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Mini Review

Covid -19 Glycoprotein can be Modified by Adding Short Oligosaccharide or o-Polysaccharide Derived from *E. coli* Making Weakness or Lose of ACE-2 Attachment

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Abstract

SARS-CoV-2 is a highly contagious and mutant pathogen. The pathogenicity depends on glycosylated spike which attack different types of cells and tissues containing ACE2 receptors. Here blocking or modified SARS-COV-2 glycoprotein by oligosaccharide derived from colon bacteria (Microbiota) or synthetic oligosaccharide (13 monosaccharide units) may of value in changing or modifier the configuration structure of spike protein. Finally may viruses lose the first step to induce pathogenesis.

Keywords: Oligosaccharide, Manno-sidases, Demon-strates

Introduction

(SARS-CoV-2), the causative pathogen of COVID-19 [1,2], induces fever, severe respiratory illness and pneumonia. SARS-CoV-2 develops a widely glycosylated spike (S) protein that bulges from the viral surface to bind to angiotensin-converting enzyme 2 (ACE2) to mediate host-cell entry [3]. The S protein is tri-meric class I fusion protein, composed of two functional sub-units, responsible for receptor binding (S1 subunit) and membrane fusion (S2 subunit) [1,2]. Remarkably, the surface of the envelope spike is dominated by host-derived glycans with each trimer displaying 66 N-linked glycosylation sites.

There are two sites on SARS-CoV-2 S that are principally oligomannose-type: N234 and N709. The predominant oligo-mannose-type glycan structure observed across the protein, with the exception of N234, is Man5GlcNAc2, which demon-strates that these sites are largely accessible to α1,2 -manno-sidases but are poor substrates for GlcNAcT-I, which is the gateway enzyme in the formation of hybrid- and complex-type glycans in the Golgi apparatus. The stage at which processing is impeded is a signature related to the density and presentation of glycans on the viral spike. For example, the more densely glycosylated spikes of HIV-1 Env and Lassa virus GPC exhibit numerous sites dominated by Man9GlcNAc2 [3-4].

Commensal bacteria and their products can indirectly protect against Influenza A virus (IAV) infection by interacting with the host's immune system in the absence of commensal bacteria, mice suffered from impaired type I/II interferon responses, CD4/CD8 T cell responses, and antibody production to IAV infection [6,7]. Moreover, mice pretreated with bacterial lipopolysaccharide (LPS), a product present on the exterior surface and shed by all Gram-negative bacteria, triggered a Toll-like receptor 4 (TLR4)-mediated antiviral response to protect the hosts from lethal infection with IAV [8,9]. In contrast, LPS was found to bind directly to the capsid protein of poliovirus, increasing cell attachment and the ability of the virions to remain infectious at elevated temperatures [10].

Additionally, LPS binding to mouse mammary tumor virus (MMTV) resulted in increased immune evasion and transmission of the virus [5]. In the case of influenza, it is unclear whether commensal bacteria and LPS are interacting directly with IAV in addition to their indirect effects on the immune system.

Conclusion

In these study we make fragments either in laboratory or taken from LPS o- side chain containing 13 oligosaccharides units then firstly, performing *in vitro* study to detect if these oligosaccharide inhibits virus entry on specific TC cells

The second trial applied on monkey as *in vivo* study to detect that if block virus entry and study the toxicity if present.

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