GSC Biological and Pharmaceutical Sciences
eISSN: 2581-3250 CODEN (USA): GBPSC2
Cross Ref DOI: 10.30574/gscbps
Journal homepage: https://gscbps.gsconlinepress.com/

(RESEARCH ARTICLE)

# EFFECTS OF POST-EXERCISE CURCUMIN SUPPLEMENTATION ON BLOOD GLUCOSE LEVELS FOLLOWING ACUTE HIGH-INTENSITY RESISTANCE EXERCISE: A RANDOMIZED CONTROLLED PILOT TRIAL

Sabila Camelia Hidayat [1], Lina Lukitasari [2] and Sundari Indah Wiyasihati [2,*]

[1] Medicine Program, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
[2] Department of Physiology and Medical Biochemistry, Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia
* Corresponding Author

ORCID Details
Sabila Camelia Hidayat: https://orcid.org/0009-0004-4031-2778
Lina Lukitasari: https://orcid.org/0000-0002-9413-2949
Sundati Indah Wiyasihati: https://orcid.org/0000-0003-3905-5327

GSC Biological and Pharmaceutical Sciences, 2026, 36(03), 057–063

Publication history: Received on 26 July 2026; revised on 05 September 2026; accepted on 07 September 2026

Article DOI: https://doi.org/10.30574/gscbps.2026.36.3.0309



## ABSTRACT

**Background:** High-intensity exercise acutely elevates blood glucose through catecholamine-driven hepatic glucose production and NF-κB-mediated inflammation. Curcumin inhibits NF-κB signalling and may attenuate post-exercise hyperglycaemia, yet its efficacy following a single high-intensity bout in non-diabetic individuals remains unexplored. This study evaluated the effect of a single oral curcumin dose administered 24 hours after acute high-intensity resistance exercise on blood glucose in healthy untrained adult males.

**Methods:** A randomized placebo-controlled pilot trial was conducted to evaluate blood glucose. Fourteen males were divided into two groups. Group K1 received placebo, and group K2 was administered 400 mg curcumin. All participants performed a single session of lower-limb resistance exercise (4 sets × 10 repetitions; 80-90% 1RM) for approximately 40 minutes (including a 10-minute warm-up). After 24 hours of exercise, placebo and 400 mg curcumin were administered as a single oral dose. After 24 hours of supplementation, blood glucose was measured at four time-points and analysed using the Wilcoxon signed-rank test and paired t-test.

**Results.** There was no significant blood glucose reduction post-exercise after a 200 mg single-dose curcumin ($p \geq 0,05$).

**Conclusion:** Single-dose curcumin did not reduce post-exercise blood glucose, though a decreasing trend warrants further investigation with higher doses and larger samples.

**Keywords:** Curcumin; Blood Glucose; Resistance Exercise; NF-κB; Post-Exercise; Healthy Lifestyle.

## 1 INTRODUCTION

Diabetes mellitus represents one of the most pressing global health crises of the 21st century, with an estimated 537 million adults living with the disease in 2021 and projections reaching 783 million by 2045[1]. In Indonesia, the prevalence has risen markedly, with approximately 19.5 million adults affected, placing the country among the top five globally for absolute diabetes burden [1, 2]. The pathophysiological hallmarks of type 2 diabetes mellitus (T2DM), insulin resistance, impaired glucose uptake, and chronic low-grade inflammation, are increasingly recognised as targets amenable to lifestyle-based interventions, particularly structured physical exercise [3].

High-intensity exercise (HIE) is widely practised among competitive athletes and recreational exercisers owing to its superior metabolic and cardiovascular adaptations relative to moderate-intensity continuous training [4, 5]. However, a paradoxical acute glycaemic response has been documented following HIE: rather than the glucose-lowering effect commonly associated with aerobic exercise, single bouts of HIE can transiently elevate blood glucose due to catecholamine-mediated hepatic glycogenolysis and gluconeogenesis that outpace peripheral glucose uptake [6, 7]. This exercise-induced hyperglycaemic overshoot, if repetitively experienced, may impair long-term glycaemic regulation, particularly in individuals predisposed to metabolic dysfunction [7].

Mechanistically, HIE promotes the generation of reactive oxygen species (ROS) and activates the nuclear factor kappa-B (NF-κB) signalling cascade in skeletal muscle, leading to upregulation of pro-inflammatory cytokines including tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β) [8]. TNF-α and related pro-inflammatory cytokines disrupt insulin receptor substrate-1 (IRS-1) phosphorylation, attenuate glucose transporter 4 (GLUT-4) translocation, and impair insulin-stimulated glucose uptake in skeletal muscle, collectively contributing to the acute post-exercise glycaemic disturbance [9].

Curcumin (diferuloylmethane; 1,7-bis(4-hydroxy-3-methoxyphenyl)-1,6-heptadiene-3,5-dione), the principal bioactive polyphenol of Curcuma longa (turmeric), has attracted considerable research interest as a multi-target anti-inflammatory and antioxidant agent [10]. Curcumin inhibits IκB kinase (IKK)-mediated phosphorylation of IκB, thereby preventing NF-κB nuclear translocation and downstream transcription of pro-inflammatory genes including TNF-α and cyclooxygenase-2 (COX-2) [11]. In skeletal muscle cell lines (L6 myotubes), curcumin has been shown to enhance GLUT-4 translocation and protein kinase B (AKT) phosphorylation, suggesting a direct insulin-sensitising effect independent of its anti-inflammatory properties, though its oral bioavailability remains a key determinant of clinical efficacy [12].

Prior clinical investigations have predominantly studied curcumin supplementation in populations with established metabolic disease (T2DM, non-alcoholic fatty liver disease, metabolic syndrome) over intervention periods of 8-12 weeks [13]. The efficacy of short-term, single-dose curcumin supplementation in the specific context of post-HIE glucose dysregulation in metabolically healthy individuals has not been investigated. Furthermore, the dosing of 400 mg/day of Longvida® optimised curcumin, a phosphatidylcholine-complexed formulation with superior bioavailability, was selected based on evidence from McFarlin et al. [14], who demonstrated significant attenuation of TNF-α following this dose after exercise-induced muscle damage.

Identifying effective nutritional strategies to mitigate post-HIE glycaemic perturbations is of public health significance, particularly given the rising prevalence of pre-diabetes and the growing adoption of high-intensity training protocols across all fitness levels. Accordingly, the present study aimed to determine whether a single oral dose of curcumin (400 mg) administered 24 hours after an acute bout of high-intensity resistance exercise could attenuate blood glucose concentrations in healthy, untrained adult males. We hypothesised that curcumin supplementation would produce a greater reduction in blood glucose from T2 to T3 compared with placebo.

## 2 MATERIAL AND METHODS

A randomized, placebo-controlled, parallel-group pilot trial was conducted at the Faculty of Medicine, Universitas Airlangga, Surabaya, Indonesia (July-August 2023; Ethics No. 118/EC/KEPK/FKUA/2022) in accordance with the Declaration of Helsinki 2013.

Fourteen healthy untrained males (age 21-30 years; BMI 18.5-24.9 kg/m$^2$) were randomly allocated to placebo (K1; n = 6) or curcumin (K2; n = 8) groups. Inclusion criteria included normal Body Mass Index (BMI), no structured resistance training for ≥6 months, and no use of anti-inflammatory or antidiabetic agents. On Day 1, following an overnight fast (≥8 hours) and baseline blood glucose measurement (T0), all participants performed a single session of lower-limb resistance exercise comprising smith machine squat and leg press (4 sets × 10 repetitions; 80-90% 4 sets × 10 repetitions at 80–90% of the pre-tested 1-repetition maximum (1RM); 60-second inter-set rest; total exercise duration approximately 40 minutes including a 10-minute warm-up), with blood glucose recorded immediately post-exercise (T1). On Day 2 (24 hours post-exercise), pre-intervention blood glucose was measured (T2); K2 then received a single

oral dose of curcumin (400 mg Longvida®, phosphatidylcholine-complexed formulation) and K1 received an identical placebo capsule. On Day 3 (48 hours post-exercise; 24 hours post-intervention), final blood glucose was measured (T3). Blood glucose was assessed via fingertip capillary sampling (EasyTouch® glucometer) at all four time-points.

Data were analysed. Normality of data was analysed using the Shapiro-Wilks test and homogeneity analysis using the Levene test. Within-group changes in blood glucose were analysed by the Wilcoxon signed-rank test (for non-normally distributed data) and paired t-test (for normally distributed data); significance threshold $p < 0.05$. The SPSS program version 24 was used to analyze the data.

## 3 RESULT

### 3.1 Participant Flow and Baseline Characteristics

Fourteen participants were enrolled and randomized: K1 (placebo, n = 6) and K2 (curcumin, n = 8). All 14 participants completed the three-day protocol; there were no dropouts or protocol deviations. All baseline characteristics were normally distributed ($p \geq 0.05$). No significant differences between-group were observed for age, body weight, height, or BMI ($p = 0.728$) (Table 1).

**Table 1: Baseline Characteristics of Study Participants**

| Characteristic | K1 (Placebo)n = 6 | K2 (Curcumin)n = 8 | p-value |
|---|---|---|---|
| Age (years) | 22.16 ± 1.60 | 23.12 ± 1.89 | 0.337 |
| Body weight (kg) | 57.91 ± 5.89 | 61.62 ± 5.64 | 0.255 |
| Height (m) | 1.66 ± 0.05 | 1.71 ± 0.07 | 0.255 |
| BMI ($kg/m^2$) | 20.81 ± 1.40 | 21.06 ± 1.17 | 0.728 |

### 3.2 Acute Glycaemic Response to Exercise (T0 → T1)

Blood glucose decreased from pre-exercise to immediately post-exercise in both groups (Table 2). Neither within-group change was statistically significant.

**Table 2: Blood Glucose Levels Before and After High-Intensity Exercise**

| Group | n | Pre-exercise T0 (mmol/L (mg/dL), mean ± SD) | Post-exercise T1 (mmol/L (mg/dL), mean ± SD) | Change (mmol/L (mg/dL)) | p-value* |
|---|---|---|---|---|---|
| K1 (Placebo) | 6 | 6.38 ± 2.27 (115.00 ± 40.90) | 5.51 ± 0.74 (99.33 ± 13.34) | -0.87 (-15.67) | 0.463 |
| K2 (Curcumin) | 8 | 7.40 ± 2.48 (133.33 ± 44.63) | 5.56 ± 1.67 (100.17 ± 30.15) | -1.84 (-33.17) | 0.069 |
| Between-group p** | - | 0.796 | 0.477 | - | - |

*Wilcoxon signed-rank test (within-group). **Mann–Whitney U test (between-group).

### 3.3 Effect of Curcumin on Post-Exercise Blood Glucose (T2 → T3)

Table 3 shows the effect of curcumin supplementation on post-exercise blood glucose between T2 and T3. At T2, the mean blood glucose level was 6.28 ± 1.05 mmol/L (113.17 ± 18.99 mg/dL) in the placebo group (K1) and 5.81 ± 1.07 mmol/L (104.67 ± 19.35 mg/dL) in the curcumin group (K2). The independent t-test showed no significant difference between groups at T2 ($p = 0.525$), indicating that both groups were comparable prior to the intervention.

The paired t-test showed no significant change in blood glucose from T2 to T3 in either group. In K1, blood glucose decreased slightly by 0.03 mmol/L (0.50 mg/dL) ($p = 0.947$), while in K2 it decreased by 0.17 mmol/L (3.00 mg/dL) ($p = 0.395$). Although the reduction in K2 was greater and in the direction expected for a hypoglycemic effect of curcumin, the difference was not statistically significant, likely due to the limited sample size in this pilot study.

**Table 3: Blood Glucose Levels Before and After Curcumin/Placebo Intervention (Primary Outcome)**

| Group | n | Pre-intervention T2 (mmol/L (mg/dL), mean ± SD) | Post-intervention T3 (mmol/L (mg/dL), mean ± SD) | Mean Diff (mmol/L (mg/dL)) | Cohen's d | p-value* |
|---|---|---|---|---|---|---|
| K1 (Placebo) | 6 | 6.28 ± 1.05 (113.17 ± 18.99) | 6.25 ± 0.27 (112.67 ± 4.84) | -0.03 (-0.50) | 0.04 | 0.947 |
| K2 (Curcumin) | 8 | 5.81 ± 1.07 (104.67 ± 19.35) | 5.64 ± 1.15 (101.67 ± 20.70) | -0.17 (-3.00) | 0.15 | 0.395 |
| Between-group p** | - | 0.525 | 0.234 | - | - | - |

Data presented as mean ± SD. *Paired-samples t-test (within-group). **Independent-samples t-test (between-group). Cohen's d: small effect < 0.5; medium 0.5–0.8; large > 0.8.

### 3.4 Blood Glucose Trajectory Across All Time-Points

Figure 2 illustrates the mean blood glucose trajectory across all four measurement time-points. Both groups showed a decline from T0 to T1, a partial recovery at T2, and a further slight decrease at T3. The decline from T2 to T3 appeared numerically larger in K2 (-0.17 mmol/L [-3.00 mg/dL]) than in K1 (-0.03 mmol/L [-0.50 mg/dL]), consistent with a potential but non-significant curcumin effect within this pilot sample.

Figure 1. CONSORT Flow Diagram. The diagram shows: Assessed for eligibility (n = 14); Excluded: none (n = 0); Randomized (n = 14); Allocated to K1 Placebo (n = 6) group, received allocated intervention (n = 6); Allocated to K2 Curcumin (n = 8) group, received allocated intervention (n = 8); Lost to follow-up: K1 (n = 0), K2 (n = 0); Discontinued: K1 (n = 0), K2 (n = 0); Analysed: K1 (n = 6), K2 (n = 8).

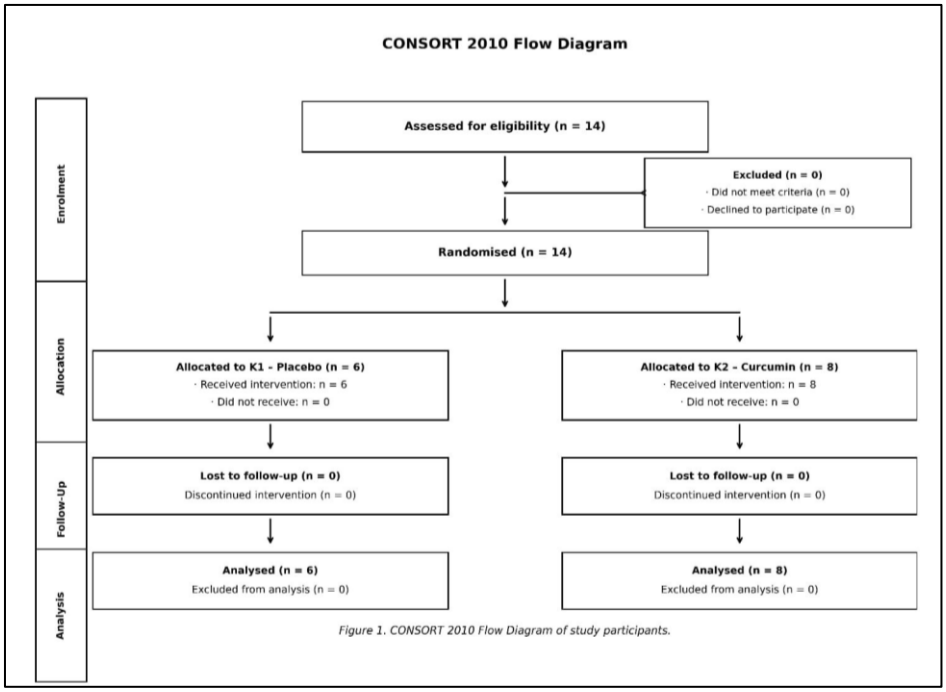


**Figure 1: CONSORT 2010 Flow Diagram.**

Figure 2. Mean Blood Glucose Levels Across Four Time-Points. Line graph with two lines (K1 placebo = dashed; K2 curcumin = solid). X-axis: T0 (Pre-exercise), T1 (Post-exercise), T2 (24h post-exercise/Pre-intervention), T3 (48h post-exercise/24h post-intervention). Y-axis: Blood Glucose (mmol/L (mg/dL)), range 4.44-8.88 (80-160). Error bars represent ± SD. Between-group p-values: T0 = 0.796; T1 = 0.477; T2 = 0.525; T3 = 0.234.

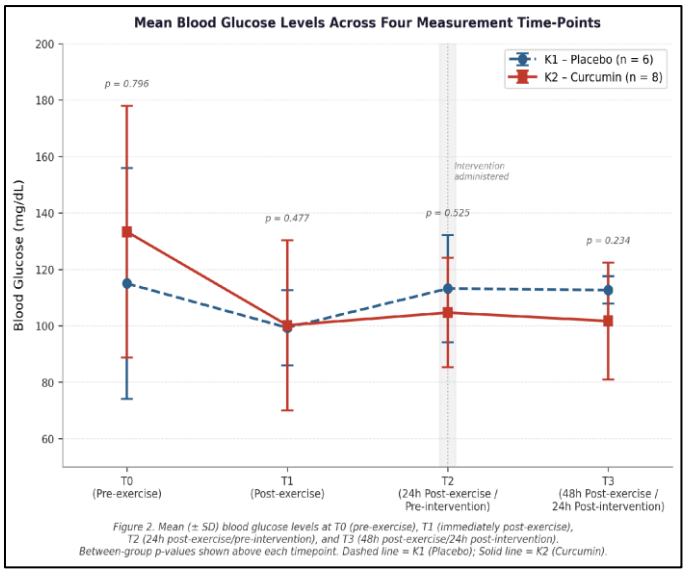


**Figure 2: Mean Blood Glucose Levels Across Four Measurement Time-Points.**

Mean (± SD) blood glucose levels at T0 (pre-exercise), T1 (immediately post-exercise), T2 (24h post-exercise/pre-intervention), and T3 (48h post-exercise/24h post-intervention). Between-group p-values shown above each timepoint. Dashed line = K1 (Placebo); Solid line = K2 (Curcumin).

## 4 DISCUSSION

### 4.1 Principal Findings

The primary finding of this randomized pilot trial is that a single oral dose of 400 mg Longvida® curcumin administered 24 hours after an acute bout of high-intensity resistance exercise did not produce a statistically significant reduction in blood glucose concentrations in healthy, untrained adult males at 24 hours post-administration. A small numerical reduction (-0.17 mmol/L [-3.00 mg/dL]) in the curcumin group versus a negligible change in the placebo group (−0.03 mmol/L [-0.50 mg/dL]) aligned with the hypothesised direction but was of very small magnitude (Cohen's d = 0.15), suggesting that the present study was underpowered to detect such an effect, rather than that the null hypothesis is definitively confirmed.

### 4.2 Comparison with Previous Studies

The absence of a significant glucose-lowering effect in healthy, metabolically normal individuals is consistent with a subgroup analysis from the meta-analysis by Musazadeh et al.[13], which demonstrated that curcumin supplementation was inefficient in controlling short-term glucose fluctuations in individuals with subclinical metabolic anomalies, such as overweight, pre-diabetes, and metabolic syndrome, and was more effective in populations with established glycaemic pathology.

The most directly comparable study is that of Naghizadeh et al. [15], who investigated 12 weeks of HIIT combined with curcumin 2100 mg/day (divided into three doses) in obese men with T2DM and hyperlipidaemia. That study demonstrated a significant reduction in fasting plasma glucose in the combined intervention group. Key methodological differences include the study population (metabolically diseased vs healthy), duration (12 weeks vs single dose), dose (2100 mg vs 400 mg), and exercise protocol (3 sessions/week vs single session), all of which likely account for the divergent findings.

Similarly, Dolati et al. [16] reported that 8 weeks of combined curcumin supplementation (500 mg/day) and aerobic treadmill exercise significantly improved glycaemic status in overweight women compared with either intervention alone. These multi-week designs align with evidence from a recent meta-analysis demonstrating that curcumin's hyperglycaemia-attenuating effects are most pronounced in sustained supplementation protocols [17].

### 4.3 Biological Plausibility and Mechanistic Considerations

The mechanistic rationale for the present study was grounded in the NF-κB/TNF-α pathway through which HIE-induced oxidative stress may impair post-exercise glucose homeostasis. Curcumin's well-characterised inhibition of IKKβ prevents IκB degradation and NF-κB nuclear translocation, thereby attenuating TNF-α expression and downstream

disruption of IRS-1-mediated glucose uptake [11, 18]. Additionally, curcumin may enhance insulin sensitivity via upregulation of peroxisome proliferator-activated receptor gamma (PPAR-γ), stimulation of glucagon-like peptide-1 (GLP-1) secretion, and inhibition of dipeptidyl peptidase-4 (DPP-4) [13]. However, these mechanistic effects are predominantly documented in models of chronic or established insulin resistance, where the inflammatory milieu is substantially more pronounced than in healthy individuals following a single exercise bout.

The bioavailability of curcumin remains a critical limiting factor. Standard curcumin exhibits poor aqueous solubility, rapid hepatic conjugation, and extensive intestinal glucuronidation, collectively yielding very low systemic plasma concentrations after oral administration [10]. The Longvida® formulation used in this study, a phosphatidylcholine-complexed, solid lipid nanoparticle matrix, has demonstrated 65-fold higher plasma curcumin levels compared with standard curcumin [14]. Despite this optimised formulation, a single 400 mg dose may be insufficient to achieve the plasma concentrations required to meaningfully inhibit NF-κB in skeletal muscle within the 24-hour post-administration window in otherwise healthy individuals.

*Limitations*

Several limitations must be acknowledged. The small sample size (N = 14) substantially limited statistical power to detect small-to-moderate effect sizes; this is a pilot study, and confirmatory trials with adequate power calculations are required. The use of non-fasting (random) capillary blood glucose at most time-points introduced measurement variability, as postprandial glucose fluctuations were not controlled; fasting plasma glucose or continuous glucose monitoring would provide more precise outcome data. The single-dose, 24-hour intervention window is unlikely to be sufficient for curcumin to exert clinically meaningful anti-inflammatory effects on glucose metabolism. No dietary control was implemented between T2 and T3, creating an uncontrolled source of variation. The absence of biomarker data (TNF-α, NF-κB, insulin, and the homeostasis model assessment of insulin resistance [HOMA-IR]) precludes mechanistic interpretation of the results. Participants were not blinded to group allocation, although the primary outcome (blood glucose) is objective.

### 4.4 Strengths

Strengths of this study include: prospective randomized design with allocation concealment; standardised and well-defined HIE protocol using established 1RM-based intensity prescription; use of a bioavailability-optimised curcumin formulation (Longvida®); four-point longitudinal glucose measurement design; and a homogeneous, well-characterised study population. This study also represents, to the best of the authors' knowledge, the first investigation of single-dose curcumin in the context of acute HIE glycaemic response in metabolically healthy individuals.

### 4.5 Implications and Future Directions

The findings suggest that a single low dose of curcumin is insufficient to modulate post-HIE blood glucose in healthy individuals. Future trials should employ: (1) higher curcumin doses (≥ 1 g/day, consistent with the subgroup analysis by Musazadeh et al. [13]); (2) multi-week supplementation protocols (≥ 8 weeks); (3) fasting plasma glucose as the primary outcome; (4) mechanistic biomarkers (TNF-α, insulin, NF-κB activity, GLUT-4 expression); (5) continuous glucose monitoring to capture the full post-exercise glycaemic profile; and (6) larger, adequately powered samples. Populations at higher metabolic risk (pre-diabetes, overweight athletes, metabolic syndrome) may demonstrate greater responsiveness to curcumin intervention.

## 5 CONCLUSION

Administration of a single oral dose of curcumin (400 mg, Longvida® formulation) 24 hours after an acute bout of high-intensity resistance exercise did not decrease in blood glucose levels in healthy, untrained adult males at 24 hours post-administration. A numerically larger glucose reduction was observed in the curcumin group compared with placebo, representing a small effect size. These pilot findings do not support the use of single-dose, low-dose curcumin as an acute glucose-modulating strategy after HIE in metabolically healthy individuals. Well-powered, multi-week randomized controlled trials with higher curcumin doses, fasting glucose measurements, and mechanistic biomarkers are warranted to definitively evaluate this intervention.

## STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

*Acknowledgments*

The authors acknowledge all study participants for their time and commitment. The authors thank the staff of the Department of Physiology and Medical Biochemistry, Faculty of Medicine, Universitas Airlangga, for technical support.

### *Disclosure of conflict of interest*

The authors declare that they have no competing interests. This study was not sponsored by any supplement manufacturer.

### *Statement of informed consent*

The written consent was obtained from all study participants involved in this study.

### *Author Contribution*

SCH: conceptualization, data collection, statistical analysis, and manuscript writing. LL and SIW: supervision, critical review, and manuscript revision. All authors read and approved the final manuscript.

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