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Concentration Levels of Dissolved Angiotensin Converting Enzyme 2 (sACE2) in Children and Adults with and without COVID-19

Olivia Arnold*

Department of Paediatrics, Stanford University School of Medicine, Palo Alto, California, USA

Opinion

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***For Correspondence**

Olivia Arnold, Department of Paediatrics, Stanford University School of Medicine, Palo Alto, California, USA

E-mail: OliviaArnold2@gmail.com

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INTRODUCTION

The coronavirus disease 2019 (COVID-19) pandemic began in December 2019 as a result of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and is to blame for illnesses and deaths worldwide. While there are several risk factors for a severe course of the disease, such as older age, male gender, obesity, and pre-existing illnesses, it is unclear which pathophysiological differences Cause the increased risk. Notably, the disease's mild course in children is notable. The angiotensin converting enzyme 2 is a key enzyme in SARS-CoV-2 infection (ACE2). It is a membrane-bound carboxypeptidase found on the surface of cells in many organs including the kidney, heart, gastrointestinal tract, and lung. ACE2 converts various peptides.

DESCRIPTION

The counter regulatory function of ACE2 to ACE in the renin-angiotensin-aldosterone system is critical (RAAS). ACE converts angiotensin I (AT1) to angiotensin II (AT2), which has a vasoconstrictive effect, whereas ACE2 converts AT1 to Angiotensin-1-9 and AT2 to Angiotensin-1-7, which have vasodilative and anti-inflammatory functions. Soluble ACE2 (sACE2) is formed by the shedding of ACE2 from the membrane by the adisintegrin-like and metalloproteinase 17 (ADAM17). We must distinguish between soluble sACE2 and membrane bound mACE2. The mACE2 protein is primarily responsible for SARS-CoV-2 cell entry. mACE2 interacts with the spike protein, which is found on the virus's surface [1-3]. The virus binds to ACE2 and initiates the fusion to the host cell after priming the spike protein with the transmembrane protease serine subtype 2.

However, the role of sACE2 in COVID-19 infection remains unknown. An in vitro study revealed that SARS-CoV-2 can also enter cells through sACE2. Endocytosis is used to enter the host cell after binding to sACE2.Differences in ACE2 distribution can provide insight into different COVID-19 courses. High sACE2-concentration has been linked to a number of diseases, primarily cardiovascular diseases, such as hypertension, diabetes, obesity, and renal disease, all of which are risk factors for severe COVID-19. As a result, high sACE2 may be a risk factor for severe COVID-19. We found that the concentration of sACE2 in the blood was related to the severity of congenital heart disease and heart failure in adults. During the pandemic, children and young adults typically had mild COVID-19 infections [4,5]. As a result, it is intriguing to investigate the pathophysiological mechanism and role of sACE2 in children as opposed to adults.

The identification of pathophysiological risk factors is important for gaining a better understanding of the infection process, protecting vulnerable groups, and developing COVID-19 therapeutic approaches. We investigated the concentration of sACE2 in children's plasma because ACE2 is a risk factor for SARS-CoV-2 infection. To date, there is little data on sACE2 concentrations in children, and data on sACE2 in children with COVID-19 is unknown. The coronavirus disease 2019 (COVID-19) pandemic is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). There are several risk factors for a severe course, including older age, male gender, and pre-existing illnesses. Pathophysiological risk factors, on the other hand, are largely unknown. Notably, the mildness of the disease in children is noticeable.

CONCLUSION

Angiotensin converting enzyme 2 (ACE2) is a key enzyme in infection and acts as a receptor for SARS-CoV-2. Differences in ACE2 distribution can provide insight into different COVID-19 courses. Our goal was to determine the role of ACE2 as a pathophysiological risk factor by measuring soluble ACE2 (sACE2) via ELISA in blood samples (lithium-heparin-plasma or serum)

from 367 people with and without COVID-19. sACE2-levels were compared between age and gender groups. sACE2-concentrations in adults and children with COVID-19 are significantly higher than in healthy individuals. sACE2-levels rise with age and are lower in children with COVID-19 than in adults. Sex has no discernible effect on sACE2-concentration. It is unknown whether sACE2 concentrations rise as a result of infection and what factors may influence this response. Finally, the increase in sACE2-concentration with age may indicate that ACE2 concentrations correspond to increased COVID-19 severity.

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