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.

control participants from the local community and health care workers ($R^2 = 0.879$) than was spike IgG antibody ($R^2 = 0.410$). Open elements (diamonds, triangles, or circles) represent high titer samples, gray-shaded elements represent positive but not high titer, and filled black elements represent negative samples to correspond to **A** to **C**. Threshold for IgG positivity denoted with horizontal dashed line. Linear regression correlations were plotted with 95% confidence intervals (dotted curves). (**G**) Scatter plot illustrating distribution of spike IgG (diamonds), RBD IgG (triangles), and NTD IgG (circles) in pediatric patients on dialysis ($n = 10$). Open elements represent positive samples and closed represent negative samples. (**H**) Distribution of neutralization for spike IgG-, RBD IgG-, and NTD IgG-positive samples.

Figure S3. Neutralizing antibodies against SARS-CoV-2 in health care workers and PCR-positive individuals from the local community by IgG antibody status. Relationship of spike IgG, receptor-binding domain (RBD) IgG, and spike peptide N-terminal domain (NTD) IgG to percentage of neutralization. NTD IgG was associated with higher neutralizing effect in the community of PCR-positive control participants and health care workers with high OD ($R^2 = 0.888$) and spike IgG antibody ($R^2 = 0.834$). No association was seen in those with low OD. Threshold for IgG positivity denoted with horizontal dashed line. Linear regression correlations were plotted with 95% confidence intervals (dotted curves). Open elements (diamonds, triangles, or circles) represent positive samples and closed represent negative samples.

Figure S4. Microneutralization correlation to SARS-CoV-2 antibody subsets. The community of SARS-CoV-2 PCR-positive control participants ($n = 17$) illustrating the relationship among levels of spike IgG, receptor-binding domain (RBD) IgG, and spike peptide N-terminal domain (NTD) IgG to one-half maximal inhibitory concentration (IC50) against live SARS-CoV-2.

Figure S5. Surrogate viral neutralization assay in different clinical cohorts. SARS-CoV-2 binds to the ACE2 receptor for host cell internalization via the receptor-binding domain (RBD) of the spike protein. Signal is produced by the absence of antibodies in the subject's serum enabling the binding of RBD to the ACE2 receptor. Signal is absent when serum contains neutralization antibodies reflecting inhibition of binding. When serum contains robust spike IgG antibodies and no neutralization antibodies, an increase in signal is demonstrated potentially from spike IgG to RBD complexes binding to ACE2. During uremia, we do not see the effect of neutralization antibodies on RBD-ACE2 binding.

Table S1. Characteristics and cumulative SARS-CoV-2 seroconversion for patients receiving dialysis and health care workers.

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Decline and loss of anti–SARS-CoV-2 antibodies in kidney transplant recipients in the 6 months following SARS-CoV-2 infection

To the editor: The dynamics of immune response to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in kidney transplant recipients (KTRs) remains largely unknown. KTRs have been reported to develop serological responses to SARS-CoV-2.[1,2] However, information about the duration and significance of antibody response in this immunocompromised population is still critically lacking. We herein report anti–SARS-CoV-2 IgG trajectory in a cohort of KTRs followed at Necker Hospital (Paris, France) between 2 and 6 months after symptomatic coronavirus disease 2019 (COVID-19) infection.

Forty-two patients (22 men [52.4%]; median age of 57.7 years; interquartile range [IQR]: 47.2–67.0), who developed COVID-19 infection between March 14 and May 2, 2020, were included. COVID-19 was defined by typical clinical symptoms associated to a positive SARS-CoV-2 polymerase chain reaction test on nasopharyngeal swab. Sera were tested for the presence

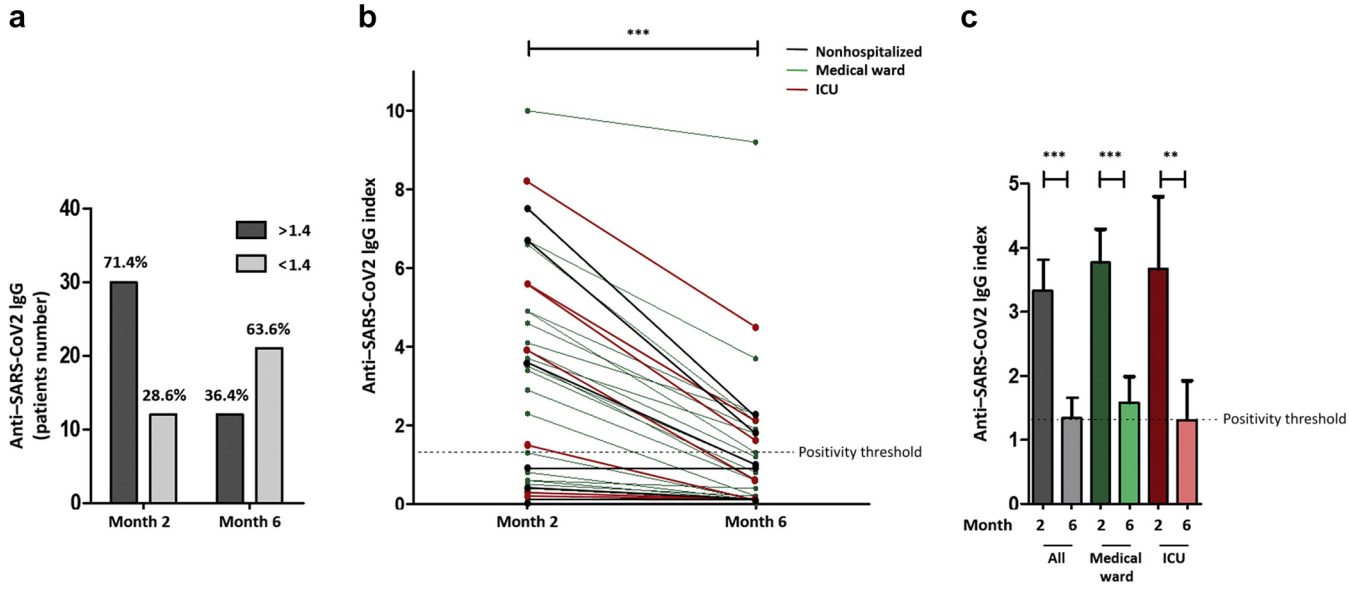

Figure 1 | Anti–severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) IgG evolution between 2 and 6 months after coronavirus disease 2019 onset in kidney transplant recipients. (**a**) A total of 71.4% of patients have a positive IgG response at month 2 while 63.6% of them have a negative or equivocal IgG serology at month 6. (**b**) Anti–SARS-CoV-2 IgG index decreases in all patients between months 2 and 6. (**c**) Median anti–SARS-CoV-2 IgG index significantly decreases between months 2 and 6 in all patients, including patients requiring hospitalization or treatment in the intensive care unit (ICU).

of anti-nucleocapsid protein IgG by a chemiluminescent microparticle immunoassay (SARS-CoV-2 IgG assay, Abbott, Abbott Park, IL) at 2 and 6 months after COVID-19 onset. According to the manufacturer's instructions, an IgG index >1.4 indicates a positive serology while an IgG index between 0.4 and 1.4 is considered to be an equivocal result and IgG index <0.4 to be a negative result. Sera were available for all patients at month 2 and for 33 of 42 patients at month 6.

COVID-19 occurred at a median time of 6.3 years (IQR: 3.1–12.7) after transplantation. In our cohort, 32 patients (76.2%) required hospitalization, including 7 (21.9%) in an intensive care unit (ICU), none of whom died.

At first serological testing (month 2), all patients had recovered and SARS-CoV-2 polymerase chain reaction was negative in 40 patients (95.2%). At month 6, SARS-CoV-2 polymerase chain reaction was negative in all except 1 patient.

Of the 42 patients, 30 (71.4%) were seropositive (IgG> 1.4) at month 2 (Figure 1a). Among the 21 of 33 patients (63.6%) who were IgG-positive at month 2 and who had available sera at month 6, 12 (57.1%) remained positive (index ≥1.4) at month 6, while 9 (42.9%) had negative or equivocal results. Overall, 21 of 33 patients (63.6%) had an IgG index <1.4 at month 6 (Figure 1a), including 14 of 24 patients (58.3%) and 4 of 7 (57.1%) who, respectively, required hospitalization and ICU stay at the time of the COVID-19 episode.

IgG index decreased between months 2 and 6 in all patients including in patients requiring hospitalization or ICU stay. Median IgG index fell from 3.6 (IQR: 1.3–5.1) at month 2 to 0.7 (IQR: 0.1–2.0) at month 6 (Figure 1b and c). Median decrease was 80.3% (IQR: 60.8%–83.3%; $P < 0.0001$). No patient relapsed from COVID-19 infection.

At month 6, there was no correlation between IgG index and initial disease severity ($P = 0.65$), post-transplantation delay ($P = 0.99$), or induction therapy by anti-thymocyte globulins ($P = 0.77$).

In conclusion, this is the first study assessing the anti–SARS-CoV2-IgG trajectory over a period of 6 months after disease onset in KTRs. Our results confirm that most KTRs develop specific antibodies against SARS-CoV-2.[1] However, antibody levels rapidly decrease in all patients and more than 60% had negative or equivocal IgG results at month 6. Interestingly, antibodies turned also negative or equivocal in patients with severe forms. Data about anti–SARS-CoV-2 antibodies' duration in the general population are controversial.[3,4] However, antibodies' decline had been mainly described in mild disease forms.[4,5] Further studies are needed to assess long-term antibody response in KTRs and its potential correlation with COVID-19 reinfections or relapses, as well as the efficacy of the vaccine in this population.

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Hyponatremia under MAP kinase inhibitors: a complex relationship between aquaporins and ERK activation

To the editor: We read with interest the recent review by Workeneh *et al.*[1] related to the epidemiologic and pathophysiological issues of hyponatremia in cancer. Our group recently reported data about hyponatremia under mitogen-activated protein kinase inhibitors in melanoma.[2] We hypothesized that B-Raf (BRAF)/mitogen-activated protein kinase kinase inhibitors could activate aquaporin-2 trafficking in tubular epithelia from intracellular sites to the luminal membrane and discussed potential mechanisms underlying the complex relationship between aquaporins and extracellular signal–regulated kinase (ERK) activation.

Several data support our hypothesis. Activated ERK has been reported to be involved in aquaporin-2 phosphorylation at serine 261, a process abolished by ERK inhibitors *in vitro*.[3] Aquaporin-2 phosphorylation at serine 261 may stabilize ubiquitinated aquaporin-2 within intracellular compartments.[3] In fact, posttranslational modifications of aquaporin-2 such as phosphorylation and ubiquitylation tightly regulate localization of aquaporin-2 within subcellular compartments and its degradation.[3] Thus, the prevention of ERK activation by BRAF/mitogen-activated protein kinase kinase inhibitors could prevent aquaporin-2 phosphorylation at serine 261 and activate aquaporin-2 insertion to the plasma membrane.

However, 2 recent studies suggest a potentially more complex interaction. The first report demonstrated a role of activated ERK in aquaporin-2 transcription in a rat model of lithium-induced nephrogenic diabetes insipidus.[4] The second report showed aquaporin-4 deregulation in association with mitogen-activated protein kinase kinase inhibition, pointing to the possibility that BRAF/mitogen-activated protein kinase kinase deregulation acts on aquaporins other than aquaporin-2.

In conclusion, the mechanisms of hyponatremia induced by BRAF/mitogen-activated protein kinase kinase inhibitors include a complex relationship between aquaporins and the activation and transcription of ERK. Understanding these mechanisms may help to improve the management of hyponatremia in the setting of cancer therapy with mitogen-activated protein kinase inhibitors.

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Validation of the ANCA renal risk score in a London cohort: potential impact of treatment on prediction outcome

To the editor: A risk score using 3 clinicopathologic parameters was developed by Brix *et al.*[1] to predict renal survival at 36 months in patients with newly diagnosed antineutrophil cytoplasm antibody–associated glomerulonephritis.[1] Smith *et al.* validated the tool in 102 Scottish patients.[2]

We sought to validate the risk score in a cohort of 178 patients with antineutrophil cytoplasm antibody–associated glomerulonephritis treated at our center between 2006 and 2019. Sixty-four (36%), 76 (43%), and 38 (21%) patients were stratified into low-, medium-, and high-risk groups, respectively. Poor renal survival at 36 months was evident in the high-risk group (55% reaching end-stage kidney disease,