length of its polypeptide chain varies slightly: from 252 amino acid residues in a rabbit to 253-254 in human. The presence of a 22-membered signal sequence at the N-terminal region of the PrPc protein molecule provides cotranslational transfer of the newly formed polypeptide chain across the membrane of the endoplasmic reticulum. When the polypeptide chain passes through the channel (SEC61) in the rough ER membrane wall, the leader signal sequence is removed, as well as the folding of the molecule into a globule and its covalent modification. Two N-glycosylation sites of the sequense Asn-Ile-Thr and Asn-Phe-Thr are located at amino acid residues 181 and 197 respectively (human prion protein). The distribution of di-, mono-, and non-glycosylated forms of prion can vary both in different organisms of the same species, and within the same organism. Glycosylated forms of the protein are also diverse, which makes it possible to isolate about 400 different PrPc glycoforms. Natural and induced mutations in the hydrophobic sequence located in the middle part of the molecule, near it, as well as in the region of the N-terminal region, lead to an increase in the proportion of CtmPrP, which causes neurodegenerative diseases. A number of facts have been established that indicate the influence of the degree of glycosylation of prion proteins on the efficiency of prion disease transmission, as well as on the formation process of various strains of the prion pathogen. To date, more than 30 possible mutations of the prion protein have been described, of which most are reliably associated with hereditary prion diseases in human. Together with the infectious isoform, they do not exceed 10-20% of all reported cases of prion diseases, the rest are the sporadic form of Creutzfeldt-Jakob disease (neurodegenerative disease of spongiform encephalopathy in humans).

Thus, prion diseases are a group of neurodegenerative diseases of animals and humans caused by infectious isoforms of one of the host proteins called prion (PrP) and encoded by the cellular genome. Currently, the term PrP is used both to denote the isoform of a protein formed during normal cell metabolism (PrPc) and its pathological (infectious) isoform (PrPd), which causes prion diseases in humans and various animal species.

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NEW APPROACHES IN DIAGNOSIS OF CYSTIC FIBROSIS

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Cystic fibrosis (CF) is an autosomal recessive disorder caused by the mutation of a gene located on the long arm of chromosome 7 that encodes for a protein of 1480 amino acids, the cystic fibrosis transmembrane conductance regulator (CFTR), which works as a chloride channel on the apical membrane of epithelial cells. This mutation results in a change of the viscosity of secretions, and the production of thick mucus that leads to malabsorption, loss of electrolytes in sweat, and alteration of pulmonary secretions. As we know along with impairment of sweat glands and mucous glands the patient also suffers from various allergic bronchopulmonary infections such as aspergillosis and are at a high risk of pneumothorax.

Purpose of the study - to find out the most prevalent methods used to diagnose Cystic fibrosis by deep surfing scientific internet sources. To explain the main diagnostic that are used for Cystic fibrosis along with their importance, methods, and comparison between their cost and effectiveness.

Immunoreactive trypsinogen (IRT) test is the Newborn Screening technique used to diagnose cystic fibrosis in newborns. This test is done by taking blood sample by pricking the heel of the infant. If the level of IRT is not abnormal, then it is possible that the newborn is not suffering from CF. But, if the infant shows signs and symptoms consistent with CF, other tests for cystic fibrosis, such as sweat chloride or CF gene mutation testing, can be considered. Such as Sweat chloride test. It is well known that level of chloride in sweat is high in patients with CF. It happens due to defective chloride transport. The sweat test detects the level of chloride that is excreted in sweat. It is used as a diagnostic technique for CF. A chloride level of more or equal to 60 mmol/L is likely to be diagnosed with CF. Next test that is commonly used for CF is a Sputum test. Patients with CF frequently suffers from respiratory infections, caused by bacteria or fungi. A

sputum/mucus CF respiratory screen or culture helps doctors to diagnose and identify these bacteria or fungi so they can use the most effective antibiotics to cure a specific infection. An additional method of CF is the X-Ray with a small dose of ionizing radiation. It helps to evaluate dilated airways, which contains mucus,and also to detect lung infections that can be treated with antibiotics. Chest x-rays are used regularly to observe changes in patients with cystic fibrosis and detect other respiratory conditions such as pneumonia and collapsed lung. To discover more detailed lung picture we can use CT scanning. Chest CT scans can show both mucus and bronchiectasis that may specify infection, inflammation, and potential lung damage. Normally, sinuses are filled with air and appear black in CT scans but in patients with CF, the sinuses can be filled completely with mucus and appear white or grey in a sinus CT scan. To find out how lungs are working it is useful to apply Pulmonary function tests (PFTs). PFTs are non-invasive tests that measures Rates of flow and gas exchange and Lung volume and lung capacity.

We can observe that each of these methods are equally important when it comes to diagnose cystic fibrosis. The patients and parents can experience anxiety due to extra testing, the waiting associated with it and the difficulty in explaining the results. All these methods offer great hopes for the patient if the patient is diagnosed early before any irreversible damage is done.

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USING OF MELATONIN AS A POTENTIAL COVID-19 PROPHYLAXIS METHOD

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One of the most terrible occurrences in recent history is the current COVID-19 pandemic. The elderly people and those with chronic inflammations are more likely to die as a result of coronavirus infection. As a result, it's critical to figure out how to boost vulnerable groups coronavirus resistance. The goal of this study is to look into the possibilities of using melatonin as a medicine to assist reduce the number of deaths and boost the body's immune system.

Melatonin (N-acetyl-5-methoxytryptamine) is a bioactive molecule that has been used to treat sleep disorders, delirium, atherosclerosis, respiratory disease, and viral infections.

Recent studies show that the effect of SARS-CoV on humans is clearly age-related. Obviously, the increased sensitivity to coronavirus in the elderly is due to their reduced levels of melatonin. Daily variations in melatonin in young people (age 26+/-2 years) were in the region of 7 pg/ml, and in people aged 84+/-2 years, the level of melatonin dropped to 2 pg/ml. So, the application of melatonin may partially alleviate age-related comorbidities exacerbating SARS-CoV-2 infection and increasing its risk.

Viral respiratory infections are associated with oxidative stress characterized by elevated levels of reactive oxygen (ROS) and/or nitrogen species (RNS). SARS-CoV induces oxidative stress; oxidative stress induces the expression of PLA2G2D phospholipase; higher expression of PLA2G2D reduces anti-viral immunity, making the virus more lethal. Melatonin possesses high antioxidant properties. It binds up to 10 free radicals per molecule, while such classic antioxidants as vitamins C and E bind just one. Also, melatonin has a high bioavailability, penetrating blood-brain barrier and placenta.

The COVID-19 societal crisis has led to massive and prolonged stress, anxiety and sleep deprivation, which shall become a subject of systemic scientific analysis. These evident factors may have a significant negative impact on people's immune systems and their ability to combat COVID-19 and other illnesses. Like the neuroendocrine system, the immune system has its own set of rhythms. Melatonin's nightly release, for example, is timed to coincide with the peak of progenitor cell proliferation before they are differentiated into granulocytes and macrophages.

Based on circadian cycles, phagocyte activity rises in tandem with the nocturnal peak of melatonin. Long-term sleep deprivation and/or chronic stress impair immune function by disrupting barrier mechanisms, suppressing phagocytosis, reducing proliferation and activity of some leukocytes, particularly CD^{4+} T cells, while increasing T-suppressors and raising oxidative stress and pro-inflammatory background, as well as increasing oxidative stress and pro-inflammatory