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# ABO blood type association with SARS-CoV-2 infection mortality: A single-center population in New York City

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## Abstract

**Background:** Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has a variable clinical course with significant mortality. Early reports suggested higher rates of SARS-CoV-2 infection in patients with type A blood and enrichment of type A individuals among COVID-19 mortalities.

**Study Design and Methods:** The study includes all patients hospitalized or with an emergency department (ED) visit who were tested for SARS-CoV-2 between March 10, 2020 and June 8, 2020 and had a positive test result by nucleic acid test (NAT) performed on a nasopharyngeal swab specimen. A total of 4968 patients met the study inclusion criteria, with a subsequent 23.1% (n = 1146/4968) all-cause mortality rate in the study cohort. To estimate overall risk by ABO type and account for the competing risks of in-hospital mortality and discharge, we calculated the cumulative incidence function (CIF) for each event. Cause-specific hazard ratios (csHRs) for in-hospital mortality and discharge were analyzed using multivariable Cox proportional hazards models.

**Results:** Type A blood was associated with the increased cause-specific hazard of death among COVID-19 patients compared to type O (HR = 1.17, 1.02–1.33, $p = .02$) and type B (HR = 1.32, 1.10–1.58, $p = .003$).

**Conclusions:** Our study shows that ABO histo-blood group type is associated with the risk of in-hospital death in COVID-19 patients, warranting additional inquiry. Elucidating the mechanism behind this association may reveal insights into the susceptibility and/or immunity to SARS-CoV-2.

**KEYWORDS**
ABO, SARS-CoV-2

## 1 | INTRODUCTION

SARS-CoV-2, first identified in Wuhan, China, as the cause of a severe pneumonia outbreak, is the agent responsible for the current ongoing pandemic, claiming more than 600,000 lives worldwide.[1] Reports from China during the early months of coronavirus disease 2019 (COVID-19) pandemic suggested higher rates of SARS-CoV-2 infection in patients with type A blood as well as enrichment of individuals with type A blood among expired COVID-19 patients.[2,3] To date, numerous studies addressing the subject have emerged, some

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corroborating original reports and others failing to detect any association.

The ABO blood group, which is the key determinant in blood transfusion compatibility, includes three carbohydrate antigens (A, B, and H) present on glycosphingolipids and glycoproteins on the extracellular surface of red blood cells (RBCs). Blood groups exhibit a simple Mendelian genetics pattern of inheritance,[4] are polymorphically expressed in individuals and across various ethnic groups, and, thus, are frequently surveyed in epidemiological studies. The distribution of ABO types varies among different ethnic groups. Globally, type O is the most common blood group overall, but significant regional variation exists. Expression of ABO blood group antigens is not limited to RBCs but is also present on epithelium and endothelium, platelets, as well as on mucins secreted by exocrine glands in some individuals (i.e., saliva).[5]

Associations between ABO types and numerous disease processes have been previously reported. For example, type O individuals have been reported to be more susceptible to the Norwalk virus,[6] and ABO type has been associated with infectivity of *H. pylori* and *P. falciparum*.[7–10] One report that surveyed a small series of health care workers who were exposed to an index patient during the 2003 severe acute respiratory syndrome coronavirus (SARS-CoV) outbreak in Hong Kong found that type O individuals appeared to have some protection from infection relative to non-O individuals.[11] Guillon et al subsequently proposed a potential mechanism for this observation, showing that anti-A isohemagglutinins were protective by inhibiting the adhesion of SARS-CoV S-protein to its receptor [angiogtension-converting enzyme 2 (ACE2)] in vitro.[12]

Amidst the 2019 SARS-CoV-2 pandemic, several brief scientific reports examining the relationship between ABO group and the risk for SARS-CoV-2 infection and some querying the association with outcome of coronavirus disease 2019 have been published. The earliest reports by Zhao[2] and Li[3] both suggested a predilection for SARS-CoV-2 to infect ABO type A individuals, whereas those with Group O were found to have the lowest risk of infection. Additionally, Zhao et al reported a higher percentage of blood type A in those who died, suggesting potentially a higher mortality rate for type A individuals. Subsequently, additional studies have been published probing the relationship between ABO type and risk of infection or disease outcome, with conflicting results.[13–18]

Here, we investigated the distribution of ABO types in the SARS-CoV-2 positive patients and assessed the effect of ABO type on mortality in COVID-19 patients presenting to Montefiore Medical Center (MMC), an ethnically diverse patient population in the Bronx borough of New York City, USA.

## 2 | MATERIALS AND METHODS

The electronic heath record system and related data warehouse were queried for all patients hospitalized or with an emergency department (ED) visit who were tested for SARS-CoV-2 between March 10, 2020 and June 8, 2020. The study was approved by the Albert Einstein College of Medicine Institutional Review Board. Patients with a positive test result by nucleic acid test (NAT) performed on a nasopharyngeal swab specimen were identified These patients were labeled as having a "COVID-19 associated ED visit or admission," if the first inpatient or ED encounter was associated with a positive SARS-CoV-2 test collected during or up to 7 days before the visit. Patients who remained hospitalized at the time of analysis, those without an ABO type on record, and patients less than 18 years old were excluded (Figure 1).

To determine if the distribution of ABO type was similar to the distribution seen in the institution prior to COVID-19, the ABO typing of patients from the last ~10 years at the medical center was determined from the Blood Bank in a de-identified fashion. Comparison of this 10-year ABO type cohort was made to the COVID-19 cohort using Chi-square.

The primary outcome analyzed was all-cause in-hospital mortality. All hospitalized patients included in the study had a minimum of 20 days follow-up period. Comparison of ABO type differences was performed using chi-square tests, 2-sample Student $t$ tests, and for non-normally distributed data, the Mann–Whitney U test. To estimate crude competing risks of in-hospital mortality and discharge by ABO type, a cumulative incidence function (CIF) was calculated. Cause-specific hazard ratios (csHRs) for in-hospital mortality and discharge were analyzed using multivariable Cox proportional hazards models to evaluate the effect of ABO type controlling for age, sex, body mass index (BMI), initial estimated glomerular filtration rate (eGFR), and initial partial pressure of oxygen (pO2). Covariates were selected a priori by literature review. The above adjustment parameter values included were those indexed within 36 h after triage/admit or up to 24 h prior to admission. Time from admission (in days) was set as the underlying time metric. Proportionality between the predictors and the hazard was verified through an evaluation of Schoenfeld residuals. To account for competing events and determine the effect of the covariates on the CIF, we estimated subdistribution hazard ratios (sdHRs) using the Fine and Gray model. A sensitivity analysis was performed to examine the effect of statistical method and predictor selection on the results and effect of the cohort inclusion groups: *Admitted* versus *Admitted and ED* patients (Tables S2–S3 and Figure S1). An additional post-hoc analysis was carried out to
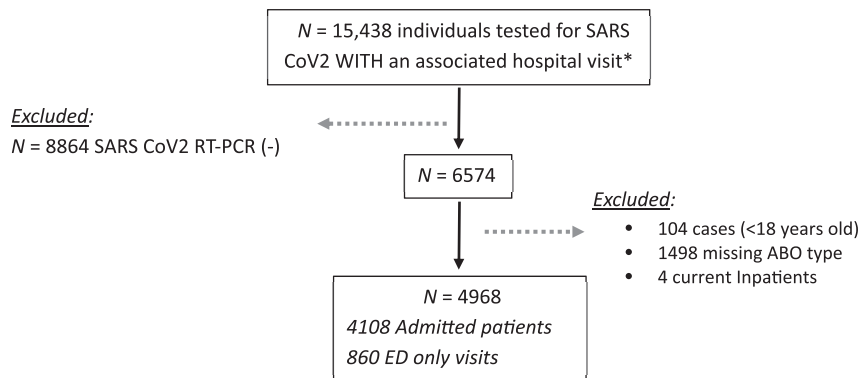
N = 15,438 individuals tested for SARS CoV2 WITH an associated hospital visit*

_Excluded:_
N = 8864 SARS CoV2 RT-PCR (-)

N = 6574

_Excluded:_
- 104 cases (<18 years old)
- 1498 missing ABO type
- 4 current Inpatients

N = 4968
_4108 Admitted patients_
_860 ED only visits_

**FIGURE 1** Study cohort

**TABLE 1** (Panel A) Characteristics of the admitted and ED cohort by ABO type; (Panel B) comparison of the ABO distribution of the admitted and ED cohort to the institutional historical distribution

| (A) n (%) | Overall cohort 4968 | Cohort characteristics stratified by blood type | | | | p value |
| --- | --- | --- | --- | --- | --- | --- |
| | | Type A 1473 (29.6) | Type AB 204 (4.1) | Type B 846 (17.0) | Type O 2445 (49.2) | |
| Mortality, n (%) | 1146 (23.1) | 381 (25.9) | 48 (23.5) | 172 (20.3) | 545 (22.3) | **.01** |
| Inpatient, n (%) | 4108 (82.7) | 1207 (81.9) | 166 (81.4) | 705 (83.3) | 2030 (83.0) | .73 |
| Male, n (%) | 2406 (48.4) | 707 (48.0) | 95 (46.6) | 421 (49.8) | 1183 (48.4) | .80 |
| Age, mean (SD) | 62.1 (17.2) | 62.6 (17.0) | 61.9 (17.0) | 62.0 (16.9) | 61.8 (17.5) | .52 |
| BMI, median (IQR) | 28.5 (24.6, 33.2) | 28.8 (25.1, 33.3) | 28.7 (25.6, 33.3) | 28.4 (24.3, 32.7) | 28.4 (24.3, 33.4) | .14 |
| eGFR, mean (SD) | 69.0 (33.1) | 68.8 (32.2) | 70.6 (31.8) | 68.0 (34.2) | 69.3 (33.4) | .68 |
| Initial PO2, mean (SD) | 94.6 (7.2) | 94.4 (7.5) | 94.7 (7.6) | 94.8 (6.6) | 94.6 (7.2) | .48 |
| (B) | Type A | Type AB | Type B | Type O | | p-value |
| Study cohort, n (%) | 1473 (29.6%) | 204 (4.1%) | 846 (17.0%) | 2445 (49.2%) | | .062 |
| MMC[a], n (%) | 296,805 (30.3%) | 37,950 (3.8%) | 154,058 (15.7%) | 489,970 (50.0%) | | |

_Note:_ Bold values indicating statistical significance.
[a]Historical distribution of blood group types from Blood Bank Laboratory Information System over the last decade.

evaluate the pattern and possible impact of missing data (Figures S2–S3). Analyses were performed using R version 3.6.2. A p value <.05 was considered statistically significant.

# 3 | RESULTS

Of the 15,438 ED and inpatient encounters including testing for SARS-CoV-2 between March 10, 2020 and June 8, 2020, 6574 tested positive and 4968 patients met the study inclusion criteria. Of included patients, 4108 (82.7%) were inpatients and 860 (17.3%) were seen only in the ER. The mean age of the study cohort was 62.1 years (SD = 17.2) and sex distribution was 2562 females to 2406 males (51.6%/48.4%). ABO blood type distribution for types A, B, AB, and O were 1473 (29.6%), 846 (17.0%), 204 (4.1%), and 2445 (49.2%), respectively (Table 1, Panel A). The ABO distribution in study subjects was similar to the ABO distribution of our general medical center population—data obtained from historical ABO types on record in medical center Blood Bank Laboratory Information System (LIS) over the last 10 years, ($X^2$ $p = .06$) (Table 1, Panel B).

We observed a 23.1% (n = 1146/4968) all-cause mortality rate in our study cohort, similar to previously published data from New York City.[19] Also, 1103/1146 (96.2%) of these were inpatient, constituting an inpatient mortality rate of 27% (n = 1103/4108).

Unadjusted, COVID-19 all-cause in-hospital mortality was found to differ by ABO type ($X^2$ $p = .01$). On post-hoc pairwise comparison using Chi-square analysis with a Bonferroni correction, the mortality difference between type A compared to type B was found to be statistically significant (adjusted $X^2$ $p = .01$), and a trend toward significance was noted in type A compared to type O (adjusted $X^2$ $p = .07$).

The cumulative incidence for all-cause mortality was found to differ by ABO type (Grays test, $p = .01$) (Figure 2). A Cox proportional hazards model with the

**FIGURE 2** Cumulative incidence function of COVID-19 mortality vs discharge by ABO type. Groups compared using Gray's test for subdistribution hazards
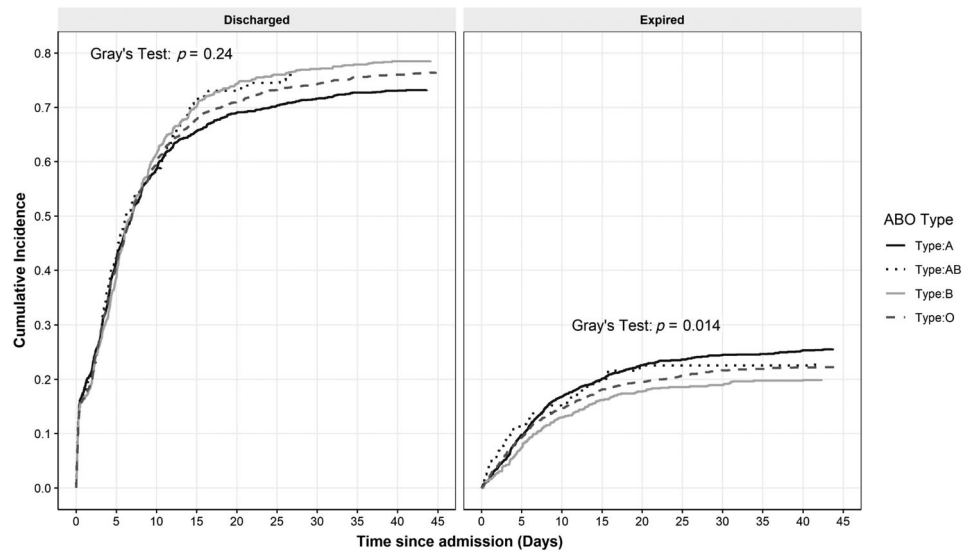


**TABLE 2** Hazard ratios (95% confidence intervals) from cause-specific and subdistribution hazard models for all cause in-hospital death and discharge

| Risk factor | Cause-specific hazard model | | Subdistribution hazard model | |
|---|---|---|---|---|
| | **All-cause mortality** | **Discharge** | **All-cause mortality** | **Discharge** |
| Type A | 1.17 (1.02–1.33), **p = .025** | 1.00 (0.93–1.09), **p** = .9 | 1.15 (1.008–1.32), **p = .037** | 0.97 (0.89–1.05), p = .52 |
| Type AB | 1.22 (0.87–1.72), p = .2 | 1.04 (0.89–1.22), p = .6 | 1.19 (0.89–1.61), p = .23 | 0.93 (0.77–1.13), p = .49 |
| Type B | 0.88 (0.74–1.05), p = .2 | 1.01 (0.92–1.11), p = .9 | 0.88 (0.73–1.05), p = .16 | 1.03 (0.94–1.13), p = .49 |
| eGFR (per 10 unit change) | 0.95 (0.93–0.97), **p < .001** | 1.06 (1.05–1.07), **p < .001** | 0.90 (0.89–0.93), **p < .001** | 1.07 (1.06–1.08), **p < .001** |
| BMI (per 5 unit change) | 1.09 (1.05–1.14), **p < .001** | 1.0 (0.98–1.03), p < 0.9 | 1.10 (1.05–1.16), **p < .001** | 0.98 (0.96–1.01), p = .21 |
| Sex [M] | 1.3 (1.15–1.48), **p < .001** | 0.84 (0.78–0.90), **p < .001** | 1.50 (1.32–1.70), **p < .001** | 0.80 (0.75–0.86), **p < .001** |
| Initial PO2[a] | Predictor stratification[a] | | 0.95 (0.95–0.96), **p < .001** | 1.06 (1.05–1.06), **p < .001** |
| Age[b c] | Predictor stratification[b] | | Quadratic time varying covariate[c] | |

*Note:* Blood type O set as reference level. Bold values indicating statistical significance.
[a]Predictor placed into three initial PO2 (Pulse Oximetry) groups (<90%, 90%–95%, >95%) and stratified.
[b]Predictors placed into age groups by quartile and stratified to meet proportional hazard assumption.
[c]Quadratic time varying covariate for age was added to fulfill the proportional hazard subdistribution assumption.

competing event of hospital discharge treated as a censored observation was used to examine the effects of ABO type, adjusting for eGFR, BMI, pO2, age, and sex on the cause-specific hazard of all-cause in-hospital expiration. Blood type A was associated with the increased cause-specific hazard of all-cause in-hospital mortality compared to type O (csHR: 1.17, 95% CI 1.02–1.33; $p = .02$) (Table 2) and type B (csHR: 1.29, 95% CI 1.08–1.54; $p = .006$) (Table S1). Increased eGFR ($p < .001$) and higher initial pO2 at presentation ($p < .001$) were associated with the *decreased* cause-specific hazard of all cause in-hospital mortality (Table 2), while increased BMI was associated with the *increased* cause-specific hazard of in-hospital mortality. Males had a higher cause-specific hazard of in-hospital mortality compared to females (csHR: 1.3, 95% CI 1.15–1.48; $p < .001$).

The cumulative incidence of patient discharge was found to not vary by ABO ($p = .24$) (Figure 2). In contrast, in the admitted only subgroup of our cohort, CIF of discharge did vary by ABO ($p = .03$) (Figure 1). eGFR, initial PO2, and sex displayed significant effects on the discharge subdistribution hazard ($p < .001$ for all factors). Neither ABO type nor BMI were found to have any effect on the hazard ratio of discharge ($p > .05$ for all factors) (Table 2).

## 4 | DISCUSSION

Here, we report an association between increased in-hospital mortality for ABO type A as compared to ABO type O individuals observed in the largest to date

monocentric cohort of COVID-19 patients. Our cohort reports on patients seen at an academic medical center geographically situated within one of the early epicenters of the COVID-19 pandemic, in New York City.

The association between COVID-19 and ABO blood group has been an area of controversy and an active topic of investigation worldwide. In the earliest report, Zhao and colleagues posit that SARS-CoV-2 has a predilection for infecting ABO type A individuals, whereas those with ABO type O were found to have the lowest risk of infection.[2] The report was based on a cohort of 2173 PCR-positive COVID-19 patients in Wuhan and Shenzhen, China. The study observed that for COVID-19-associated hospital deaths, there was an overrepresentation of ABO type A individuals relative to the ABO distribution of "normal people" from Wuhan and Shenzhen. However, other authors have rightly suggested that the study's control group is less than ideal.[13] Li et al found a similar association between ABO type and COVID-19 disease at a different Wuhan hospital, Central Hospital of Wuhan.[3] In this study, the ABO type distribution among COVID-19 patients was compared to the Wuhan City population's ABO distribution that had been previously reported by Zhao et al[2]; however, no association with ABO type and mortality was apparent. Other studies on this subject matter have corroborated that ABO type A individuals may be at increased risk of infection and that ABO type O may be protective.[2,3,14,17] However, the choice of a reference population in these studies varied and potentially not ideal for studies of this nature: some studies used blood donors, others a COVID-19 (−) subgroup, others a historical population for comparison. Interestingly, a genome-wide association study (GWAS) reported enrichment in ABO type A and a depletion in ABO type O genotypes in COVID-19 patients from Italy and Spain,[20] although blood donors were used as a reference population which has the potential to skew results.

In agreement with Dzik et al,[13] and in contrast to most previously published reports,[2,3,16,17] we report no enrichment in a particular blood group in our COVID-19 patients as compared to the historical blood bank records of the population treated at MMC over the last 20 years. While Zietz et al and Dzik et al included their institutional historical ABO data as controls, the former observed "higher odds of testing positive for ABO type A," while the latter observed no difference in distribution in those who tested positive[13,16]. Of note, all studies to date investigated significantly fewer patients than are represented in our cohort.

A relationship between ABO type and disease infectivity is not novel. There exists a large body of robustly confirmed historical literature showing an association of infectivity of various pathogenic microorganisms with particular blood groups.[21,22] Additionally, ABO blood group is well known to be associated with the levels of von Willebrand factor (vWF), with blood group O individuals having the lowest vWF levels.[23,24] As thromboembolic events, hypercoagulability, and endothelial dysfunction are well-recognized manifestations of COVID-19,[25] the effect of ABO type on mortality may in part be explained by this association. Thus, investigating the incidence of thromboemboli in COVID-19 patients as it relates to ABO type may be informative.

To our knowledge, an association of ABO type A with increased mortality in COVID-19 has not been previously reported within a cohort design following COVID-19 patients.[3,13,14,26,27] It may be possible that other studies were underpowered to detect it, especially if they lacked the cohort size and mortality event rate observed in the present study.[19] While Zhao and colleagues did report a higher risk of death for ABO Type A, it was based on the blood group analysis in the expired cases (n = 206), with regional ABO distributions serving as the controls.[2]

Our analysis is not free of limitations. As any observational study, results may be influenced by unmeasured confounding factors. First, during the study period, there were various public and institutional policies, patient triaging practices, and SARS-CoV-2 testing algorithms that likely influenced the composition of the patients presenting and admitted over time. This, combined with known clinical variation in symptomatology and disease severity, limits the generalizability of our study, particularly for mild or asymptomatic disease and in outpatient settings. Relatedly, in addition to patient heterogeneity, there was temporal heterogeneity in the management of COVID-19 patients coinciding with secular trends in survival (data not shown). Treatment and intervention practices were not captured in our analysis. However, with the exception of convalescent plasma, ABO group was not pertinent to clinical decision-making for most of the COVID-19 therapies our cohort was exposed to. Furthermore, less than 15% of the 127 convalescent plasma units issued during the inclusion period were ABO nonidentical (but compatible), with all recipients being type O except one, who was type B. Nonetheless, we cannot exclude the role of stochastic treatment imbalances potentially serving as an additional source of residual confounding. Second, 23% of our originally identified cohort did not have ABO typing in our system. It is suspected that this may have biased our cohort toward patients with distinct transfusion-relevant conditions or medical histories (e.g., prior obstetric or surgical history), but with an uncertain impact on our analysis. However, the failure to find a significant difference in ABO distribution between our study cohort and our multiyear center-wide patient population is of some reassurance. In supplemental competing risk survival analyses, we included diabetes and hypertension

(data not shown); however, since other co-morbidities have also been associated with increased COVID-19 mortality, this adjustment is unlikely to completely account for comorbid risks. Interpretation of our hypertension- and diabetes-adjusted survival analyses is further complicated by significant listwise deletion secondary to missingness. Similarly, while we performed a logistic regression sensitivity analysis (Tables S2–S3), our models do not include various laboratory parameters (i.e., D-Dimer, CRP, etc), as these initial values were often missing within our cohort; such laboratory results would be necessary for mediation analyses exploring possible intermediate factors involved in our findings.

Racial disparities in COVID-19 outcomes in the US are well-recognized[28] and blood type frequencies vary by ethnicity. Relevantly, Leaf and colleagues reported an increased representation of ABO type A among critically ill COVID-19 patients only in the white, non-Hispanic cohort, and not the Black or the Hispanic cohort.[15] Unfortunately, ethnicity information was not readily available for the present study. Thus, it remains unclear to what extent the sociodemographic structure of our cohort—representing the patient-base of a private safety net hospital serving an ethnically diverse urban catchment area—impacts our results. Of note, recent data from MMC that reported on the earliest cohort of patients did not uncover an increase in COVID-19-positive in-hospital mortality among Black and Hispanic inpatients relative to White inpatients.[29] It remains possible that severity at presentation, geographic proximity to our medical center versus other major hospital systems in the tri-state area, residential living situation (e.g, retirement homes, skilled nursing, or assisted living), and ethnicity are correlated in such a way as to confound the association between ABO group and outcomes. We hope to explore this possibility further in a future analysis. Generalizability to other regions is circumscribed for similar reasons.[28] The impact of follow-up time on our analysis is likely negligible as only four patients remained hospitalized by the day of final follow-up. Finally, outcome assignments are limited to encounters within a single health center. Patients discharged alive or never admitted from the ED could have been subsequently admitted and experienced a terminal event at another institution.

It is premature to say whether ABO type will prove to be of any clinical significance in the spread, diagnosis, or management of COVID-19 patients. Further inquiry, ideally in the form of prospective investigation, would be needed to validate these findings. If indeed present, the observed effects of ABO group may be mediated by anti-glycan antibodies or may be a direct consequence of varied glycosylation patterns/functions of various host/viral proteins. Further preclinical studies would be helpful in suggesting possible mechanisms underlying the association.[30–32]

## CONFLICT OF INTEREST
None.

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## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.