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Neutralising antibodies after COVID-19 vaccination in a cohort of UK haemodialysis patients

Edward J Carr¹, Mary Wu¹, Ruth Harvey², Emma Wall¹, Gavin Kelly¹, Saira Hussain¹, Michael Howell¹, George Kassiotis¹, Charles Swanton¹, Sonia Gandhi¹, David LV Bauer¹,³, Haemodialysis COVID-19 consortium, Crick COVID Immunity Pipeline¹, Roseanne Billany⁴, Matthew Graham-Brown⁴, Joseph Beckett⁵, Katherine Bull⁶, Sushma Shankar⁶,⁷, Scott Henderson⁸, Reza Motallebzadeh⁸,⁹, Alan D Salama⁸, Lorraine Harper¹⁰,¹¹, Patrick B Mark¹²,¹³, Stephen McAdoo¹⁴,¹⁵, Michelle Willicombe¹⁴,¹⁵, Rupert Beale¹,³ & ⁸

Affiliations

¹ The Francis Crick Institute, London, UK
² Worldwide Influenza Centre, The Francis Crick Institute, London, UK
³ Genotype-to-Phenotype UK National Virology Consortium (G2P-UK)
⁴ Department of Cardiovascular Sciences, University of Leicester, Department of Renal Medicine, University Hospitals of Leicester NHS Trust, UK and NIHR Leicester Biomedical Research Centre, Glenfield Hospital, Leicester, UK
⁵ Transplantation Research & Immunology Group, Nuffield Department of Surgical Sciences, University of Oxford, UK
⁶ Nuffield Department of Medicine, University of Oxford, UK
⁷ Oxford Transplant Centre, Nuffield Department of Surgical Sciences, University of Oxford, UK
⁸ UCL Dept of Renal Medicine, Royal Free Hospital, London, UK
⁹ Research Department of Surgical Biotechnology, Division of Surgery and Interventional Science, University College London, UK
¹⁰ Institute Applied Health Research, College of Medical and Dental Sciences, University of Birmingham, Birmingham
¹¹ Department of Nephrology, University Hospitals Birmingham NHS Foundation Trust, Birmingham
¹² Glasgow Renal and Transplant Unit, Queen Elizabeth University Hospital, Glasgow, UK
¹³ Institute of Cardiovascular and Medical Sciences, University of Glasgow, UK
¹⁴ Department of Immunology and Inflammation, Faculty of Medicine, Centre for Inflammatory Disease, Imperial College London, UK
¹⁵ Renal and Transplant Centre, Imperial College Healthcare NHS Trust, Hammersmith Hospital, London, UK

Correspondence to: Edward J Carr edward.carr@crick.ac.uk and Rupert Beale rupert.beale@crick.ac.uk

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[should not include abstract, references, figures or tables]

Word count for abstract: 0
[no abstract]
Vaccination against COVID-19 induces highly protective immune responses in the great majority of people. As some countries switch from suppression to acceptance of transmission of SARS-CoV-2 within a largely vaccinated adult population, vulnerable patient groups that have not mounted adequate immune responses to vaccination may suffer significant morbidity and mortality. There is an urgent need to identify such patient groups and to optimise medical advice and vaccination strategies for them.

In-centre haemodialysis patients (IC-HD) represent a particularly vulnerable group. During the first wave of the COVID-19 pandemic (1 March 2020 to 30 August 2020), there were 4,666 cases and 1,373 deaths in IC-HD patients reported to the United Kingdom’s Renal Registry (1), a case fatality rate of 29%. In the UK, whilst IC-HD patients were treated as ‘clinically extremely vulnerable’, they were unable to fully ‘shield’ due to mandatory life-sustaining attendance at HD (typically three 4-hourly sessions per week), and instances of in-unit transmission have been shown by sequencing viral isolates (2).

Vaccine responses are substantially attenuated in haemodialysis patients. For example, the subunit hepatitis B vaccine had to be re-formulated for HD patients with a higher antigenic dose (3). There is uncertainty that either an mRNA or an adenoviral-vectored vaccine could provide clinical protection in the IC-HD population, or how long that protection lasts given the known waning of SARS-CoV-2 antibodies after natural infection (4).

The majority of IC-HD patients were vaccinated by their dialysis care team, as part of the Joint Committee on Vaccination and Immunisation (JCVI) priority group 4 (3), resulting in rapid delivery of doses to this at-risk population (appendix, p2). Phase 3 studies of authorised vaccines in the UK either excluded IC-HD or did not report their ‘renal disease’ subgroups (5–7). While multiple reports regarding anti-S antibodies (reviewed recently (8)) in IC-HD patients have been published, they do not widely report the levels of neutralising antibodies (nAbs) to the prevalent variants of concern (VOCs), which have emerged as the crucial serological correlate of protection (9,10).

To assess the induction of nAbs in IC-HD patients, after vaccination with BNT162b2 (Pfizer-BioNTech, international nonproprietary name: tozinameran) or AZD1222 (Oxford-AstraZeneca, international nonproprietary name: ChAdOx1-S [recombinant]), we are curating a meta-cohort of HD patients from around the UK (appendix, p 2). This is a multi-centre cohort study, allowing comparison of the antibody responses after vaccination between pre-specified cohorts of interest (appendix, p 14). We have used our previously reported high throughput live virus neutralisation assays (11,12), against a variant with a spike identical to the virus first identified in Wuhan, China (wildtype), a
variant with an Asp614Gly mutation isolated during the UK's first wave, and three VOCs: Alpha (B.1.1.7, first isolated in Kent, UK), Beta (B.1.351, first isolated in South Africa) and Delta (B.1.617.2, one of several variants described in India in early 2021 and now predominant). Here, we report the first interim analysis of this study, testing the hypothesis that the neutralising antibody response to either vaccine is non-inferior, using sera drawn pre-vaccination, at a median of 28 days after dose 1 [IQR 26-35], and at a median of 33 days [IQR 26-48] after the second dose, in 178 IC-HD patients (appendix, p 2). Three centres had available data for this analysis: Oxford, Leicester and Royal Free Hospital (appendix, p 5). Whilst there were differences with the deployment of vaccines - two centres predominantly administered AZD1222, one centre predominantly BNT162b2 - there were no significant differences in age (median 63.2 vs 63.1 years), gender (34% vs 37.3% female), ethnicity, the presence of diabetes or the immunosuppression state of AZD1222 and BNT162b2 recipients (appendix, p 5).

We focused initially on seronaïve patients (n=108) - defined by pre-vaccination sera that lacked detectable anti-S IgG by ELISA, or nAbs against wildtype or D614G and who had never returned a positive PCR prior to commencing vaccination - and assessed nAb responses 33 days after two vaccine doses of either AZD1222 or BNT162b2 (appendix, p 2,3). We found that BNT162b2 induced nAb titres (nAbTs) across all 5 variants (median NAbT IC$_{50}$=582, 327, 174, 136, 267 against wildtype, D614G, Alpha, Beta and Delta respectively; appendix, p 3). For AZD1222 the response was markedly reduced compared to BNT162b2, and may fall below the likely correlate of protection from severe disease against Alpha (>4 fold reduction, falling below the limit of detection of IC$_{50}$>40), Beta (>3 fold reduction, falling below the limit of detection), or Delta (>6 fold reduction, falling below the quantitative range) variants (appendix, p 3). Stratifying the nAbTs better illustrates the differing distributions of responses with patients with low (<40), medium (40-256) and high (>256) titres after two doses of AZD1222 compared to BNT162b2 (P<0.001 by ANOVA for vaccine effect in ordered logistic regression; appendix, p 3, 7). The corresponding analysis for infection-experienced patients revealed smaller differences between AZD1222 and BNT162b2, with AZD1222 achieving median nAbT IC$_{50}$>150 for all variants (appendix, p 10-11), suggesting a potential for adenoviral-vectored vaccines in certain settings. A similar pattern of improved responses in infection-experienced IC-HD patients, in anti-S titres rather than neutralising antibody, has been reported for the single-dose adenoviral-vectored vaccine, Ad26.CoV.2 (13).

Next, we sought to compare with the healthy individuals we have already reported from the Legacy study. As a control group, we selected Legacy participants who had never reported COVID symptoms (likely infection and sero-naïve) and had received two doses of either vaccine (appendix, p 4, 8-9). We found that an mRNA vaccine performed
similarly in IC-HD as in healthy volunteers (both infection naive), despite the age
difference between the cohorts (appendix, p 8). As expected, we found an attenuated
response in the IC-HD AZD1222 recipients (appendix, p 8).

Given the ability of BNT162b2 to induce nAbTs across all variants in IC-HD, we wanted
to assess other vaccine response associations. The response to BNT162b2 exhibits
age associated waning (age grouped as greater or less than 65; (appendix, p 12)), this
is not discernible in the AZD1222 response due to its low titres and BNT162b2 showed
a gender effect, absent in AZD1222 (appendix, p 12). Stratifying by diabetes found no
effect (appendix, p 12). As expected, immunosuppressed patients showed attenuated
responses (appendix, p 12).

There are several limitations to our study, most importantly the potential for confounding
factors to exist between HD centres. However, it is unlikely that the same confounder
would be present between several different centres since they are physically split over
more than one site (a hub – satellite model), and the hub and satellite have used
BNT162b2 or AZD1222, but share medical, nursing staff, HD protocols and a single
dialysis supplier. We were able to restrict our analysis to a single centre with a mix of
BNT162b2 (n=48) and AZD1222 (n=12) sero naïve patients, and recapitulated our prior
findings (appendix p 12). Whilst we have stringently tried to exclude prior antigenic
exposure in our sero naïve group (by anti-S ELISA, by nAbT to relevant variants, and
PCR data where available), we cannot fully exclude the possibility that there were
infections in early 2020, before widespread PCR and whose patients either did not
generate an antibody response, or their response had waned below the level of
detection in our baseline sampling.

We draw several conclusions from this interim report on a subset of the full UK cohort.
Firstly, an mRNA vaccine induces nAb titres in IC-HD patients comparable to healthy
controls. This represents an important initial step in improved vaccinations in IC-HD for
other pathogens. We note that there is a mRNA influenza vaccine in phase 1/2
development, and IC-HD are a cohort of patients that stand to benefit from a novel
influenza vaccine. Secondly, two doses of either vaccine consolidates antibody
immunity in infection-experienced individuals. A caveat to this conclusion is presence of
survivor bias for individuals infected in the first wave. Thirdly, AZD1222 alone in
sero naïve individuals induces sub-optimal nAbT against all VOCs, including the Delta
variant dominant in the UK and globally. Fourthly, the very high proportion of previously
infected IC-HD patients may obfuscate calculations of vaccine efficacy if based on
epidemiological parameters alone. Overall, our data highlight an urgent need for similar
studies assessing vaccine responses in at-risk populations.
Whilst delivery of any approved vaccine will likely mitigate morbidity and mortality, the optimal strategy for IC-HD patients yet to start a vaccination course remains to be determined. Our data suggest two doses of mRNA vaccine or a heterologous boosting strategy are likely to offer the broadest VOC nAb coverage. The UK’s JCVI has announced, in principle, booster doses for many vulnerable groups (14). The precise start date for this programme, which vaccines are used, and the ordering of the groups is under review. Internationally, most countries with pre-existing IC-HD vaccination strategies (Israel, USA, Canada, France, Germany, Portugal), have used two doses of mRNA (8) and there are now three studies reporting a third dose of BNT162b2 in 132 IC-HD patients in France showing further augmentation of responses (15–17). We suggest that IC-HD patients should be prioritised for a third dose, particularly AZD-1222 recipients that have not already survived infection.

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References


Appendix for

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Affiliations

¹ The Francis Crick Institute, London, UK
² Worldwide Influenza Centre, The Francis Crick Institute, London, UK
³ Genotype-to-Phenotype UK National Virology Consortium (G2P-UK)
⁴ Department of Cardiovascular Sciences, University of Leicester, Department of Renal Medicine, University Hospitals of Leicester NHS Trust, UK and NIHR Leicester Biomedical Research Centre, Glenfield Hospital, Leicester, United Kingdom
⁵ Transplantation Research & Immunology Group, Nuffield Department of Surgical Sciences, University of Oxford, UK
⁶ Nuffield Department of Medicine, University of Oxford, UK
⁷ Oxford Transplant Centre, Nuffield Department of Surgical Sciences, University of Oxford, UK
⁸ UCL Dept of Renal Medicine, Royal Free Hospital, London, UK
⁹ Research Department of Surgical Biotechnology, Division of Surgery and Interventional Science, University College London, UK
¹⁰ Institute Applied Health Research, College of Medical and Dental Sciences, University of Birmingham, Birmingham
¹¹ Department of Nephrology, University Hospitals Birmingham NHS Foundation Trust, Birmingham
¹² Glasgow Renal and Transplant Unit, Queen Elizabeth University Hospital, Glasgow, UK
¹³ Institute of Cardiovascular and Medical Sciences, University of Glasgow, UK
¹⁴ Department of Immunology and Inflammation, Faculty of Medicine, Centre for Inflammatory Disease, Imperial College London, UK
¹⁵ Renal and Transplant Centre, Imperial College Healthcare NHS Trust, Hammersmith Hospital, London, UK

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Crick COVID Immunity Pipeline consortium membership list
Figure 1: Study design and defining seronaïve patients

(A) Study design. Dates of vaccine administration and serum sampling times are shown in the top and bottom panels respectively. N=178 patients. Demographics in Supplementary Table 1.

(B) The proportion of patients defined as seronaïve at the time of first vaccination. Seronaïve was defined as (i) no detectable anti-S IgG by ELISA (143 patients of 178 had no anti-S IgG), no positive PCR results before first dose (117 patients) and no detectable neutralising antibodies to either wildtype SARS-CoV-2 or SARS-CoV-2 carrying the D614G spike mutation at baseline (108 patients). Seronaïve demographics in Supplementary Table 2.
Figure 2: Neutralising antibody responses after two doses of AZD1222 or BNT162b2 in seronaïve haemodialysis patients

(A) Live virus microneutralisation titres against SARS-CoV-2: wildtype, the D614G spike mutant, and VOCs - alpha, beta and delta - 33 days after two doses in seronaïve haemodialysis patients comparing AZD1222 and BNT162b2 responses (AZD1222 n=56, BNT162b2 n=59).

(B) Data as in (A) plotted with stratification of titres into three categories. An ordinal logistic regression model: IC50_binned ~ variant * vaccine was fitted. ANOVA P<0.001 is indicated by *** for the vaccine term (see also Supplementary Table 3 for ordinal logistic regression).

In (A), the medians are plotted as a black diamond. Note that the median is below the quantitative range (IC50<40) in some instances. The estimated fold-decrease between AZD1222 and BNT162b2 is shown in (A), where the AZD1222 median IC50<40, it was assigned an value of 40 for a conservative estimate of fold-decrease, and no confidence intervals are calculated.
Figure 3: Neutralising antibody responses after two doses of AZD1222 or BNT162b2 in seronaïve haemodialysis patients compared to never-symptomatic healthy individuals.

(A) Microneutralisation titres, comparing two doses in seronaïve haemodialysis patients (IC-HD) with two doses in never-symptomatic healthy individuals (Legacy) for AZD1222 and BNT162b2. Legacy demographics are shown in Supplementary Table 4.

(B) Data as in (A) stratified into three bins of neutralizing antibody. An ordinal logistic regression model: IC50_binned ~ variant * cohort was fitted for each vaccine separated. ANOVA P<0.001 is indicated by *** for the cohort term (see also Supplementary Table 5-6 for ordinal logistic regression).

In (A), the medians are plotted as a black diamond. Note that the median is below the quantitative range (IC₅₀<40) in some instances.
Supplementary tables 1-7

Supplementary table 1: Demographics of the whole interim report cohort, grouped by vaccine

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<td>n = 84</td>
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<td>M</td>
<td>62 (66%)</td>
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<td>B</td>
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<td>15 (17.9%)</td>
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<td>C</td>
<td>17 (18.1%)</td>
<td>68 (81%)</td>
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Supplementary table 2: Demographics of the seronaïve cohort

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<td>12 (22.6%)</td>
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For supplementary tables 1 and 2, P values are t tests for single level continuous variables (eg age) and ANOVAs for higher levels (eg ethnicity). The Chi2 tests for categorical data (eg gender). Apart from dialysis centre, the cohorts of AZD1222 and BNT162b2 are matched for age, gender ethnicity, diabetes and immunosuppressed status.
Supplementary table 3: Ordered logistic regression model of effect of variant and vaccine type on neutralising antibody titres 33 days after 2 doses in seronaïve IC-HD patients, relating to Figure 2B. Model: ic50_binned ~ variant * vaccine

| FACTOR                              | COEF  | SE    | WALD Z | PR(>|Z|) |
|-------------------------------------|-------|-------|--------|---------|
| VARIANT (VS WILDTYPE)               |       |       |        |         |
| D614G                               | -1.169| 0.3587| -3.26  | 0.0011  |
| ALPHA                               | -1.751| 0.3731| -4.69  | <0.0001 |
| BETA                                | -1.952| 0.3812| -5.12  | <0.0001 |
| DELTA                               | -1.559| 0.3745| -4.16  | <0.0001 |
| VACCINE (VS AZD1222)                | 1.2487| 0.3844| 3.25   | 0.0012  |
| BNT162B2                            | -1.751| 0.3731| -4.69  | <0.0001 |
| INTERACTION (VARIANT * VACCINE)     |       |       |        |         |
| D614 * BNT162B2                     | 0.3755| 0.5316| 0.71   | 0.4801  |
| ALPHA * BNT162B2                    | 0.321 | 0.5318| 0.6    | 0.5462  |
| BETA * BNT162B2                     | 0.2872| 0.5332| 0.54   | 0.5902  |
| DELTA * BNT162B2                    | 0.6437| 0.5395| 1.19   | 0.2328  |

ANOVA

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Supplementary table 4: Demographics comparison between IC-HD and Legacy cohorts

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<th>LEGACY</th>
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<td></td>
<td>n = 108</td>
<td>n = 162</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>F</td>
<td>40 (37%)</td>
<td>102 (63%)</td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>68 (63%)</td>
<td>60 (37%)</td>
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</tbody>
</table>

Supplementary table 5: Ordered logistic regression model of effect of variant and vaccine type on neutralising antibody titres after 2 doses of AZD1222 in seronaive IC-HD patients or Legacy participants, relating to Figure 3B. Model: ic50_binned ~ variant * cohort

AZD1222 recipients

| FACTOR                        | COEF | SE    | WALD Z | PR(>|Z|) |
|-------------------------------|------|-------|--------|---------|
| VARIANT (VS WILDTYPE)         |      |       |        |         |
| D614G                         | -1.5474 | 0.3934 | -3.93  | <0.0001 |
| ALPHA                         | -2.2325 | 0.4032 | -5.54  | <0.0001 |
| BETA                          | -2.4547 | 0.4099 | -5.99  | <0.0001 |
| DELTA                         | -2.0145 | 0.4061 | -4.96  | <0.0001 |
| COHORT (VS IC-HD)             | 1.3244 | 0.4055 | 3.27   | 0.0011  |
| LEGACY                        | -1.5474 | 0.3934 | -3.93  | <0.0001 |
| INTERACTION (VARIANT * COHORT) |      |       |        |         |
| D614 * LEGACY                 | -0.7819 | 0.5519 | -1.42  | 0.1566  |
| ALPHA * LEGACY                | 0.4837  | 0.5645 | 0.86   | 0.3915  |
| BETA * LEGACY                 | -0.6531 | 0.569  | -1.15  | 0.251   |
| DELTA * LEGACY                | -1.1898 | 0.5631 | -2.11  | 0.0346  |

ANOVA

Wald Statistics

<table>
<thead>
<tr>
<th>Factor</th>
<th>Coef</th>
<th>SE</th>
<th>Wald Z</th>
</tr>
</thead>
<tbody>
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<td>&lt;0.0001</td>
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<tr>
<td>Cohort (incl. Higher Order Factors)</td>
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</tr>
<tr>
<td>Interaction</td>
<td>11.31</td>
<td>4</td>
<td>0.0233</td>
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</table>
Supplementary table 6: Ordered logistic regression model of effect of variant and vaccine type on neutralising antibody titres after 2 doses of BNT162b2 in seronaïve IC-HD patients or Legacy participants, relating to Figure 3B. Model: ic50_binned ~ variant * cohort

| FACTOR                      | COEF    | SE     | WALD Z | PR(>|Z|) |
|-----------------------------|---------|--------|--------|---------|
| VARIANT (VS WILDTYPE)       |         |        |        |         |
| D614G                       | -0.8896 | 0.4069 | -2.19  | 0.0288  |
| ALPHA                       | -1.7099 | 0.4081 | -4.19  | <0.0001 |
| BETA                        | -2.0401 | 0.4066 | -5.02  | <0.0001 |
| DELTA                       | -1.0378 | 0.4056 | -2.56  | 0.0105  |
| COHORT (VS IC-HD)           | 1.0603  | 0.3972 | 2.67   | 0.0076  |
| INTERACTION (VARIANT * COHORT) |       |        |        |         |
| D614 * LEGACY               | -0.2846 | 0.5249 | -0.54  | 0.5877  |
| ALPHA * LEGACY              | 0.1135  | 0.5207 | 0.22   | 0.8275  |
| BETA * LEGACY               | -0.3623 | 0.5169 | -0.7   | 0.4834  |
| DELTA * LEGACY              | -1.7592 | 0.5183 | -3.39  | 0.0007  |

ANOVA

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>COEF</th>
<th>SE</th>
<th>WALD Z</th>
</tr>
</thead>
<tbody>
<tr>
<td>VARIANT (INCL. HIGHER ORDER FACTORS)</td>
<td>123.77</td>
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<td>&lt;.0001</td>
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<tr>
<td>COHORT (INCL. HIGHER ORDER FACTORS)</td>
<td>32.72</td>
<td>5</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>INTERACTION</td>
<td>19.6</td>
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<td>6.00E-04</td>
</tr>
</tbody>
</table>

Whilst there is a significant cohort effect, there is also (unlike for AZD1222) an opposing interaction effect is seen with Delta, such that the two cohorts have equivalent Delta responses.
Supplementary table 7: Ordered logistic regression model of effect of variant and vaccine type on neutralising antibody titres after 2 doses of either vaccine in seropositive IC-HD patients, relating to Supplementary Figure 1. Model: ic50_binned ~ variant * vaccine

SEROPOSTIVE (at baseline) patients

| FACTOR                        | COEF  | SE   | WALD Z | PR(>|Z|) |
|-------------------------------|-------|------|--------|---------|
| VARIANT (VS WILDTYPE)         |       |      |        |         |
| D614G                         | -0.4671 | 0.4525 | -1.03  | 0.302   |
| ALPHA                         | -0.9416 | 0.4472 | -2.11  | 0.0352  |
| BETA                          | -1.0489 | 0.4464 | -2.35  | 0.0188  |
| DELTA                         | -0.6607 | 0.4588 | -1.44  | 0.1498  |
| VACCINE (VS AZD1222)          | 1.2509  | 0.6961 | 1.8    | 0.0723  |
| BNT162B2                      | -0.4671 | 0.4525 | -1.03  | 0.302   |
| INTERACTION (VARIANT * VACCINE) |    |      |        |         |
| D614 * BNT162B2               | 0.428   | 0.9735 | 0.44   | 0.6602  |
| ALPHA * BNT162B2              | 0.1352  | 0.8817 | 0.15   | 0.8782  |
| BETA * BNT162B2               | -0.1859 | 0.8598 | -0.22  | 0.8288  |
| DELTA * BNT162B2              | 0.0612  | 0.9061 | 0.07   | 0.9461  |

ANOVA

Wald Statistics Response: ic50_binned

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>COEF</th>
<th>SE</th>
<th>WALD Z</th>
</tr>
</thead>
<tbody>
<tr>
<td>VARIANT (INCL. HIGHER ORDER FACTORS)</td>
<td>11.16</td>
<td>8</td>
<td>0.1930</td>
</tr>
<tr>
<td>VACCINE (INCL. HIGHER ORDER FACTORS)</td>
<td>25.20</td>
<td>5</td>
<td>&lt;.0001</td>
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<tr>
<td>INTERACTION</td>
<td>0.56</td>
<td>4</td>
<td>0.9678</td>
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Supplementary figure 1: Live-virus microneutralisation antibody titres in infection-experienced IC-HD patients

(A) Live virus microneutralisation titres against SARS-CoV-2: wildtype, the D614G spike mutant, and VOCs - Alpha, beta and Delta - 33 days after two doses in seronaïve haemodialysis patients comparing AZD1222 and BNT162b2 responses (63 patients in total (AZD1222 n=38; BNT162b2 n=25).

(B) Data as in (A) plotted with strafication of titres, P < 0.001 from denoted by *** (ANOVA of regression model; see also Supplementary Table 9 for ordinal logistic regression).
Supplementary figure 2: Comparing nAbT responses by age group, gender, diabetes and immunosuppression in seronaïve IC-HD patients.

NAbTs are compared 33 days after two doses in seronaïve haemodialysis patients. The data is grouped by age (18-65 or >65 years old, A), gender (B), the presence of diabetes (C), or the presence of immunosuppression (D) and each vaccine is shown separately. P values from ANOVA for the effect of age ($P=0.76$, $P<0.0001$), gender ($P=0.72$, $P=0.17$), diabetes ($P=0.99$, $P=0.29$), or immunosuppression ($P<0.0001$, $P=0.02$), performed on ordinal linear regression models are provided. (AZD1222 model: ic50_binned ~ age * variant, BNT162b2 model: ic50_binned ~ age * variant, with the variable ‘age’ changed for each panel to gender, diabetes or immunosuppression as indicated).
Supplementary figure 3: Comparing nAbT responses by age group, gender, diabetes and immunosuppression in seronaive IC-HD patients

(A) Live virus microneutralisation titres against SARS-CoV-2: wildtype, the D614G spike mutant, and VOCs - alpha, beta and delta - 33 days after two doses in seronaive haemodialysis patients comparing AZD1222 and BNT162b2 responses in a single centre (AZD1222 n=12, BNT162b2 n=48).

(B) Data as in (A) plotted with stratification of titres into three categories. An ordinal logistic regression model: IC50_binned ~ variant * vaccine was fitted. ANOVA P<0.001 is indicated by *** for the vaccine term.
Supplementary Methods

Study objectives and design

We are performing a cohort study of 1,200 IC-HD patients across the UK. The study has several objectives:

1. Confirm the immunogenicity of BNT162b2 and AZD1222 in IC-HD patients, including the generation of neutralising antibodies.
   a. Confirm augmentation of the antibody response with the second dose of vaccine
   b. Assess the longevity of the antibody response, including neutralising antibody.

2. Compare the profiles of neutralising antibodies generated between BNT1262b and AZD1222 IC-HD recipients.

3. Compare the profiles of neutralising antibodies generated by either vaccine between different age groups, different genders, different ethnicities, and different primary renal diseases.

4. Compare the profiles of neutralising antibodies generated between patients with and without diabetes or with and without immunosuppression.

5. Exploratory / discovery phase, where novel patterns / correlations are identified to provide hypothesis for testing in other cohorts / specifically targeted studies.

For any cohort comparison we expect, given the nature of the UK’s IC-HD population (its ethnicities, the frequencies of diabetes, immunosuppression) to be able to assemble groups of >100 patients for each comparison.

We planned serum collections were before vaccination, 28 days after each vaccination, and 6 & 12 months after commencing vaccination.

Clinical cohorts

Three haemodialysis centres are included in this interim report, and one healthy control cohort. In centre haemodialysis patients were included if they were able to consent into their local study and were clinically eligible to receive the available vaccine. Home haemodialysis patients and peritoneal dialysis patients were not included. The data shown is censored for individuals who received two doses of vaccine, and had available neutralising antibody titres at the first three study time points (baseline, ~28 days after vaccine 1, and ~33 days after vaccine 2). Anonymised (coded only against a research identifier) sera and phenotype data were provided for central analysis: age, gender, ethnicity, diabetes, immunosuppression, primary renal disease, alongside the dates of vaccine, vaccine manufacturer and the dates of serum sampling. Ethnicity was recorded as Asian, Black, Mixed, White or Other (in line with UK government advice at the time of commencing the study).

https://webarchive.nationalarchives.gov.uk/20210224165417/https://design-system.service.gov.uk/patterns/ethnic-group/). Diabetes was recorded as Y/N, and we
defined immunosuppression as Y/N as in Billany et al. (1). Individuals were vaccinated intramuscularly as part of their usual care, with either 0.5mL [not less than $2.5 \times 10^8$ infectious units] AZD-1222, ChAdOx1-S [recombinant] (Oxford-AstraZeneca) or 30ug BNT162b2 (Pfizer-BioNTech), at the interval indicated in Figure 1.

**Leicester cohort (IC-HD)**
Patient samples were collected as part of the study “PHENOTYPING SEROCONVERSION FOLLOWING VACCINATION AGAINST COVID-19 IN PATIENTS ON HAEMODIALYSIS”, with REC approval from (West Midlands - Solihull Research Ethics Committee, REC: 21/WM/0031) sponsored by the University of Leicester and included consent for samples to transfer to the Francis Crick Institute. This work was conducted locally with support from the NIHR Leicester Biomedical Research Centre and funding from the Leicester Hospitals Charity, University Hospitals of Leicester NHS Trust. Data from these patients have been published previously (1).

**Royal Free Hospital cohort (IC-HD)**
Patients were consented to join the UCL-RFH biobank approved study "ANALYSIS OF ANTI-SARS COV2 IMMUNE RESPONSE". The UCL-RFH Biobank has been given a favourable ethics opinion for conduct in the NHS by the Wales research ethics Committee 4 (REC: 16/WA/0289). This work was conducted locally with funding support from The St Peter’s Trust, Royal Free Charity.

**Oxford cohort (IC-HD)**
Patients were consented to join the Oxford Radcliffe Biobank approved study “Immunological responses to COVID-19 vaccines in transplant and haemodialysis patients” (ref: ORB 21/A014). The Oxford Radcliffe Biobank has a favourable ethics opinion from the South Central Oxford Committee C (REC: 19/SC/0173). This work was conducted locally with funding support by the Oxford Transplant Foundation and the Oxfordshire Health Services Research Committee, part of Oxford Hospitals Charity.

**Legacy cohort (Healthy volunteers)**
The Legacy cohort (NCT04750356) has been described recently(2,3). It comprises of healthcare workers from University College London Hospital and scientists from the Francis Crick Institute, London. The Legacy study was approved by London Camden and Kings Cross Health Research Authority (HRA) Research and Ethics committee (REC: 20/HRA/4717) and sponsored by University College London. The full dataset was kindly made available by the Legacy team for analysis in this report. Please see Wall et al. for access details (2,3).

**Serological Analysis and live-virus neutralisation**
All serum samples were collected during routine IC-HD sessions from the HD circuit, without additional venepuncture. Sera were separated from blood in local laboratories and stored frozen. Sera were shipped to the Crick on dry ice, and barcoded whilst frozen. All serological analyses, including in-house anti-Spike IgG ELISA and live-virus microneutralisation were performed as described previously (4).
Data analysis, statistics

Data analysis was performed in R/Rstudio, using Rmarkdown. Anonymised data wrangling used a mix of base R and tidyverse. As previously (2,3), IC_{50} values above the quantitative limit of detection of the assay (>2560) were re-coded as 5120; IC_{50} values below the quantitative limit of the assay (< 40) but within the qualitative range were re-coded as 10 and data below the qualitative range (i.e. no response observed) were re-coded as 5. IC_{50} values are shown on a log2 scale throughout. NAbT are compared between vaccines, age groups, gender, diabetes (as a categorical variable) or immunosuppression using unpaired Mann-Whitney tests. 95% confidence intervals of the fold changes of median NAbT were estimated using bootstrap and boot.ci, with type="basic“ argument, which does not assume normality. Where the median is below the quantitative range of the assay and estimated effect is shown using the lower bound of the quantitative range (IC_{50}=40), and confidence intervals are not reported. Stratified IC_{50} NAbT were compared using ordinal logistic regression, from the rms package, using the model: IC_{50} binned ~ variant * vaccine, or IC50b binned ~ variant * cohort to compare AZD1222 Legacy with IC-HD recipients, and BNT162b2 Legacy with IC-HD. The ordinal regression was necessary due to non-random censoring of the IC50s at low levels of response (a fully parametric model would be biased, and a dichotomisation into responders/non-responders is less powerful). Plots were generated using ggplot2 and ggpubr packages.

Data Sharing

All R code to reproduce all figures and analyses is freely available at (https://github.com/EdjCarr/Crick-HD-AZD-BNT-VOCs-2021-07/). The public dataset omits dialysis centre, age and dates, to ensure no individual participant can be unique. The Legacy data are already available as outlined in their original publications (2,3).

Ethics

This work is covered by the following REC approvals: REC: 21/WM/0031, REC: 16/WA/0289, REC: 19/SC/0173, REC: 20/HRA/4717, as described in the cohort descriptions above. Within REC: 21/WM/0031, central processing in the Crick was included.

Role of the funding source

This work was supported by Kidney Research UK, NKF, PKD charity, Kidney Wales and several Kidney Patient Associations [Exeter, North Staffs and South Cheshire, Northamptonshire, South Eastern and Wessex], the MRC and core funding from the Francis Crick Institute, which receives its funding from Cancer Research UK, the UK Medical Research Council, and the Wellcome Trust. The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.
The corresponding authors had full access to all the data and the final responsibility to submit for publication.

**Contributors Statement**

Edward J Carr - Investigation, data curation, data analysis, Writing - original draft, review and editing. Has access to and has verified underlying data

Mary Wu - Investigation, Methodology, Resources, Writing – review & editing,

Ruth Harvey - Investigation, Methodology, Resources, Writing – review & editing,

Conceptualization

Emma C Wall - Investigation, Data Curation, Resources

Gavin Kelly - Formal Analysis, Validation

Saira Hussain - Investigation, Resources

Michael Howell - Project administration, Supervision, Writing – review & editing,

Conceptualization

George Kassiotis - Writing – review & editing, Conceptualization

Charles Swanton - Supervision, Funding acquisition, Project administration, Writing – review & editing, Conceptualization

Sonia Gandhi - Supervision, Funding acquisition, Methodology, Project administration, Writing – review & editing, Conceptualization. Has access to & has verified underlying data.

David LV Bauer - Methodology, Formal Analysis, Visualization, Writing – review & editing, Conceptualization.

Haemodialysis COVID-19 consortium - Investigation, Resources

Crick COVID Immunity Pipeline - Investigation, Data Curation, Resources

Roseanne Billany - Investigation, Data Curation, Resources

Matthew Graham-Brown - Supervision, Resources, Conceptualization

Joseph Beckett - Investigation, Data Curation, Resources

Katherine Bull - Investigation, Data Curation, Resources

Sushma Shankar - Supervision, Resources, Conceptualization

Scott Henderson - Investigation, Data Curation, Resources

Reza Motallezadeh - Supervision, Resources, Conceptualization

Alan D Salama - Supervision, Resources, Conceptualization

Lorraine Harper - Conceptualization

Patrick B Mark - Conceptualization

Stephen McAdoo - Conceptualization, Supervision, Funding acquisition

Michelle Willicombe - Conceptualization, Supervision, Funding acquisition

Rupert Beale - Supervision, Funding acquisition, Project administration, Writing – review & editing, Conceptualization, has access to & has verified underlying data.

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Supplementary references

Seroprevalence of antibody to S1 spike protein following vaccination against COVID-
4.

activity against SARS-CoV-2 VOCs B.1.617.2 and B.1.351 by BNT162b2 vaccination.


Haemodialysis COVID-19 consortium

Sherna F Adenwalla1, Paul Bird1, Christopher Holmes1, Katherine L Hull1, Daniel S March1, Haresh Selvakandan1, Jorge J Silva1, Julian W Tang1, Joanna Hester2, Fadi Issa2, Martin Barnardo3, Peter Friend3, Andrew Davenport4, Catriona Goodlad4, Vignesh Gopalan4, Theerasak Tangwonglert4, Hans J Stauss5, Alex G Richter6, Adam F Cunningham7, Marisol Perez-Toledo7, Gemma D Banham8, Nadya Wall9

1 Department of Cardiovascular Sciences, University of Leicester and Departments of Renal Medicine and Clinical Virology, University Hospitals of Leicester, UK
2 Transplantation Research & Immunology Group, Nuffield Department of Surgical Sciences, University of Oxford, UK
3 Oxford Transplant Centre, Nuffield Department of Surgical Sciences, University of Oxford, UK
4 UCL Dept of Renal Medicine, Royal Free Hospital, London, UK
5 Institute of Immunity & Transplantation, Royal Free Hospital, London, UK
6 Institute of Immunology and Immunotherapy, College of Medical and Dental Sciences, University of Birmingham and UHB NHS Foundation Trust
7 Institute of Immunology and Immunotherapy, College of Medical and Dental Sciences, University of Birmingham
8 Department of Nephrology, University Hospitals Birmingham NHS Foundation Trust, Birmingham
9 Institute Applied Health Research College of Medical and Dental Sciences, University of Birmingham and UHB NHS Foundation Trust, Birmingham

Crick COVID Immunity Pipeline consortium membership list

Bobbi Clayton
Sina Namjou
Vanessa Silva
Meghan Poulten
Philip Bawumia
Murad Miah
Samuel Sade
Mauro Miranda
Tom Taylor
Ilenia D'Angelo
Mercedes Cabrera Jarana
Mahbubur Rahman
Janet Abreu
Sandeep Sandhar
Neil Bailey
Simon Caidan
Marie Caulfield
Mary Wu
Ruth Harvey
Lorin Adams
Caitlin Kavanagh
Scott Warchal
Chelsea Sawyer
Mike Gavrielides
Jag Kandasamy
Karen Ambrose
Amy Strange
Titilayo Abiola
Nicola O'Reilly
Philip Hobson
Ana Agau-Doce
Emma Russell
Andrew Riddell
Svend Kjaer
Annabel Borg
Chloë Roustan
All The Francis Crick Institute, London, UK