Impact of previous left atrial ablation procedures on the mechanism of left atrial flutter: A single-centre experience

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Abstract

Introduction: Left atrial flutter predominantly occurs after surgical or ablation procedures but this entity has also been recently reported in individuals without previous interventions. The use of high-density electroanatomical mapping systems (HDM) has improved the understanding of underlying mechanisms beyond entrainment maneuvers and substrate analyses. We aimed to evaluate the mechanism of left atrial (LA) flutters in preablated vs ablation-naïve individuals and sought to assess the efficacy of empiric ablations sets in these groups.

Methods and results: We included 55 patients admitted for ablation of LA flutter between July 2017 and August 2019. On the basis of HDM analyses the arrhythmia mechanism was determined with consecutive ablation targeting the suspected critical isthmus.

Mean age was 69.8 ± 10.7 years, with 26 of 55 (47.3%) male patients. Thirty-nine (71%) patients had previously undergone LA ablation. Arrhythmia mechanisms differed between preablated and ablation-naïve patients as anatomical structure-related LA flutters (perimitral, roof-dependent, within-pulmonary veins) were more frequent in the preablated cohort compared to ablation-naïve individuals (74.4% vs 43.8%; \( P = .03 \)). In ablation-naïve patients, most flutters (9 of 16, 56.3%) were related to low-voltage areas at the anterior/posterior wall. Acute termination rates were high (>90%) in both groups. Empirical mitral isthmus or roof lines showed a potential higher success rate in preablated patients.

Conclusion: We identified different mechanisms of LA flutters in preablated vs ablation-naïve patients. In ablation-naïve patients, most tachycardias involved low-voltage areas rather than anatomical structures. Using HDM, acute success rates were high. Hypothetical linear ablations were less successful in ablation-naïve individuals, further highlighting the need to identify the specific individual tachycardia mechanism in these patients.

Keywords: high-density mapping system, left atrial flutter, perimitral, reentry
1 | INTRODUCTION

Left atrial (LA) flutter is believed to be mainly attributed to cardiac interventions, like pulmonary vein isolation (PVI) or surgical valve repair. Less frequent, LA flutter occurs in patients without previous cardiac interventions, in those cases commonly associated with structural heart disease due to arterial hypertension, diabetes, coronary artery disease, or heart failure.\(^1\) Despite macroreentry as the suspected mechanism in most cases of LA flutters it is unclear whether there are differences in arrhythmia mechanism in patients with and without a history of previous cardiac procedures. Recent advances in three-dimensional (3D) high-density mapping (HDM) systems enabling high-resolution maps of activation circuits have led to a profound understanding of the underlying arrhythmia mechanism beyond entrainment-mapping manoeuvres.\(^2\) Therefore, with the increasing number of patients presenting with LA flutters we sought to identify the mechanism of the flutter form depending on a history of previous LA ablation procedures. As HDM is still not the widespread standard for flutter ablation procedures, we aimed to evaluate whether there are specific patients preferably being treated by HDM guidance for ablation of LA flutter.

2 | METHODS

We conducted a retrospective cohort study at the West German Heart and Vascular Center Essen, Germany. We identified 55 consecutive patients with any type of macroreentrant LA flutter admitted for radiofrequency ablation (RF) with an HDM system between July 2017 and August 2019. Patient clinical characteristics including outcome data were extracted from the patients’ hospital records including sex, age, antiarrhythmic therapy, CHA2DS2-VASc score, left ventricular ejection fraction, and LA diameter. Patients were divided into two groups: patients who had undergone a prior LA ablation procedure (preablated group) and patients without prior LA ablation therapy (ablation-naïve group). Procedural data for HDM-guided ablations were obtained during the procedure. The methods were carried out in accordance with the relevant guidelines and regulations of the University of Essen Medical School. The protocol was approved by the institutional review board and the local ethics committee (registration number: 19-8714-BO). All patients gave written informed consent before ablation.

2.1 | Procedure and mapping protocol

The details on procedure preparation and mapping are provided in the online supporting information. In brief, a 3D electroanatomical map of the LA with simultaneous local activation mapping was performed using the HDM system by two experienced operators (RW and JS) with an experience of more than 350 LA procedures each. The components of this HDM system have been described previously.\(^3,4\) There was an intention to not perform entrainment mapping to prevent a potential early termination of the tachycardia or conversion into a different form during the pacing manoeuvres. The goal was to assess the mechanism and potential reentry circuit by HDM mapping to consecutively target the critical isthmus by RF ablation to proof the suspected arrhythmia mechanism.

2.2 | Analysis of interobserver and intraobserver variability

For assessment of interobserver variability, the generated maps were reanalyzed by an independent physician (NV) with extensive experience in 3D mapping on an offline panel blinded to the initial result of the mapping process. Intraobserver variability was reassessed by the initial analyst (SK) by reanalyzing the HD maps after a mean period of 585 ± 408 days with respect to the dominant mechanism, also blinded to the initial result.

2.3 | Methodology of flutter classification

The first LA flutter form (present at the beginning of the procedure) was categorized into (a) fixed anatomical structure-related, including (i) perimital flutter, (ii) roof-dependent flutter, (iii) flutter within the PVs, and (b) non-fixed anatomical structure-related flutter forms, comprising localized reentries involving low-voltage/slow conduction areas. All flutter forms occurring spontaneously or converting during RF delivery following the predefined ablation approach were documented.

2.4 | Ablation protocol

All procedures were performed with an open-irrigated single-tip catheter (3.5 mm irrigated tip; Biosense Surround Flow, Biosense Webster, Diamond Bar, CA) by point-by-point RF delivery, with a maximum delivered energy of 35 W (30 W at the posterior wall). Each RF ablation was limited to 240 seconds, delivered by a 500 kHz ablation unit (Stockert EP shuttle; Biosense Webster, Inc, Baldwin Park, CA). The periprocedural anticoagulation regime has been previously described.\(^5\)

After analysis and identification of the mechanism of atrial flutter, RF energy was delivered according to the predefined protocol:

- RF deliveries targeted the most favorable critical isthmus of the arrhythmia. The RF delivery aimed at connecting the ablation lesion targeting the critical isthmus with either preexisting anatomical structures (ie, mitral valve annulus) or with iatrogenic scars (eg, left PV ostia after PVI) which led to the creation of linear ablation lines. The complete conduction block across the ablation line was verified by 3D electroanatomical mapping with the multipolar mapping catheter, either in sinus rhythm (SR) or by differential pacing with the coronary sinus (CS) or ablation catheter when indicated.\(^6\)
RF ablation was performed until (a) the tachycardia terminated or (b) converted into a second form, identified by a significant change in CS activation or in combination with alteration of surface electrocardiogram (ECG) morphology. Termination of the tachycardia was considered as ablation success if it occurred directly during RF delivery with or without prior prolongation of tachycardia cycle length (TCL) and in absence of a premature atrial beat (<90% TCL).

After conversion, a detailed remap (see detailed mapping protocol, online Supporting Information) and reablation were performed until termination in sinus rhythm could be achieved or the procedure had to be stopped at the discretion of the physician (long duration, safety concerns). If rhythm degenerated into atrial fibrillation (AF) during RF delivery, electrical cardioversion was performed. Empiric PVI was only performed if there was clear documentation of AF before the procedure. In preablated patients, all PVs and in the ablation-naïve group only PVs that were involved in the tachycardia mechanism (eg, in case of within-PV flutter) were checked for complete isolation and reisolated in case of persistent atrio-PV conduction.

### 2.5 | Objectives and endpoints

The aim of the present study was to assess the impact of a LA pre-ablation history on the dominant mechanism in LA flutters. Flutter forms were divided into “classical” fixed anatomic structure-related (eg, mitral isthmus or roof-dependent) and non-anatomic structure-related flutters (involving localized scar and low-voltage/slow conduction areas). In a second step, we sought to empirically assess the efficacy of predefined ablation sets in the two groups. Third, we analyzed the midterm arrhythmia recurrence between preablated and ablation-naïve patients which was defined as the occurrence of any atrial tachycardia during 720 days after the index procedure after a 90-day blanking interval.

### 2.6 | Evaluation of potential empirical linear ablation sets

Empirical linear ablation is sometimes used as an additional tool in AF ablation or in ablation of LA flutters in case of nonavailability of the HDM system, failed activation mapping and/or complex arrhythmia mechanism. On the basis of the underlying mechanism of the respective tachycardia, we sought to assess whether one of three standardized ablation strategies or a combination of those could potentially terminate/convert the tachycardia. The following three lesion sets were investigated: (a) anterior mitral isthmus line (deployed between anterior/anterolateral mitral isthmus and the left superior pulmonary vein (LSPV) including isolation of the LSPV) as previously described; (b) roof line (shortest ablation connection of LSPV and right superior pulmonary vein including isolation of both superior PVs), and (c) sole PVI.

### 2.7 | Clinical outcome

According to our local clinical routine, clinical follow-ups (FUs) were performed at 3, 6, and 12 months after PVI, and additionally in a 6-month interval if applicable, including physical examination, 12-lead ECG and a 7-day Holter-ECG. Any symptomatic atrial tachycardia greater than 30 seconds (AF, atrial flutter, focal tachycardia) on a Holter-ECG recording or on a 12-lead ECG was considered a recurrence. We applied the established 90-day blanking period with respect to early recurrences. Palpitations without ECG documentation of AF were not considered a recurrence. Antiarrhythmic drugs were discontinued after 90 days of the blanking period. FU was censored at 2 years.

### 2.8 | Statistical analysis

Continuous variables are expressed as mean ± standard deviation. Normally distributed data were compared using the unpaired Student t test and non-parametric variables using the Mann-Whitney U test, respectively. Descriptive statistics assessing the accordance between the raters was performed for interobserver and intraobserver variability. A \( P < .05 \) was considered statistically significant. Statistical analyses were performed using SPSS for Windows (version 20.0; SPSS Inc, Chicago, IL).

### 3 | RESULTS

All patients presented with LA flutter at the beginning of the procedure, which enabled mapping of at least one flutter form in 55 of 55 (100%) patients.

Out of the 55 patients, 39 patients (71%) had a history of a prior LA ablation, while 16 patients (29%) presented with LA flutter without any LA preablation history. Within the 39 patients with a LA preablation, \( n = 20 \) had a history of PVI only and \( n = 19 \) patients a history of a combination of PVI and linear line ablations, respectively (Figure 1). Baseline patient characteristics were comparable between the two groups and listed in Table 1 in detail.

### 3.1 | Procedural data including details of LA mapping process

A total of 55 activation maps was assessed for the initial flutter form. Procedural parameters including procedure duration, LA (mapping) time, and cumulative RF time were not significantly different between preablated and ablation-naïve individuals (see Table 1).

### 3.2 | Mechanism of macroreentry depending on preexisting LA procedure

Figure 2A depicts the distribution of flutter mechanisms stratified by the presence of a preexisting LA ablation procedure.
In the preablated group, the majority of flutter forms involved fixed anatomical structures (29 of 39 patients, 74.4%). In the group of ablation-naïve individuals, we found a higher percentage of flutter forms not related to fixed structures compared to flutters related to fixed anatomical structures (56.3% vs 43.8%; Figure 2A). Our analysis revealed a significant difference between the two groups with respect to the underlying arrhythmia mechanism (P = .03). Looking at the reentries in detail, we found that perimital reentry was the most common mechanism in preablated patients (14 of 39 patients, 35.9%), while the localized non-anatomical reentry was the most common form in non-preablated patients (9 of 16, 56.3%) (Figure 2B). Video S1 (online Supporting Information) visualizes perimital flutter in a preablated patient in a counter-clockwise rotation direction. Our results demonstrate a trend for a different pattern of the underlying reentry mechanism between the groups with respect to a preexisting history of a prior LA ablation (Figure 2B; P = .07).

In ablation-naïve individuals, the majority of reentries (9 of 16, 56.3%) were not related to anatomic structures, predominantly involving low-voltage areas at the anterior or posterior LA wall (Video S2, online Supporting Information).

### 3.3 Acute success of RF ablation of mapped arrhythmias

Including the consecutive second and third flutter forms resulting from conversion during ablation a total of 78 LA flutters were analyzed with a mean of 1.4 flutter forms per patient per procedure. Figure S1 (online Supporting Information) depicts a flowchart with a detailed illustration of the terminated and converted arrhythmia forms after ablation in preablated and ablation-naïve individuals. Overall, 36 of 55 (65.5%) flutters of the initial clinical arrhythmia could be directly terminated into SR by the first ablation

### TABLE 1 Baseline characteristics and procedural parameters

|                         | Overall (n = 55) | Preablated (n = 39) | Non-preablated (n = 16) | P value |
|-------------------------|-----------------|---------------------|-------------------------|---------|
| **Baseline characteristics** |                 |                     |                         |         |
| Male sex, n (%)         | 26 (47.3)       | 16 (43.0)           | 10 (62.5)               | .23     |
| Age at procedure, years | 69.8 ± 10.7     | 68.9 ± 8.9          | 72.1 ± 14.2             | .33     |
| LVEF, %                 | 51.7 ± 10.2     | 52.9 ± 8.1          | 48.6 ± 14.0             | .16     |
| LA diameter, cm         | 4.7 ± 1.0       | 4.7 ± 0.9           | 4.6 ± 0.9               | .69     |
| Hypertension, n (%)     | 40 (72.7)       | 29 (74.4)           | 11 (68.8)               | .74     |
| Coronary heart disease, n (%) | 20 (36.4) | 14 (35.9)           | 6 (37.5)                | .91     |
| Stroke/TIA, n (%)       | 6 (11.0)        | 3 (7.7)             | 3 (18.8)                | .34     |
| CHA2DS2-VASc            | 3.4 ± 1.5       | 3.4 ± 1.4           | 3.3 ± 1.6               | .76     |
| AAD at time of ablation | 15 (27.3)       | 10 (25.6)           | 5 (31.3)                | .87     |
| Class III, n (%)        | 11 (20)         | 7 (18.0)            | 4 (25.0)                |         |
| Class I, n (%)          | 4 (7.3)         | 3 (7.7)             | 1 (6.3)                 |         |
| Cycle length, ms        | 284.2 ± 51.1    | 281.8 ± 52.7        | 290.0 ± 48.1            | .59     |
| **Procedural parameters** |                 |                     |                         |         |
| Procedure time, min     | 242.0 ± 71.4    | 233.1 ± 65.2        | 263.7 ± 63.3            | .18     |
| LA time, min            | 158.4 ± 57.4    | 157.5 ± 58.5        | 160.7 ± 56.9            | .86     |
| LA mapping time, min    | 219.9 ± 9.0     | 217.9 ± 9.4         | 222.2 ± 8.3             | .83     |
| RF time, min            | 35.2 ± 18.5     | 34.4 ± 16.7         | 37.6 ± 22.8             | .56     |

Abbreviations: AAD, antiarrhythmic drugs; LA, left atrial; LVEF, left ventricular ejection fraction; RF, radiofrequency; TIA, transitory ischemic attack.
approach. In $n = 18$ patients the initial LA flutter converted under RF delivery into a second flutter form, while in $n = 5$ patients a conversion into a third form could be observed requiring further RF deliveries. In total, two LA flutters could not be terminated by endocardial RF delivery (potential epicardial substrate involving the reentry circuit) and were finally cardioverted into SR at the discretion of the physician.

Table S1 (online Supporting Information) lists the individual mechanism of flutters in the predefined cohorts in detail.

### 3.4 | Clinical outcome

Recurrence rates were comparable between preablated and ablation-naive individuals (35.9% vs 25%; $P = .72$) after 2 years of FU. Figure 3 depicts the Kaplan-Meier survival estimation for the two cohorts over the 2-year observation period. With respect to arrhythmia recurrence during the FU period, it is of note that 100% (4 of 4) of patients with recurrence in the ablation-naive group and only 35.7% (5 of 14) in the preablated cohort presented with atrial flutter as recurrent arrhythmia. The majority of recurrences in the preablated group, 9 of 14 patients (64.3%), could be attributed to AF.

### 3.5 | Safety

With respect to safety, one major stroke occurred due to thromboembolic occlusion of the left cerebral media artery in a patient admitted for LA flutter ablation (ablation-naive group). Two cases of hematoma at the venous access site in the femoral vein (both preablated cohort) without the need for transfusion or surgical intervention were observed.

### 3.6 | Evaluation of potential empirical linear ablation sets with respect to first tachycardia termination/conversion rate

In this analysis, we evaluated hypothetical success rates of classical PVI and linear ablation strategies distinguishing between ablation-naive and preablated patients. Figure 4 illustrates the hypothetical success rates for termination/conversion by deploying standard PVI and/or linear ablation lines in both patient cohorts.

Overall, PVI alone could have terminated/converted about 16.4% of flutter forms in preablated and ablation-naive patients, respectively. Comparing patients with a prior ablation history with ablation-naive patients, there was a clear difference suggesting that PVI alone would have terminated the flutter forms in 31.2% of the ablation-naive group and in 12.8% in the preablated cohort, respectively. On the basis of the predominant perimital mechanism in the preablated cohort, a hypothetical anterior mitral isthmus line would have terminated/converted about 56% of all flutter forms in the preablated individuals compared to 25% of patients in the ablation-naive cohort. Applying hypothetical dual combinations of different ablation sets, it appears that the combination of anterior mitral isthmus line plus PVI increases the success rate up to 64% in preablated and 50% in ablation-naive patients, respectively. A triple combination of “PVI + anterior line + roof line” further increased the
success rate primarily in the preablated cohort: up to 87.2% compared to 62.5% in the ablation-naïve cohort. In general, the ablation of predefined linear lesions showed significantly higher acute success rates in preablated than in ablation-naïve patients consistent with our previous findings with respect to the underlying reentry mechanism. Finally, looking at our real success rates by HDM-guided ablation, we could terminate more than 90% of all flutter forms independent of the preablation history (Figure S1).

4 | DISCUSSION

Treatment of LA flutter is a challenge warranting a profound understanding of the underlying and often complex arrhythmia mechanism. While significant experience is already available for the mechanisms underlying LA flutter after ablation or surgery, little is known about atypical flutter forms in ablation-naïve patients. Our study provides a systematic analysis assessing distinct mechanisms of LA flutters in preablated vs ablation-naïve individuals. We used HDM to elucidate the underlying mechanism of abnormal atrial activation and observed that macroreentry around anatomically fixed structures was the dominant mechanism in preablated individuals, while anatomical structure-independent (localized) reentries represented the predominant mechanism in ablation-naïve patients. In the latter, reentry was in most cases related to slow-conducting low-voltage areas at the anterior and posterior wall of the LA.

4.1 | Underlying causes of atypical LA flutter

The incidence of LA flutters has spread exponentially which can be attributed to the increase in cardiac interventions (especially LA
ablations) in the last years. With respect to AF ablation and its development of ablation strategies, the reported incidence of LA flutters after AF ablation increased up to 30% and even up to 50% in case of additional substrate modification for persistent AF. In our present experience, we observed that nearly one-third (16 of 55; 29%) of patients with LA flutter did not undergo any LA ablation in the past. This is a high number when compared to previous reports where the rate of ablation-naïve patients in LA flutter cohorts was around 10%.

As reentrant flutter forms always require anatomic barriers with a protected isthmus region favoring slow conduction, critical sites for reentrant tachycardias are suggested to be associated with atrial remodeling. Besides iatrogenic creation of those anatomic barriers during ablation or surgical procedures, they are especially found in atria of individuals with structural heart disease, like coronary or rheumatic heart disease, hypertrophic or dilated cardiomyopathy, or even in the absence of any heart disease. This could be linked to a recently reported phenomenon called "atrial myopathy" which is described as "any complex of structural, architectural, contractile, or electrophysiological change affecting the atria with the potential to produce clinically relevant manifestations." It can be speculated that those atrial alterations especially play an important role in ablation-naïve individuals where structural changes are involved in creating an arrhythmogenic milieu. Compared to overall baseline characteristics of published LA flutter cohorts, our ablation-naïve cohort tended to be older, with a lower mean left ventricular ejection fraction (72.1 years [48.6%] vs 56 years [53%] and 61 years [55%], respectively) pointing at the potential impact of cardiac structural alterations on the high prevalence of ablation-naïve individuals in our flutter cohort.

4.2 | Distinct mechanisms of atrial flutter in preablated vs ablation-naïve individuals

Our results concerning the mechanisms of LA flutter in preablated patients are in line with the published literature showing that perimital macroreentry is the dominant mechanism in LA flutters. Previous studies report frequencies of perimital reentry in 41.1% up to 76.9% in flutter cohorts. We observed a rate of 36% perimital flutters in the preablated cohort. However, the rate of perimital flutters in our ablation-naïve cohort was extremely low (2 of 16 patients, 12.5%). Our results clearly suggest that flutters related to fixed anatomical structures (perimital, roof-dependent, within-PVs) were much more common in preablated patients than in those without preablation. It appears that LA preablated patients constitute a group predisposed to perimital and roof-dependent flutter forms. To which extent the preceding ablation contributes to slow conduction favoring reentry formation around the mitral valve or the roof remains unclear. One hypothesis is that LA ablation produces slow conduction areas, with gaps in ablation lesions creating an arrhythmogenic milieu.

In contrast to the preablated individuals, localized flutter forms unrelated to fixed anatomical reentries were much more common in ablation-naïve individuals, representing 56% of all flutter forms. Most localized flutters were located at the anterior wall (12 of 16 flutters, 75%).

4.3 | Acute and midterm success rates

Using HDM we were able to terminate all flutter forms into SR except for two patients (all-over success rate, 96.3%; 97.4% and 93.8% in the preablated and in the ablation-naïve cohort, respectively). The comparison to data from literature is difficult as most papers do not differentiate between preablated and ablation-naïve individuals. Available literature reports on acute termination rates between 82% and 86%. Both studies performed ablations without HDM support. Two studies relying on HD activation sequence mapping demonstrate LA flutter termination rates of almost 100% which goes in line with the high success rates in our study.

With respect to midterm outcome we show that arrhythmia recurrence rates are comparable between preablated and ablation-naïve individuals (recurrence rates: 14 of 39 (35.9%) and 4 of 16 (25%), respectively, after 2 years. This is consistent with the reported midterm outcomes in literature between 18% and 49%.

The only literature available specifically investigating patients without any prior cardiac intervention reports on 11 patients with no recurrence during the 12 months FU. However, the patient number in these subgroups is too small to allow any valid conclusion on the different outcomes in preablated vs ablation-naïve individuals.

4.4 | Empirical linear ablation

As our acute termination rates were high, we assume that we actually correctly identified the critical isthmus in most patients. As we identified different mechanisms of LA flutters in preablated and ablation-naïve patients, we analyzed whether hypothetical empiric ablation lines would be able to terminate the distinct flutter form. We postulate significant lower (hypothetical) success rates with linear ablation approaches in ablation-naïve patients as the mechanism in those patients was less likely macroreentry involving fixed anatomical structures. Interestingly, when deploying PVI, anterior and roof line, a termination/conversion success rate of more than 87% could be achieved in the preablated individuals, while only 63% of flutters would have been terminated/converted in the ablation-naïve patients.

4.5 | Relevance and potential clinical implications

On the basis of our results we hypothesize that linear ablation sets at standardized locations can terminate many LA flutter forms especially in preablated patients, but still do not reach success rates of
HDM systems. In ablation-naïve patients a HDM-guided approach with identification of the individual tachycardia mechanism should be the preferred strategy since arrhythmia mechanisms are distinct and complex in these patients. As we did not perform empiric PVI in the ablation-naïve cohort except for PVs involved in the reentrant mechanism, there is a need for studies assessing the impact of an empiric PVI in those patients presenting with atrial flutter without a history of AF.

5 | LIMITATIONS

Although we present one of the largest studies on ablation outcomes in LA flutters the patient number is low, especially in the subgroup (n = 16) of ablation-naïve individuals. Additional verification of the arrhythmia mechanism (eg, by entrainment maneuvers) was not performed. Further, midterm outcome was evaluated by regular in-office FU and Holter-ECG recordings. No continuous 24 hours telemetric ECG surveillance was performed to rule out asymptomatic arrhythmia recurrence.

6 | CONCLUSION

Our study provides evidence for distinct mechanisms of LA flutters in preablated vs ablation-naïve patients showing significantly higher rates of tachycardias involving low-voltage and slow-conducting areas in ablation-naïve patients. Use of HDM-guided ablation resulted in very high acute success rates in both groups. Nevertheless, based on different dominant mechanisms, linear ablation approaches seem to be still reasonable in preablated patients, while LA flutters in ablation-naïve individuals are probably less accessible to this approach highlighting the need to identify the specific individual tachycardia mechanism in these patients.

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AUTHOR CONTRIBUTIONS
All authors significantly contributed to the following items: (a) substantial contributions to the conception and design or the acquisition, analysis, or interpretation of the data. (b) substantial contributions to the drafting of the articles or critical revision for important intellectual content. (c) Final approval of the version to be published. (d) Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the article are appropriately investigated and resolved.

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**SUPPORTING INFORMATION**

Additional supporting information may be found online in the Supporting Information section.

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