The publisher regrets the following corrections to the running head, the Prediction Models for External Validation, the legend of Figure 1, the Discussion, and the Author Contributions of the above-stated article. The corrections have also been incorporated in the published article online. The publisher would like to apologize for any inconvenience caused.

The running head is Y Zhang et al.: External Validation of IgAN Prediction Model.

Prediction Models for External Validation
The published prediction models for validation were derived as follows\textsuperscript{11}:

Predicted risk (time $t$) = $1 - S_0(t)^{\text{Exp (linear predictor)}}$.

(1) For the full model without race:

Linear predictor $= -0.320 \times \text{squ}(\text{eGFR}) - 8.8 + 0.002 \times (\text{MAP} - 97) - 0.035 \times \log(\text{proteinuria}) - 0.09 + 0.004 \times (\text{MAP} \times \log(\text{proteinuria}) - 8.73) + 0.201 \times M1 - 0.035 \times E1 + 0.084 \times S1 + 0.700 \times T1 + 1.237 \times T2 + 0.101 \times T1 \times \log(\text{proteinuria}) - 0.321 \times T2 \times \log(\text{proteinuria}) - 0.017 \times (\text{age} - 38) + 0.118 \times \text{RASB} + 0.166 \times \text{RASB} \times \log(\text{proteinuria}) - 0.266 \times \text{immunosuppression}.

$S_0(t) = 1.0003754 - 0.1131641 \times [(t + 0.1) / 100]^2 + 0.0964763 \times [(t + 0.1) / 100]^2 \times \log([t + 0.1] / 100)]$.

(2) For the full model with race:

Linear predictor $= -0.351 \times \text{squ}(\text{eGFR}) - 8.8 - 0.0002 \times (\text{MAP} - 97) - 0.093 \times \log(\text{proteinuria}) - 0.09 + 0.006 \times (\text{MAP} \times \log(\text{proteinuria}) - 8.73) + 0.155 \times M1 - 0.131 \times E1 + 0.097 \times S1 + 0.607 \times T1 + 1.189 \times T2 + 0.109 \times T1 \times \log(\text{proteinuria}) - 0.339 \times T2 \times \log(\text{proteinuria}) - 0.016 \times (\text{age} - 38) + 0.246 \times \text{RASB} - 0.225 \times \text{immunosuppression} - 0.396 \times \text{Chinese race (if } t \leq 36 \text{ months)} + 0.818 \times \text{Chinese race (if } t > 36 \text{ months)} - 0.431 \times \text{Other race}$.

$S_0(t) = 0.9964303 + 0.04392517 \times [(t + 0.1) / 100]^{0.5} - 0.1257002 \times [(t + 0.1) / 100]$.

where log is the natural log function.

DISCUSSION

The 2 reported full prognostic models without and with race\textsuperscript{11} provided us with a useful prediction tool for clinical IgAN progression. In this study, we further evaluated model performance using an external cohort from China and Argentina. For the full models without race and with race, good discrimination (C-statistic $>0.80$) was observed and the models fit well. The survival curves of patients stratified by percentiles of linear predictor were well separated. Our external validation provides further evidence that the clinical and pathologic variables used in the model appeared to be sufficient for patient discrimination. The full model without race showed acceptable calibration. Although the full model with
Figure 1. (a) Enrollment flowchart and (b) cumulative incidence of the primary outcome in the current cohort. (a) The flowchart for patient enrollment. Overall, 1275 of the original 1476 patients remained in the final cohort, including 1169 Chinese patients and 106 Argentinian patients. Among the excluded patients, 92 had other forms of glomerulopathy, 59 were <18 years old, 12 had end-stage renal disease (ESRD) at the time of renal biopsy, 22 were without an available MEST score, and 16 lacked medication information. (b) The primary outcome was 50% decline in estimated glomerular filtration rate or ESRD. MEST, M = mesangial hypercellularity, E = endothelial hypercellularity, S = segmental sclerosis, T = tubular atrophy and interstitial fibrosis; RASB, renin-angiotensin system blocker.

| Race       | Number of Patients |
|------------|--------------------|
| Chinese    | 1169               |
| Argentinian| 106                |

race showed a similar regression slope on linear predictor (0.89), it seemed to overestimate the prognostic risk over 3 years.

Compared with the vigorous progress in model development, only about 25% (32 of 127) of them have been validated and few have been used in clinical practice.\textsuperscript{13} Although there is an increasing number of clinical prediction models for IgAN, most were performed in single ethnicity and few were approved to accurately identify high-risk patients.\textsuperscript{25–30} Considering that the diagnosis of IgAN depends on renal biopsy, a prediction model with histologic variants would help increase the model accuracy. However, although there were some prediction models developed with pathologic variables, these models either developed with a relatively earlier cohort or in a single population. Moreover, some of these models used different pathologic scoring systems that are not widely used. For example, Goto et al. derived a prediction model based on the Japanese population, with both clinical variables and pathologic variables that were not internationally committed.\textsuperscript{31,32} Chen et al. used the XGBoost system and stepwise Cox regression to develop a prediction model, based on Chinese patients at different centers.\textsuperscript{33} The predictors included demographic, clinical, and pathologic variables. However, this model was based on a single ethnic population, and external validation based on a non-Asian population is not available. In this context, 2 full models integrating clinical variables and Oxford MEST histologic scores were derived and validated in 2 multiple ethnic cohorts.\textsuperscript{11} The well-established factors for disease progression of IgAN, including eGFR, proteinuria, blood pressure, Oxford MEST histologic scores, age, and use of RASB/immunosuppressant, could be “easy” and consistently obtained in clinical practice, demonstrating its potential in clinical practice.

In this study, we have further performed external validation of the full models in a Chinese-Argentinian cohort from a relatively “new era.” Notably, compared with the reported cohorts, there was a significantly higher rate of RASB initiation before
diagnosis (30% vs. 70%). This strengthens the analysis because our current cohort is much more representative of patients receiving the current treatment regimens. We found that the prediction models consistently performed well for discrimination in our validation cohort. Our subgroup analysis suggests that survival curves of different risk groups were quite well-separated in both models. Accordingly, the eGFR decline slope was relatively larger with risk increasing. This was consistent with the worse predictor variables, such as more proteinuria, more Oxford MEST lesions, and lower eGFR, across the risk groups. In this way, the “simple, robust” models validated in our cohort could be applied to improve risk prediction, which was approved to both increase treatment allocation to patients at high risk of disease progression and avoid treatment in patients with nonprogressive disease.34 Moreover, for the full model without race, the calibration was acceptable. However, for the full model with race, it seemed to overestimate the prediction risk over 3 years in our cohort. Considering that, for a given high-risk clinical decision, a well-calibrated model providing a wider risk stratification is likely to have greater clinical utility, we suggest using the full model without race for further assessment of setting the thresholds, which is used to estimate the benefits and costs of specific interventions.

The strength of this study is that we did external validation based on a Chinese/Argentinian population from a relatively “new era,” which enabled us to validate the performance of the full models in the populations with treatment under current guidelines. However, there are also some limitations. First, our final cohort excluded patients who did not have a renal biopsy performed or for whom the Oxford MEST histologic scores were not available, meaning that we may have missed some very high-risk patients because the Oxford MEST histologic scores were hard to evaluate due to few glomeruli in the biopsy.7 Second, there was a large proportion of Chinese patients in our Chinese/Argentinian validation cohort; the model performance in other new ethnic populations is still needed. Furthermore, recalibration of the full model with race is to be evaluated in the future. Third, a limitation of the prediction model is that, taking into account that IgAN is an entity (not a disease) with a long-term evolution, the model offers only short-term prognosis, up to 8 years at the most.

In summary, we externally validated the full prediction models to risk stratify patients after an initial diagnosis of IgAN. The prediction models showed good performance on personalized risk assessment, which will help allocate immunosuppression to those patients at high risk of disease progression and avoid treatment in those with nonprogressing disease.

**AUTHOR CONTRIBUTIONS**

YMZ and JCL designed the study; LG, ZW, and HT acquired and cleaned the data; YMZ, JWW, LE, and SJB analyzed and interpreted the data; and YMZ and LG drafted the manuscript. All authors assisted in revision of the work and approved the final version submitted for publication.