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Journal of Antivirals & Antiretrovirals

Mini Review

Antivirus Therapy: Using Annihilating Inside Liposomes as a Bait for SARS-CoV2

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ABSTRACT

This article refers to the treatment devised by the author in order to treat the SARS-CoV-2 pandemic. There is talk of the properties of the virus, its genome and the proteins it encodes, as well as various articles, in order to highlight possible treatment aspects to be excluded, namely a treatment that targets endogenous adherents rather than the virus itself, due to high toxicity, as well as an RNAi treatment. Finally, the very treatment with references to the technique of liposomes as a means of transporting drugs and the technique of annihilating genes as a technique of oligonucleotides is introduced.

Keywords: SARS-CoV2; Antivirus; Liposomes

INTRODUCTION

ISARS-CoV-2 virus is an RNA virus with genetic material monoclonal RNA, which infects humans and is presumed to also infect bats, through the Angiotensin Converting Enzyme 2 (ACE 2) receptor and with the help of the diamembranal ion protein S (spike protein), consisting of 2 subunits, S1 and S2, using the human protease of the TMPRSS2 host. The genetic material of the virus consists of 20 genes and it was discovered that much of its genome, both in encoders and in mamcodeen regions, undergoes mutations, making this virus highly unpredictable. The symptoms caused by the virus come mainly from the respiratory system, mainly early-stage coughing and subsequent pneumonia. Diarrhoea has even been recorded. It is important to report that there is heterogeneity among sufferers, with the largest patient populations belonging to the elderly or people with underlying diseases. People of younger age or people without an underlying disease who do not belong to the previous population groups exhibit severe symptoms, at rates of 2%-3% of the total patient population of this age group.

METHODOLOGY

Several articles have been researched to provide information useful for the pathology of SARS-CoV-2, with the ultimate goal of devising an effective treatment for the virus, which will provide the least toxicity possible [1-5].

RESULTS

SBefore any prospect of fighting the virus is analyzed, its pathogenicity, genome and structure, especially its pathogenicity elements, must be studied. As mentioned, the virus genome regularly undergoes mutations as one of the articles studied showed that a total of 86 different samples of the virus identified 93 different mutations, which negate the prospect of a wave of virus outbreaks and sound scars for possible future currents of cases from mutated strains of the same virus, despite finding a vaccine, in influenza virus standards. Moreover, the importance of the oligonucleotide technique proposed by the author has been applied experimentally through the technique of intervening RNA (RNAi) against influenza A virus, which creates the potential for application of treatment in a different naturally scheme. Research has also shown that serine TMPRSS2 protease is just as important as protein S for virus infectivity, which identifies this protease as a potential target, but with greater toxicity. On the other hand, the use of RNAi techniques has demonstrated that it has much less toxicity to the host than a treatment that inhibits endogenous adhemes and therefore the treatment recommended is to modify the virus, with the aim of inactivation, by inhibiting substrates of the virus, against host substrates, as is the case with antiviral drugs. Another article studied revealed that the virus's Protein N has the ability to inhibit RNAi molecules and therefore such an operation against the virus would be impossible. For this reason treatment should focus on other types of RNA oligonucleotides. Conventional treatments with existing drugs are limited to the use of the combination of hydroxychloroquine and azithromycin, but no positive results are obtained in all clinical studies. In addition, it has emerged that hydroxychloroquine is more active than

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Received: October 09, 2020; **Accepted:** October 23, 2020; **Published:** October 30, 2020

Citation: Labrou D (2020) Antivirus Therapy: Using Annihilating Inside Liposomes as a Bait for SARS-CoV2. J Antivir Antiretrovir. 12:001.

chloroquine in virus inhibition in in vitro experiments, so an even more drastic modification of the lateral groups of chloroquine may be an even more effective treatment. After all, there are rumours about the discovery of the potency of chloroquine phosphate in China, as various sources reveal. The problem with this virus is that it is immunomodulatory and thus able to escape detection and in addition the inflammatory reaction it causes is so extensive that it needs immediate treatment. Clearly we could use drugs that have immunomodulatory properties, such as various cytokine synthesis inhibitors, as well as enzyme inhibitors that are active for the virus to enter the host cell. However, inhibition of endogenous agents certainly results in side effects, and if their presence in the body is intense, then toxicity is important in many systems. For instance, using ACE2 blockers, such as captopril, will have the following side effects: severe dry cough, hypotension, headache, angioedema, dizziness. Up to a certain point the use of those drugs is useful. However, as the disease progresses, blood pressure will not be easily regulated, so side effects would be more severe and the drug would be useless. Since inhibition of endogenous agents is quite unfavourable, what if we modified the virus itself? And clearly one could claim that this is done through vaccines. Yes, but there is always the risk of reactivation of the strain and in addition the vaccine is a preventive means. I refer to in vivo therapy on the virus itself. This technique is a technique of modification, commonly, innovative. I call this treatment antivirus therapy. My goal is to inactivate the virus so that it is not further infectious. This can be achieved by genetic modification of the virus. To this end, annihilating genes will be prepared, i.e., RNA oligonucleotides, whose incorporation into the genetic material of the virus will lead to inactivation of the genes of the virus that produce proteins that have the ability to give pathogenicity in the virus, in this case the genes that make up the virus S protein subunits. These oligonucleotides cannot pass through the virus membrane on their own, so we should direct the virus to them. This can be achieved by the induction of oligonucleotides into liposomes. A double liposome will be used, inside which oligonucleotides will be placed. The liposome will be 500-1000 nm in dimensions. The inner membrane of the liposome will carry the receptor converting enzyme of angiotensin 2 acting as bait for the virus, while the outer double layer will not bring macromolecules to its surface. The use of the outer membrane is done because in administration per os the receptor as a protein will degrade, so we need a protective agent. Liposomes will be absorbed into the gastrointestinal tract and pass a 1st-pass metabolism. In general liposomes have high rates of bioavailability, so this route of administration is possible. Once the drug enters systemic circulation we will have degradation of the outer membrane and thus reveal the target receptor for the virus, which will be attached to it and the liposome will act essentially as bait, preventing binding in cells. Once the binding is done, the virus will be intractable and enter the inside of the liposome, where we will have fusion of oligonucleotides and the genetic material of the virus and thus inhibit synthesis of the Protein S. is now dormant. This treatment will not eliminate the rates of the virus in the blood, except to maintain its rates at manageable levels in order for the immune system to act. It is not yet clear whether the treatment will also lead to a suppression of the generalized inflammatory reaction, which is under investigation.

DISCUSSION AND CONCLUSION

It is generally concluded that SARS-CoV-2, due to high mutagenicity, has the ability to be resistant to certain treatments and perhaps in the future produces new strains starting a new epidemic wave. However, this treatment will enable the disease to be treated, as will its alternative assumptions in a short period of time, without the risk of toxicity or death. The contribution will also be made to the treatment of other diseases, which have a viral cause, because with appropriate modifications the use of this treatment can be extended to other viruses. It is a matter of knowing the mechanisms of infectivity of the virus and of course its goon idiom.

REFERENCES

1. Phan T. Genetic diversity and evolution of SARS-CoV-2. Infect Genet Evol. 2020; 81:104260.
2. Hoffman M, Kleine-Weber H, Schroeder S, Kruger N, Herrler T, Erichsen S, et al. SARS-CoV-2 cell entry depends on ACE2 and TMPRSS2 and is blocked by a clinically proven protease inhibitor. Cell. 2020; 181(2):271-280.
3. Zhang H, Tang X, Zhu C, Song Y, Yin J, Ertl HCJ, et al. Adenovirus-mediated artificial MicroRNAs targeting matrix or nucleoprotein genes protect mice against lethal influenza virus challenge. Gene Ther. 2015; 22(8):653-662.
4. Kannan P, Ali SS, Sheeza A, Hemalatha K. COVID-19 (Novel Coronavirus 2019)-Recent trends. Eur Rev Med Pharmacol Sci. 2020; 24(4):2006-2011.
5. Xueting Y, Fei Y, Miao Z, Cheng C, Huang B, Niu P, et al. In vitro antiviral activity and projection of Optimized dosing design of hydroxychloroquine for the treatment of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Clin Infect Dis. 2020; 9:237.