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Adrenaline Intolerance in ASD curing from Autoimmune Pathogens — Sebaceous Immunobiology in Autoimmune Pathogen Research

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Indicator	Baseline	Reference Range	Unit	Method
Adrenaline Intolerance in ASD curing from Autoimmune Pathogens — Sebaceous Immunobiology in Autoimmune Pathogen Research				
HIV	0.08	0.00-1.00	S/CO	Chemiluminescence

Abstract

The review is a summative of the clinical trials numbered NCT05711810 and NCT05839236 on ClinicalTrials.gov, with the sole participant's recovery from autoimmune pathogens who was also diagnosed as neurodivergent during the first interventional trial. The review seeks to bridge the literature gaps between psychiatry and the medical sciences on neurodiversity, focusing on immunobiology. It chooses the concept of adrenaline intolerance during the final phase of the recovery process to summarize the clinical evidence. The first part of the review synthesizes the key locations of autism spectrum disorder (ASD)'s neurological differences to neurotypical individuals. With the anatomic overview, the second part reviews the relevance to the immune system and implications in immune reflex. The third part examines the neuroatypical hormonic paths based on the ASD participant's data, whereby the final recovery process with hypolipidemic agent intervention posed a contradiction between the neuronal needs and autoimmune needs of the participant's internal conditions. The review predicts that the contradiction between clinical observation and medical literature offers a new window into the study of sebaceous immunobiology.

Keywords: *chromatophores; degeneration; immunology; ions; lipids; observational trial; oxidative stress; sebaceous immunobiology; sebum*

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Evidence in Context

What Know: The clinical trials numbered NCT05711810 and NCT05839236 on ClinicalTrials.gov, with the sole participant's recovery from autoimmune pathogens who was also diagnosed as neurodivergent during the first interventional trial. The review seeks to bridge the literature gaps between psychiatry and the medical sciences on neurodiversity, focusing on immunobiology.
What New: The review predicts that the contradiction between clinical observation and medical literature offers a new window into the study of sebaceous immunobiology.

To view

Introduction

The antigen-independent regulation of sebaceous gland function by immune cells is recently put into focus as sebaceous immunobiology [1]. Sebum lipids, predominantly triglycerides and fatty acids, adds up to 57.5% of total lipids, and the major fatty acid desaturase in human sebaceous glands is stearoyl-CoA D-6 desaturase [2]. It induces rapid oxidation and degradation of linoleic acid, along with its derivatives in sebaceous gland cells [2]. The degradation chain not only yields antibacterial and antifungal products, but also products that activate the receptor domain [2].

Sebaceous immunobiology is especially relevant to endocytosis. The causal relations have been less studied. Epidermal growth factor receptor (EGFR) is induced into cell cytoplasm by endocytic trafficking induced by human sebocytes, during hypoxic stress [3]. It is inferred from the evidence that sebaceous immunobiology is interlinked with apoptosis pathways. With the close associations between immunobiology and neurology, suitable study designs for sebaceous immunobiology have been insufficient.

With a sole participant study design on COVID-19 post-vaccination autoimmune pathogen, the scientific value of the participant's autism spectrum disorders (ASD) in response to autoimmune pathogens has emerged from the medical observations during clinical monitoring. Summative of the clinical trials numbered NCT05711810 and NCT05839236 on the participant's neurodiverse case, the review navigates the trials' basic science implications [4, 5]. The interventional trials mainly adopted inhibition therapies to the neurodivergent case to boost recovery and detoxication of the autoimmune pathogens; the interdisciplinary approach amongst psychiatry, neurology, immunology, and pharmacokinetics has been adopted for the review.

Review

Neurodiversity

Neurodiversity is a recent topic in the medical sciences that originated from psychiatry. Neurodiversity is comparable to biodiversity within the human species, and the statistically insignificant group under the evolutionary perspective may reflect the dynamic human-environment interactions that can further co-determine the evolutionary paths of the human race. Both genetic and prenatal conditions have been reported to contribute to neurodiversity, and propositions on epigenetic studies in gene-environment interactions have been raised [6].

Albeit the review has limited the scope in terms of the medical sciences, cognitive science aspects are not irrelevant to neurodiversity phenomena. Institutional and organizational behaviors are mass psychologically driven. The multivariable among human activities, collection decisions, focus and predictability of nature, intersubjective interpretations, etc., all pose the possible uncomprehensiveness in collecting scientific and human knowledge concerning the orthodoxes of behaviors from their implications in the natural sciences.

Within the framework of rationality, the review does not take The responsive cognitive elements into the discussions of neurological and immunological sciences. There is a scientific consensus that persons with ASD are more prone to viral pathogens [7]. The review takes an alternative perspective that persons with ASD may be more sensitive to pathogenic signals. Traditional views of ASD vulnerability are based on normative pathogens but do not consider autoimmune pathogens. The baseline of the case from autoimmune pathogens is set on the medical history tested on Feb. 8, 2022, whereby the chemiluminescence results on human immunodeficiency virus (HIV) are seen in Table 1 [8].

Table 1: The virus-specific false positive result of the participant approximately one year before the clinical trial.

Indicator	Baseline	Reference Range	Unit	Method
HIV	0.08	0.00-1.00	S/CO	Chemiluminescence

Contributed to the HIV gp41 structural similarity of SARS-CoV-2 Spike 2 protein, virus-specific HIV tests with chemiluminescence and other methods have reported false-positive results [8-10]. Regarding autoimmune pathogen detection, however, HIV tests could have provided a better indicator than COVID-19-specific tests, whereby the participant has been testing negative for COVID-19 after the vaccinations without any risky contacts.

Nerve Compression in ASD

The nerve compression clinical symptoms are the natural nerve terminal inhibition activities in response to autoimmune pathogens. Twin studies in ASD upon phenotypes suggest both genotype and prenatal environment play a role in developing neurodiverse conditions [7]. The participant's birthmother experienced the loss of twin daughters before bearing the participant, contributed by the virological leak in the hospital in Chongqing in the 1980s upon labor. Apart from mutations resulting from developmental differences in the Central Nerve System (CNS) compared to neurotypical persons, the main developed anatomic neurodivergence has been psychiatrically located to be the differentiations in neuronal [dis]connectivity [7]. In the case the review based upon, the disconnectivity is located on the cervical plexus. While the disconnectivity can be clinically define as nerve compression due to the diagnostic purposes in medical treatment during the recovery process' necessities in lipid degradation, it is regarded as the indicator of nerve terminal inhibition.

The main differentiation between ASD and neurotypical persons resides with the vagus nerve (VN). The VN belongs to the Autonomic Nervous System (ANS) under the subcategory of Parasympathetic Nervous System (PNS), with the other two main subcategories of Sympathetic Nervous System (SNS) and Enteric Nervous System (ENS) [11]. The ANS is classically considered a system that cannot be voluntarily influenced, and that passively balances homeostasis [12]. The SNS and PNS regulate vegetative function by acting in opposition to each other [11]. However, the logical premise may be shaken by the voluntary activation of the SNS exceptions with techniques involving the breathing methods [12]. The exceptions suggest voluntary controls in the ANS may change the homeostasis by conscious practices and, in turn, influence an individual's internal nuclear dynamics.

Selective serotonin reuptake inhibitor (SSRI) was introduced as soon as the notice of participant's ASD. SSRIs exert action by inhibiting the serotonin reuptake, thereby increasing serotonin activity while having little effect on other neurotransmitters [13]. The logical induction, after adopting angiotensin-converting-enzyme inhibitor (ACEI) in reducing the adverse event risks and sudden death risks of the participant from autoimmune pathogens, was made by the notice of vaccine viral entery through muscle subcutaneous tissues, intruding the lipid barrier with proximity. The sebaceous gland is currently considered not only as an important endocrine organ, along with the keratinocyte origin of the lipids, but it is also acknowledged to be immunobiological in stem cell homeostasis and coordinating innate immunity [1, 14]. Furthermore, preproenkephalin agonist and μ-opioid receptor (MOR) stimulate the consumption of high-fat food and are correlated to dopamine levels and MOR genetic expression [15].

Cervical Spinal Nerves

Motor and sensory information is conducted by the cervical spinal nerves via efferent and afferent fibers respectively, to and from the CNS [16]. The inhibited / compressed nerves in the ASD participant are symptomatically narrowed down to the C1 and C2 cervical nerves, whereby C1 gives rise to the nerves to the geniohyoid and to the thyrohyoid [16]. The diagnosis coincides with the participant's medical history, who frequently had inflammation in the upper respiratory tract and suppurative tonsillitis during growth.

The Trigemino-Cervical Complex (TCC) rooted in C2 *on a par* with occipital nerve contributes to migraine, headache, and neck pain [17]. The participant's migraine started after ACEI and proton-pump inhibitor (PPI) treatments and shifted to neck pain in the second trial of lipid detox treatments. The involvement of the afferent stimuli from neuropeptide-containing trigeminal nerve, together with the clinical evidence in myoelectrogram suggest a correlation between migraine symptoms and myoelectric activity changes in the trigeminovascular system [18, 19]. Furthermore, the effectiveness of SSRIs in assuaging the symptoms suggests the correlations to innate immunological changes. This partially explains the participant's involuntary defibrillation responses while quietly in bed before sleep by the end of the NCT05711810 trial, when the HIV testing baseline dropped to 0 [19].

While the migraine started not long after the emergence of throat pains, the pain's similarity in motif with the co-occurrence of headache and neck pain can be associated with neuronal signaling on infection risks [17]. This assertion is a common phenomenon regardless of neurotypical or neurodiverse in many respiratory tract infections. Therefore, the motifs of C1 and C2 inhibitions in the TCC in the particular case are further reviewed with the introduction of SSRI.

As evidenced by the hematology tests, the introduction of SSRI did not substantially influence the platelet binding activities [19, 20]. The neural-immune interaction via the ANS, in the case of serotonin, fulfilled the correlations with receptors' presence in immune cells and immunoregulatory effects, but without the local association of neurotransmitter-specific nerve Fibers with immune cells nor exclusive neurotransmitter supply of the immune target cells/organ by neurons [20]. Since sebaceous immunobiology is antigen-independent, it is hypothesized serotonin's associations with immunology reside with sebaceous immunobiology and not the traditional narrower definitions of immunology. The special characteristics of serotonin that it can be taken up by noradrenergic terminals on smooth muscle cells, similar to the adrenal medulla, corroborates with the clinical experience in terms of adrenaline intolerance from the internal functions with dopamine levels of the ASD participant [20]. The neuromuscular junction is the focus on the associations between sebaceous immunobiology and neurological regulations. The question is raised; did the participant's migraine result from neurosarcoidosis from the signaling pathways between neurological regulations and sebaceous immunobiology?

Peripheral Serotonin in Endocytosis and Exocytosis

Apart from the central functions of serotonin, peripheral serotonin is known to affect vascular tone, cell regeneration, heart functions, hemostasis, immuno-modulation, organ development, intestinal motility, etc., stored in platelets in high concentration [20]. Serotonin stimulates monocytes and lymphocytes, and influences the secretion of cytokines, a major contributor to SARS-CoV-2 lung fibrosis [20, 21]. The antigen-independent effects of serotonin in immunology mainly function through antigen-dependent leukocytes. Even though not conclusive enough in scientific consensus, it is certain that serotonin takes part in reactive oxygen species (ROS) regulations, possibly through neutrophils [20].

Not only monocytes/macrophages and lymphocytes take up serotonin-by-serotonin transporter (SERT), but also T-cells and B-cells. In contrast, platelets take up and store plasma serotonin released from intestinal enterochromaffin cells and function after stimulation on inflammation with 5-HT2A receptor [20]. Monocytes/macrophages express serotonin receptors and are believed to express serotonin synthesis (TPH) and monoamine oxidase (MAO). At the same time, neutrophil (and eosinophils) recruitment during inflammation is enhanced by activation of serotonin receptors, including the presence of platelet serotonin [20]. Whereas serotonin functions in a positive role in anti-inflammatory immune responses, serotonin may inhibit the anti-viral capacities of the leucocytes by inhibiting interferon-γ-induced antigen-presenting macrophage capacity, affecting the phagocytic activity of neutrophils, driving apoptosis of Burkitt lymphoma cells, and influencing the adaptive immune response of B-cells [20].

Even though serotonin modulates anti-inflammatory cytokine secretion, which is the main cause in COVID-19 severe cases, lipopolysaccharide-induced pro-inflammatory cytokine production is not influenced [20, 21]. Besides, dopamine inhibitors increase sebaceous secretion considerably, and there is a strong correlation between the SARS-CoV series' immunological pathogens and lipid sedition [22, 23]. The phenomenon suggests the ASD case's natural neuronal advantage against the SARS-CoV series, born in the areas where the virus was first known. Moreover, the inhibition of C1 and C2 cervical nerves of the ASD case coincides with the serotonin bacterial, viral, and autoimmune pathways [16].

This incentivizes further review of the intersection of sebaceous immunobiology with endocytosis and exocytosis in the blood pathogenesis of the SARS-CoV series [24, 25].

The main entry mechanism of the complex autoimmune pathogens of SARS-CoV-2 is through endocytosis with membrane fusion [26]. The acidic cytoplasm for fusion and genome entry into the cytosol through endosomes of SARS-CoV-2 is partially guided by S1 protein's ACE2, or both ACE2 and TMPRSS2, targeting, in receptor-mediated endocytosis [26, 27]. Even though on a molecular level, Ca^{2+} and nicotinic acid adenine dinucleotide phosphate (NAADP) have been reported to be the main contributing factors, proton dynamics and homeostasis are still the transmembrane domain's main regulators [27, 28]. SARS-CoV-2's autoimmune contributor S2 is enabled by protonation to collapse toward the folded-back, postfusion trimer of hairpins and pull together the viral and host-cell lipid bilayers, causally explaining the low-density lipoproteins cholesterol (LDL-C) correlations in critical and severe COVID-19 cases [26, 29].

Positrons' influences on proton homeostasis, electrostasis, and hydrostasis for exocytosis are theoretically the direction for sebaceous immunobiology concerning the autoimmune pathogen. Current medical resource conditions limited the interventional solutions to inducing apoptosis with internal medicine in order to discontinue viral replications and decrease the viral load and concentration, and bafilomycin A1 does not have human trials yet for discretion. Substantial cell kill capacities by ^{18}F β^+ emission have been reported i*n vitro* with β^+ irradiation of LNCaP C4-2B cells [30]. Continuous distribution of radiation damage, however, can occur with positron therapy's sub-KeV resonance by a higher density of several electrons and ions, as a result of greater localization of energy deposition and resulting in a higher probability of inducing direct DNA double-strand breaks [30].

There is no primary literature on positrons' functions in exocytosis, but positron emission tomography has been adopted for real-time and *in situ* observations in relevant studies [31]. Molecularly, fusion pore in exocytosis differs in classical and compound events [32]. Classical exocytosis undergoes discrete fusion with single secretory granules' extrusion, and compound exocytosis aggregates granule-granule fusions before focused secretion in the transmembrane domain [32]. The two types influence hormone concerns in secretion intervention for detoxication. Macroscopically, it is conceptualized as in-put and out-put signaling in dendrites around the cell bodies [33]. Exocytosis in normative immune responses is a pro-inflammatory factor, and the mast cells exhibit compound exocytosis [32]. In the meantime, tryptophan hydroxylase 1 is present in mast cells, which can be synthesized into 5-hydroxytryptamine (serotonin) [20].

The implications that the changes in hydrostasis during compound exocytosis may influence ROS and apoptosis, with fusion pore considerations between endocytic acidification and exocytic alkaline tendencies, diversified the methodological approaches to the autoimmune pathogen [34]. With statistical pharmacokinetic rationales, extracellular vesicles have been the focus for solutions in endocytotic and exocytotic domains [35].

Vacuolar-type H^+-ATPase inhibition has been the main purpose in both clinical and theoretical internal medicine solutions, including PPIs, bafilomycin, (hydroxy)chloroquine, etc., and the detailed efficacy and safety concerns concentrate on proton-coupled electron transfer in the transmembrane extracellular vesicle ingredients [35].

Albeit the adverse event in hindsight may have risked neurosarcoidosis in the clinical trial NCT05711810, carcinogenesis risks by the viral pathogen do not falsify the validities of the interventions. Discrete *in vitro* and *in vivo* investigations have reported PPI intervention's inhibition effects on tryptophan hydroxylases and monoamine oxidase-A, underlying the adverse event of migraine discovering the participant's ASD [36]. With the hibernating viral concentrations in sebum lipids, endocytic trafficking in EGFR cell cytoplasm can be the main cause of the autoimmune pathogen's carcinogenesis capacities, apart from the natural accumulation in LDL-C [3, 29]. From the other perspective, the participant's neurodiverse conditions in the compression of C1 and C2 cervical spinal nerves naturally prevented endocrine concentration of ROS in transmembrane proton activities, explaining the more-than-1-year prolonged cardiac event from being fully vaccinated. Further reviews must associate both intracellular and extracellular signals in the apoptotic pathway with the ANS [37].

Autonomous Nerve Interactions with Autoimmune Pathogen

Innate immunity is intervened by the ANS through efferent pathways, and homeostatic control centers locate in the hypothalamus and brainstem. ASD's main differences are VN's afferent transmissions to the brainstem, resulting in stronger CNS impulses to ANS with inhibitory afferent feedback [38]. The adrenal glands more sensitive influences to total testosterone, free testosterone, and dehydroepiandrosterone in ASD have been clinically tested, implying less tolerance from adrenal medullae's epinephrine and norepinephrine release during mass sympathetic discharge, which typically occurs during the "fight-or-flight" response and exercise [38, 39].

Enhanced function of MOR regulation in ASD sebaceous immunobiology and neuroendocrine system is expected [40]. The S2-induced autoimmune pathogen has hypothermia-like characteristics for its competition for ROS, explaining the neurosarcoidosis risks [41, 42]. There is an ambiguity in the ASD natural catecholamine imbalance's potential positive or negative contributions to the curing process in the interventions. Still, the J wave shifts in the case contributed positively to the risk reduction in neurological infection with migraine adverse event and later autonomous cardiopulmonary resuscitation with involuntary defibrillation [42, 43]. The phenomena could have been induced by the intervention designs in transmembrane fusogenic inhibitions for autophagy acceleration [44].

S2's homogenous HIV-1 gp41 is not responsible for macrophage infection [45]. The release of the viral core into the cell and fusion between the viral and cellular membranes are facilitated by HIV-1 gp41, and in the case of SARS-CoV-2 vaccine poisoning, the antigen-dependent autoimmune infection is similar to a combination of leukemia and HIV [45].

Protecting the autophagy process cures the antigen-targeted HIV-like autoimmune dysregulation, and ensuring the transmembrane integrities of macrophages cures the leukemia-like autoimmune dysregulation. The differences between ASD and neurotypical in terms of HIV on CNS infection further imply that ASD nerve compression better protects the nervous system but may have higher risks of carcinogenesis if sebaceous immunobiology does not function well during autoimmune pathogens [45].

ASD's ANS is semi-autonomous, differentiated from the vegetative nervous system naming rationale from classical neurotypical interpretations. With less direct feedback from the endocrine system, catecholamine influences on ANS and its interactions with CNS have stronger correlations to hormone synthesis by autonomous exercises in ASD persons, thus more sensitive as well from the environmental factors [46]. Therefore, autoimmune pathogen interventions' neurosarcoidosis risks in ASD persons are dose-dependent. Cell-autonomous stress responses in brain-skin signaling and lipopolysaccharide-induced profiles of innate immune responses will provide further insights into differentiated immunology in ASD persons [44, 46, 47].

Conclusion

Contrary to traditional beliefs in ASD immune inferiority, prima facie evidence suggests ASD's immunological differences have better integrities against autoimmune pathogens and preservation of nervous systems. This difference can be largely environmentally adaptive with the case studied before the review, while its epigenetic origins have not been cover in the review.

The vagus nerve differences in ASD exchanged organ-specific signaling in inflammatory events with statistical response strategies. It is not sure if the ASD mutation is transitory according to environmental factors or more inherent in gene-specific alterations. The ANS flexibility, however, provided by natural nerve compressions sheds an intriguing window on sebaceous immunobiology with natural inhibition on the afferent pathways.

There may be meaningful differences in ASD electrostasis from neurotypical persons. The differences may be immunological and neurological, contributing to the cardiac differences in the electrostasis and influences on innate hydrostasis. The correlations may reside with myeloid cells and T cells in the syntheses and pathways, causing the differences in innate immune responses concerning opioid receptors.

Neuronal receptor inhibition therapy may be a research direction for autoimmune pathogen cure in neurotypical persons. The case from the clinical trials that incentivized the review has indicated profound adverse effects from COVID-19 vaccination in response to the autoimmune viral pathogen. From the clinical solutions and the study, it is inferred that the ASD immunological differences contributed by natural neural compression and inhibition can be learned from dealing with neuronal infection risks in neurotypical persons and detoxication of SARS-CoV-2 S2 protein.

Sebaceous immunobiology has the potential to advance immunological research and cancer research. The peripheral review on sebaceous immunobiology suggests its interconnected actions in innate immunity signaling and autophagy. Its antigen-independent functions show complementarity with nervous system regulations in classical innate immunity. There is a possible connection between ANS and CNS in regulating and coordinating the two immunological systems, and sebaceous immunobiology's role in the blood-brain barrier remains to be discovered.

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Conflict of Interest
The author declare no conflict of interest.

Data Availability
Data from the previous interventional trial that paved the rationales for the review is publicly available on Open Science Framework with the doi: 10.17605/OSF.IO/2MGJK.
The study protocols with statistical analysis plans and informed consent forms are openly available on ClinicalTrials.gov and can be accessed with the URLs: https://clinicaltrials.gov/ct2/show/NCT05711810 and https://clinicaltrials.gov/ct2/show/NCT05839236.

Consent Statement / Ethical Approval
Informed consent with confidentiality policy has been obtained from the participant.
The ethics committee formed for the study has approved its publication.

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