding, I would say— Well, in the case of coronavirus infections, with SARS-CoV-2, based on the recent epidemiology that we have, we can practically conclude that a very large majority of people have been infected, exposed to the virus. But that a large number of these people would not present symptoms, or at least not easily detectable ones. Is it like this because people's immune systems have stopped the virus from spreading to enough places in the body to cause these malfunctions? Or, at the same time, are there people who, in terms of this system—which seems extremely complex, with enormous ramifications in all sorts of cellular pathways and in all sorts of different organs—are there people who would have a better capacity to manage this kind of dysfunction?

Dr. Jean-Marc Sabatier
Yes, absolutely. So in my opinion, it is precisely the people with relatively severe forms of COVID—even fatal forms—who are essentially the people who are vitamin D deficient. Vitamin D plays a very important role in this system because it acts upstream of the system, as it is a renin inhibitor. And renin is the enzyme that transforms angiotensinogen into angiotensin 1. And this angiotensin 1 is the precursor of angiotensin 2, which over-activates when it is in excess because of the presence of viral or vaccine spike protein, which over-activates the AT1R receptors. So you should know that indeed, people who are vitamin D deficient or insufficient—that is to say with levels lower than 30 nanograms of calcidiol per ml; for people who are deficient, it is lower than 12 nanograms of calcidiol per ml—at this point, there is a very harmful effect, precisely because the spike protein, viral or vaccinal, will over-activate the AT1R receptors, which will go into overdrive.

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So there will be a disruption, an overactivation of the renin-angiotensin system, and vitamin D will not be there to thwart this system since it would have a braking effect on this system.

And we should be aware, of course, that there is a genetic polymorphism, if you will, of the renin-angiotensin system. We don't all have the same renin-angiotensin system. If someone is Caucasian, Indian, Asian, African, they will have different renin-angiotensin systems. In other words, globally, we all have the same elements of the renin-angiotensin system, but there is a biodistribution of the receptors that is not the same. And then there are also variants at the level of the receptors and of the molecules as well. Now, for example, 35 variants of the AT1R receptor are known. So there is a polymorphism which is very important at the level of the RAS [renin-angiotensin system] that can actually also explain the differences in the occurrences which can be observed in people. We should be aware that this renin-angiotensin system is also not the same in the same person throughout his life. In other words, when you are a baby, you do not have the same renin-angiotensin system as when you are a child, a teenager, an adult, or a very old person. It constantly evolves throughout your life. And then, we should also be aware that a woman does not have the same renin-angiotensin system as a man. Why? Because, among other things, the ACE2 receptor, is encoded by a gene which is located on the X chromosome, which is the common sex chromosome. The AT1R receptor, which is responsible for COVID diseases, is encoded by another chromosome, which is chromosome 3.

But, in any case, what is certain is that there are comorbidities which make us more sensitive to an over-activation of the renin-angiotensin system when we have this system

that is already out of order. In other words, when you have comorbidities—for example, when you are hypertensive, when you have an autoimmune disease, when you have cancer—that means you already have a problematic renin-angiotensin that is dysregulated. And therefore, the vaccine injection can have a much more harmful effect on such a person. Likewise, a SARS-CoV-2 infection can cause a much more severe case of COVID precisely because these people are susceptible. We also know of genes that make someone more susceptible to developing serious forms of COVID. For example, there is a gene called HLA-B27. We know that people who have this gene have a greater risk of having a severe form of COVID.

So you have other genes that are involved. For people who have this HLA-B27 gene, it is interesting to know that, in the situation of infection with HIV or the hepatitis virus, it has a protective effect. Who knows why, but it does not behave the same depending on the microbes. Anyway, there are genes which strongly affect outcomes. Of course, in this gene polymorphism, that is very important. There are other diseases, you know, in people who have problems like Marfan's disease, for example, with a defect in the production of fibrillin-1, or people who have Ehlers-Danlos disease, for example, who have a problem producing collagen since they have a collagen-deficient gene. When they are infected with the virus or receive a vaccine injection, these people develop more severe forms precisely because they have a problem.

Commissioner Massie
Monsieur Sabatier, I will try to focus the discussion a little with a question for you, which has to do with the fact that, well, this spike protein— According to your knowledge of coronaviruses, and given its preferred target with the ACE2 receptor, is it unique in the coronavirus family or does it exist in many other coronaviruses?

Dr. Jean-Marc Sabatier
No, it's found in coronaviruses. It's not unique at all. For example, you know that SARS-CoV-2 is a beta-coronavirus, from the sarbecovirus family. So it's an enveloped virus with spike protein and then you have a single-stranded arm positive-sense ribonucleic acid.

Commissioner Massie
My question, more specifically, is this: Is the interaction of this spike with this receptor new?

Dr. Jean-Marc Sabatier
So it recognizes the ACE2 receptor. We need to be aware that the 2002 epidemic was also an infection with a coronavirus. It's SARS-CoV, now called SARS-CoV-1. So the target of this coronavirus was also the ACE2 receptor, in other words, the angiotensin-converting enzyme 2. The MERS-CoV of the 2012 epidemic, on the other hand, was different.

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It's also a coronavirus, but it was targeting another receptor, which is CD26; it's a DPP4—a dipeptidyl peptidase-4—which is another receptor. So we are aware of different types of receptors.

But you also have, for example, the cat FIP [feline infectious peritonitis] virus, which also disrupts the renin-angiotensin system, and which, in fact, causes exactly the same diseases

in cats that we see as COVID diseases in humans. It also disrupts the renin-angiotensin system, but it recognizes another receptor: it recognizes the spike protein of the cat coronavirus, of the cat FIP virus, and it recognizes another receptor called CD13, that is aminopeptidase N; and in fact, in this case, it will hinder the degradation of angiotensin 3. This angiotensin 3 will be found in excess since the spike protein of this coronavirus has fixed on the APN receptor, on the CD13 receptor. And so this excess of angiotensin 3 will also over-activate the cat's AT1R receptor and will cause COVID-type diseases in cats. We find exactly the same pathologies with hypertension, thrombosis, and pleural effusions. So if you will, you have a whole family of receptors, and obviously, there are other receptors that are targeted by coronaviruses.

Commissioner Massie
Going back to the treatment of COVID with this alleged magic wand, which was the gene injection for the expression of the spike protein, you mentioned briefly that you thought it was probably not very wise to choose this antigen in the platform, notwithstanding the quality of the gene platform that was chosen. The choice of this protein was misguided. My question is, how long have we had sufficient knowledge to conclude that—when we made the choice to use this protein—we should have known that there would be problems in choosing this target for vaccine platforms?

Dr. Jean-Marc Sabatier
As early as 2002, in fact, because the 2002 SARS-CoV virus also targeted the ACE2 receptor, so we already knew that it was a receptor that was harmful. In addition, there has been work done since 2002 on SARS-CoV. There were facilitating epitopes that had been highlighted: that is to say, regions of the spike protein that contain facilitating epitopes; in other words, regions that will stimulate the immune system—in particular the production of antibodies which will, in fact, not neutralize the virus, but on the contrary, facilitate infection by the virus SARS-CoV. However, these domains are also found on the spike protein of SARS-CoV-2. So the vaccine designers could have already known that these regions were potentially harmful in the case of vaccination with a messenger RNA that codes for the spike protein of SARS-CoV-2.

In addition, this spike protein has other problems. The spike protein of SARS-CoV-2 has an RGD motif. It has isotypes that the SARS-CoV-1 spike protein does not have. And we know that this RGD motif is a small piece of the protein which is made up of three beads; these three amino acid residues make up RGD, or arginine-glycine-aspartic acid. We know that it can be very dangerous because it is a motif that recognizes membrane integrins. It has been shown that the spike protein of SARS-CoV-2 is capable of recognizing membrane integrins, among other things: in other words, capable of triggering activity in the cell. And it recognizes, among other things— And this was described experimentally—this spike protein of SARS-CoV-2 is able to recognize membrane integrins which are called alpha v beta 3 and alpha 5 beta 1. And this is serious because these integrins can also be recognized by collagen. But hey, these critical sites are hidden within the collagen, and also happen to have these RGD motifs which are hidden, and these are critical motifs. In fact, when the spike protein, if you will, binds to these membrane integrins, it activates a system called caspase-3 and induces cell death, or apoptosis.

Additionally, we know that there is another danger. In this spike protein of SARS-CoV-2, there is a furin site which happens to have a particular affinity for human cells. And so we knew that compared to SARS-CoV, it was going to increase the infectious capacity of SARS-CoV-2 and the harmful effects of this spike protein. And further, concerning your question

on the vaccine platform, it is bad because, you know, at the level of this messenger RNA, this vaccine messenger RNA has also been completely modified to be very stable. It received, for example, a polyadenylation tail in order to stabilize it.

[00:35:00]

The nucleotides have also been changed. You know, you have as a basis ATGC: adenine, thymine, guanine, cytosine. They modified uracil because, when it has ribose on it, it becomes uridine. They modified it to be a pseudouridine. And that's serious all on its own. It's playing sorcerer's apprentice because we only have a decade of hindsight regarding this pseudouridine. And above all, we don't really know what it does because we don't really understand all the enzymatic systems that process the pseudouridine found in these messenger RNA vaccines, especially when the uridine is replaced at the stop: UAA, UAG and UGA codons. When they are replaced, there are no stop codons—which means, if you will, that the system is unable to adequately recognize pseudouridines.

This means that when these vaccine messenger RNAs are translated, there is the possibility that the ribosomes are also capable of making mistakes: that the transfer RNAs are capable of making mistakes and of introducing a different amino acid than the amino acid found in the primary structure of the spike protein of SARS-CoV-2. This has been demonstrated experimentally. And furthermore, if ever the messenger RNA could, in one way or another, integrate into the genome of the host cell—which has not been completely ruled out either since there are systems that could apparently do this, such as a system called SINE-1 LINE-1—you would have a system that is, in fact, an RNA-dependent DNA polymerase activity that could actually make DNA from RNA. At the moment, these polymerases—DNA polymerase, RNA-dependent and RNA polymerase, RNA-dependent—we know that they are also not capable of correctly reverse transcribing a pseudouridine. This means that they can make a mistake. And if indeed the gene that codes, for example, for the spike protein or for the virus genome is effectively incorporated into the human genome, at that point, there may be mutations. So this platform is not ready. In other words, it is too stable. And the fact that it is too stable also leads to the fact that it is capable of producing a lot of spike proteins whereas, normally, a natural messenger RNA would quickly degrade.

Commissioner Massie
Monsieur Sabatier, can I interrupt you here? Because the explanations you give are excellent for a scientist like me. That's fantastic, but I suspect there are a lot of people in the room for whom these explanations are perhaps a bit too sophisticated. I would like to perhaps underline two points concerning vaccine strategy.

You have experience in vaccine development. What you said, in many words, is that this vaccine approach with the messenger RNA platform and the choice of target, which is the spike protein, is very misguided for a large number of reasons that you have listed. I am going to ask you a question that will go one step forward. From what we know about coronaviruses, is even hoping to develop a vaccine that could control the infection like the one we had a possible approach? And if it is possible, what would you suggest as a vaccine strategy?

Konstantinos Merakos
Excuse me, Monsieur Sabatier. Just a moment, sorry to interrupt you. Can you just speak a little slower for the translator, just speak a little slower? That's all. Thank you so much. You can continue, sorry.

Dr. Jean-Marc Sabatier
Yes, absolutely. It is quite possible to produce a vaccine against SARS-CoV-2, one that is a real vaccine and not a pseudo-vaccine. In fact, a vaccine must meet two demands, two essential criteria: It must first be effective, and then it must be innocuous to a certain degree for the people who receive these vaccine injections. However, the current vaccines that we are offered meet neither criterion. In other words, they are ineffective since they do not prevent the infection of the individual who is going to be "vaccinated," in quotation marks, and then in the event of infection, they do not prevent transmission from the person who has been vaccinated to the person who is not vaccinated.

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So there is already a lack of effectiveness. And furthermore, it is not harmless precisely because this vaccine spike protein is capable of causing the renin-angiotensin system to over-react, thus triggering COVID diseases. So what should have been done, and what the designers should have done when producing this spike protein that they modified— Just to remind you, at the level of two beads out of the 1,273 beads, the beads in a 986-987 position, they did that for a very simple reason actually; it was because they wanted to maintain this spike protein in a prefusion conformation. In other words, they wanted to expose a domain which is called the RBD, or the "Binding Domain" receptor, which is the domain of the spike protein that is able to recognize the receptor ACE2. So they wanted to expose this domain of the spike protein so that the immune system would be able to mobilize against it, and in particular, to produce neutralizing antibodies against it. Except that there is still a problem, since the spike protein is able to recognize this receptor, and that is why it is very harmful.

So in order to make a vaccine that is not harmful, it would be necessary to produce a spike protein analogue and to make sure that this structural analogue, modified on one, or even several beads— It would be necessary to make sure that this analogue of the spike protein was unable to recognize the ACE2 receptor—and that way, the spike protein would be somewhat safe. It is not certain, but at least it would not be as toxic as it is at present. Why? Quite simply because this spike protein analogue would not be able to bind to the ACE2 receptor. So there would actually be no disturbance at the level of the degradation of angiotensin 2 or angiotensin 1-7; and that way, there would be no dysregulation of the renin-angiotensin system, and there would be no overactivation of the AT1R receptor, which is the cause of COVID diseases. So that would be important. At the same time, they should have already removed the domains from this spike protein, in other words, the portions of the spike protein which are known to contain facilitating epitopes.

So just to remind you, the facilitating epitopes are the regions of the spike protein that stimulate the immune system—in particular the B lymphocytes, which, when they differentiate into plasma cells, will produce antibodies directed against this region. But these antibodies will not be neutralizing. They will do the opposite of neutralizing antibodies. In other words, they will not have the expected effect; they will have the opposite effect. That is to say, they will facilitate the infection of the host by the SARS-CoV-2 virus, quite simply because these antibodies will bind to the spike protein of the virus. And there are innate immune cells—especially macrophages and dendritic cells—which have a receptor on the surface that is called the Fc Gamma R2A receptor. These will, in fact, recognize the antibody-virus complex.

Commissioner Massie
Monsieur Sabatier, if I may interrupt you. Your explanations are once again very detailed. And my question was, well, actually, you answered it. This is not the type of vaccine that we should have developed. We could have potentially chosen a better target by modifying it. And the second step is the delivery platform. Do you think it's safe to use a genetic platform rather than a protein platform, as is suggested in some of the vaccines that exist at this moment? Do you think it would be safer or more effective to favour these protein platforms rather than genetics?

Dr. Jean-Marc Sabatier
So in a few words: without a lot of data as at the beginning, it was already somewhat logical and normal to choose the envelope glycoprotein of a microbe because that is what is usually done. But let's say that given the history of SARS-CoV, they could have already seen that there were problems with this spike protein. They could perhaps have targeted another antigen of the virus, in particular the N protein—an internal protein, the nucleocapsid protein—since that one is highly conserved, and can produce neutralizing antibodies or stimulate a cellular response that is neutralizing. So that's another antigen that could have been targeted.

To me, the current messenger RNA vaccines are not at all good. In my opinion, it would take at least another decade for them to be perfected because we have no perspective on them. We have no perspective at all. We may say that these vaccines, these messenger RNAs, these vaccine platforms have been studied since the '80s—which is true, they have been studied since the '80s—but the work that has been done on them is not all conclusive since we don't know much. It is not known how stable these messenger RNAs really are.

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We do not know if they are able to produce 5, 10, 20, 100 spike proteins, et cetera, since they are very stabilized. We don't know what their biodistribution will be. We don't even know exactly how they will be translated. We don't even know, in fact, which amino acids are really going to be found in the spike proteins produced due to the presence of these pseudouridines, among other things. The lipid nanoparticles, too, which are used precisely in order to allow the penetration of these messenger RNAs, are not ready either because we know that they are also toxic in themselves, that they are picked up by the various organs, including the reproductive organs. They can be picked up by the brain, by the lymph nodes, by the liver, by the spleen—in fact, by many organs.

Commissioner Massie
If you allow me once again, if I may summarize your thoughts, you are saying that what we have at the moment are prototypes which are ineffective and dangerous.

Dr. Jean-Marc Sabatier
Yes, absolutely.

Commissioner Massie
That there would potentially be—

Dr. Jean-Marc Sabatier
We are lacking sober reflection.

Commissioner Massie
—the possibility of developing something better, but we are far from the mark.

Dr. Jean-Marc Sabatier
Yes, we should have taken inspiration from vaccination trials that have been carried out in cats. Because the cat FIP coronavirus—which is an alpha coronavirus, but is made exactly the same, is an enveloped virus with a spike protein, which disrupts the renin-angiotensin system—there were vaccination trials that were done on it, and those vaccination trials were not successful. So we know already that coronaviruses are not very easy targets. And as for messenger RNA vaccines, in my opinion, we don't have enough perspective on them at all. And I think that, personally, it was madness to vaccinate billions of people with a platform that is, in fact, still experimental; that is to say, we don't have years of hindsight on it.

Therefore, the other "anti-COVID" vaccines, in quotes— Whether they are: attenuated virus vaccines; adenoviruses like Sputnik, Janssen, AstraZeneca; or inactivated virus vaccines, Sinopharm, Sinovac, Chinese vaccines; or even vaccines with recombinant spike proteins like Novavax, the Sanofi vaccine, they also pose a problem because the spike protein is, in fact, there. And the problem is that the spike protein, in itself, is harmful. It should have been modified so as to not be harmful because it might eventually no longer be harmful. But that would be the first thing to study before launching large-scale vaccination trials, especially for a disease that is not very lethal. It would have been better to carry out early outpatient treatments, for example, by treating with an active form of vitamin D.

Commissioner Massie
So as we speak, in the situation we are in, you advise against vaccination with the vaccines we currently have. Does that sum it up a bit?

Dr. Jean-Marc Sabatier
Yes, these vaccines are harmful in themselves. They can cause COVID pathologies for the reasons I have given you. And then, it goes beyond that. There are a certain number of booster vaccine injections which are planned, up to ten, I believe. There is a strong push for booster vaccines. But that's madness because this spike protein affects immunity; because by disrupting the renin-angiotensin system, it affects innate immunity, since the renin-angiotensin system drives innate immunity. So that means the monocytes, the macrophages, the dendritic cells, the granulocytes and eosinophils, basophils, neutrophils, and the "natural killer" cells with the mast cells.

And so the dysregulation of the RAS affects innate immunity, and this innate immunity is what launches the specific adaptive immunity—which is based on the B lymphocytes and the T lymphocytes—and it therefore also disrupts the adaptive immunity that launches itself about four days later. And by disrupting innate immunity, what happens is that it induces a complete disruption of the immune system—since innate immunity launches adaptive immunity. And when we disturb the two, at that moment, it provokes an immunodeficiency, that is to say that it induces AIDS: an acquired immunodeficiency syndrome. And it's a type of AIDS which has nothing to do, of course, with HIV; it's an

immunodeficiency. And this immunodeficiency is accentuated by booster vaccinations since we exceed the immune system's threshold of organized criticality by injecting too many antigens—that is to say, too many spike proteins—either in the form of messenger RNA, indirectly, which produces the spike protein, or by directly injecting the spike protein— well, we induce this deficiency of the immune system.

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And it goes beyond that, since we can provoke the triggering of autoimmune diseases. Because innate immunity commands the recognition of self and non-self proteins, and therefore, when it is dysfunctional, it can recognize a self-protein as foreign—for example, as microbial—and can then initiate autoimmune diseases.

Commissioner Massie
Thank you very much, Monsieur Sabatier. In the interest of time, I will ask my colleagues and commissioners here if they have any questions for you. We have to move on to our next witness soon, who is waiting in line. Do you have any questions you'd like to ask, Ken? I'm going to translate and then if you could answer in French afterwards because the translator will make it possible to give the answer to the Commissioner and the audience will be able to hear. What's your question?

Commissioner Drysdale
Good afternoon. What you've been talking about so far has to do with a properly manufactured theoretical vaccine. Can you comment? We've had a lot of testimony about manufacturing issues with the vaccine. Can you comment on what additional effects manufacturing errors or manufacturing defects might have?

Dr. Jean-Marc Sabatier
Yes, absolutely, because, apparently, the vaccine batches—in particular for messenger RNA vaccines—do not appear homogeneous. That is to say, we can find messenger RNAs which are truncated, with batches that are not equivalent. And of course, when we inject messenger RNAs that are truncated, we also produce spike proteins that are truncated. So that means that we produce different types of spike proteins and that can be problematic, precisely because we know that the spike protein has harmful effects. And it can also be problematic to present fragments of spike protein since certain fragments of this spike protein can perhaps bind to specific receptors. Because in fact, we always talk about the ACE2 receptor when it comes to the spike protein, but there are also other receptors that have been described. For example, DC-SIGN, neuropilin-1: there are a number of receptors that are potentially targeted by this vaccine spike protein. This means that fragments can affect cellular functioning or can affect the functioning of physiological pathways. And so it's problematic. Normally, there should be very homogeneous batches of vaccines.

Commissioner Massie
Thank you for your reply. Do you have any other questions, Ken? Janice? Okay.

We thank you very much, Monsieur Sabatier, for this testimony and, indeed, for having contributed to enlightening us on this whole issue of vaccines. It will help us in our reflection and in the recommendations that the Commission will try to make for the future. We thank you very much.

Dr. Jean-Marc Sabatier
It is I who thanks you. Sorry for being a bit long.

Commissioner Massie
I will pass you on to our attorney, who will conclude this testimony.

Konstantinos Merakos
So Monsieur Sabatier, thank you once again for your testimony and for all the valuable information you have given us today. And, on that note, the Commission wishes you a good day. But I think it's an evening at home because you are six hours ahead of us.

Dr. Jean-Marc Sabatier
That's right, it's 9 p.m.

Konstantinos Merakos
So we thank you and wish you a wonderful evening.

Dr. Jean-Marc Sabatier
Thank you and I wish you success.

Konstantinos Merakos
Thank you so much.

[00:54:21]

Final Review and Approval: *Erin Thiessen, October 30, 2023.*

The evidence offered in this transcript is a true and faithful record of witness testimony given during the National Citizens Inquiry (NCI) hearings. The transcript was prepared by members of a team of volunteers using an "intelligent verbatim" transcription method, and further translated from the original French.

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