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EC DIABETES AND METABOLIC RESEARCH
Editorial

Hyperglycemia and Covid-19: State of the Art

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Received: August 25, 2020; **Published:** August 29, 2020

Coronavirus disease infection-2019 (COVID-19) poses a twofold danger of patients with diabetes. Diabetes has been reported as a contributing factor for the seriousness of the condition, but in cases where food intake is lower and more nuanced, patients prefer to regulate glucose [1]. COVID-19 is caused by coronavirus SARS-CoV-2 (severe acute respiratory syndrome coronavirus-2), which has spread exponentially in several countries across the world [2,3]. Older patients and individuals of all ages with severe underlying health problems in conjunction with obesity could be at elevated risk of COVID-19 acute disease. While general obesity is a risk factor for several diseases, many clinical trials have shown that visceral fat accumulation is most strongly linked to specific health conditions, such as cardiovascular disorders, insulin resistance and type 2 diabetes mellitus [3-5]. In addition, effective cardiometabolic monitoring of patients suffering from extreme COVID-19 disease could be appropriate [6].

Not a strategy for COVID-19 but good eating patterns increases immune system function, encourages immunometabolism and is a modifiable factor in the progression of chronic disease strongly associated with COVID-19 deaths [7].

Concentration of glycosylated severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) viral particles in the lung epithelium, concentration of glycosylated angiotensin-converting enzyme receptor 2 (ACE2) in the lung epithelium will describe COVID-19 infection and its severity; the degree and regulation of the pulmonary immune response to the spike protein SARS-CoV-2 at about day 8 to 10 after the start of the symptom and could be linked to both [8]. In COVID-19, SARS-CoV-2 binding of ACE2 also indicates that chronic unregulated hyperglycemia, and not only a history of diabetes mellitus, could be significant in disease pathogenesis [9]. A documented history of diabetes (DM) and ambient hyperglycemia have been reported as independent risk factors for SARS morbidity and mortality [10].

Different clinical findings in SARS and preclinical studies in the NOD diabetic mouse may provide a potential cause for a correlation between hyperglycemia and ACE2 rates in the seriousness of COVID-19 disease [11]. Possible changes in ACE2 glycosylation as well as viral spike protein glycosylation, all likely caused by unregulated hyperglycemia, may alter both the binding of the viral spike protein to ACE2 as well as the degree of immune response to the virus [11]. This indicated a transient hyperglycemia process caused by a transient inflammation of the islet cells of the pancreas by SARS-CoV by linking the SARS-CoV to the ACE2 present on islet cells, resulting in a transient insulin-dependent diabetes mellitus that resolved with disease resolution [11].

Through lowering rates of glycosylated ACE2 concentrations in the intended lung tissue by glycemic regulation, this may likely decrease the amount of glycosylated viral binding sites in the lung and thereby theoretically relieve some of the COVID-19 disease inflammation and symptoms [12]. It further indicates a potential paracrine loop explanation for COVID-19 infection, where the virus infects the pancreas and liver, resulting in glycosylated ACE2 hyperglycemia and upregulation in the liver, and more virus attachment and inflammation [13]. Hence low glycemic regulation may render the disease more serious. This also indicates a potential paracrine loop theory for

COVID-19 infection, where the virus infects the pancreas and lung, contributing to glycosylated ACE2 hyperglycemia and upregulation in the lung and more virus attachment and inflammation. Therefore, weak glycemic regulation could render the disease more serious.

Presumably elevated and abnormally glycosylated ACE2 in the lungs, nasal airways, tongue, and oropharynx in unregulated hyperglycemia may also act as enhanced viral binding sites for SARS-CoV-2 resulting in increased susceptibility to COVID-19 infection and higher seriousness of disease [14]. It is most possible that it is the amount of glycosylated ACE2 receptor and not just the amount of ACE2 itself, that is responsible for the attachment and fusion of the virus [15].

If correct, this supports improved glycemic regulation in patients with pre-diabetes and diabetes as a possible method for preventing the progression of COVID-19 and rising symptom frequency.

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Volume 4 Issue 9 September 2020