

# The five-point Likert scale for dyspnea can properly assess the degree of pulmonary congestion and predict adverse events in heart failure outpatients

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**OBJECTIVES:** Proper assessment of dyspnea is important in patients with heart failure. Our aim was to evaluate the use of the 5-point Likert scale for dyspnea to assess the degree of pulmonary congestion and to determine the prognostic value of this scale for predicting adverse events in heart failure outpatients.

**METHODS:** We undertook a prospective study of outpatients with moderate to severe heart failure. The 5-point Likert scale was applied during regular outpatient visits, along with clinical assessments. Lung ultrasound with  $\geq 15$  B-lines and an amino-terminal portion of pro-B-type natriuretic peptide (NT-proBNP) level  $>1000$  pg/mL were used as a reference for pulmonary congestion. The patients were then assessed every 30 days during follow-up to identify adverse clinical outcomes.

**RESULTS:** We included 58 patients (65.5% male, age  $43.5 \pm 11$  years) with a mean left ventricular ejection fraction of  $27 \pm 6\%$ . In total, 29.3% of these patients had heart failure with ischemic etiology. Additionally, pulmonary congestion, as diagnosed by lung ultrasound, was present in 58% of patients. A higher degree of dyspnea (3 or 4 points on the 5-point Likert scale) was significantly correlated with a higher number of B-lines ( $p=0.016$ ). Patients stratified into Likert = 3-4 were at increased risk of admission compared with those in class 1-2 after adjusting for age, left ventricular ejection fraction, New York Heart Association functional class and levels of NT-proBNP  $>1000$  pg/mL (HR = 4.9, 95% CI 1.33-18.64,  $p=0.017$ ).

**CONCLUSION:** In our series, higher baseline scores on the 5-point Likert scale were related to pulmonary congestion and were independently associated with adverse events during follow-up. This simple clinical tool can help to identify patients who are more likely to decompensate and whose treatment should be intensified.

**KEYWORDS:** Heart Failure; Dyspnea; Likert Scale.

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## INTRODUCTION

Dyspnea is defined as the subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. This condition is caused by interactions among multiple physiological, psychological, social and environmental factors and may induce secondary physiological and behavioral responses (1). The objective quantification of dyspnea in heart failure (HF) patients is limited by the subjective reporting of this common symptom. Moreover, there is no consensus regarding the

best way to measure and quantify the subjective sensation of "shortness of breath", making this task extremely difficult for patients and healthcare professionals (2).

The 5-point Likert scale (5PLS) for dyspnea is a psychometric instrument for the measurement and grading of dyspnea (1,3,4). Certain authors recommend the use of this scale to assess patients with acute decompensated HF (5-7). Others state that the best way to measure dyspnea involves evaluating quality of life; however, the questionnaires used for this type of assessment are lengthy and require more experienced and well-trained researchers. Thus, such questionnaires are usually not well suited for daily clinical practice. Taken together, these issues justify the use of simpler instruments to properly grade dyspnea (8-10).

Lung ultrasound (LUS) through B-line evaluation (formerly referred to as ultrasound lung comets) has been proposed as a simple, noninvasive and semiquantitative tool for the assessment of extravascular lung water (11,12). A recent study (13) of chronic HF outpatients demonstrated

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the reliability of this noninvasive method in identifying clinically silent pulmonary edema and predicting adverse outcomes (14). In HF, the amino-terminal portion of pro-B-type natriuretic peptide (NT-proBNP) is a powerful neurohormonal predictor of prognosis and elevated left ventricular (LV) filling pressures, and higher levels are correlated with greater degrees of pulmonary congestion (15-17).

To date, no study has evaluated the intensity of dyspnea quantified by 5PLS for the estimation of significant pulmonary congestion (SPC) in chronic HF outpatients (18). The aim of this study was to determine whether dyspnea graded using a 5PLS can be used to objectively define SPC and predict the occurrence of adverse events in chronic HF outpatients, in comparison with the use of LUS, NT-proBNP levels and New York Heart Association (NYHA) functional class.

## ■ MATERIALS AND METHODS

### Study design and population

Single-center cross-sectional study followed by cohort study of 58 patients with systolic HF who were followed at a pre-transplant outpatient clinic at the Cardiology Institute of Rio Grande do Sul, Brazil, between November 2011 and January 2012. The inclusion criteria were as follows: 1) age >18 years; 2) a diagnosis of LV systolic HF for more than 6 months, regardless of the cause, as defined by the Framingham criteria (19) and satisfying the European Society of Cardiology's guidelines (20); 3) moderate to severe systolic HF (ejection fraction (EF) <45%); 4) no prior diagnosis of pulmonary fibrosis; 5) the absence of congenital heart disease; and 6) no recent admission due to decompensated HF in the previous 6 months.

Clinical assessment, NT-proBNP analysis, LUS, echocardiography and chest X-ray (CXR) were independently performed by different physicians at the first clinical appointment (index evaluation), with a maximum 5-hour time delay between the first and last examinations. Following a brief explanation provided by a nurse, all patients filled out a 5PLS immediately before and after undergoing a 6-minute walk test (6MWT) in accordance with standardized methodology.

### The 5PLS for dyspnea

The 5PLS for dyspnea is a self-administered psychometric instrument for measuring and grading the experience of breathing discomfort that has been validated for use in acute decompensated HF patients (1-4). This scale can be used to verify changes in the intensity of shortness of breath between two different time points. The scale includes the absence of dyspnea (a score of 1), mild shortness of breath (a score of 2), moderate shortness of breath (a score of 3), severe shortness of breath (a score of 4) and the worst possible shortness of breath (a score of 5). All patients filled out the 5PLS without any interference after a brief explanation provided by a nurse.

### LUS, NT-proBNP measurement and clinical assessment

LUS was used to assess the presence of B-lines (12,21). A B-line was defined as a discrete laser-like vertical hyper-echoic reverberation artifact that arises from the pleural line, extends to the bottom of the screen without fading and

moves synchronously with lung sliding (22). We analyzed the anterior and lateral hemithoraces, scanning along the parasternal, midclavicular, anterior axillary and mid-axillary lines, as previously described (22). A total of 28 chest sites were scanned, and the total number of B-lines was recorded by an independent investigator. A B-line number  $\geq 15$  was considered as the cut-off for SPC (13).

An NT-proBNP analysis was performed using the Elecsys® 2010 analyzer (Roche Diagnostics, Mannheim, Germany). An NT-proBNP level >1000 pg/mL was used as the cut-off for decompensated HF (17,23).

### Statistical analysis

Continuous variables are expressed as the mean  $\pm$  standard deviation or as the median (25<sup>th</sup>-75<sup>th</sup> percentiles), as appropriate. We used Student's t-test for continuous variables with symmetric distribution and the chi-square test or Fisher's exact test for categorical variables. For asymmetrical variables, the Mann-Whitney test and ANOVA were used for repeated measures. The correlation between the 5PLS score and the number of B-lines was assessed using non-parametric Spearman's correlation analysis. Additionally, multivariate analysis was used to verify the differences between the different 5PLS scores, and logistic regression models were employed. First, separate analyses were performed for individual variables to allow for the largest possible sample size. Second, stepwise models were run using all of the potential risk factors identified in the univariate analysis. Finally, a model was run using a backward stepwise (likelihood) method that included variables meeting the following entry criteria: a *p*-value of 0.05 and a removal value of 0.20 in the prior regression analysis. A two-tailed *p*-value <0.05 was considered to be statistically significant. The cumulative incidence of hospital admission during follow-up was analyzed individually and collectively using the Cox proportional hazard regression model. Kaplan-Meier survival analysis with the log-rank test was also used. All statistical analyses were performed using IBM SPSS Statistics version 19.0.0 (Chicago, IL, USA).

### Ethics

The study protocol was approved by the ethics committee of our institution (protocol 4720/12). All of the participants provided informed consent, and the study complied with the Declaration of Helsinki.

## ■ RESULTS

The main characteristics of the patients are listed in Table 1. The mean age was  $49 \pm 11$  years, and 47.2% of the patients had idiopathic dilated cardiomyopathy. The mean LV EF, as determined by the bi-planar Simpson method, was  $27 \pm 4\%$ , and the left atrial volume indexed to body surface area was  $53.8 \pm 27$  mL/m<sup>2</sup>. The patients were followed up for a median period of  $106 \pm 12$  days (inter-quartile range: 89-115 days).

SPC was diagnosed in 44.8% of subjects, based on an NT-proBNP level >1,000 pg/mL. In total, 58% of patients had SPC, as diagnosed by LUS ( $\geq 15$  B-lines).

When the 5PLS for dyspnea was applied during rest, 44.8% of patients had a score of 1, 32.7% had a score of 2, 17.2% had a score of 3, and 5.17% had a score of 4. None of

**Table 1 - Primary patient characteristics.**

Characteristic	N= 58 (%)
Age	49 ± 11
Caucasian	46 (79)
Male	38 (65)
Body mass index	28 ± 4
Heart rate (beats per minute)	74 ± 12
Systolic arterial pressure (mmHg)	112 ± 17
Diastolic arterial pressure (mmHg)	71 ± 11
Coronary artery disease	16 (27)
Myocardial infarction	15 (26)
Hypertension	24 (41)
Dyslipidemia	26 (49)
COPD	1 (2)
Diabetes mellitus	12 (20)
Minnesota score	43 ± 22
Cardiomyopathy etiolog	
Idiopathic	29 (50)
Post-ischemic	17 (29)
Hypertension	6 (10)
Myocarditis	4 (7)
Other	2 (4)
NYHA functional class	
I	15 (26)
II	24 (41)
III	13 (23)
IV	6 (10)
Left ventricular ejection fraction (%)	27 ± 0.9
Left atrial volume (mL/m <sup>2</sup> )	54 ± 27
Electrocardiogra	
Sinus rhythm	46 (79)
Atrial fibrillation	10 (17)
Pace	2 (3)
QRS ≥ 150 ms	35 (60)
Creatinine/CC	0.9 ± 0.3/109 ± 40
Chronic renal failure	7 (12)
Urea (mg/dL)	48 ± 27
Potassium (meq/L)	4.4 ± 0.5
Sodium (meq/L)	139 ± 4
Hematocrit (/)Hemoglobin (g/dL)	40 ± 4/13 ± 1.4
β-blocker	56 (96)
Target dose	22 (38)
ACE inhibitor	38 (65)
Target dose	12 (20)
Angiotensin II receptor blocker	15 (26)
Target dose	9 (15)
Aldosterone antagonist	42 (72)
Diuretics	42 (72)
Digoxin	34 (58)
Calcium antagonists	3 (5)
Nitrates	10 (17)
Hydralazine	4 (0.7)
Statin	27 (46)
Aspirin	24 (41)
Coumarin	10 (17)
Amiodarone	8 (14)
Pace/ICD	9 (15)
CRT	4 (7)

The data are presented as the mean ± standard deviation or n(%).

ACE: Angiotensin-converting enzyme; CC: Creatinine clearance; COPD: Chronic obstructive pulmonary disease; CRT: Cardiac resynchronization therapy; ICD: Implantable cardiac defibrillator; NYHA: New York Heart Association.

the patients had a score of 5 points on the scale when it was administered during rest.

The mean NYHA functional class was higher for those patients who had higher scores on the 5PLS for dyspnea ( $p < 0.001$ ). The mean NYHA functional class was 3.67 for Likert=4, 3 for Likert=3, 2 for Likert=2 and 1.87 for

Likert = 1. There were no significant differences in mean NT-proBNP levels between groups with different perceptions of dyspnea ( $p = 0.16$ ). After the 6MWT, there was a significant increase in the mean score on the 5PLS for dyspnea compared with the mean score at rest ( $p < 0.0001$ ) (Figure 1).

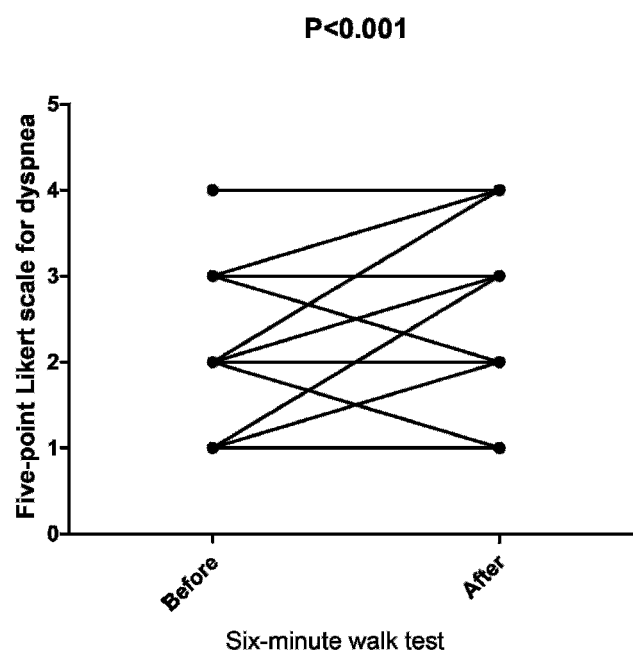
When separated based on their score on the 5PLS for dyspnea, patients who had a score of 4 had more B-lines identified by LUS than in the other groups ( $p = 0.016$ ) (Figure 2).

Adverse events occurred in 15 patients during follow-up. Twelve of the patients were hospitalized with pulmonary congestion. In addition, two were hospitalized with acute myocardial infarction (AMI), and one experienced an implantable cardiac defibrillator (ICD) shock due to ventricular tachycardia.

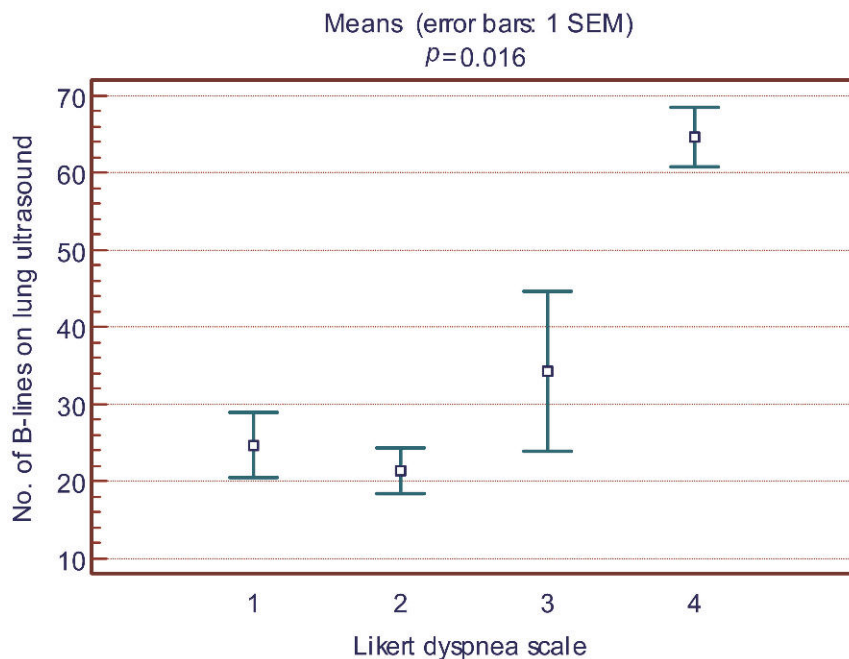
Adverse event-free survival was higher in patients with lower scores on the 5PLS for dyspnea (log-rank  $p < 0.001$ ). Patients with Likert scores of 3 or 4 were at higher risk of emergency room admission during follow-up than were those with scores of 1 or 2 after adjusting for factors such as age, LV EF, NYHA functional class and levels of NT-proBNP >1000 pg/mL (HR=4.9, 95% CI 1.33-18.64,  $p = 0.017$ ) (Figure 3).

## DISCUSSION

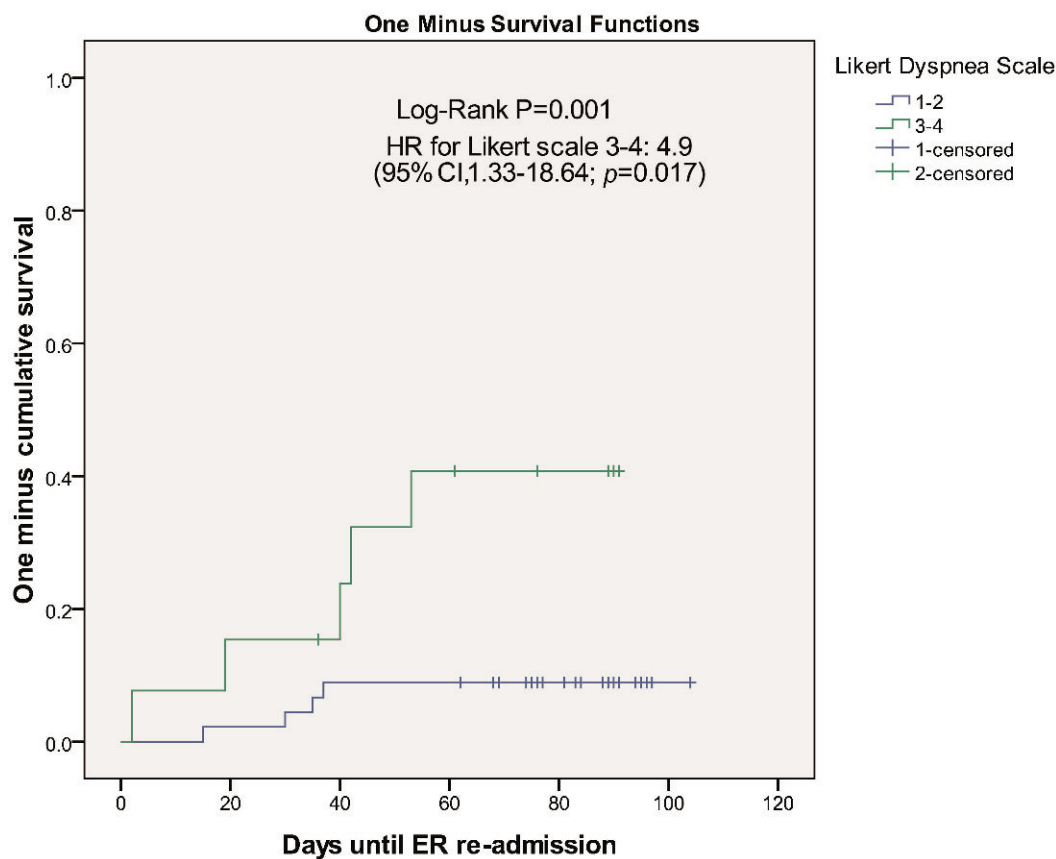
Our results demonstrate that there is an objective relationship between the score on the 5PLS for dyspnea, which assesses the severity of symptoms perceived by the patient, and higher levels of extravascular lung water, as assessed based on LUS and elevated LV filling pressure and NT-proBNP levels. However, a previous study that investigated the relationships between patient history, physical examination results, CXR findings and pulmonary capillary pressure in 42 patients with HF who were referred for assessment for a heart transplant showed that the clinical, X-ray and hemodynamic assessments failed because they



**Figure 1 - Five-point Likert scale for dyspnea before and after effort.**



**Figure 2** - Number of B-lines estimated by lung ultrasound in patients with different dyspnea scores measured by the 5-point Likert scale for dyspnea. ANOVA:  $p=0.016$ . The Student-Newman-Keuls test for all pairwise comparisons revealed a difference between those patients who had a score of 4 on the 5-point Likert scale for dyspnea and those with other scores ( $p<0.05$ ).



**Figure 3** - Kaplan-Meier 1-cumulative survival curve for the emergency room admission of patients stratified into two groups based on the 5-point Likert scale for dyspnea: patients with scores of 1-2 and patients with scores of 3-4.





yielded conflicting results (19). These authors justified this conclusion based on the fact that patients with an elevated pulmonary capillary pressure may show no congestion during a clinical or radiological exam, and the absence of congestion on an X-ray is associated with a better pulmonary capacity. This discordance among clinical, radiological and hemodynamic results has implications for the management of patients with HF and could lead to inaccurate diagnoses and inadequate treatment (24). Echocardiography can also be used to noninvasively measure LV filling pressures (12); the ratio of the early diastolic mitral inflow velocity to the early diastolic velocity of the mitral annulus ( $E/e'$ ) is one of the most frequently used parameters for the assessment of LV pressures (13,14). Both natriuretic peptides and  $E/e'$  can be used to identify hemodynamic congestion in systolic and diastolic HF patients, a condition that precedes pulmonary congestion. Furthermore, pulmonary congestion is usually, but not always, related to high LV filling pressure, which may explain the absence of a correlation with NT-proBNP levels (25).

HF causes functional, metabolic and histological changes in the inspiratory muscles. In 30-50% of patients, these muscles are weak, with a consequent reduction in functional capacity, a worse clinical prognosis and a lower quality of life for these individuals. Furthermore, there is a relationship between the autonomic control systems of these patients and intolerance to exercise, in addition to weakness of the inspiratory muscles, which, according to previously performed studies, can be improved with physical training (25). This phenomenon plays an important role in the sensation of shortness of breath during and after exercise.

We believe that the 5PLS can transform the sensation of dyspnea (subjective data) into objective data because in our patient population, we were able to demonstrate an excellent relationship between the Likert scores and the pulmonary ultrasound findings used to quantify the amount of free fluid in the lungs of this cohort of patients with advanced HF. However, this relationship was not observed when the scale was used after exercise. This finding may be due to the muscle component of dyspnea, which plays a major role in the chronically ill patient population, especially after physical activity. Thus, the severity of dyspnea measured by the Likert scale is only directly correlated with pulmonary congestion when used to assess patients at rest.

Perhaps the most striking finding of our study is that patients who had grade 3 or 4 dyspnea (defined by the 5PLS for dyspnea as moderate and strong, respectively) had a greater number of hospital admissions. This correlation remained statistically significant even after controlling for clinical factors that are already known to predict adverse outcomes, such as the NYHA functional class, the EF and NT-proBNP levels. In our view, dyspnea must be measured objectively when obtaining a patient's history during the initial clinical exam because dyspnea is the most common and significant symptom of distress in patients with HF. This symptom is still an important therapeutic target, and its measurement should influence treatment (7,8). Despite the reduced number of patients receiving the target dose of HF medication, similar to what has been reported in the literature, no relationship was found with the occurrence of adverse events.

Another important observation is that the Likert scale can even help to distinguish patients who are in the same functional class (e.g., NYHA III) by objectively grading the intensity of dyspnea at the same level of effort. This distinction can independently predict clinical outcomes during follow-up, as demonstrated by multivariate analysis.

Patients with HF generally present signs and symptoms of pulmonary systemic congestion when they are admitted to the hospital. However, high LV filling pressures may be present long before the development of clinical pulmonary congestion that results in hospital admission. Thus, it is necessary to manage and reassess pulmonary and systemic congestion at each clinical encounter (26).

The 5PLS for dyspnea used in this study is easy to apply and can be used by any healthcare professional (e.g., nurses, physicians, physician's assistants and respiratory therapists) in HF outpatient clinics. This simple score for the assessment of dyspnea has an objective correlation with pulmonary congestion and has prognostic implications.

One limitation of this study is the small size of the sample, which included only patients with moderate to severe systolic dysfunction. This limitation should be taken into account when interpreting the results. Moreover, the lack of a gold standard to verify the presence or absence of pulmonary congestion in the outpatient environment is a further limitation of the study. However, the utilization of the NT-proBNP level and the  $E/e'$  relationship, rather than invasive measures, made it possible to perform the study during routine outpatient visits with patients in a stable clinical condition. Additionally, the change in the 5PLS score after the 6MWT was not tested in comparison with other methodologies to determine whether the score was correlated with increased extravascular lung water after the exercise test.

In our cohort of HF patients, the 5PLS for dyspnea was able to predict adverse clinical events, even when controlling for potential biases (NYHA functional class, EF, age and NT-proBNP levels). Higher scores on the 5PLS for dyspnea were associated with unfavorable clinical outcomes in patients with HF. The scale was also capable of quantifying pulmonary congestion objectively. Healthcare professionals can use this instrument to better assess patients and improve their care, thereby potentially reducing the rate of hospital admissions.

## ■ AUTHOR CONTRIBUTIONS

Weber CK performed patient evaluations and data collection. Miglioranza MH participated in study design and conception, database construction and manuscript writing. Moraes MA performed patient evaluations and data collection. Sant'anna RT performed patient evaluations and data collection and wrote the first draft of the manuscript. Rover MM performed patient evaluations and data collection and participated in study design. Kalil RA participated in study design. Leiria TL participated in study design and data review, wrote the final manuscript and performed the statistical analysis.

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