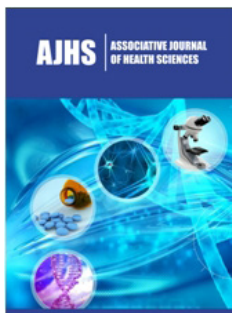


Hypothesis for COVID-19 Vaccine Detoxification

Yang Pachankis*


Independent Researcher, 1001 Biqing N Rd, Chongqing, China

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***Corresponding author:** Yang Pachanki, Independent Researcher 28-4 Bldg. 2, Dexinyua 1001 Biqing N. Rd. Chongqing, 402762, China

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Abstract

The article puts forth the null hypothesis and secondary alternative hypothesis for a newly designed clinical trial. The alternative hypothesis utilizes the clinical trials characteristics for a parallel behavioral trial in determining the correlative role of chromatophores in human immunobiology. The clinical trial is designed with the sebaceous immunobiology concept, and the detoxification ideally reduces the acidification activities in SARS-CoV-2 viral entry and reactive oxygen species' concentrations in the viruses' hibernation in low-density lipoprotein cholesterol altogether.

Keywords: Chromatophores; Degeneration; Immunology; Ions; Lipids; Observational trial; Oxidative stress; Sebaceous immunobiology; Sebum

Background

The concept of sebaceous immunobiology is relatively new, and the regulation of sebaceous gland function by immune cells is antigen-independent [1]. It was observed in vitro that human sebocytes during hypoxic stress induce endocytic trafficking and induces epidermal growth factor receptor (EGFR) into cell cytoplasm [2]. With the quantitative dynamics of reactive oxygen species (ROS), the involvement of sebaceous immunobiology in endocytosis not only suggests the possibilities of correlations to apoptosis pathway, but also the possibilities for chromatophores' influences on natural detoxification and the human immune reflexes [3]. The phenomenon was observed in proton motive force (PMF) dynamics with relation to adenosine triphosphatase (ATPase), making a correlation between antigen-independent sebaceous immunobiology and antigen-dependent cardiac activities [1,3]. Current studies on the chromatophores' influences focus on skin-brain neuroendocrine activities with solar entropic activities, and the classical studies focus on microbe bacteria [4,5]. Even though COVID-19 also induces bacterial infections, the research hypothesis mainly aims to fill in the gap on autoimmune viral pathogen in the microbiome spectrum.

The Low-Density Lipoprotein Cholesterol (LDL-C)'s correlations to COVID-19 vaccine injuries incentivized further studies on the phenomenological roles of chromatophores in human physiology. Ethnic differences in COVID-19 pathogenic risks have raised more awareness on the potential immunological roles of chromatophores against autoimmune pathogens, and the studies will focus on sebum in immunological chains [6-8].

Vaccine poisoning entry path and LDL-C's correlations to COVID-19 prognosis and severity call for sebaceous immunobiology studies. Evidence emergence from the pilot interventional trial seeded the null hypothesis, that SARS-CoV-2 vaccination poisoning induces SARS-CoV-2 Spike 2 protein hibernation in human host in LDL-C, for another clinical trial, after the elimination of sudden death risks from cardiac adverse events of the participant [9-11].

It has been traced that SARS-CoV-2 entered the participant's blood system already after the first vaccine shot with subcutaneous hemorrhage, second vaccine shot with arthritis from old minor surgery, and months after being fully vaccinated with relatively comprehensive physical examinations; hematology results did not show prominent platelet over-size half a year after 3-dose full vaccination, with earnings from platelet distribution width [12]. Minor chemiluminescence was detected with the HIV-test but was only regarded as minor

laboratory bias. Only retrospectively, it is confirmed to be the baseline characteristics at the conclusion of the interventional trial with the ClinicalTrial.gov number NCT05711810 [12,13].

The evidence suggests endocytic fusogenicity did not happen during the vaccination, but caused the pericarditis symptoms that informed the medical intervention. Furthermore, the participant’s neurodiverse conditions never emerged with neurologically caused physiological symptoms until the intervention started. It was inferred from the trial hematology data that worsened blood acidification signified by eosinophils absolute number and percentage underlay the adverse event, and preliminary review indicates to the role of peripheral serotonin in respiratory immune

reflex [14,15]. Its continued symptoms after the stabilization of cardiac conditions imply that sebaceous immunobiology can be more associated with the neurological responses than the leukocyte disorders.

Since the participant’s neurodiverse conditions are mainly associated with the vagus nerve and oxidization is also associated with the symptom emergence with the ATPase readjustments, the order of symptom disappearance between neurological conditions and detoxification from vaccine poisoning can further edge in the causal inference between sebaceous immunobiology and oxidization stress. A truth table has been organized and seen in Table 1.

Table 1: Statistical testing truth table for the trial NCT05839236.

	Healing	Before or After	Null Hypothesis	Alternative Hypothesis 1	Alternative Hypothesis 2
Symptomatic Signs	F	-	-	F	-
	F	A	F	F	-
	T	B	T	T	F
	T	A	T	T	T

Since platelet binding must proceed after initial rapid acidification in SARS-CoV-2 entry, it is highly likely that the correlation exists between oxidization and symptomatic autism spectrum disorder (ADS), i.e., the neurological changes with the EGFR’s cell cytoplasm and S2 protein fusogenicity in the endocytic domain [16-18] The behavioral studies in the new trial, therefore, may be able to contribute new knowledge on neurodiversity’s implications to immunology and immune reflex.

Material and Methods

The clinically relevant behavioral trial adopts the participant with ADS and the clinical trial medicines as materials. Atorvastatin Calcium Tablets and Chinese herb compounded Anti-Viral Granules are used together with Trimetazidine Hydrochloride to monitor the symptomatic behavioral changes under the biomedical detoxification in sebaceous immunobiology. With the case’s special conditions of ADS compared to wider neurotypical population, and the background from family history suggesting possible microbiome-induced adaptive genetic mutation contributing to neurodiversity, there is the possibility that the adaptive genetic mutation is protective to the autoimmune pathogen in the areas where SARS-CoV were first known to humans. Therefore, sebaceous changes under medication are regarded as interventional probing responses in the observational facet of the trial. Questionnaires and self-reporting are adopted for behavioral surveys, and the particular case’s ASD is regarded as the natural inhibition with nerve compression located to be the C1 and C2 cervical nerves [19].

The method adopted serves as the secondary alternative hypothesis for the clinical trial. The behavioral trial hypothesizes that SARS-CoV-2 acidification strategizes oxidizing the endocytic domain for getting out of hibernation and entering the host’s upper immune system through EGFR cytoplasm, and the respiratory

and neurological pathogenesis partially result from hypoxia [20]. With the gaps between the first known SARS-CoV outbreak in Guangzhou during 2002 and 2003, causal inferences between neurodegenerative diseases and pharmacokinetic correlations in clinical trials between 2003 and 2019 will be potentially renewed with the role of chromatophores with the complex symptomatic results from SARS-CoV pathogen.

Result

The secondary alternative hypothesis from the analysis suggests spike protein platelet binding and / or further pathogenic activities depend on ROS [21]. The implication is consistent with chromatophores in ethnic differences in the roles of COVID-19 severity, where possibly the lowest chromatophores ethnicity has the least risks in severe COVID-19 outcomes and the slightly different trends between Asians and black ethnicity may have resulted from viral and host ROS competition [8].

Cytokine storms in COVID-19 lung fibrosis could have resulted from ROS concentration in the immunological tract [22]. Albeit SARS-CoV series’ spike proteins target across the immunological duality in human physiology, both the Spike 1 and Spike 2 proteins’ features in targeting human physiology concentrate on ROS-rich areas. With Spike 1 protein’s angiotensin-converting enzyme 2 targeting that takes a faster route across the blood-brain barrier pounding between carotid artery and the lower respiratory tract through cardiac contraction, the immune tract’s less defense filtering from stem cells could have led to the respiratory symptoms that constitute the long mis-categorization of the virus.

Discussion

The secondary alternative hypothesis explains the clinical trial’s alternative hypothesis’ rationale. The objective for the

alternative hypothesis is to bypass the lipid degeneration activities in the blood system that can acidify the blood environment, and to detox the inactive or mildly active viruses and proteins out of the endocrine system and respiratory tract. Due to the limited availability of medicines, the choice of Chinese-medicine compound antiviral drug mainly reduces the risks for over-capacity and over-stimulation on internal organs during detoxification excretion, and proton pump inhibitor will be discretely continued from the previous trial's evidenced positive effects.

It is possible that the sebum and chromatophores regulate the ROS across the immune system and have an influence on immune reflex. The inference from the analysis may not be able to be determined by the clinical trial's study design, but the correlative evidences may accumulate just as the detail in its possible correlations with PMF [3]. Even though lipids are generally considered non-polar, they may have a polar activity with the ROS and PMF correlations that influence the physiological complex and viral infection patterns. Further studies into the molecular diffusion and kinetics via density and ions may shed new light on the questions involving chromatophores.

Further reviews into sebaceous immunobiology and neurodiversity will be conducted for precision. Sebaceous immunobiology is a relatively new concept, and the review will focus on physiological correlations for further causal inferences. At the end of the trial NCT05711810, the participant started to have involuntary defibrillation reactions when resting at bed before sleep. The positive health outcome's causal relations have not yet been fully studied nor phenomenon well-explained. The antigen gaps between sebaceous immunobiology and the blood-brain barrier may offer new insights into the phenomenon.

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Conflict of Interest

The author declares no conflict of interest.

Data Availability

1. Data from the previous interventional trial that paved the rationales for the hypothesis is publicly available on Zenodo with the doi: 10.5281/zenodo.7883407.
2. Registration of the two clinical trials with study protocols and statistical analysis plans can be accessed on ClinicalTrials.gov with the URLs: <https://clinicaltrials.gov/ct2/show/NCT05711810> and <https://clinicaltrials.gov/ct2/show/NCT05839236>.

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