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# Modulation of the immune system for treatment of atherosclerosis

## Propositions

1. Polyclonal stimulation of Tregs with IL-2 complexes is not a viable way to enhance tolerance to a specific antigen. (This thesis)
2. Adjuvant properties of the Apolipoprotein B100 derived peptide p210, would explain the divergent immunological effects dedicated to vaccination with p210. (This thesis)
3. The opposing, anti-inflammatory and pro-inflammatory effects of the activation of the unfolded protein response in cells of immune origin and non-immune cells, are a logical consequence of different roles of immune cells and non-immune cells respectively. (This thesis)
4. An optimal atherosclerosis vaccine should act tolerogenic and target a variety of plaque antigens. (This thesis)
5. CTLs in vivo are not the relentless killers as they pretend to be in vitro. (Halle et al. 2016)
6. There has been a disproportional focus on ApoB100 as an auto-antigen in atherosclerosis, while autoantibodies against many extracellular matrix proteins and heat shock proteins are found in atherosclerosis. (Merched et al. 2016)
7. While advances in omics techniques clearly provide opportunities, the big volumes of data associated with it pose significant statistical and interpretative challenges for the researcher, let alone for a peer reviewer. (Angerer et al. 2017)
8. The effect of aggressive therapeutic lowering of LDL-cholesterol levels is limited and comes with tradeoffs. (Ma et al. 2019)
9. Implementation of general immunosuppressive therapies to treat atherosclerosis could potentially act like anti-vax v2.0.
10. As atherosclerosis development impacts the immune system, immunological changes observed after successful treatment of atherosclerosis are not necessarily causal to reduced atherosclerosis.
11. The strict word counts enforced by journals promote salami slicing, and shallow context, while the days that physical space restriction due to printing on paper were relevant are long gone for most journals.
12. A better understanding of the working mechanism of preclinically tested pharmaceuticals could improve the success rate in clinical trials, but is not necessarily the focus of small biopharma companies.

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