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.

using the Chronic Kidney Disease Epidemiology Collaboration equation.[1] Looking at the renal function dynamics in figure 1 of their appendix,[1] if the patient's eGFR had decreased to 35–40 mL/min per 1·73 m² (equivalent to an SCr of 107–121 µmol/L), then there is no clear evidence for acute kidney injury based on reported data.

It is surprising that two of the 12 patients (cases 17 and 48) with a reported history of chronic kidney disease had eGFR values of 60 mL/min per 1·73 m² or more, which is not consistent with the KDIGO definition of chronic kidney disease, unless there is persistent albuminuria.[3] Acute kidney injury was reported in the other ten patients with chronic kidney disease.[1] In contrast, the seven patients who did not have acute kidney injury were not reported to have chronic kidney disease.[1] Underlying chronic kidney disease is a risk factor for acute kidney injury.[4]

In the absence of specific therapeutic options, application of the KDIGO supportive care guidelines (eg, regular monitoring of urinary output and SCr, and avoidance of nephrotoxins) could reduce the incidence and severity of acute kidney injury in COVID-19.[5]

I declare no competing interests.

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Authors' reply

We thank Kay Choy for the nterest in our Correspondence,[1] in which we described an association between severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) renal tropism and acute kidney injury in autopsy cases with COVID-19 diagnosis.

We analysed data from a large autopsy series of 63 patients. Since an individual's disease course, comorbidities, and complications of severe COVID-19 disease are highly variable, we depicted three different cases as supplemental information exemplifying this variation.[1] The first example had initially stable renal function and presented an abrupt decline before death, shortly after COVID-19 diagnosis (case 50). The second example presented with declining renal function before COVID-19 diagnosis, which aggravated over the following weeks (case 52). The third example had signs of acute kidney injury shortly after admission and later a positive respiratory swab for SARS-CoV-2 (case 45). As we expected, these cases have sparked some interesting discussions.

Case 45 was used by Choy as an example that merits clarification regarding the adherence to Kidney Disease: Improving Global Outcomes (KDIGO) guidelines for the acute kidney injury definition. This patient developed an increase in serum creatinine from 102·57 µmol/L at admission to 197·18 µmol/L within 48 h, meeting KDIGO criteria for acute kidney injury.[2] Notably, this patient was not tested for COVID-19 due to respiratory symptoms, but was instead diagnosed following a routine diagnostic procedure on the ward. Given the complex clinical context of COVID-19 and the dynamic nature of indications for testing, establishing the link between acute kidney injury and SARS-CoV-2 infection remains challenging. In this case, we chose to use temporal proximity to COVID-19 diagnosis as a key defining parameter. Although there is no available consensus or guidelines to conclusively define COVID-19-associated acute kidney injury, the high frequency of acute kidney injury among patients with SARS-CoV-2 infection might serve as a catalyser for such discussions.

Another important point is the definition of chronic kidney disease. Cases 17 and 48 indeed presented with an estimated glomerular filtration rate (eGFR) above the threshold for chronic kidney disease according to the KDIGO guidelines.[3] However, the possibility of a careful organ examination allowed us to grade structural kidney changes associated with chronic kidney disease (ie, fibrotic parenchymal remodelling, thinned kidney cortex, or decreased organ weight), which clearly indicated abnormalities of kidney structure as per KDIGO guidelines.[3] In contrast, determination of kidney function using a single measurement of eGFR can be limited by multiple causes (eg, case 48 had a measured body-mass index of 17; hence eGFR could be overestimated). Thus, autopsy studies provide a unique opportunity to extend clinical definitions with additional layers of structural information.

In summary, although the KDIGO guidelines have provided the framework for reliable and reproducible nomenclature of acute kidney injury and chronic kidney disease, autopsy studies can add further anatomical and pathological information and help to identify renal tropism and COVID-19-related acute kidney injury.[1,4,5] The association of SARS-CoV-2 infection and kidney injury opens potential new avenues for early diagnostics, prediction, and prevention of COVID-19-related kidney disease.[6]

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One Health or Planetary Health for pandemic prevention?

For more on the **Arctic Monitoring and Assessment Programme** see https://www.amap.no

It is a well accepted narrative that the disruption of forests, rapid urbanisation, and population growth are driving zoonotic events simply by increasing close contact between people and animals.[1] However, these predictor variables (eg, urbanisation, land usage, human population density) only explain about 30% of the total variation in zoonotic potential.[2] With advancing technologies, it has been shown that vertebrate RNA viruses, despite transmitting between species, have co-evolved with their hosts for millions of years, which only strengthens the theory that zoonotic diseases caused by RNA virus spillover, such as COVID-19, are linked to human activities and transformation of ecosystems.[3]

Genomic analysis cannot be used to predict spillover and pandemics, mainly because of the scarcity of empirical data and the reliance on reviewing studies and databases. This setback implies that our notion of urbanisation and population growth as the most important drivers of spillover is only part of the story. Attempts to overcome this scarcity by use of complicated statistical methods only compensate somewhat for the missing links and inadequate research efforts. Such a situation shows a clear gap in knowledge on what the other drivers of spillover are. Dedicated studies for establishing these drivers are scarce, given that reducing close contact with animals, including restricting global wildlife trade, is not enough.[4]

A considerable framework shift is needed to change this situation. We therefore ask for the comprehensive integration of planetary health across scientific communities, with clear governance and interdisciplinary links to climate change, which is currently the most immediate threat to the biosphere of our planet. WHO and the UN should drive such integration.

The approach could be inspired by the Arctic Monitoring and Assessment Programme, which is currently the sole international programme that has a true holistic combined planetary approach with clear links to the UN. This programme incorporates ecosystem dynamics including wildlife, infectious diseases, hazardous substances, food supply, and human health, and this type of holistic interdisciplinary programme is the only way to to prevent pandemic zoonotic outbreaks and achieve the UN Sustainable Development Goals.

To date, One Health alone fails to show that it can actually deal with pandemics in practice. John Amuasi and colleagues make excellent suggestions in their Correspondence about a COVID-19 One Health Coalition.[5] However, responding to the COVID-19 pandemic and avoiding zoonitic outbreaks in the future will necessitate a complete change in the intensity of human-managed ecosystems, with decreased deforestation and species extinction, including a reversal of the world's climate tipping points.[6]

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Authors' reply

One Health recognises the fundamental interconnectedness and complex interdependence of all living species and their shared environment.[1] Thus, a view that One Health is focused on human–animal interaction alone, primarily addresses the risk of zoonotic events, and that these events occur as a result of urbanisation, land usage, and increasing human population density, therefore suggesting that One Health is sufficient to address pandemic risks, is flawed. Nevertheless, there is abundant evidence that disease agents of zoonotic origin account for over half of all emerging or re-emerging human infectious diseases,[2] often with pandemic potential. We also know that