

Celivir: A Viral Therapy for the Autoimmune Celiac Disease

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Abstract With an average of 1/200 affected people around the world, celiac disease still lacks a cure. Here a concept of a viral therapy with certain design degrees of freedom is suggested to the scientific community with the hope that a well experienced and equipped research team is able to endeavour such project. Also, safety concerns of the viral therapy about escape mutations and tropism are covered. These ideas can be extended to any autoimmune disease with an etiological alteration of the memory T-cells, paving the way to safer alternatives to the immunological reboot.

Keywords: celiac, autoimmunity, viral therapy, escape mutation

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1. Introduction

The goal of this short paper of opinion is to share a strategy with the scientific community for a cure for the celiac disease in particular, and for autoimmune diseases grounded on modified memory T-cells in general. The suggestion conceives a new potential line therapy, with the hope that a research team with the necessary resources is able to tackle the challenge.

The idea is to use a modified virus with a RBD (receptor binding domain) spike epitope similar to gluten targets of implicated memory T-cells. We shall call this special spike design from now on “glutespike”. The objective is to selectively infect and destroy the altered memory T-cells related with the autoimmunity, and guarantee the safe conditions of avoiding the risk of tropism as well as accidental mutations.

For this purpose, it is necessary to identify the preferred epitopes of the gluten antigen at which altered T-cells specifically dock, and then to reproduce this epitope in the RBD of the glutespike. A combined strategy, similar to sarscov2 (from now on easier than SARS-CoV-2 typing), is to use the extra glutespikes to attach specifically to the membrane of these memory T-cells [1] [2], so that they tend to attack each other the altered T-cells as if they were infected. This assures that specific autoimmune memory T-cells responsible of the celiac disease will be completely erased. In order to avoid a cytokine storm it is key to accomplish that the glutespike RBD does not attach to other important cells such as EC, SMC, platelets and healthy immunological cells. Also, the number of the glutespikes per virus should be relatively slow to avoid the storm and the corresponding side effects, and therefore is a degree of freedom of the problem to be optimized. These

techniques must be tested in vitro and, if successful, in animal models.

In order to avoid at the same time the risk of tropism and accidental mutations, the RBD must have a synchronized mechanism of two structures that must co-work for the docking, so that the virus would be incapable of adopting positive mutations since the probability of mutation would be as low as $\epsilon^2 \lll 1$ (one structure cannot adopt mutation alone if the complementary part do not adopt the specific compatible co-mutation that makes the couple still functional). An example of this mutation inhibition can be observed in the 6HB structure (helical bundle) of the sarscov2 spike-S2, responsible of the fusion, in which one of the two helices of the bundle, namely the whole HR2 structure, indeed keeps the same aminoacid chain than the one of its parent SARS-CoV (which points this predecessor as the origin of the sarscov2 with an unavoidable ID mark [3]). Regarding tropism, it is so specific the coordination of multiple synchronized binding bases for the attachment that it impedes the glutespike to dock to the rest of cells. In simple words is to comply necessarily with A key and B key simultaneously to allow the docking operation, so that the mechanism is so selective that cannot be found in the rest of cell types.

As a matter of fact, this viral strategy can be employed to efficiently treat all the autoimmune diseases that shares a common etiology of an altered memory T-cell response. This solution would be remarkable less risky than completely rebooting the immunological system as it is done for other autoimmune diseases such as Crohn [4].

References

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