NEURODEGENERATIVE TREATMENT

Revolutionizing Neurodegenerative Disease Treatment: Unlocking the Power of mRNA

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Treatment Introduction

We have compiled and listed a provisional (through Reinhold Cohn & Partners) six short sequences of mRNA sequences, designated to treat neurodegenerative diseases such as Parkinson ,ALS, Alzheimer's and Multiple Sclerosis by intramuscular (IM) injection of formulation.

Each such mRNA sequence is an Amino Acid comprised of 4 Amino Acids in addition to an opening and closing Codons, totaling at 6 codons altogether.

Unlike existing treatments, the treatment is not only designated to slow the progression of the degeneration but has potential to reverse the process by stimulating the immune system and resume the autophagy of the misfolded proteins – resulting in cellular regeneration.

Executive summary

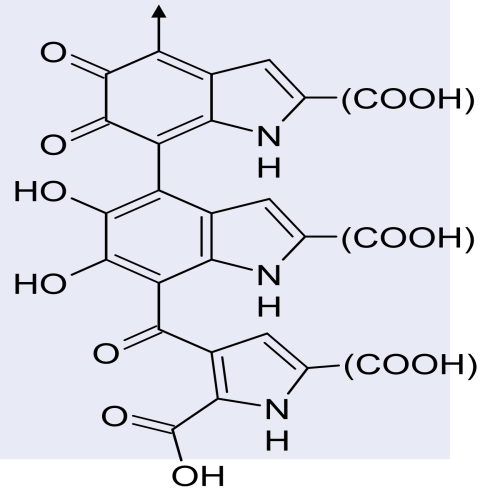
After investing over two decades researching the human body, focusing on brain and nerve system,

Ilana Rogel has compiled six short sequences interwoven in genes across all chromosomes.

These sequences stand out due to their unique vibration, distinct from the rest of the genome.

These sequences are involved in activating the melanin pigment in all its forms:

EUMELANIN, PHEOMELANIN & NEUROMELANIN

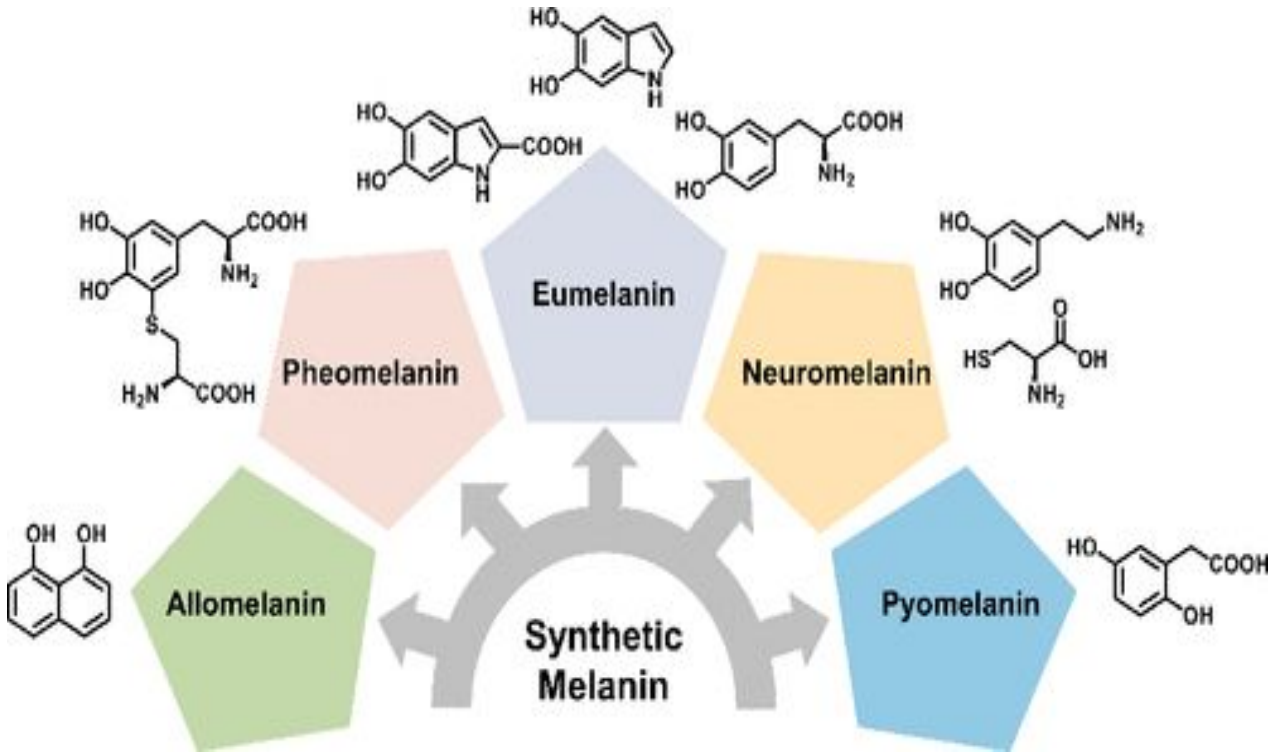


Executive summary

These sequences are dominant during the first 40 days of gestation and remain physiologically relevant throughout one's life.
Melanin, as a pigment, is responsible over various directed response types, motor skills, types of movements (facultative), and more.

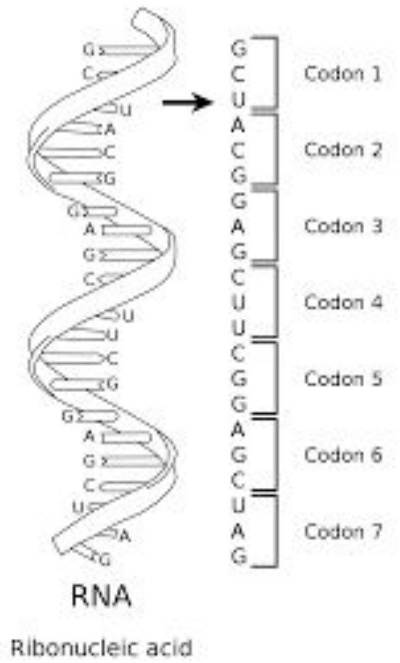
Melanin affects the absorption and emission of photon that stimulate electrons which in turn create reactions in various proteins composed of amino acids.

Neural crest is the origin of melanin-containing cells (melanocytes).
Melanin's is synthesized from the aromatic amino acid: **TYROSINE**.



American Chemical Society

Decoding Genomic Sequences: Understanding Codon Variability



Our hereditary material is composed of 4 letters, 4 molecules called nucleotides represented in the DNA: **Thymine (T)**, **Adenine (A)**, **Cytosine (C)**, and **Guanine (G)**.

DNA consists of 2 strands when represented the genome in stable state.

In RNA, **Thymine** (T) is replaced with **Uracil** (U).

RNA serves as an intermediate stage between DNA and protein synthesis, representing a variable state. Therefore, the lifespan of an RNA molecule is short until protein synthesis occurs, at which point it perishes.

Each three letters in the genome (out of 4 possibilities) are called 'codon'.

In the genome, there are 64 codons representing 20 amino acids which represent the basic structural units of proteins.

In each codon, the first 2 letters are identical and chemically represent the same amino acid.

The 3 letter, on the other hand, can end in one of 4 possibilities: **U, C, A, or G.**

Decoding Genomic Sequences: Understanding Codon Variability

Thus, for a single amino acid, there can be multiple expression possibilities depending on the third letter.

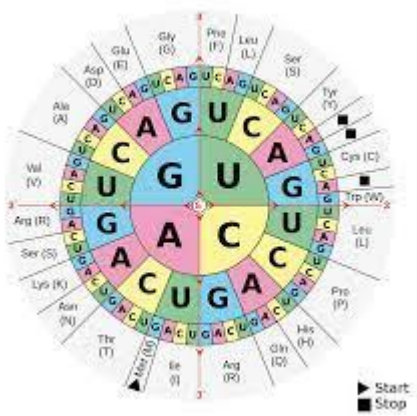
Until recently, it was widely assumed that the third nucleotide was not significant and did not change the protein sequence carrying no impact on gene function in the genome.

My research demonstrates that changing the third letter of the codon, although encodes the same amino acid, can lead to mutations that result in various brain pathologies.

This research sheds light on the role of melanin pigment and its implications on brain functions.

As such, the research may lead to groundbreaking advancements in our understanding of Melanin's role in neurological diseases.

Silent Mutations:
The Foundation of Our Innovative mRNA Sequences



Research Findings:

- A study published in the journal Nature* (June 2022) found that a significant portion of “silent” mutations had a substantial impact on an organism's survival and adaptation.
- About 75.9% of “silent” mutations caused significant harm to the survival of yeast strains, challenging the previous notion of their neutrality.
- Silent mutations, once considered inconsequential, can influence protein production and cellular function. This research highlights the importance of researching silent mutations, especially in the context of auto-immune and neurogenerative diseases.

* https://www.nature.com/articles/s41586-022-04823-w

Unlocking the Potential: Harnessing Silent Mutations in Our mRNA Sequences

- Our research demonstrates that when the third nucleotide in the codon is substituted with a different nucleotide, even though it results in the same amino acid, leads to **mutations associated with various brain pathologies**.
- It appears that the third nucleotide in the codon has a long-range impact on the types of melanin crucial for gene expression, through fluorescence spectroscopy – a chemical "brightness" with a distinct emission from the full spectrum.
- The clear perception is that as a result of different radiation absorption and emission, there is a prominent difference in protein folding, and response to charged molecules and ions such as Sodium, Potassium, Chloride, Calcium, and more. All reactions also manifest in the water molecule.
- The difference between a normal state and a mutation is expressed in the misfolding of proteins, which impairs protein stability, its specific function, and, most importantly, affects communication modes and neural signals.

Unlocking the Potential: Harnessing Silent Mutations in Our mRNA Sequences

- Misfolding does not allow for 'hybrid resonance,' creating resonance while interacting critically for the formation of equilibrium.
- Misfolding leads, among other things, to pathologies that ultimately result in the collapsed destruction of nerve cells in the brain and spinal cord.
- Our research opens new paths for understanding the crucial role of genetic mutations and protein folding in brain health and disease. We believe that our findings holds the potential to revolutionize the field of biotechnology and lead to innovative therapies for neurological disorders.

Impact of Misfolded Proteins on Neurological Disorders

In the world of genetics and neurobiology, the distinction between a properly folded protein and a misfolded one carries profound consequences. Misfolded proteins can disrupt the stability, intended function, and, most notably, interfere with various forms of cellular communication and neural signaling.

Misfolding Hinders Crucial Processes:

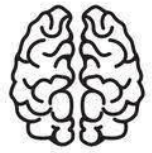
1. **Loss of Structural Integrity:** Misfolded proteins lack the structural stability essential for their proper function. This instability can result in a cascade of cellular dysfunction.
2. **Impaired Interactive Dynamics:** Incorrectly folded proteins fail to facilitate essential interactions required for equilibrium and balance.
3. **Neurological Damage:** Misfolded proteins are implicated in several pathological conditions.

Impact of Misfolded Proteins on Neurological Disorders

1. **Beta-Amyloid Plaques:** Misfolding of proteins, particularly Beta-Amyloid, leads to the formation of toxic aggregates called plaques, a hallmark of conditions like Alzheimer's disease.
2. **APO E4-Related Cognitive Impairment:** Molecular-level disruptions associated with the APO E4 gene variant lead to cognitive deficits and are linked to neurodegenerative diseases.
3. **TAU Protein Disfunction:** In neurodegenerative disorders, such as Alzheimer's, the Tau protein misfolds, forming twisted tangles (Tangles), disrupting neuronal connections.
   In turn the tangles damage the Axonal Axons, responsible for inter-neuronal communication in the brain which can lead to severe damage in neural circuits.

Our research aims to shed light on the intricate relationship between genetic mutations, misfolded proteins, and neurological disorders, paving the way for innovative treatments and improved patient outcomes.

Cutting-Edge Experiment with Organoids



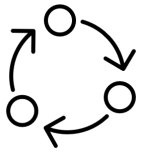
Our current experiment utilizes organoids as a powerful tool.
Organoids are miniature, organ-like structures that closely resemble the original organs.
They are generated from tissue-specific cells using stem cells with self-renewal and defined differentiation capabilities.

In 2019, groundbreaking research successfully cultivated human brain organoids. These organoids represent three-dimensional microanatomy and function closely approximating the original organ.
An additional study recently published in the journal NATURE, provides a breakthrough in proving the mutation at the third nucleotide carries immense weight (June 2022).
These two elements, organoids and the proof of the third nucleotide, accelerate our research on melanin pigment and its implications for the future of medicine and human ability to cure neuro-degenerative conditions.

The investigation of melanin pigment pathways towards known pathologies using organoids that are not subject to limiting regulations.

Proof of Concept Milestones

Synthesis of the 6 Amino Acid short mRNA sequences.



Stabilization of the mRNA sequences to be used for cellular culture and IM injection in Organoid (preparation of mRNA sequences for cellular penetration)

IM Injection of the protein to an infected cellular culture.

We are looking for Pre-Seed partners able to support and help facilitate the POC.

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