

DOI: 10.21767/2573-0282.100035

Inhibition of Viruses by Metadichol®: A Novel Nano Emulsion Lipid

Raghavan PR*

Nanorx Inc, Chappaqua, New York, USA

*Corresponding author: Raghavan PR, Founder and CEO, Nanorx Inc, Chappaqua, New York, USA, Tel: 9146710224; E-mail: raghavan@nanorxinc.com

Received date: December 14, 2016; Accepted date: January 13, 2017; Published date: January 18, 2017

Citation: Raghavan PR (2017) Inhibition of Viruses by Metadichol®: a Novel Nano Emulsion Lipid. *Pediatric Infect Dis* 2:35. doi: 10.21767/2573-0282.100035

Copyright: © 2017 Raghavan PR. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

The inevitable, but unpredictable, appearance of new infectious diseases has been recognized for centuries well before the discovery of causative infectious agents. Today, however, despite advances in development of therapeutics, and vaccines the ease of world travel and increased global interdependence have added layers of complexity to containing these infectious diseases that affect not only the health but the economic stability of societies. Viruses and bacteria and other pathogens impose enormous pressures on their human hosts, and combatting these pathogens is fundamental to the propagation of a species. Innate immunity provides the foundation for pathogen resistance.

Keywords: Zika; Ebola; Dengue; Chikungunya; H1N1; Respiratory viruses, Metadichol; Inverse agonist; Protean agonist; Malaria

Introduction

The world is being ravaged by viruses at regular intervals and there is no long term solution insight. Our approach for a long term solution was focused on prevention by enhancing innate immunity towards that goal we developed Metadichol® [1]. Its constituents are food ingredients that are free of toxic, mutagenic, or teratogenic properties [2,3]. We recently showed that [4,5] Metadichol exhibits potent, broad spectrum viral inhibitory activity in Vero and MDCK cells infected with Dengue, Ebola, Marburg, Influenza A (H1N1), Chikungunya and Human Respiratory Syncytial viruses. In addition, we tested the efficacy of Metadichol® in preventing cell death caused by Adenovirus, Tacaribe Mammarenavirus, Rift Valley Fever virus, SARS coronavirus, Japanese Encephalitis virus, West Nile virus, and Yellow Fever virus (Figure 1). Also presented a case study of two patients diagnosed with Dengue Fever who were successfully treated with Metadichol® (see attached supplement) which confirmed the inhibition of dengue virus that was demonstrated in-vitro.

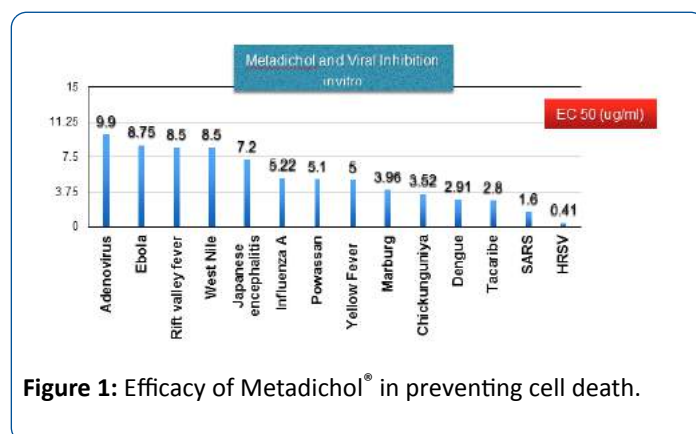


Figure 1: Efficacy of Metadichol® in preventing cell death.

Commercially available antiviral therapeutic compounds block replication processes shared by the virus and infected target cells [6]. Such compounds are potentially toxic, mutagenic, and/or teratogenic for the host and can induce drug-resistant viral mutant sub-strains. Metadichol fills a need as an efficacious new antiviral compounds that is free of such deleterious effects. In addition, it is a broad spectrum as it also inhibits parasites and also bacteria like MRSA.

Discussion

Lipids play key role as immune response modulators. Many of the receptors that activate the innate immune system have evolved to recognize and react to the hydrophobic portions of molecules should these become suddenly exposed [7]. These properties have been applied in the treatment of autoimmune and inflammatory diseases [8,9]. Metadichol is a Nano emulsion of long-chain lipid alcohols (C-26, C-28 and C-30), which are commonly known as Policosanol's. Metadichol has a particle size of less than 60 nm. It binds to the vitamin D receptor (VDR) as an inverse agonist. It is the only known inverse agonist of VDR in medical literature. Calcitriol (1,25-Dihydroxy Vitamin D) is the natural ligand for the VDR and acts as an agonist. Metadichol acts a inverse agonist but more likely is a Protean agonist. Protean agonists act as both positive and negative agonists on the same receptor, depending on the degree of constitutive activity that is present. If there is no constitutive activity, the agonist would be a positive agonist. When

constitutive activity is present, the protean agonist would be an inverse agonist [10].

Viruses have evolved strategies to knock out the activation of the VDR and stop it from producing antimicrobials that would otherwise kill the intracellular microbes. For e.g., Persistent Epstein-Barr virus infection down regulates VDR >10 fold [11]. By binding to the VDR the innate immune system is compromised. Then the cytokine release causes the adaptive immune system to start working extra hard and try and deal with this problem that the innate immunity was handling. Metadichol by binding to the VDR reactivates immune function competitively disrupts this process. In addition to VDR binding, Metadichol shares cross-reactivity with other nuclear receptors [12,13], which may explain its activity against a wide range of viruses.

Conclusion

Metadichol is ready for large scale Clinical testing in areas which are ravaged by viruses. Once proven on large populations, Metadichol could be used as a preventive nutritional supplement in countries where viral fevers are widely prevalent. Metadichol is being sold as a nutritional supplement in a few Asian countries for the last two years and is extremely well tolerated. So far no reports of any adverse side effects. Metadichol is made from renewable sources and could serve as safe and cost effective solution to mitigating viral diseases that threatens humanity.

References

1. https://www.researchgate.net/publication/310297987_In_vitro_Inhibition_of_Zika_Virus_by_MetadicholR_A_Novel_Nano_Emulsion_Lipid
2. Alemán CL, Mas R, Hernandez (1994) A 12-month study of policosanol oral toxicity in sprague dawley rats. *Toxicol Lett* 70: 77-87.
3. Alemán, CL, Mas Ferreiro (1994) Carcinogenicity of policosanol in sprague-dawley rats: A 24-month study. *Teratog Carcinog Mutagen* 14: 239-249.
4. Aleman CL, Puig MN, Elías EC (1995) Carcinogenicity of policosanol in mice: An 18-month study. *Food Chem Toxicol* 33: 573-578.
5. Raghavan PR (2016) Inhibition of Dengue and other enveloped viruses by metadichol®, a novel Nano emulsion Lipid, *Journal of the Science of Healing Outcomes* Vol 8: 19-25
6. Keynote presentation at the Influenza congress (2016) Berlin Germany pp. 13.
7. Shugar D (1972) *Virus-cell interactions and viral antimetabolites*, Ed. Shugar, D. (Academic, New York), Vol. 22, p. 193-207.
8. Prusoff WH (1972) in *Virus-cell interactions and viral antimetabolites*, Shugar D Edn., (Academic, New York), Vol. 22, p. 135-148.
9. Seong SY, Matzinger P (2004) Hydrophobicity: an ancient damage-associated molecular pattern that initiates innate immune responses. *Nat Rev Immunol* 4: 469.
10. Sands J, Auperin D, Snipes W (1979) Extreme sensitivity of enveloped viruses, including herpes simplex, to long-chain unsaturated monoglycerides and alcohols. *Antimicrob Agents Chemother* 15: 67-73.
11. Snipes W, Person S, Keller G, Taylor W, Keith A (1997) Inactivation of lipid-containing viruses by long-chain alcohols. *Antimicrob Agents Chemother* 11: 98-104.
12. Neubig RR (2007) Missing Links: Mechanisms of Protean Agonism. *Mol Pharmacol* 71: 1200-1202.
13. Yenamandra SP, Lundin A, Arulampalam V (2009) Expression profile of nuclear receptors upon Epstein-Barr virus-induced B-cell transformation. *Exp Oncol* 31: 92-96.