ACCESS VIA NERVE SYSTEM BY SARS-COV-2 SPIKES

Antonino Messina*

Former Assistant University of Anatomo-Pathological,

School of Lausanne and Geneva, Switzerland

ABSTRACT

Purpose: The aim of this study is to propose the nerve pathway and its mechanism for penetration of Coronavirus.
Introduction: The virus RNA polarity and the nanoparticle spike polarity of the Coronavirus's surface may interact, through their electro-chemical properties, with the Golgi apparatus and the terminal endings of the body's peripheral nerve axons. Many patients who have recovered from Covid-19 suffer from loss of taste, numbness of the tongue and alteration of smell, sublingual microangioma, contracture of the masseter muscle, decreased mouth opening, polyarthragia, mild soreness of the entire body and cognitive problems. C*ontagion could starts from buccal mucosa and nerve endings of olfactory* **(First site of penetration).**
Materials: The peripheral stimulation of nerve endings promotes electrical migration along the parasympathetic cells on the Vagus nerve and on the sympathetic system, interesting the entire nervous pulmonary trunk. This mechanism also involves the diaphragm, rib muscles and the innervation of the interstitial cellular neurofibrillar tangles of the alveolar surface of the lung with consequent inflammatory response (as allergic-hyperallergic reaction), cell adhesions, microvascular thrombosis, microvascular haemorragic lesions and finally blockage of oxygen exchange **(Second site of penetration).**
Our research has mathematically evaluated the numerical intensity of the environmental accumulation of the Coronavirus, corresponding to thousands of million billions of Covid-19 virus particles having, like CNT particles, electrical polarity and countless possibilities for mutation.
Conclusion: Owing to the strong electrical activity of CNT particles, we suppose that it could be possible to neutralize the particle spikes in laboratories (white chamber) to render the coronavirus harmless.

Keywords: Nanoparticles; Covid-19; CNTs; Neuro-mechanism; Contagion; Spike mechanism

***Correspondence to**: Antonino Messina, Former Assistant University of Anatomo-Pathological, School of Lausanne and Geneva, Switzerland, E-mail: messinaaf@gmail.com

Received: Jan 05, 2021; **Accepted:** Jan 30, 2021; **Published:** Feb 05, 2021

Citation: Antonino Messina (2021) Access via Nerve System by Sars-Cov-2 Spikes. J. Nanomed. Nanotech. Nanomat. 2(1): 105.

INTRODUCTION

Today we know that Covid-19, with its aggressive clinical manifestations, is not only located in the lungs and that the complete mechanism for infecting the alveolar tissue of the lungs is not yet fully known [1].

Also the ways in which the electrical polarity of the Coronavirus spikes may affect neuronal electrical activity in the interstitial pulmonary nervous system and its cellular function are not still studied.

The electro-chemical conductance of RNA coronavirus provided by electrical polarity [2,3] demonstrates that the potential health risk (inflammatory response) of its particles may be like that of CNT particles, through inhalation, skin, mucosal walls, eyes, gastrointestinal absorption etc. [4,5]. It is almost certain that infection may occur through penetration of mucosal contact by Golgi apparatus, buccal and oral mucosa by Frank nervous action and the Alderman nerve of the outer ear **(First sites of the neural penetration mechanism for Covid-19).**

From clinical observations we know that Covid-19 can be responsible for persistent and worsening pneumonia, as well as for a temporary loss of the olfactory and taste senses (terminal peripheral nervous endings and Golgi mucosal contact points). Many patients, after their recovery, suffer from numbness of the tongue, alteration of smell, sublingual microangioma, contracture of the masseter muscle, decreased mouth opening, polyarthragia, mild soreness of the entire body or cognitive problems (Figure 1) [6].

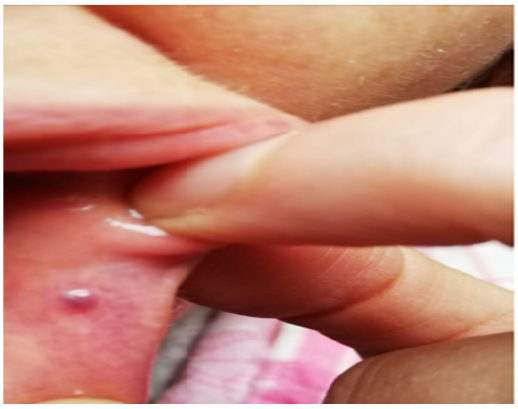

Figure 1: Post-Covid sublingual microangioma; a new clinical finding in a 57- year-old woman ; many patients who have recovered from Covid-19 suffer from loss of taste, numbness of the tongue and alteration of smell. *It is obvious that contagion could starts from the mucosa via Golgi apparatus and the endings of the olfactory and taste nerves.* These nerve endings are stimulated by the electric polarity of the nanoparticle spikes, which promote electrical migration along the parasympathetic nerve cells on the Vagus nerve and the sympathetic system of the entire pulmonary trunk. This mechanism also involves the diaphragm, rib muscles and the innervation of many organic molecules of the nerve interstitial alveolar surface of the lung with consequent inflammatory response (as allergic-hyperallergic reaction), cell adhesions, microvascular thrombosis, microvascular haemorragic lesions or similar microvascular pathological alterations like to this microangioma of the buccal mucosa

On the basis of the clinical evidence, we think that the action from nerve endings stimulation could promote cellular migration on axonal cells up to the cervical ganglia of the parasympathetic (Vagus nerve) and sympathetic trunk (Figure 2).

Through the Vagus neurons and the sympathetic system, by means of its electro-chemical interaction with different organic molecules, the electrical virus stimulation may produce an additional inflammatory reaction in the nerve fibers (like the Zoster virus).

Consequently, we believe that the electrical polarity of the Coronavirus RNA and its polarized spike particles [2,3] may be able, like CNT particles, to produce, at the level of the interstitial neurofibrillary tangles of the alveolar wall of the lung, an inflammatory response (as allergic- hyperallergic reaction), cell adhesions, microvascular thrombosis, microvascular haemorragic lesions [6,7].

It is even possible that patient deaths could be due to partial pulmonary palsy by depressive pulmonary parasympathetic action owing to the effect of the strong electrical properties of the virus particles on lung innervation.

This hypothesis might be studied to gain better knowledge of the internal electrical mechanism of the particle spikes of the virus which could act in addition to the effect of the curare administered for orotracheal intubation in intensive care units.

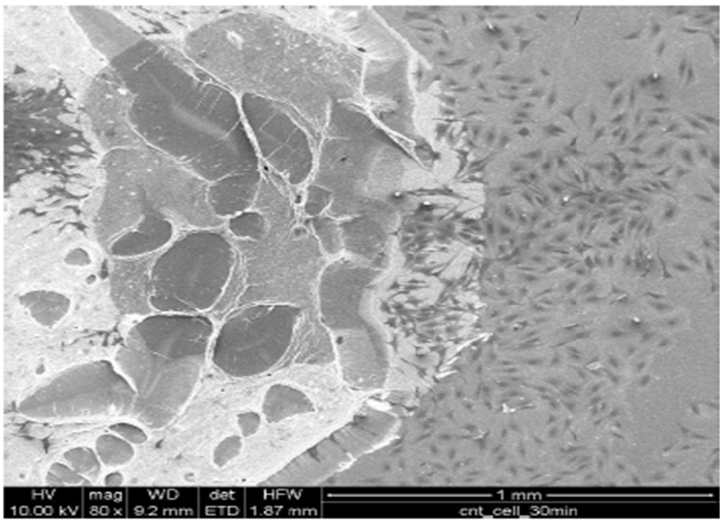

Figure 2: SEM exam for basic research on nerve regeneration in which CNTs with negative polarization attract nerve cells and stimulate cell regeneration.*Thousands of million billions of* Covid-19 *virus particles*, like CNT particles, may be able to attack and promote cellular and molecular interaction in sensitive peripheral nerves of the body through the Golgi apparatus and the nerve endings of the buccal mucosa, *the olfactory and Frank taste nerve and Alderman nerve of the outer ear*. Such axonal stimulation due to the electric polarization of Coronavirus RNA and of its polarized particle spikes could promote cellular migration on nerve cells up to the cervical ganglia of the parasympathetic (Vagus nerve) and sympathetic trunk. Through the Vagus neurons and the sympathetic system, by means of own electro-chemical interaction with different organic molecules, the virus could produce (like the Zoster virus) inflammation of neurofibrillary tangles of the interstitial alveolar wall of lungs: this mechanism also involves the innervation of diaphragm and rib muscles of the breathing system

Methods and Materials

To gain insight into the contact surface structure of the alveolar wall and evaluate the total anatomic extension of the interstitial pulmonary surface of the body, together with the quantity of Coronavirus particles acting on the alveolar site of the lung, we have applied a mathematical estimation of the nanoparticles of the virus in Covid-19 and determined how they could act [8].

It is known that the virus has an RNA genome of 26,000 to 32,000 letters in length and an electric polarity at its end; it has a spherical shape and it is surrounded by a fatty envelope and, as observed under the electron microscope, and its surface is covered by a crown of spikes (in reality a complete envelope full of nanoparticle protein spikes [2,9].

Also the club-shaped spikes have an electric polarity at their end and are made up of nanoparticle proteins which are utilized as harpoons to penetrate the interstitial wall of the alveoli of the lung. They have two action tools: the binding domain (hexapeptite), a kind of grappling hook that grips onto host cells, and the proteolytic cleavage site (proteinases S1, S2, S2'), a molecular opener that allows the nanoparticles of the virus to crack open and enter host cells [4,9,10].

In order to scientifically evaluate how the match occurs between the nanoparticles of the coronavirus spikes and the interstitial alveolar tissues of the lungs we have done a mathematical evaluation of this material and the physical situation.

- First, alveolar contagion occurs in relation to the number of breaths a person takes in one minute, 20; in an hour 1, 200; in a day 28,800.
- The total extension of the alveolar interstitial surface of the lung is estimated to be of the order of 100 m^2, corresponding to $10^6 cm^2$ (approximately equal to 100mq).
- The virus itself, whose appearance is fairly close to a sphere, has a diameter close to 100 nm, (A nanometer representing the billionth part of a meter). From this, we can compute the surface covered by a single virus on a bi-dimensional projection of the interstitial alveolar tissues of lungs:

$$S_v = \pi R^2 = \pi \cdot (55 * 10^{-9})^2 \approx 9.50 \times 10^{-15} m^2 = 9.50 \times 10^{-11} cm^2$$

- In simple words, this means that to cover even just a third of the 10^6 Cm^2 of the interstitial surface, approximately particles would be needed: hundreds of millions of billions of particles.
- In simple words, this means that, to cover even just a third of the 106 cm2 of the interstitial surface, approximately 1017particles would be needed: hundreds of millions of billions of particles
- In terms of volume, similarly, where we estimate the lungs' volume to be of the order of 6000 cm^3, we see that the volume of a virus nanoparticle is of the order of:

$$V_v = \frac{4}{3}\pi R^3 = \frac{4}{3}\pi \cdot (55 * 10^{-9})^3 \approx 6.969 \times 10^{-22} m^3 = 6.969 \times 10^{-16} cm^3$$

- Purely in words, once again, this means that, to cover even just a third (of the 6×10^3 cm^3) of the volume of the lungs (approximately 10^{19} particles) would require tens of thousands of millions of billions of particles!

  These mathematical and physical data are the real measurement of the mechanism of lung infection and are crucial for environmental transmission.

- The data on the very large alveolar surface of the human lung correspond to 1,000,000 cm^2 and to cover almost half of its surface would require some thousands of millions billion of virus particles.
- Considering the amount of space contained within the human lung of about 6000 mL, corresponding to 6000 cm^3 we understand the large possibility offered by the human body to invasion by nanoparticles of SARS-Cov-2!

In addition, we cannot exclude that the millions of toxic nanoparticles (club-shaped protein spikes having an electric polarity) may act like CNT particles, able to attack and promote cellular and molecular interactions. (Figure 2).We even believe that the electrical activity of the virus's spike nanoparticles could interface through their electro-chemical properties with different organic molecules and consequently be responsible for the interstitial inflammatory reaction at the

neurofibrillary tangles in the alveolar wall of lung.

*The action promoting cellular migration to nerve cells and interacting with the interstitial nerve plexus of the alveolar structure of the lung could be responsible for producing nanoparticle spike adhesions and persistent interstitial pneumonia (**Second site of the nerve pathway penetration mechanism**)* [8-10].

The ways in which Covid-19 affects neuronal electrical activity on the pulmonary nerve system and its cellular function have not yet been studied.

Environmental pollution and the intense accumulation of the Coronavirus allows for the first penetration of the virus nanoparticles which might start from the Golgi apparatus, the olfactory and Alderman nerve of the outer ear, or the taste nerve (Frank action). This nerve penetration leads to an interstitial pneumonia as well as to a partial and temporary loss of the olfactory and taste senses and a multiple and multiform lesions as sublingual angioma, contracture of the masseter muscle, decreased mouth opening, polyarthragia, mild soreness of the entire body or cognitive problems, now clinically and definitively ascertained (Figure 1) [2,6,8].

This action, in our opinion, by promoting cellular migration through electro-chemical interactions with different organic molecules of nerve cells, reaches along their axons the cervical ganglia of the parasympathetic and sympathetic trunk (First nerve pathway of the penetration mechanism). Through the vagus neurons of the parasympathetic system, and together with the sympathetic system, the virus could also produce nerve fiber inflammation and degeneration (like the local dermal and nerve myelin necrosis produced by the Zoster virus) resulting in additional pulmonary nerve plexus lesions up to a partial or complete palsy.

DISCUSSION

The mechanism by which the coronavirus penetrates the alveolar tissue is not yet fully understood. On the basis of recent clinical observations, we know that many patients, after their recovery from Covid-19, suffer from numbness of the tongue, alteration of smell, sublingual microangioma, contracture of the masseter muscle, decreased mouth opening, polyarthragia, mild soreness of the entire body, weakness and general fatigue and some cognitive problems.

Therefore, we must seriously consider that involvement of the parasympathetic (Vagus nerve) and sympathetic nerve system represents the coronavirus's first and second means of contagion and penetration.

But the ways in which Covid-19 affects neuronal electrical activity in the parasympathetic (Vagus nerve) and sympathetic nervous system and particularly the pulmonary nerve trunk and its cellular neurofibrillary tangles function in the alveolar wall, have not yet been studied.

In our opinion following penetration of the Golgi apparatus and other peripheral nerve endings located in the olfactory and taste mucosa, the electrical polarity of Coronavirus particle spikes can promote electrical migration on parasympathetic nerve cells along the vagus nerve and the sympathetic system of the entire pulmonary trunk, ultimately determining massive interstitial pneumonia and death.

According to this hypothesis many patient deaths from Covid-19 may be due in part to the strong electrical properties of virus spike particles, with their electrical polarity acting on the interstitial structure of the lung's alveolar nerve system, resulting in local inflammation from the local allergic- hyperallergic reaction and finally leading to an interstitial degenerative fibrosis of the pulmonary alveolar wall [2,5,7,10].

In order to defeat the pandemic, we have obtained a lot of antibodies by changing the RNA chain of the Coronavirus to make new vaccines. However, the DNA can change its structure frequently and it is possible that now we have obtained an instable or useless vaccine.

The aggressive nature of the Coronavirus and particularly the electric polarity of its nanoparticle spikes can be only rendered harmless or neutralized by working on the particle spikes in laboratories (white chamber) in order to obtain a definitive immunological state of human body.

We are convinced that SARS-Cov-2 is a conveyer and the spike particles are tools for attacking nerve extremities like Golgi apparatus and many mucosal and dermal nerve endings or the interstitial neurofibrillary

In our opinion the Coronavirus is like a battleship, that is to say a conveyer provided with a polarized RNA and countless polarized spikes, the cannons.

The new vaccines try to change the polarized RNA structure, that is, to immobilize the motor of the battleship by stopping its navigation and its diffusion of the contagion, producing at the same time a lot of antibodies without obtaining from the Coronavirus a stable immunization due to possible continuous changing of its DNA.

Another solution, more stable and reassuring to stop the conveyer-ship aggressivity, may be obtained by the destruction of the electric polarity of the cannon-particle spikes. This action could render the conveyer harmless, a real innocuous Coronavirus.

Our hypothesis is then to try to use CNT particles (which have negative polarity) in white laboratories to clean up or neutralize the polarity of the virus spikes.

This would render the coronavirus harmless so that the cannon spike proteins cannot attack the interstitial neurofibrillary tangles of the lung's alveolar walls (the virus might produce only an innocuous cold) (Figure 3).

Figure 3: Green plant prototype similar to Coronavirus image. Our hypothesis is to try to use CNT particles (which have negative polarity) in white laboratories to clean up or neutralize every virus spikes (S1,S2,S2') polarity. *This would render the coronavirus really harmless so that the spike proteins cannot attack the interstitial*

We hope additionally that the pulmonary anatomo-pathological data, together with the mathematical evaluations of coronavirus nanoparticles, can improve

tangles in the alveolar wall of lung alveolar complex contained into the membrane wall for exchange of oxygen with carbon dioxide, together with arteries, veins and pulmonary lymphatics.

neurofibrillary tangles of the lung's alveolar walls (the virus might produce only an innocuous cold).

Lastly, given the strong electrical properties of the virus particles acting on the interstitial structure of the lung's alveolar nerve (Vagus nerve system) [6], we must consider that the need to administer many different drugs and use curare to intubate patients to blow oxygen into the lungs may worsen the paresis or palsy of the Vagus pulmonary nerve system and facilitate patient deaths. In our opinion this could explain the great number of deaths not scientifically certified by research on extensive international clinical care protocols and ascertained by systematic autoptic research on the cause of death.

CONCLUSION

The latest mutation of the SARS-Cov-2 virus can render the current vaccine ineffective because derived and produced by the alteration of the viral RNA structure. Instead and alternatively, new insights into the internal mechanism, evidenced by the possibility of electrical molecular interaction of CNTs with different molecular particles of Covid-19 spike proteins, might stimulate further studies in laboratories and lead to a very stable coronavirus vaccine.

The nervous pathway of contagion and body penetration might also be the first way to have introduced the virus into the human body many months before the explosion of the Covid-19 pandemic. In addition, to add value to the scientific fight against the Coronavirus, we want point out the necessity for the World Health Organization to establish a unique and meticulous treatment protocol for all Nations to apply to all local environmental pandemic situations.

the possibility of studying the virus's activity on the body.

REFERENCES

1. Van Doremalen N, Bushmaker T, Morris DH (2020) Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. N Engl J Med.
2. Auewarakul P (2005) Composition bias and genome polarity of RNA viruses. Virus Res. 109 (1): 33-37.
3. Cruz PM, Santos E, Velazquez Cervantes MA, Leon Jarez M (2020) Covid-19 una emergencia de salud publica mondial. Rev. Clin. Esp.
4. Tellier R, Li Y, Cowling B, Tang J (2019) Recognition of aerosol transmission of infectious agents;a commentary. BMC Infectious Disease. 19: 101.
5. Snyder-Talkington BN, QianY, Castranova V, Guo NL (2012) New perspectives for in vitro risk assement of multiwallet carbon nanotubes:Application of coculture and bioinformatics. J Toxicol Environ Health B Crit Rev. 15: 468-492.
6. Conti G, Bariatti R (1953) Zeitschrift fur Zellforschung und Mikroskopische Anatomie. 38: 148- 177.
7. Messina A, Mangione A, Basirico L, Messina JC (2015) Basic Research for Peripheral Neural Regeneration by CNTs. A Preliminary "*in vitro*" Study. J nanomed Nanotechnol . 6(5): 1-4.
8. Messina A (2020) New hypothesis of cleaning of coronavirus nanoparticle spikes. J Nanomed Nanotech Nanomat. 1 (1): 1-5.
9. Usui K, Ichihashi N, Kazuta Y, Yomo T (2014) Effects of ribosomes on the kinetics of Qb replication; FEBS Letters. 588(1): 117-123.
10. Grubaugh ND, Ladner JT, Lemey P, Pybus OG, Rambaut A, et al. Tracking virus outbreaks in the Twenty-first Century. Nat Microbiol. 4(1):10-19.