Celivir: A Viral Therapy for the Autoimmune Celiac Disease

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Received XX; Revised XXXX; Accepted XXXX

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


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 \( e^2 \ll 1 \) (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 glutespikes 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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