Review

May a Clinical Implementation of the United Immune System Concept Help Delay the Onset of Degenerative Proteinopathies?

Theodor-Nicolae Carp *

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Review

May a Clinical Implementation of the United Immune System Concept Help Delay the Onset of Degenerative Proteinopathies?

Theodor-Nicolae Carp

Watford, Hertfordshire, England, United Kingdom of Great Britain; theodore.nicholas100@gmail.com

Abstract: Degenerative proteinopathies constitute a set of molecular diseases that are caused by the misfolding of specific proteins, leading them to change their biochemical configuration and become toxic for entire systems of organs. Such protein toxicity induces the lysing of an increasing number of proteins that have a biochemically 'wild-type' version, gradually and eventually leading to a complete shift in the ratio between such 'wild-type' and 'altered' versions of such proteins, which directly precedes the clinical onset of such diseases. Proteinopathies not only involve neurodegenerative illnesses, but also a disease that leads to a progressive rate of blindness. Sadly, all such impairments that are neurodegenerative in nature may only receive palliative treatment, given that they are caused by aggregated proteins that start damaging and destroying entire neuronal systems, which leads to impairments in the neuro-muscular and ultimately to the inability of the patients to perform vital functions, like breathing and deglutition. There is neither a cure, nor a definitive method in which the progression of the illness can be stopped at the present time. Consequently, all neurodegenerative diseases have mortality rates of 100% and clinical approaches aim to reduce the suffering of such patients. Nonetheless, there seems to be a glimmer of hope regarding future prophylactic approaches that could delay the onset of many types of proteinopathies. Namely, an immune application could support efforts of clinical suffering delay and attenuation in an unprecedented manner. At the same time, it is necessary to emphasise upon realistic scenarios, that it remains virtually impossible to delay the onset of proteinopathies to the point of the patient reaching the average number of years in life expectancy without experiencing clinical symptoms yet. Initially, clinicians developed and tested a nasal spray containing a substance known as protollin, which stimulates a restricted extent of adaptive lymphocyte recruitment and transport to the central nervous system areas affected by initial stages of protein aggregation, activating a substantial number of microglial cells and preventing the lysis of numerous astrocytes, which in turn start lysing a number of beta-amyloid protein aggregates together without inducing pathophysiology, given the stage in which the patients have not experienced any clinical manifestation of the neurodegenerative disease yet. In case of an unsuccessful attempt to bring protollin above the threshold levels of clinical safety and efficacy, an immunostimulatory and immunomodulatory substance containing a low concentration of a mixture of recombinant Type I & III Interferons, innate and adaptive lymphocytes, perhaps themselves priorly treated with such IFN glycoproteins, would probably remain a vital candidate for an effective, yet probably still restricted delay of onset of various proteinopathies that could be neurodegenerative and optically degenerative. An existent success rate of the clinical test allows the opening of a window of opportunity regarding an increased efficacy of such adaptive lymphocyte approach, by including recombinant Type I and Type III Interferons into such a nasal spray, which could also enter adaptive lymphocyte and further improve their structural integrity and their multi-lateral functionality. Moreover, a low dose of protollin, Type I Interferons and Type III Interferons could be inserted in combination into adaptive T-Lymphocytes to optimise their defence mechanisms and immune functions, potentially bringing a considerable immunising effect against microbial diseases like HIV-induced, retroviral AIDS. Such an approach could create a stable and wide "highway bridge" of connection between innate and adaptive immunity, aiming for the best version of an immune contribution toward a considerable delay of proteinopathy clinical onset. Overall, there may be a requirement for a bi-lateral update of immunological research covering therapeutics and vaccine

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development; an immune system based optimisation that builds a stable and wide bridge of connection more directly between pre-cytokine and post-cytokine immune activation, and overall between innate and adaptive immune departments; and a pathogen-based optimisation that either eliminates or partially activates genes suppressive of Type I and Type III Interferon-encoding genes, helping enliven the concept of "United Immune System" as well, though less directly than the immune system-based potential approach.

Keywords: nervous system; optical system; proteinopathy; United Immune System; innate immunity; adaptive immunity; Interferon System; lymphocyte system; pre-cytokine; cytokine; post-cytokine; immunotherapy; Therapeutic Adjuvants; Protein misfolding; nervous system; optical system

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Introduction

The Nervous System (NS) coordinates all bodily functions and consists of the Central (CNS) and the Peripheral Nervous System (PNS), as well as of the white and the dark matter. The white matter consists of the neuronal axons, whilst the dark matter consists of the neuronal cellular bodies. The Nervous System (CNS) and the Optical System (OS) consist of cells that play vital roles with regards to linkage with vital muscular networks and processes of sensory production respectively. Given the fact that the Nervous System overall controls the essential functions of all other systems of organs, and that the source of an optimal nervous system functioning lies in the CNS, it is the normal functioning of the CNS - from the neuronal cells, which constitute the dark matter - that ultimately plays a vital role in the sustainability of human life itself, via functions that include breathing and deglutition, which are performed by both striated and smooth muscles. There are three major types of human proteins that play roles in the maintenance of neuronal stability and good functioning: alpha-synuclein, beta-amyloid and tau, and they are involved in the pathogenesis of three neurodegenerative diseases: Alzheimer's Disease (AD), Parkinson's Disease (PD) and Amyotrophic Lateral Sclerosis (ALS). The pathogenesis of Alzheimer's Disease mainly implicates the aggregation of the beta-amyloid protein, which starts to progressively damage and destroy widespread parts of the dark matter in the CNS, and once clinical disease is onset, immune responses are also induced, as helper CD4+ and cytotoxic CD8+ T-Lymphocytes are activated and recruited, leading to the activation of microglia. Nonetheless, by such time of immune activation, it is already too late for the proteinopathy to be reversed. Usually, the development of immune responses contributes to the process of pathophysiology, rather than even slowing down its process by the slightest, and such developments overall explain why the only available type of treatment for neurodegenerative proteinopathies can only be palliative in nature. A different context applies to optical degenerative proteinopathies, as they do not directly threaten the survival rate. The optical system consists of cells that play vital roles in the development of the sensation of vision, which occurs in the occipital lobe of the encephalon. In the optical system, there are proteins that facilitate the functioning of two types of retinal cells: cells with cones, which are responsible for colourful vision, and cells with rods, which are responsible for vision that lacks colour. Such proteins are known as copies of Rhodopsin (Rho), which is encoded by the Rhodopsin gene (RHO). A misfolding of the Rhodopsin protein is caused by a single-nucleotide polymorphism (SNP) event in the RHO gene, and it involves a change of biochemical configuration from a healthy version to a toxic one. The toxic version of the Rho protein starts damaging and destroying an increasing number of cells with rods, leading to a progressive loss of vision from the peripheral side to the central side, and the name of the clinical disease is known as Retinitis Pigmentosa. Usually, patients develop a full clinical version of Retinitis Pigmentosa around the age of 40, though some SNP events lead to faster disease onset and others to a slower disease onset.

The topic of SNP variation is central also in the area of evolution, as there has been an ongoing strive between human immunity adaptation and microbial polymorphism, both being based upon

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SNP events. The primary and most proximal target of microbial evolution seems to be the human genes that encode major Type I and Type III Interferon subtypes, which further indicates the important role they play in the proper bridging between innate and adaptive immune activation. It is important to note the difference between Type I & Type III Interferons, and Type II Interferons, given the fact that the first are known to activate major types of pro- and anti-inflammatory cytokines, whilst the latter is known to be activated in various manners by pro- and anti-inflammatory cytokines. It may be important to regard Type I and Type III IFNs as pre-cytokine innate immune elements, and Type II IFNs as post-cytokine innate immune elements. Such a new development of the immune hierarchies could offer a more detailed resolution of the overall hierarchical tree that makes the immune system both in its structure and in its function. Once a development as such has been made, it could be well applied into a potential optimisation of other systems of organs, given that an ongoing positive evolution of the immune system can play a major role for the improvement of other bodily functions. In the present case, it is important to note that proteinopathies do have immunological implications, hence the need for researchers to recruit helper CD4+ and cytotoxic CD8+ T-Lymphocytes, perhaps as well as B-Lymphocytes, which are generally known to produce IgM and IgG immunoglobulins and aid the activity of important T-Lymphocyte subtypes in immune responses against various toxins, to slow down the process of neurodegeneration, perhaps as well as visual degeneration, by decreasing the number of functional misfolded and aggregated proteins like alpha-synuclein and rhodopsin. Furthermore, if such an experiment is successful, then it may be possible over a long extent of time for the immune system to gain a degree of "memory" against such toxic proteins, meaning that even pathogen-derived immunisation approaches could be developed and merged with immune system-based vaccination approaches that would be possible to develop after a given extent of time in the future. In short, this could mean that, by the end of the 21st century, it will become possible for vaccines against neurodegenerative diseases and blindness to be successfully developed and rolled out, perhaps through a thorough analysis and application of the "United Immune System" concept into clinical research and testing, to determine whether a statistically significant probability of an actual medical application exists.

Discussion

Human immunotherapy- and vaccine-based research and innovation concerns two areas of biology: immunology and microbiology. Likewise, vaccines have been developed based on both major immune elements and on wholly deactivated or lysed pathogenic agents. Whilst pathogen-based vaccine research may need a restricted extent of further research, perhaps via genetic editing and activation or elimination of genes that are only responsible with the suppression of human and animal genes encoding Type I and Type III IFNs, immune system-based vaccines may need a slightly more extensive amount of innovation, as the current version of the average bridge between innate and adaptive immune system seems to be weak enough for several polymorphic pathogens to use as weak points in their evolutionary path of adaptation to immune changes via SNP alterations. Namely, pathogenic agents have primarily targeted Type I and Type III IFN-encoding genes, both directly and indirectly, causing both the cytokine system and the adaptive immune system to receive the necessary signals of infection later and even much later than the necessary point of time, causing the microbial count/load to increase without significant immune restriction, which itself is causative of the development of chaotic immune responses, with disrupted balances between pro- and anti-inflammatory pathways, which in turn are known to involve less effective antimicrobial responses. Sometimes, such disturbances lead to the development of autoimmune reactions that bring clinical consequences as well. Likewise, the clinical research community may be witnessing the midst of an evolutionary situation in which the progression of human and animal immunity is at least partly prevented by the process of transient innate immune silencing. Given the fact that Type I and Type III IFNs represent pre-cytokine elements that constitute a major part of innate immunity, it may be that a better sensitisation of IFNs I and III represents the most proportional solution to such an evolutionary problem for the overall immune system. Such a sensitisation process can be both direct and indirect. A direct methodology would involve a sensitisation of Type I and Type III IFN-encoding

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genes (INGs), perhaps through a heightened phosphorylative stimulation of the transcription factors that activate such genes, whilst an indirect methodology would involve the usage of deactivated and harmless microbial agents that only contain the interferon-suppressive genes as partially activated genetic compounds, perhaps to indirectly stimulate a faster sensitisation process of the activation of genes encoding IFN I and III (Carp T. et al., 2024).

Traditionally, it has been widely theorised within the broad scientific communities that the two main sectors of the human and animal immune systems are broadly distinct from one another in both structure and function. Namely, the signalling processes contained within the innate immune system, which consists of a first and a second line of immunity, have been regarded as fast and broad-spectrum, lacking specificity and "memory", whilst the signalling processes contained within the adaptive immune system, which consists of a third-line immunity, have been regarded as slow and narrow-spectrum, containing specificity and a "memory". Nonetheless, it has been only in the recent years that scientists discovered there is a significant interpolation between the innate and the adaptive immune systems, as it was observed that the innate immune system develops considerable specific signals and contains a "memory", and that the adaptive immune system has traits lacking specificity. Such a discovery makes it possible for clinical researchers to create immune system-based vaccine candidates for various infectious, oncological and protein-related diseases, that also contain innate immune elements as significant contributory elements. For example, it may not only be T-Lymphocytes that can be used as immune system-based vaccine candidates, but many other immune elements, such as Type I and Type III Interferons, plasmacytoid dendritic cells and natural killer cells, perhaps also in combination with such Interferon glycoproteins. Furthermore, it has become possible even for natural and adaptive lymphocytes to be treated with a low dose of Type I and Type III Interferons before their use in a vaccine candidate that contains all such immune elements in a combination. As years have passed by, it has been observed that critical locations of the evolutionary battleground between various microbial agents and host immune systems are located within the first line of the host immune system, as microbes have been undergoing single-nucleotide polymorphic events within genes that are both directly and indirectly responsible for the suppression of Type I and Type III Interferon synthesis and signalling from the interferon-encoding genes (INGs). Indirect methods of interferon suppression as such have involved the production of non-structural proteins (NSPs) 10, 14 and 16 by the microbial genome, which are implicated in a double methylation of the 5′ end of viral genomes, causing important Pattern Recognition Receptors (PRRs) not to detect Pathogen-Associated Molecular Patterns (PAMPs) and resulting in the host cell not to be able to distinguish between cellular nucleic acids and viral nucleic acids. Consequently, the signalling cascade responsible for the activation of the INGs is not activated, and this leads to the cell's inability to dispose of Damage-Associated Molecular Patterns (DAMPs), which represent toxic substances released by the microbial agent. Direct methods of such interferon suppression have involved the production of non-structural proteins (NSPs) 1 and 2, which are involved in the prevention of the translation process of Type I and Type III Interferons, by cleaving and disintegrating the mRNA molecules that encode them in the cytoplasm of the infected cells. Furthermore, such NSPs also prevent the exocytosis and paracrine of ISG protein products, disrupting the process of a healthy balancing between the synthesis of pro- and anti-inflammatory cytokines.

As novel epidemics and pandemics continued to emerge in the world, scientists reached a point of better ability to thoroughly build an evolutionary tree covering the ability of such microbes to transiently and sometimes even permanently suppress various areas of the host immune system. The emergence of the HIV-induced AIDS pandemic has brought the final straw of research opportunity, as it has been discovered that the virus has gained an evolutionary ability to suppress the central elements of the immune system - helper CD4+ and cytotoxic CD8+ T-Lymphocytes - and not only the peripheral elements of it - Type I and Type III Interferons. With the occurrence and distribution of AIDS cases in the world, evolutionary scientists became able to detect a pattern of microbial evolution, which in fact aims to completely conquer the "intelligence" of human and animal immunity. Namely, the fact that HIV first suppresses Type I and Type III Interferon-based signalling before it suppresses the lymphocyte system, shows that the virus has "led by example" in the overall

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evolutionary process of hijacking and "outsmarting" the evolutionary process of the entire immune system. Given the recent thorough assembly of an immunological tree based deeply on both its structure and function, clinical researchers may be able to successfully counteract the long-term evolutionary strategy developed with each round of microbial SNP event, by inducing a higher sensitisation of the genes that encode major subtypes of Type I and Type III Interferon glycoproteins. A potential positive aspect from the present hypothesis is the fact that major central elements of the immune system can be treated with such interferon glycoprotein to aid in a better immune acquisition against microbial agents like HIV retroviruses. Specifically, it was observed that an early Type I Interferon-based stimulation of helper CD4+ T-Lymphocytes (Itell H. L. et al., 2023), as well as of cytotoxic CD8+ T-Lymphocytes (Cabral Piccin et al., 2023), resulted in a better immune protection of such T-Lymphocytes against HIV, which indicates the existence of an immunisation adjuvant and possibly even whole immunising effect by Type I Interferon glycoproteins for such adaptive lymphocytes, and a similar outcome could apply for Type III Interferons also. If such a scenario occurs, then helper CD4+ and cytotoxic CD8+ T-Lymphocytes treated with a low dose of Type I and Type III IFNs, and/or possibly with a low dose of substances displaying similar immunostimulatory and immunomodulatory effects as such IFN glycoproteins, could become "super-lymphocytes", ultimately helping the evolutionary process of human immunity become boosted in front of advanced evolving methods displayed by various polymorphic viruses and bacteria (Carp T., 2024). Furthermore, following the occurrence of the SARS-CoV-2-induced COVID-19 pandemic, scientists continued to test nasal sprays containing a low dose of Type I and sometimes also Type III Interferon glycoproteins, and the existence of outstanding positive results following clinical trials confirms the foundational role that such innate immune elements play in the induction of an overall immune response that contains a balanced ratio between pro-inflammatory and anti-inflammatory signals and activities. Moreover, it was determined that the sensitivity and quality of Type I and Type III Interferon-based signalling substantially depends on age and underlying health condition, and an existing history of immune diseases or autoimmunity also particularly impacts the sensitivity and quality of interferon-based signalling and induced immune responses (Smits, H. H., and Jochems, S. P., 2024). Overall, it may only be a thorough understanding of the structure and function of the evolutionary battleground between host immunity and polymorphic microbes that may help clinical researchers innovate for novel immunotherapies and vaccine candidates in the most potential manner in such a context of a continuous microbe-immune strive for domination.

Protollin represents a substance introduced in a nasal spray in a relatively low concentration that has recently crossed the threshold level of success considerably in the first phase of a randomised, double-blinded clinical trial. A low dose of Protollin is known to stimulate the recruitment and transport of adaptive lymphocytes from the ophthalmological system - which constitutes the base of the humoral immunity that is significantly involved in a bridging to the adequate activation of adaptive immunity - to important areas of the central nervous system, by the crossing through the blood-brain barrier (BBB). The "low dose" passage contains two potentially key medical terms in the present context, given the fact that an immunostimulatory and immunomodulatory process ought to be clinically "silent" from the moment recruited immune cells cross the BBB into the CNS fluid, and the same ought to be applied for the Retinitis Pigmentosa disease in case of a confirmed prophylactic application against it as well, given the sensitivity of the retinal tissue toward any clinically visible form of immune activity. Nonetheless, it has been discovered that microglia become activated during a process of photoreceptor lysis, which indicates the existence of a firm relationship between the optical system and the nervous system, and perhaps also the existence of a significant overall relationship between the ophthalmological system, the optical system and the nervous system (Karlen S. J. et al., 2020). During the first phase clinical trial that implicated the testing of protollin, it was shown that the substance successfully recruited and induced the transport of such adaptive immune cells to the encephalon, having activated a considerable count of microglial cells, prevented the lysis of astrocytes and helped disintegrating a visible concentration of both soluble and insoluble beta-amyloid aggregates, without inducing clinical illness, as the patients in cause have not experienced a clinical development of the neurodegenerative disease yet (Frenkel D. et al., 2008).

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Likewise, the substance is now due for further clinical trials and researchers are awaiting the collection of results that will confirm or infirm a threshold-level of safety and efficacy for both neurodegenerative and optical degenerative proteinopathies. Several scientists have regarded the substance of protollin as a "vaccine candidate against Alzheimer's Disease" (Frenkel D. et al., 2005). Hence, a similar application could be done for the retinal proteinopathy of Retinitis Pigmentosa, given the much-more-proximal site of the Retina in relation to the nasal cavity, compared to the various areas of the encephalon. Furthermore, Type I and Type III Interferons could positively interact with protollin, bringing a useful immunostimulatory and immunomodulatory effect upon both the innate and the adaptive lymphocyte system. Such a combination could make a useful vaccine candidate against such proteinopathies, and given the stimulatory effect that protollin brings upon important subtypes of lymphocytes, a candidate as such could have considerable applications for other diseases in which the immune system is at least partially implicated. At the same time, a recruitment of helper CD4+ and cytotoxic CD8+ T-lymphocytes does occur in latter stages of the molecular pathogenesis of Alzheimer's Disease, activating the microglial cells in the encephalon. Nonetheless, such a process brings an opposite effect to the mechanical induction of microglial responses that occurs before the symptomatic onset of the neurodegenerative disease, contributing to the pathophysiology process in this case. Perhaps such a phenomenon reflects the one in which delayed and exaggerated Type I and Type III IFN-based immune responses rather contribute to the pathogenesis and severity of various infectious diseases. The similar pattern of a double-edged sword shared by protollin and Type I & Type III Interferons may further indicate the similar immunising nature that protollin and the pre-cytokine interferon glycoproteins share (Chen X. et al., 2023). Likewise, it may even be that a low dosage of protollin can be inserted into adaptive T-lymphocytes with a low dosage of Type I and Type III Interferon glycoproteins, and such a combination of methods could further help T-lymphocytes obtain a more optimal immune function, as well as a more increased defence system against environmental hazards of microbiological nature. In other words, the administration of a low-dose combination of Type I IFNs, Type III IFNs and protollin into adaptive lymphocytes could constitute a considerable immunostimulatory, immunomodulatory and even completely immunising approach against infectious diseases like the HIV-induced AIDS.

The matter of SNP-related evolutionary strive between human immunity and microbial polymorphism could be serious enough for bioinformaticians and biostatisticians to develop novel softwares and update existing computerised systems in order to increase the speed and capacity of data covering the evolutionary history of both human and animals, and of polymorphic microbes, as well as the developed pharmaceutical solutions against past epidemics caused by such polymorphic microbes, by means of increasing the resolution of statistical projections of future public health-related crises, as well as catalysing the process of vaccine-based and therapeutic research. It may also be that an update of computer softwares responsible with bioinformatics and biostatistics-related projections is necessary, by means of the creation and adaptation of artificial intelligence-based softwares into entire systems of human and animal genetic data, which often contain integrated engines that are responsible for the storage and transfer of various digital data between multiple formats, such as HL7 and XML. There may be an example of a health corporation that stores relevant genetic and evolutionary data regarding evolved microbial, animal and human genomes by means of various genetic testing methods that include Real-Time Quantitative Polymerase Chain Reaction (RT-qPCR) - Sonic Healthcare UK. Such a health corporation has recently created an artificial intelligence-based software known as franklin.ai, which has originally been developed for the processing of genetic tests for the detection of various cancer forms, and that has recently helped various laboratories increase the storage of genetic data that are normally collected from patients genetically tested for various molecular diseases (Smith S. C. et al., 2024). In the present scenario, AI softwares like franklin.ai may be adapted to analyse all past genomic moments of adaptation via diverse events of SNP occurrence, which could significantly increase the speed of statistical projection with regards to any future epidemic and even pandemic illnesses. Such an AI-based catalysis of scientific, statistical and clinical research efforts would have a higher probability of long-term efficacy if both front-end and back-end methods of computer programming are consistently used in the

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process of collecting biological and statistical data found in databases like FASTA, MEGABLAST, GBLAST-N, UniProt and SPSS respectively, thereby highlighting the major role that automated, AI-based softwares play in the catalysis of scientific and medical research. It may likewise be important to bring a wider inclusion of artificial intelligence-based methods of research whilst continuing to deeply rely on scientific intelligence, wisdom and intellectual efforts that are based upon the concept of thinking outside of the box, which can only be done by the reach of a consistent emotional and psychological state of humility (Carp T., 2024).

Conclusion

A successful approach of immune optimisation, potentially by updating immunisation and immunotherapeutic approaches to better match the present evolutionary context, through the analysis and implementation of the "United Immune System" scientific concept into clinical practice through realistic methods, would likely bring a considerable effect upon the branches of clinical research that concern proteinopathies causative of neurodegenerative and optical degenerative illnesses. Given the present stage of research concerning the substance of protollin, it is important for the product to undergo further clinical testing in order for scientific and medical researchers to confirm whether it brings an above-threshold level of efficacy whilst checking whether the level of induced adverse reactions remains under-threshold. Interestingly, protollin has been indicated to be a nasal vaccine candidate against Alzheimer's Disease, which may represent a window of opportunity for a future testing of scientific projections that human recombinant Type I and Type III Interferons themselves represent nasal vaccine candidates against various infectious diseases, including those caused by polymorphic microbial agents. Furthermore, an existing probability that protollin is an immunising agent against proteinopathy may also suggest that both protollin and interferon glycoproteins from the First and Third interferon classes may constitute, both individually and in combination, vaccine candidates against proteinopathies inductive of neurodegeneration and blindness, though more evidence may need to be obtained to either confirm or infirm such propositions. Given the fact that both protollin and interferon glycoproteins that are part of the first and third classes of interferons share a double-edged sword-like nature, it remains statistically probable that a low dosage of Type I and Type III Interferons constitute major immune system-based vaccine candidates that may deserve more generalistic clinical testing, perhaps even in combination with a low dosage of the protollin substance itself. In case protollin does not cross the threshold levels of safety and efficacy following further, a low dosage of human recombinant Type I and Type III Interferons and adaptive lymphocytes - perhaps treated with a low dose of such IFN glycoproteins themselves beforehand, could still represent an outstanding immunological candidate helping delay the onset of degenerative proteinopathies, and would additionally still likely constitute an immunising approach against both proteinopathies and infectious diseases caused by various polymorphic microbes. It remains essential to stick to all scientific data that bring a p-value testing of null hypothesis lower than 0.05 by means of outfiltering any pieces of data that are based on erroneous information. Given the fact that the discussion covers the topic of immune stimulation and modulation, it may be important to emphasise upon a detailed assembly of structural and functional trees that covers host immune systems, the evolutionary past of microbes causative of significant infectious disease and the history of the overall battle between host immunity and such microbial agents, in order to reach the utmost resolution of the immune context, which could in turn significantly aid in the innovation of specific immunotherapeutic agents to considerably delay the onset of degenerative toxic protein-related diseases in the long run. Such efforts, combined with artificial intelligence-based catalysis of statistical projections regarding the efficacy rate of such proposed prophylactic and potentially immunising methods, could ultimately aid in the delay of a considerable proportion of proteinopathy cases, for a considerable extent of time, which could overall represent an important first-step in the combat of such clinical diseases currently lacking an effective symptomatic treatment, let alone a cure.

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