TPM Vol. 32, No. S6, 2025
ISSN: 1972-6325
https://www.tpmap.org/

THE POTENTIAL OF MARINE COMPOUNDS IN NEURODEGENERATIVE DISEASE TREATMENT

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Abstract
Clinical trials with the drug candidates that inhibit protein aggregation are in progress. Disease progression can be prevented by use of these drugs. These drug candidates hold the promise to substantially reduce the occurrence of such diseases if given pre-symptomatically Prevalence of neurodegenerative diseases is increasing with increase in the age of the population. Due to their multi systemic nature several challenges have occurred for the potential treatment of neurodegenerative diseases. The existing drug repositioning research on Neurogenerative therapeutics integrated an integrative strategy of a number of computational repositioning approaches. Network-based analysis was conducted to model the complex interactions between biological molecules and pathways to identify probable drug targets for both the diseases of our concern. Specifically, we constructed protein-protein interaction networks and signalling/regulatory pathways associated with the pathology through a variety of pathway databases and protein-protein interaction resources. By network topology analysis, we found proteins which can potentially function as key intervention points upon perturbation by drug compounds.
Keywords: Marine, Compounds, Neurodegenerative, Disease, Treatment.

1. INTRODUCTION

Human neurodegenerative disorders form a heterogeneous group of intractable and debilitating conditions associated with progressive loss and dysfunction of identified neuronal populations eventually resulting in cognitive, behavioural and physical impairment. Neurodegenerative conditions are divided on the basis of clinical presentation, e.g. movement disorders (e.g. Parkinson's (PD), Huntington's (HD), motor neuron diseases (MND)) and dementias (e.g. Alzheimer's disease (AD), fronto-temporal dementia (FTD)); neurons affected, e.g. dopaminergic (PD), GABAergic (HD) or motor neurons (MND); mode of origin (familial or sporadic) [2]. Age is one of the most powerful determinants for the development and progression of neurodegenerative disease onset that are a new socio-economic burden in countries whose populations have increasing life expectancy [1]. Over 10 million people suffer from neurodegenerative diseases each year, and this figure will grow by 20 % within the next 10 years [9]. The polyglutamine (polyQ) disorders form a family of at least 9 most common inherited neurodegenerative disorders that include six Spinocerebellar ataxias (SCAs), spinal and bulbar muscular atrophy

(SBMA), dentatorubral-pallidoluysian (DRPLA) and Huntington's disease (HD) [3]. The polyQ disorders are autosomal dominant disorders, except for SBMA which is X-linked in inheritance and involves the male hormone receptor. The etiological mutation of polyQ diseases is an unstable CAG trinucleotide repeat expansion in the specific disease gene. [17]. Local concentration amplification of protein that catalyzes the protein fibril nucleation results as a consequence of protein adsorption at the nanoparticle surface. But the protein fibrillation is inhibited because of the strong binding or high particle/protein surface area [6]. Peptide fibrillation of peptides having higher tendency towards fibril formation is retarded while peptide fibrillation of peptides having lower tendency towards fibril formation is promoted by the same surface with weak monomer. Coincident occurrence of hydrophobic surfaces and hydrodynamic flow has also been shown to increase protein aggregation while the not much effect is found when only one of these two factors is present [10].

2. REVIEW OF LITERATURE

Severe neurodegenerative disorders like Alzheimer's disease (AD) and Parkinson's disease (PD) are marked by the misfolding and aggregation of amyloid-beta (Aβ) and alpha-synuclein (αsynuclein) proteins into oligomeric and fibrillar pathogenic forms [13]. Aggregation of proteins may trigger downstream phenomena like oxidative stress, mitochondrial dysfunction, inflammation, and cellular transport defects that in turn contribute to neurodegeneration [4]. Understanding the mechanisms of protein misfolding and cell-to-cell transmission could enable the design of specific therapies that inhibit aggregation or trigger clearance systems. Neuroinflammation due to abnormally activated microglia and astrocytes is a recurrent observation in areas of neurodegeneration in the majority of diseases. Inflammatory mediators like cytokines and chemokines have the capacity to kill or damage neurons directly. Anti-inflammatory drugs that target immune pathways may be able to limit neuronal loss, but to date, trials have only been partially successful, indicating that inflammation may be both cause and effect. Moreover, mitochondrial damage and impaired energy production are also involved in most neurodegenerative diseases. Disease proteins are able to directly impair mitochondrial membranes or dynamics. Oxidative damage and calcium dysregulation likewise damage mitochondria with time, causing neuronal dysfunction [5].

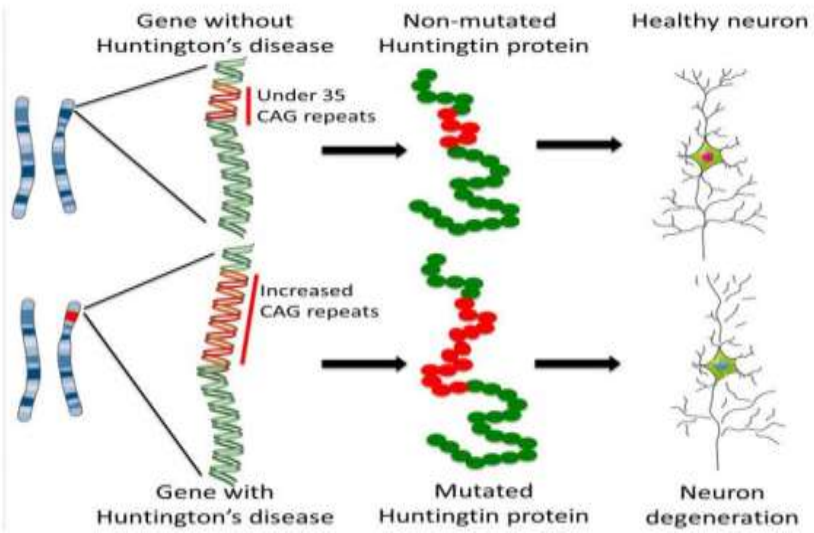


Figure 1: Schematic representation of the formation of mutant Huntingtin protein

The drug development process for AD and PD has been beset by a high failure rate in clinical trials, primarily due to safety concerns or absence of therapeutic effect. It is a costly and time-consuming effort to design new drugs from scratch to address each of these targets in isolation. Traditional drug discovery is a long process that lasts 10-15 years from discovery to approval and costs over $2 billion on average. Discovery of drug compound (2-3 years), screening assays (0-1 years), lead optimization (1-3 years), ADMET profiling and safety reviews (1-2 years) and drug development and clinical trials (5-6 years), and finally registration and licensing (1-2 years) of the new drug in the market [18]. This is an unpredictable and risky process since most drug candidates do not pass clinical trials [8]. In contrast, drug repositioning/repurposing i.e., new use of already approved drugs, can provide a cost and time-saving way to bring a new therapy to AD and PD patients. Repositioning relies on existing evidence and clinical knowledge, thus facilitating the interrogation of compounds with documented safety profiles and the discovery of drugs that specifically act against molecular targets of disease, which may result in discovering a drug less toxic and more targeted in its action [11].

3. MATERIALS AND METHODS

Amyloid hypothesis' can be applied to describe the formation of amyloid aggregates or senile plaques at extracellular space of neuronal network leading to pathologic process by activating various molecular entities and disrupting the cellular homeostasis terminal staging the neuronal death. Senile plaque process is actually triggered by amyloid beta (Aβ) monomers that are produced due to cleaved APP. It is a glycosylated N-terminus extending to extracellular neuron cellular space and C-terminus towards cytoplasmic core integral membrane. APP isoforms vary from 695-770 amino acids and possesses a function of regulating synaptic formation, repair and iron transport.

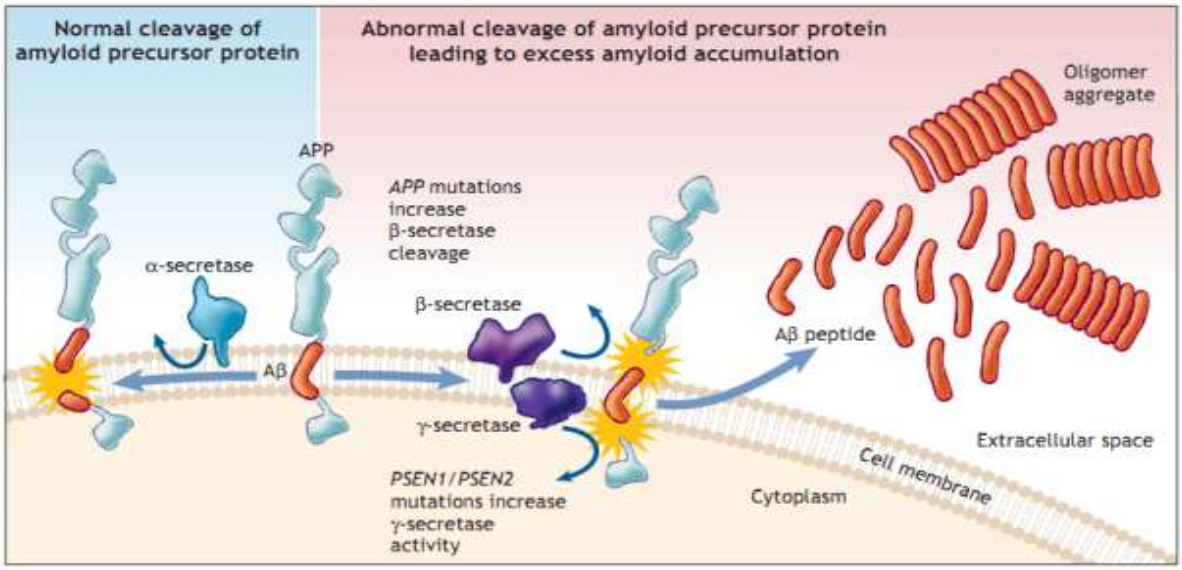


Figure 2: proposed diagram

Formation of Aβ monomers with specification of length numbers of amino acid chains formed on cleavage of CTF99 by γ-secretase [15]. Aβ monomers vary in length 43, 45, 46, 48, 49, and 51 which are later cleaved into toxic forms 40 and 42 and are the reason for formation of varying monomers on account of γ-secretase as it can cleave at more than a single site [14]. These monomers also exhibit aggregation pattern further developing to higher order structure responsible for the senile plaques' formation. The growth cycle includes lag, log, growth or elongation and stationary phase [12]. The structure evolves from oligomer formation, protofibrils, and matured fibrils eventually culminating to give rise to the senile plaques that form the Aβ pools. Fig. 2 symbolizing process of amyloid aggregation prolongation gives an insight into how the single strand peptide structures the aggregation. Aβ oligomers are soluble in nature, they are generated by the monomers which become aggregated to form dimers, trimers, tetramers, pentamers, up to decamers [7].

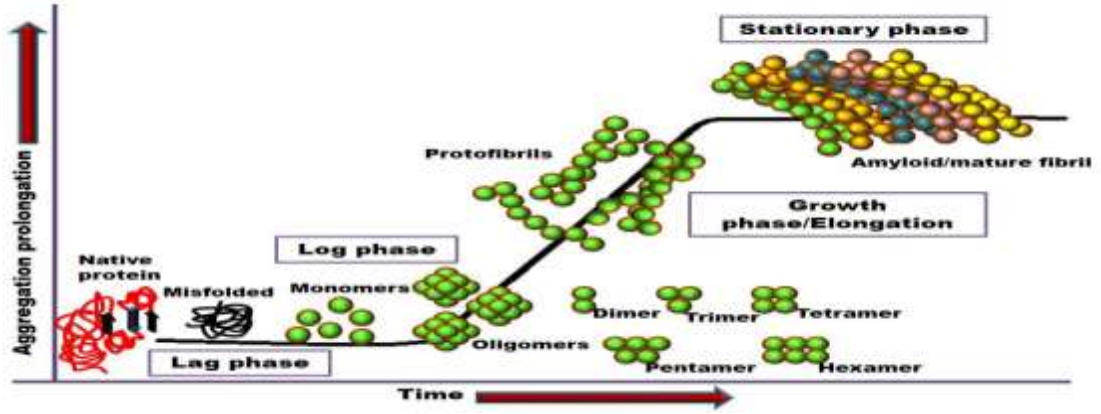


Figure 3: Representation of amyloid beta aggregation formation

Drug repositioning relies on a number of computational approaches such as signature matching, machine learning models, network-based, literature-mining, and structure-based investigations. Signature matching compares drug molecular signatures with disease molecular signatures to find possible drug-disease associations.

4. RESULT AND DISCUSSION

The development of transgenic rodent models of research that express some of the pathological characteristics of AD has given a tremendous boost to the process of drug discovery, and has also raised several intriguing questions about the disease process per se. One must never forget, however, the potential dangers of uncritical translation from mouse/rat to human. The fact that no animal model at present replicates all of the human AD features reflects the constraints involved with modelling a condition in humans which takes decades to develop and overwhelmingly presents as a higher cognitive dysfunction [16].

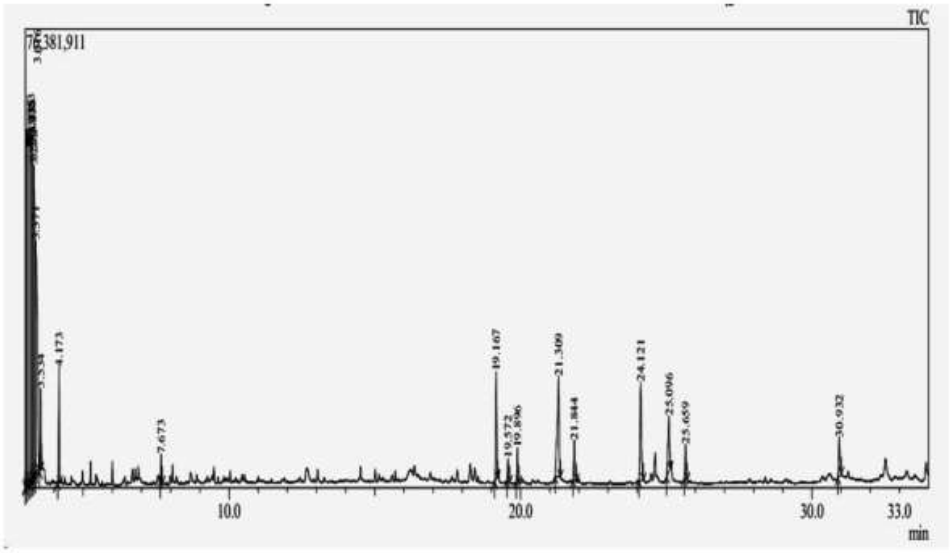


Figure 4: GC-MS analysis of M. neriifolia ethanolic leaf extract

The technology has to develop in many new forms every day in the health sector. The length of human lives extends to the scope such as biotechnology, pharmaceuticals, information technology, medical device and equipment development.

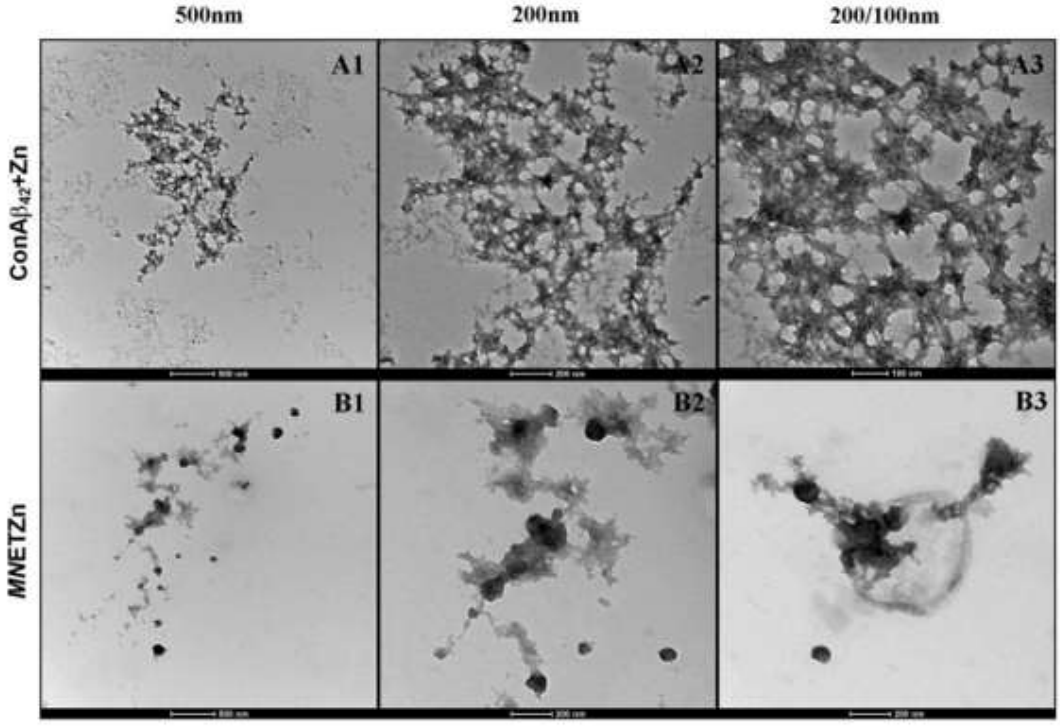
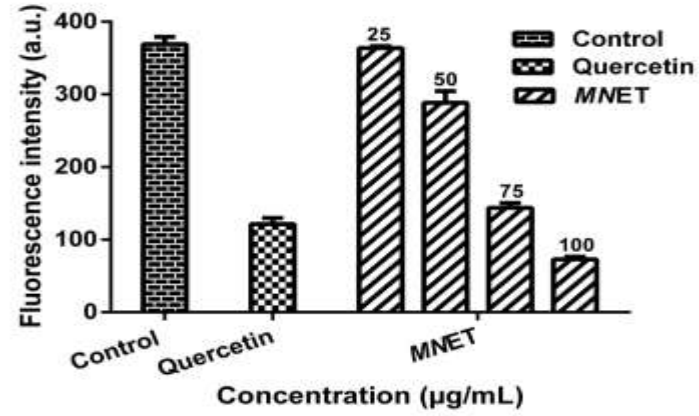


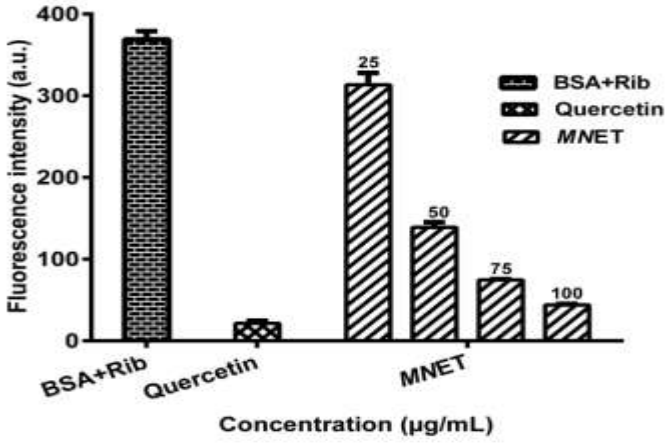
Figure 5: Control assay

The development of the healthcare field is astounding ranging from small advancement like adhesive plaster bandages and ankle braces to large the likes of MRI technology, artificial limbs, and limbs with robots. The lengthiness of life as well as cure process is the main factor to remember in health care technology growth. The

development in technology helps to detect the disease at the earliest stage and provides treatment accordingly. The scientists, research workers, and medical interns are creating new technologies and diagnosis techniques to rescue the disease-afflicted people from uncured diseases and also in testing and identifying the new medicines/drugs that help to cure the disease.



(a)



(b)

Figure 6: Performance comparison of Feature Extraction Techniques

Medical technology is the different procedures, processes, and equipment utilised in medical practice. These could include treatments and diagnostic methods in clinics and hospitals. Also included are medications and devices for treatment and diagnosis. Medical technology has offered tremendous benefits to physicians and patients alike. Technology has also helped in aiding people in developing machines and equipment that effectively diagnose the illness of the patient. This has eliminated routine procedures like unnecessary physical checkup and evaluation in an attempt to determine the true disease of the patient. Physicians no longer need to take the traditional palpation routine and naked-eye viewing. They use extremely advanced machinery in an attempt to avoid human error and good evaluation of patients.

5. CONCLUSION

In the human body, the brain is the main element in the functioning of the system. If destroyed, then the person is as good as dead. Due to some disorders, the human brain gets exposed to more diseases. In the 1:6 ratio of the population, the person is suffering mostly from the brain-related problems. The disorder or brain injury of the one of brain tissues, neurons and nerves is the neurodegenerative diseases. It's those diseases which upset the communication of the brain with other organs of the human body. Having all this in consideration ameliorate results MNET is an efficient natural source for excavating the drug moieties that may possess the drug like properties. Possible phytochemicals and they are in great demand that may be a possible candidature for the treatment of AD. This determines the result MNET has good antioxidant activity and polyphenolic content.

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