

Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active. compared to females had a higher proportion of cells with expression (Supplementary Fig. 2C). However, we caution against the generalizability of these findings due to the confounding variables that may modulate *ACE2* or *TMPRSS2* expression such as smoking and age. It is not clear if *TMPRSS2* and *ACE2* expression is regulated by the same process, but their expression levels are positively correlated in lung cell lines (Supplementary Fig. 3).

In summary, we found a small percentage of prostate hillock and club cells that co-express *TMPRSS2* and *ACE2*. Whether differences in *TMPRSS2* and *ACE2* expression mediate SARS-CoV-2 pathogenesis and whether androgen signaling can affect COVID-19 disease remain to be studied; sex differences in *TMPRSS2* expression alone may not drive the higher burden of SARS-CoV-2 disease among men. Further research into *TMPRSS2* expression and its modulation within the lung and other relevant cell types that may impact *ACE2* and SARS-CoV-2 pathogenesis is needed.

Conflicts of interest: The authors have nothing to disclose.

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## Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j. eururo.2020.04.065.

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# Severe Acute Respiratory Syndrome Coronavirus 2 Infection in Renal Failure Patients: A Potential Covert Source of Infection

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COVID-19, a highly infective disease caused by a newly identified coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2; previously 2019-nCoV), is spreading around the world [1]. Increasing evidence has

confirmed the human-to-human transmission. A special group of COVID-19 patients is comorbid with chronic kidney disease (CKD) [2]. In patients with CKD, innate and adaptive immune function impairment would result in increased

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susceptibility to bacterial and viral infections. Therefore, SARS-CoV-2 infection of these dialysis patients has attracted our attention.

## High risk

As reported in our previous study, 41.30% of COVID-19 patients were likely infected in hospital. Most renal failure patients were immunocompromised due to uremia and needed routine dialysis in hospital two to three times per week. Therefore, renal failure patients should be more susceptible to SARS-CoV-2 than the normal population. We noticed that, as chest computed tomography scans show, approximately 20-30% of dialysis patients were diagnosed with suspected infection in multiple dialysis centers in the early period of COVID-19 outbreak. Intriguingly, most of these suspected cases have no obvious clinical symptoms, including fever, fatigue, and dry cough. However, these asymptomatic patients may be a potential source of infection, transmitting the virus to healthcare providers, their family members, and other patients. Therefore, SARS-CoV-2 infection in dialysis patients should be taken more seriously due to a high risk of asymptomatic transmission.

## Diagnosis

Healthcare-associated pneumonia is a common infectious problem encountered in hemodialysis patients [3,4]. Moreover, many dialysis patients suffer concurrently from pulmonary edema, which may mimic pneumonia, in terms of both presenting with an abnormal chest radiograph and producing a similar clinical symptom. Nucleic acid test could provide a valuable support, but the sensitivity of the current nucleic acid tests needs to be improved. Given all of the above, the precise diagnosis of COVID-19 infection is more difficult in dialysis patients than in the normal population.

## Treatment

Another concern is the pharmacokinetics in these renal failure patients. Many antiviral drugs are eliminated by hepatic metabolism and renal excretion. There are few studies on the pharmacokinetics of antiviral drugs in patients with renal dysfunction. In addition, hemodialysis could take away some drugs and reduce their concentration in the blood. Taken together, an adjusted or even supplemental dose of those antiviral drugs should be administered.

In addition, a previous study has indicated that renal dysfunction in patients with pneumonia is associated with a risk of severe infection [5]. A higher mortality rate should be observed in renal failure patients infected with the coronavirus. Indeed, SARS-CoV-2 infection can induce acute renal injury, and this might superimpose the patient with an underlying renal problem [2].

In conclusion, SARS-CoV-2 infection should be monitored intensively for dialysis patients due to immunorepression, difficulty in diagnosis, and additional concern regarding the use of antiviral drugs. Our report should prompt experts and medical workers to pay special attention to the SARS-CoV-2 infection of dialysis patients.

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