Perspective

Patients with respiratory problems receiving glucome are evaluated

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ABSTRACT

Glycomedicine, a new field of medicine, is now beginning to demonstrate the significance of carbohydrates and their interactions with macromolecules in the pathophysiology of numerous diseases. Their contributions to the emergence of respiratory system illnesses are not an exception. In this review, we sought to ascertain the most recent theories regarding the roles of glycans, glycation products, and their receptors in the pathogenesis of diseases affecting the respiratory system, as well as the potential applications of these biological molecules as therapeutic targets for the treatment of respiratory diseases.

Research in the field of glycoscience demonstrates that glycan's are crucial to the development of respiratory system disorders. Therefore, levels of AGE and their receptors (RAGE) are separate risk factors for illnesses of the respiratory system.

Key Words: Glycan's; Undernutrition; *Pleural disease; Interventional pulmonology*

INTRODUCTION

Glycome is a comprehensive collection of an organism's carbohydrates that includes both complex glycan macromolecules and free glycans like hyaluronic acid. Proteins, lipids, and nucleic acids that have been altered by interactions with sugars or that have formed complexes with them are also of interest to glycoscience. Glycation is a non-enzymatic post-translational change of biological components that interact with carbs. On the other hand, enzymes participate in the glycosylation process. The initial description of non-enzymatic glycosylation of proteins used the example of glycated hemoglobin found in the blood of healthy individuals. The greatest research has been done on this kind of glycated protein, both in healthy individuals and in diverse diseases and disorders. Glycation of proteins has historically been linked to diabetes. Advanced Glycation End products (AGE) have been described as having a significant impact on the etiology of cardiovascular, renal, and retinal damage in diabetes mellitus. N-glycan profiles can be used to track biochemical changes in persons with concomitant pathology and type diabetes mellitus.

Plasma N-glycan profiling can forecast the emergence of the metabolic syndrome and a less-than-ideal state of health. However, there is growing evidence that protein glycation plays a significant role in the development of other disorders that do not involve hyperglycemia. While AGEs directly harm joint tissues, glycated albumins lose their antioxidant function and have a role in the development of rheumatoid arthritis. Paraoxanase-1's activity is inhibited by glycation, which raises the risk of arterial hypertension, thrombosis, and the development of atheromatous plaques. It has been consistently demonstrated that the degree of AGE accumulation in tissues closely correlates with the severity of carotid atherosclerosis. Age, gender, diabetes, or metabolic syndrome have no bearing on this variable. Another study has found a link between the rate of AGEs buildup and the risk of age-related macular degeneration. Thus, the degree of protein glycation can serve as a predictor for cardiovascular disorders. However, because there are differences in this area between researchers, it is unclear in which circumstances the degree of AGEs'

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accumulation connects with other risk factors. The accumulation of amyloid caused by protein glycation can increase the risk of amyloidosis and neurological disorders. The accumulation of amyloid caused by protein glycation can increase the risk of amyloidosis and neurological disorders. The interaction of proteins with glyoxal and the subsequent non-enzymatic glycosylation of those proteins increases the production of amylin (IAPP), which is a key factor in the onset of type II diabetes. Immunoglobulins (IgG, IgM, IgA, IgD, and IgE) interact with carbohydrates in a variety of ways that result in irreversible alterations. IgM is twice as vulnerable to these changes as IgG. Since the Fc fragments of altered immunoglobulins suffer modifications that prevent immunocompetent cells from recognizing them, their functional activity declines. Specific alterations in the IgG N-glycan profile have been linked to the onset of dementia and diabetic retinopathy and can be utilized as indicators of these disorders. In general, aging and changes in N-glycan structure are related. Senescence monitoring in real-time is possible using this knowledge. Additionally, among persons aged and older, low and moderate physical activity had a better impact on cognitive performance than severe physical activity. Protein structural degradation, aggregation, and oligomerization are caused by AGE transformation. Cell lesions and death are unavoidably brought on by these mechanisms. Glycation eventually led to senescence and the emergence of numerous age-related illnesses. The glycation of proteins is a broad process that is pathogenetically linked to the progression of illnesses affecting numerous organ systems. Diseases of the lungs are not an exception. A non-invasive marker that corresponds to the degree of AGE deposition in tissues is skin autofluorescence. This index is evaluated by researchers using the AGE-Reader equipment. This tool exposes skin to UV rays, causing autofluorescence. When glycation-end products glow in spectral diapason, this indicator can be used. In a group of individuals with normal spirometry who have no diseases, a poor health status, or clinical conditions that can impair respiratory function, there is a direct correlation between smoker index, age, systolic blood pressure, the prevalence of arterial hypertension, and the degree of AGE-accumulation in the skin. The intensity of skin autofluorescence was inversely associated with both the FEV1/FVC (Tiffanie index) and the estimated glomerular filtration rate at the same time. The findings of the research discussed to support the hypothesis that the degree of glycation of proteins and other macromolecules in reasonably healthy individuals is a significant marker of cardiovascular diseases and a predictor of respiratory ailments. The number of AGE-metabolites in healthy non-smokers' tissues is inversely correlated with their degree of physical activity (greater than minutes per day for at least three days per week). It's also significant that newborns fed infant formula show skin autofluorescence that is more intense than those who were breastfed. Another study's findings on the buildup of AGE in a healthy Chinese population confirm that smokers have more of these metabolites than non-smokers do. Age and skin tone affect the growth of autofluorescence intensity.

Additional research is required to confirm the comparability of these indicators in the Caucasian population as well as the degree to which skin tone affects test results with the AGE-Reader and other comparable devices. Variables were done using the Chi-square test. Numerous investigations have demonstrated a strong correlation between AGE buildup and a decline in lung parenchyma function. This fact has been seen in both COPD patients and smokers. This observation appears to be related to the systemic inflammation that accompanies COPD and causes oxidative damage as well as the activation of the inflammatory cascade. Chronic systemic inflammation is brought on by AGE products adhering to the right receptors (receptors for AGE e RAGE). When these receptors are activated, nitro chemicals, active oxygen radicals, and pro-inflammatory cytokines are released. Immunoglobulins are AGE receptors that are found on the surface of cells. These immunoglobulins have a broad spectrum of ligand recognition capabilities. Even in healthy individuals, small levels of RAGE are expressed in a variety of tissues. It appears that more research needs to be done on these receptors, which carry out a number of as-yet-unidentified tasks in the lung tissues. Therefore, individuals of the same age and smoking history who have COPD and a lower FEV1 are found to have a considerably higher degree of AGE accumulation and enhanced RAGE expression.

It is significant to notice the existence of a different type of AGE product receptor, namely a soluble receptor for AGE (stage). This receptor is found in plasma and shows signs of being anti-inflammatory. The intensity of the systemic inflammatory response and endothelial dysfunction in respiratory illnesses, primarily COPD and emphysema, as well as tissue levels of RAGE and AGE products, have all been found to be inversely linked with sRAGE levels in several investigations. RAGE was identified by some researchers from lung cancer samples. There is currently no conclusive evidence to support the use of these receptors as immunohistochemistry indicators of lung cancer. Increased RAGE expression is linked to metastasis and a bad prognosis for prostate, rectal, stomach, and breast cancers, according to research, though. The degree of RAGE expression dysregulation that occurs during tumorigenesis is closely correlated with the size and invasiveness of lung cancers. In Lewis adenocarcinoma, high-mobility group box protein 1(*HMGB1*) activation inhibits apoptosis and increases cell growth.

On the other hand, compared to tumors with lower levels of RAGE expression, A549 cells (alveolar adenocarcinoma cells) develop more slowly in lung malignancies with high RAGE expression. However, cancer cell migration and metastasis are also linked to RAGE hyperexpression. RAGE has an impact on this process as well, as rising expression causes levels of valentine and N-cadherin to rise while E-cadherin levels fall. Expression of this type of glycoprotein is usually linked to a higher risk of cancer, metastasis, recurrence, and poor survival. In addition, a link between high RAGE expression and elevated tumor-associated macrophage activity has been discovered. 45 These cells, which make up the tumor microenvironment, also contribute to the development and metastasis of the tumor. Possible targets for antineoplastic therapy include tumor-associated macrophages. Thus, the glycation of macromolecules, AGE buildup, and RAGE expression are the most likely risk factors for the emergence of more malignant tumors. As a result, additional research on RAGE will aid in predicting and validating risk in cancer cases based on the levels of RAGE expression. Furthermore, it's expected that by modifying these characteristics, anticancer therapy will be able to be more effective. RAGE activation boosts the expression of the

MUC5AC (mucin 5AC, oligomer mucus/gel-forming) gene in the airways of mice with toluene diisocyanate-induced asthma. Mucin-5AC production and mucus secretion in the airways are encoded by MUC5AC. This gene's expression is frequently shown to be higher in people with COPD and bronchial asthma. RAGEs' part in the bronchial asthma pathogenesis, however, is still mainly unknown. According to research conducted on laboratory mice, RAGE is one of the main mediators of respiratory tract hypersensitivity, increased mucus secretion, and bronchus remodeling. Additionally, RAGEs were discovered to be implicated in ovalbumin-induced airway inflammation in animal models of asthma, which were used in this work as a substitute for human asthma. RAGE mediates type II immunological inflammation, according to another study, by inducing the release of pulmonary IL-33 in response to allergen exposure. ILC2 buildup is facilitated by IL-33 (type 2 innate lymphoid cells). Thus, in cases of allergic respiratory disorders, the RAGE level strongly correlates with the severity of the inflammatory response, triggering an inflammatory cascade and encouraging the buildup of immune cells in the lung tissues. According to research, people with COVID-19 who have low levels of IgG fucosylation and sialylation start to experience antibody-dependent cellular cytotoxicity (ADCC). 57 Additionally, in patients with severe COVID-19, inadequate sialylation may result in a complement activation pathway independent of lectins. On the other hand, the severity of a coronavirus infection is strongly correlated with abnormally high levels of glycosyltransferase and glycosidase expression in B-cells. It appears that the development of severe forms of COVID-19 and an increase in the activity of ADCC-associated pro-inflammatory cytokines are related to excessive glycosylation and insufficient IgG fucosylation and sialylation processes. By affecting certain aspects of the pathogenesis, it may be feasible to lessen the severity of the course of new coronavirus infection. Therefore, the buildup of AGE products and the degree of RAGE expression in tissues have the potential to be employed as biomarkers for determining the risk, onset, and severity of specific diseases. Despite the increased interest in glycomics, it's crucial to note that many elements of the involvement of glycated products and glycoproteins in the etiology of illnesses are currently understudied.

To achieve widespread incorporation into clinical practice, scientific study in glycobiology must be expanded and intensified. The development of pathological diseases is discussed in the recent scientific literature with regard to N-glycans (glycans linked to asparagine), as well as AGE products and AGE receptors. Thus, it was discovered that N-glycans on the surface of human embryonic stem cells are changed into more developed cellular structures during differentiation.