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Improving trauma mortality prediction: Stress Hyperglycemia Ratio-Integrated Index versus traditional glucose-integrated Stress Index in a retrospective cohort study

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Abstract

Background: Stress hyperglycemia is a common response to trauma and critical illness, but conventional glucose measurements fail to distinguish stress-induced from chronic hyperglycemia. The serum glucose-to-potassium ration, or stress index (SI), is a readily available marker that may reflect the severity of the physiologic stress response. The stress hyperglycemia ratio (SHR) is defined as the ratio of an acute glucose measurement to the patient's estimated chronic glucose level, typically derived from hemoglobin A1c. This study evaluates a novel stress hyperglycemia ratio-integrated index (SHRI) against the traditional glucose-intergrated SI for predicting trauma outcomes.

Methods: A retrospective cohort study analyzed 3197 trauma patients admitted to a Level I trauma center (2019–2023). SHRI was calculated as the stress hyperglycemia ratio (SHR = admission glucose/estimated average glucose from HbA1c) divided by serum potassium. SI was defined as glucose/potassium. Optimal cutoffs were established via receiver operating characteristic analysis. Propensity score matching controlled for confounders was performed to compare the patients in the SHRI-high and SHRI-low groups.

Results: Inhospital mortality was 4.5% (144 patients). Nonsurvivors had higher SHRI (median 0.39 vs. 0.31, $p < 0.001$) and SI (53.17 vs. 43.85, $p < 0.001$), but lower HbA1c (6.05% vs. 6.50%, $p = 0.003$) and diabetes prevalence (45% vs. 58%, $p = 0.003$). SHRI > 0.41 predicted mortality with superior discrimination (AUC 0.81 vs. 0.73 for SI, $p < 0.05$) and was associated with fourfold higher mortality. After matching, SHRI-high patients maintained higher mortality (8.9% vs. 3.9%, $p = 0.015$), whereas matched SI-high versus SI-low mortality was similar (4.8% vs. 4.0%, $p = 0.70$).

Conclusions: SHRI provides superior prognostic information compared to SI by accounting for individual baseline glycemic status. This novel index could improve risk stratification and potentially guide management in trauma settings.

Cheng-Shyuan Rau and Shao-Chun Wu equal contribution to the first authorship.

KEYWORDS
glycemic status, mortality prediction, stress hyperglycemia, trauma

1 | INTRODUCTION

Hyperglycemia is a common acute response to severe trauma and critical illness, often termed stress hyperglycemia. This transient elevation in blood glucose results from a surge of catecholamines, cortisol, and inflammatory mediators during physiological stress.[1,2] Numerous studies have linked stress-induced hyperglycemia with adverse outcomes in critically ill patients, including higher rates of organ failure, longer hospital stays, and increased mortality.[1,2]

Importantly, the prognostic impact of hyperglycemia appears to differ by chronic glycemic status. Acute hyperglycemia in patients without diabetes (i.e., true stress hyperglycemia) is associated with disproportionately higher mortality risk compared to similar glucose levels in patients with preexisting diabetes.[2,3] For example, one cohort found that hospitalized COVID-19 patients without diabetes who developed hyperglycemia had a ~20% mortality rate versus only ~1.8% in normoglycemic nondiabetics.[2] In contrast, diabetic patients may tolerate higher glucose without the same relative risk increase.[2]

These observations highlight a key limitation of using absolute blood glucose alone as a prognostic marker—it fails to distinguish stress hyperglycemia from chronic hyperglycemia due to diabetes.[2] In trauma and critical care settings, relying on raw glucose values could misclassify high-risk patients; moderate hyperglycemia in a nondiabetic might signal severe stress, whereas an equivalent glucose in a diabetic patient might simply reflect baseline glycemic elevation.[2,3]

To address this, the stress hyperglycemia ratio (SHR) has been proposed as a novel index of relative hyperglycemia. The SHR is defined as the ratio of an acute glucose measurement to the patient's estimated chronic glucose level, typically derived from hemoglobin A1c (HbA1c).[4] By normalizing acute glucose to the individual's baseline glycemic control, SHR is intended to quantify the excess glycemia attributable to stress. Recent studies have demonstrated that SHR is a powerful predictor of outcomes across various acute conditions including acute coronary syndromes,[4] ischemic stroke,[5] acute kidney injury,[6] chronic kidney disease,[7] pulmonary embolism,[8] and sepsis.[9]

Parallel to SHR, researchers have explored simple composite indices of metabolic stress that combine hyperglycemia with other perturbations. One such measure is the serum glucose-to-potassium ratio, sometimes referred to as a “stress index (SI)” in critical illness. The pathophysiologic rationale is that acute stress not only raises glucose but often lowers serum potassium through catecholamine-mediated intracellular shifts and insulin release. Prior studies in acute brain injury and stroke have reported that an elevated glucose/potassium ratio on admission is associated with poor outcomes.[10] In trauma, Katipoğlu et al. observed that nonsurvivors of blunt abdominal injury had significantly higher glucose/potassium ratios than survivors.[11] Many studies suggest the SI is a readily available marker that may reflect the severity of the physiologic stress response.[12,13] However, like raw glucose, the SI does not account for chronic glycemic differences. This limitation motivates the rationale for replacing glucose with SHR in the SI formula. By using SHR in place of absolute glucose, a modified index (which we term SHRI—stress hyperglycemia ratio index, defined as SHR divided by potassium) might better isolate true stress hyperglycemia effects on outcomes.

In this study, we evaluated the clinical utility and prognostic performance of this novel stress hyperglycemia ratio-integrated index (SHRI) against the conventional glucose-based SI in a large cohort of trauma patients. We hypothesized that SHRI would outperform SI in predicting inhospital mortality after trauma by incorporating individual baseline glycemic status.

2 | METHODS

2.1 | Study design and patients

The study was authorized by the Institutional Review Board (IRB) with the approval number of 202500605B0 prior to its implementation. The patient consent is waived due to the retrospective design in accordance with IRB regulations. After excluding patients less than 20 years old, patients with burns, patients with hanging injuries, patients with drowning, and patients with incomplete data including glucose, HbA1c, and K, we conducted a retrospective cohort study of 3197 adult trauma patients (age $\geq$ 20) admitted to a Level I trauma center between 2009 and 2023 (Figure 1). Patient data included demographics, comorbidities, injury characteristics, laboratory results, treatments, and outcomes. Key baseline comorbidities extracted were history of diabetes mellitus (DM), hypertension, coronary artery disease, cerebrovascular accident (CVA/stroke), congestive heart failure, and end-stage renal disease. Injury severity was characterized by the Glasgow Coma

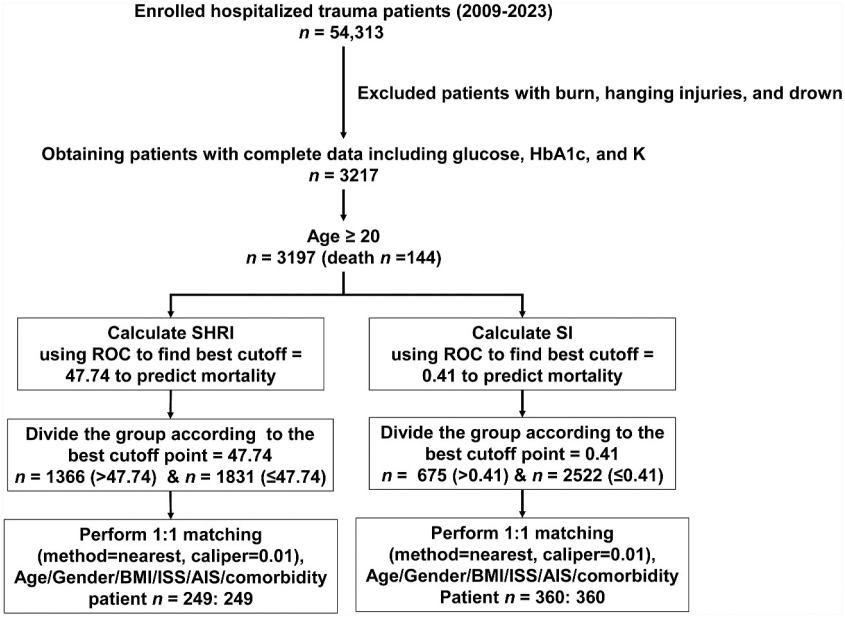

FIGURE 1 Patient inclusion flowchart and stratification based on SHRI and SI thresholds. Study flowchart showing the patient selection process from 54,313 hospitalized trauma patients to the final matched cohorts based on SI and SHRI cutoff values. AIS, Abbreviated Injury Scale; BMI, body mass index; HbA1c, hemoglobin A1c; ISS, Injury Severity Score; K, potassium; SHRI, stress hyperglycemia ratio-integrated index; SI, stress index.

Scale (GCS), Injury Severity Score (ISS), Trauma and Injury Severity Score, and Abbreviated Injury Scale (AIS) scores for each body region.

2.2 | Stress Hyperglycemia Ratio and Stress Index definitions

The SHR was calculated as the ratio of admission glucose to estimated average glucose derived from HbA1c. Specifically, we used the formula: SHR = Admission glucose (mg/dL)/[28.7 × HbA1c (%) – 46.7]. The traditional SI was defined as the ratio of admission glucose to admission potassium (K) in serum, that is, SI = Glucose (mg/dL)/K (mEq/L). We then defined the novel stress hyperglycemia ratio index (SHRI) as SHRI = SHR/K, effectively substituting SHR in place of raw glucose.

2.3 | Data groupings and matching

Optimal cutoff values for predicting mortality were derived for each index using receiver operating characteristic (ROC) curve analysis on the whole sample. The Youden index was used to select the threshold that maximized sensitivity + specificity. This yielded a cutoff of SI = 47.74 and SHRI = 0.41 for inhospital mortality prediction. Using these cut points, we stratified patients into "high" versus "low" groups for each index. To control for potential confounders, we performed a 1:1 propensity score matching between high and low groups for each index. We matched patients on a range of baseline factors including age, gender, body mass index, major comorbidities, and trauma severity indicators.

2.4 | Statistical analysis

Continuous variables were compared using the nonparametric Mann–Whitney *U* test, and categorical variables were compared by chi-square test or Fisher's exact test. We assessed discrimination for mortality using ROC curve analysis, calculating the area under the ROC curve (AUC) for SI and SHRI in the whole cohort. AUCs were compared using DeLong's test. We also evaluated sensitivity, specificity, and accuracy at the optimal cutoff points for each index.

3 | RESULTS

A total of 3197 trauma patients were analyzed of whom 144 (4.5%) suffered inhospital mortality (Table 1). The median age was 69 years (IQR 59–77) and 51% were male. 57.4% of patients had a history of DM. The median injury severity was relatively moderate (ISS 9[5–16] overall), though nonsurvivors had much more severe

TABLE 1 Baseline characteristics and outcomes of trauma patients stratified by survival status.

Variables	Total $n = 3197$	Survival $n = 3053$	Mortality $n = 144$	*p*-value
SHRI	0.31 [0.25, 0.39]	0.31 [0.25, 0.39]	0.39 [0.28, 0.49]	<0.001
SI	44.17 [33.59, 60.00]	43.85 [33.33, 59.68]	53.17 [38.26, 72.39]	<0.001
SHR	1.20 [0.99, 1.48]	1.19 [0.98, 1.46]	1.45 [1.13, 1.76]	<0.001
Sugar, mg/dL	168.00 [129.00, 231.00]	167.00 [129.00, 229.00]	193.50 [153.25, 248.50]	0.001
HbA1c, %	6.50 [5.80, 7.50]	6.50 [5.80, 7.60]	6.05 [5.68, 7.10]	0.003
K, mEq/L	3.90 [3.50, 4.30]	3.90 [3.50, 4.30]	3.80 [3.20, 4.20]	0.002
Age, years	69.00 [59.00, 77.00]	69.00 [59.00, 77.00]	71.00 [60.00, 80.00]	0.093
Male, *n* (%)	1639 (51.3)	1559 (51.1)	80 (55.6)	0.333
BMI	24.40 [21.78, 27.40]	24.50 [21.80, 27.40]	23.10 [20.48, 25.91]	0.001
GCS	15.00 [15.00, 15.00]	15.00 [15.00, 15.00]	10.00 [4.00, 15.00]	<0.001
ISS	9.00 [5.00, 16.00]	9.00 [5.00, 16.00]	20.00 [16.00, 25.00]	<0.001
TRISS	0.97 [0.94, 0.98]	0.97 [0.94, 0.98]	0.87 [0.45, 0.94]	<0.001
CVA, *n* (%)	368 (11.5)	351 (11.5)	17 (11.8)	>0.999
HTN, *n* (%)	1925 (60.2)	1835 (60.1)	90 (62.5)	0.626
CAD, *n* (%)	391 (12.2)	359 (11.8)	32 (22.2)	<0.001
CHF, *n* (%)	77 (2.4)	74 (2.4)	3 (2.1)	>0.999
ESRD, *n* (%)	167 (5.2)	158 (5.2)	9 (6.2)	0.708
Temperature, °C	36.50 [36.20, 37.00]	36.50 [36.20, 37.00]	36.40 [36.00, 36.90]	0.018
Heart rate, times/min	86.00 [75.00, 98.00]	86.00 [75.00, 98.00]	90.50 [74.75, 107.00]	0.112
SBP, mmHg	156.00 [135.00, 181.00]	156.00 [135.00, 181.00]	157.50 [121.00, 181.00]	0.452
DBP, mmHg	87.00 [76.00, 99.00]	87.00 [76.00, 99.00]	82.00 [68.00, 96.25]	0.001
Resp rate, times/min	18.00 [18.00, 20.00]	18.00 [18.00, 20.00]	18.00 [16.00, 20.00]	0.467
AIS (head/neck), *n* (%)				<0.001
AIS = 0	1882 (58.9)	1855 (60.8)	27 (18.8)	
AIS = 1	77 (2.4)	73 (2.4)	4 (2.8)	
AIS = 2	74 (2.3)	73 (2.4)	1 (0.7)	
AIS = 3	286 (8.9)	276 (9.0)	10 (6.9)	
AIS = 4	707 (22.0)	658 (21.5)	45 (31.2)	
AIS = 5	173 (5.4)	118 (3.9)	55 (38.2)	
AIS = 6	1 (0.0)	0 (0.0)	1 (0.7)	
AIS (face), *n* (%)				0.165
AIS = 0	2890 (90.4)	2766 (90.6)	124 (86.1)	
AIS = 1	70 (2.2)	67 (2.2)	3 (2.1)	
AIS = 2	230 (7.2)	213 (7.0)	17 (11.8)	
AIS = 3	7 (0.2)	7 (0.2)	0 (0.0)	
AIS (thorax), *n* (%)				0.113
AIS = 0	2747 (85.9)	2633 (86.2)	114 (79.2)	
AIS = 1	66 (2.1)	63 (2.1)	3 (2.1)	
AIS = 2	107 (3.3)	100 (3.3)	7 (4.9)	
AIS = 3	180 (5.6)	165 (5.4)	15 (10.4)	

TABLE 1 (Continued)

Variables	Total $n = 3197$	Survival $n = 3053$	Mortality $n = 144$	p-value
AIS = 4	85 (2.7)	80 (2.6)	5 (3.5)	
AIS = 5	12 (0.4)	12 (0.4)	0 (0.0)	
AIS (abdomen), *n* (%)				0.783
AIS = 0	3069 (96.0)	2928 (95.9)	141 (97.9)	
AIS = 1	5 (0.2)	5 (0.2)	0 (0.0)	
AIS = 2	73 (2.3)	72 (2.4)	1 (0.7)	
AIS = 3	35 (1.1)	34 (1.1)	1 (0.7)	
AIS = 4	14 (0.4)	13 (0.4)	1 (0.7)	
AIS = 5	1 (0.0)	1 (0.0)	0 (0.0)	
AIS (extremity), *n* (%)				<0.001
AIS = 0	1222 (38.2)	1127 (36.9)	95 (66.0)	
AIS = 1	71 (2.2)	70 (2.3)	1 (0.7)	
AIS = 2	904 (28.3)	878 (28.8)	26 (18.1)	
AIS = 3	997 (31.2)	976 (32.0)	21 (14.6)	
AIS = 4	3 (0.1)	2 (0.1)	1 (0.7)	
AIS (external), *n* (%)				<0.001
AIS = 0	3014 (94.3)	2882 (94.4)	132 (91.7)	
AIS = 1	138 (4.3)	130 (4.3)	8 (5.6)	
AIS = 2	28 (0.9)	26 (0.9)	2 (1.4)	
AIS = 3	9 (0.3)	9 (0.3)	0 (0.0)	
AIS = 4	1 (0.0)	0 (0.0)	1 (0.7)	
AIS = 5	6 (0.2)	5 (0.2)	1 (0.7)	
AIS = 6	1 (0.0)	1 (0.0)	0 (0.0)	
DM, *n* (%)	1835 (57.4)	1770 (58.0)	65 (45.1)	0.003
ICU stay, days	0.00 [0.00, 4.00]	0.00 [0.00, 3.00]	8.00 [3.00, 18.25]	<0.001
Inhospital stay, days	8.60 [5.00, 15.90]	8.50 [5.00, 15.70]	10.30 [3.80, 23.57]	0.486

Note: This table compares demographic, clinical, and biochemical parameters between survivors and nonsurvivors in the full cohort ($n = 3197$), highlighting key differences in stress hyperglycemia indicators and injury severity. Data presented as median [IQR] or *n* (%). The continuous data are expressed in median [IQR].

Abbreviations: AIS, Abbreviated Injury Scale; CAD, coronary artery disease; CHF, congestive heart failure; CVA, cerebrovascular accident; DBP, diastolic blood pressure; DM, diabetes mellitus; ESRD, end-stage renal disease; GCS, Glasgow Coma Scale; HbA1c, hemoglobin A1c; HTN, hypertension; ICU, intensive care unit; ISS, Injury Severity Score; SBP, systolic blood pressure; SHR, stress hyperglycemia ratio; SHRI, stress hyperglycemia ratio index; SI, stress index; TRISS, Trauma and Injury Severity Score.

trauma (ISS 20 [16–25] in nonsurvivors vs. 9[5–16] in survivors, $p < 0.001$) and lower admission GCS (median 10 vs. 15, $p < 0.001$). Nonsurvivors also had higher rates of critical head injuries (38% with AIS Head ≥ 5 vs. 3.9% in survivors).

There were marked differences in glucose-related metrics between survivors and nonsurvivors. Admission glucose was significantly higher in patients who died (median 193.5 mg/dL) than in those who survived (167 mg/dL, $p = 0.001$). However, median HbA1c was lower in nonsurvivors (6.05% vs. 6.50%, $p = 0.003$). This indicates that many nonsurvivors had relatively normal long-term glycemic control but still experienced high acute glucose—a signature of stress hyperglycemia.

In line with this, the SHR was substantially elevated among nonsurvivors (median 1.45 [IQR 1.13–1.76]) versus survivors (1.19 [0.98–1.46], $p < 0.001$). By contrast, a history of diabetes was less common in nonsurvivors (45% vs. 58% in survivors, $p = 0.003$). The SI was also higher in nonsurvivors: median SI 53.17 (38.3–72.4) versus 43.85 (33.3–59.7) in survivors ($p < 0.001$). Nonsurvivors presented with slightly lower

serum potassium on average (3.8 vs. 3.9 mEq/L, $p = 0.002$), consistent with a more pronounced stress response. Combining these factors, the SHRI showed the greatest disparity: median SHRI was 0.39 [0.28–0.49] in nonsurvivors compared to 0.31 [0.25–0.39] in survivors ($p < 0.001$).

We determined the prognostic performance of SHRI versus SI for mortality. The AUC for SHRI was higher than that for SI, indicating better discrimination. Using the optimal thresholds, we found that both indices stratified mortality risk, but SHRI did so more sharply. Only 1.8% of patients with low SHRI (≤0.41) died compared to 12.0% mortality in those with high SHRI >0.41 (χ^2 $p < 0.001$). In contrast, SI > 47.74 identified a group with 6.3% mortality versus 3.1% in SI ≤ 47.74 ($p < 0.001$). Thus, a high SHRI carried roughly a fourfold higher death rate than low SHRI, whereas high SI carried about a twofold higher death rate than low SI.

As shown in Table 2, before matching, 675 patients (21.1%) were SHRI-high and 1366 (42.7%) were SI-high. The SHRI-high group had much higher raw glucose (median 247 vs. 154 mg/dL) and lower K (3.6 vs. 3.9) than SHRI-low, but no significant difference in diabetes prevalence (55.9% vs. 57.8%, $p = 0.384$). In contrast, the SI-high group included a disproportionate number of diabetic patients (67.6% vs. 49.8% in SI-low, $p < 0.001$) and had higher HbA1c (7.3% vs. 6.1%, $p < 0.001$). This indicates that many SI-high patients were chronically hyperglycemic (diabetic) without necessarily having a large acute glucose surge, whereas SHRI-high patients truly experienced a big jump over their baseline. For instance, median HbA1c in the SHRI-high group was actually lower than in SHRI-low (6.3% vs. 6.5%, $p < 0.001$), reflecting that SHRI-high status often came from a normal baseline and high stress glucose.

Matched cohort analysis revealed that, after 1:1 matching, we obtained 360 pairs of SHRI-high versus SHRI-low patients and 249 pairs of SI-high versus SI-low patients, with balanced baseline characteristics. In the matched SHRI cohorts, outcomes diverged significantly despite similar injury severity and comorbidity profiles. Inhospital mortality remained higher in SHRI-high (8.9%) versus SHRI-low (3.9%, $p = 0.015$). SHRI-high patients also required more intensive care—for example, need for intensive care unit [ICU] admission was greater and ICU length of stay was longer (median 5.0 vs. 2.0 days, $p < 0.01$).

Moreover, even under matched conditions, SHRI-high patients showed evidence of a heightened physiological stress response. For example, heart rate on admission was significantly elevated (median 88 vs. 82 beats/min, $p < 0.001$ after matching), and they tended to have slightly lower blood pressure and higher respiratory rate. These subtle differences suggest that for two patients otherwise similar in injuries and health status, the one with an out-of-proportion stress hyperglycemia (high SHRI) may manifest more tachycardia and systemic stress, correlating with worse outcomes.

In the matched SI cohorts (Table 3), matched SI-high versus SI-low patients had similar mortality (4.8% vs. 4.0%, $p = 0.70$) after controlling for confounders. This implies that much of the crude mortality difference by SI (pre-match) was attributable to the higher prevalence of severe injuries and comorbidities in SI-high patients rather than SI itself.

Finally, the ROC analysis (Figure 2) showed SHRI had a higher AUC (0.81) than SI (0.73) for mortality prediction (difference $p < 0.05$). At their respective optimal cutoffs, SHRI > 0.41 identified mortality with sensitivity ~79% and specificity ~72%, whereas SI > 47.74 had lower sensitivity (~65%) and specificity (~61%). The positive predictive value of SHRI > 0.41 was modest (~12% given the low overall mortality rate), but the negative predictive value of SHRI ≤ 0.41 was extremely high (>98%), meaning a trauma patient with a low SHRI had an excellent chance of survival.

4 | DISCUSSION

In this cohort of 3197 trauma patients, we found that SHRI provides superior prognostic information on mortality compared to the conventional glucose-based SI. Patients who did not survive their injuries exhibited significantly higher SHRI than survivors, even though they had lower HbA1c and fewer were diabetic—a classic pattern of acute stress hyperglycemia. By accounting for individual baseline glycemic control through HbA1c, SHRI identified high-risk trauma patients who would have been missed or underestimated by glucose alone.

In ROC analyses and matched comparisons, SHRI outperformed SI in discriminating mortality risk. Notably, SHRI > 0.41 was associated with about a fourfold increase in death rates and remained predictive of worse outcomes even among patients matched for injury severity and comorbidities. In contrast, the prognostic impact of a high SI (> 47.7) diminished after controlling for confounders like diabetes and ISS. These findings suggest that incorporating the SHR into a composite index (SHRI) yields a more robust marker of critical illness severity in trauma than the traditional glucose/potassium ratio alone. This study revealed that as the stress hyperglycemia has been shown to predict trauma complications and mortality independently of injury severity,[14,15] SHRI contributes additional prognostic information beyond traditional trauma scoring systems.

Our results align with growing evidence that stress hyperglycemia is an important determinant of trauma and critical care outcomes.[16,17] Prior studies often did

TABLE 2 Comparison of trauma patients stratified by stress index >47.74 versus ≤47.74 before and after propensity score matching.

	Before match			After match		
N	**SI > 47.74** ***n* = 1366**	**SI ≤ 47.74** ***n* = 1831**	***p*-value**	**SHRI > 47.74** ***n* = 249**	**SHRI ≤ 47.74** ***n* = 249**	***p*-value**
SHRI	0.40 [0.34, 0.48]	0.27 [0.22, 0.31]	<0.001	0.39 [0.34, 0.45]	0.27 [0.22, 0.30]	<0.001
SI	63.82 [54.66, 79.46]	34.85 [29.11, 40.90]	<0.001	61.49 [53.44, 75.37]	35.94 [29.19, 41.54]	<0.001
SHR	1.49 [1.26, 1.77]	1.05 [0.90, 1.21]	<0.001	1.46 [1.25, 1.76]	1.06 [0.91, 1.22]	<0.001
Sugar, mg/dL	242.00 [203.00, 306.00]	135.00 [115.00, 159.00]	<0.001	236.00 [195.00, 300.00]	142.00 [116.00, 170.00]	<0.001
HbA1c, %	7.30 [6.30, 8.60]	6.10 [5.60, 6.80]	<0.001	7.20 [6.30, 8.40]	6.30 [5.80, 6.90]	<0.001
K, mEq/L	3.80 [3.40, 4.10]	4.00 [3.60, 4.35]	<0.001	3.90 [3.50, 4.20]	4.00 [3.70, 4.50]	<0.001
Age, years	67.00 [58.00, 76.00]	70.00 [60.00, 78.00]	<0.001	70.00 [63.00, 77.00]	70.00 [63.00, 78.00]	0.89
Male, *n* (%)	734 (53.7)	905 (49.4)	0.018	136 (54.6)	136 (54.6)	>0.999
BMI	24.79 [22.10, 27.73]	24.20 [21.50, 27.10]	<0.001	24.30 [22.10, 26.80]	24.50 [22.30, 27.11]	0.876
GCS	15.00 [14.00, 15.00]	15.00 [15.00, 15.00]	<0.001	15.00 [15.00, 15.00]	15.00 [15.00, 15.00]	0.667
ISS	9.00 [9.00, 16.00]	9.00 [4.00, 16.00]	<0.001	9.00 [4.00, 9.00]	9.00 [4.00, 9.00]	>0.999
TRISS	0.97 [0.93, 0.98]	0.97 [0.94, 0.98]	<0.001	0.97 [0.97, 0.98]	0.97 [0.97, 0.98]	0.728
CVA, *n* (%)	130 (9.5)	238 (13.0)	0.003	14 (5.6)	14 (5.6)	>0.999
HTN, *n* (%)	805 (58.9)	1120 (61.2)	0.214	171 (68.7)	171 (68.7)	>0.999
CAD, *n* (%)	148 (10.8)	243 (13.3)	0.043	10 (4.0)	10 (4.0)	>0.999
CHF, *n* (%)	33 (2.4)	44 (2.4)	>0.999	249 (100.0)	249 (100.0)	-
ESRD, *n* (%)	61 (4.5)	106 (5.8)	0.113	14 (5.6)	14 (5.6)	>0.999
DM, *n* (%)	923 (67.6)	912 (49.8)	<0.001	184 (73.9)	184 (73.9)	>0.999
Temperature, °C	36.50 [36.20, 36.90]	36.50 [36.20, 37.00]	0.01	36.50 [36.20, 37.00]	36.50 [36.20, 36.90]	0.891
Heart rate, times/min	90.00 [79.00, 102.00]	83.00 [73.00, 96.00]	<0.001	86.00 [77.00, 98.00]	84.00 [74.00, 95.00]	0.063
SBP, mmHg	158.00 [135.00, 185.00]	155.00 [135.00, 180.00]	0.033	161.00 [143.00, 189.00]	157.00 [137.00, 181.00]	0.097
DBP, mmHg	88.00 [76.00, 100.00]	86.00 [76.00, 98.00]	0.075	88.00 [76.00, 100.00]	86.00 [75.00, 97.00]	0.591
Resp rate, times/min	18.00 [18.00, 20.00]	18.00 [18.00, 20.00]	<0.001	18.00 [18.00, 20.00]	18.00 [18.00, 20.00]	0.368
AIS (head/neck), *n* (%)			<0.001			>0.999
AIS = 0	740 (54.2)	1142 (62.4)		191 (76.7)	191 (76.7)	
AIS = 1	29 (2.1)	48 (2.6)				
AIS = 2	29 (2.1)	45 (2.5)				
AIS = 3	134 (9.8)	152 (8.3)		7 (2.8)	7 (2.8)	
AIS = 4	321 (23.5)	381 (20.8)		46 (18.5)	46 (18.5)	
AIS = 5	112 (8.2)	61 (3.3)		5 (2.0)	5 (2.0)	
AIS = 6	1 (0.1)	2 (0.1)				
AIS (face), *n* (%)			0.031			>0.999
AIS = 0	1213 (88.8)	1677 (91.6)		248 (99.6)	248 (99.6)	
AIS = 1	32 (2.3)	38 (2.1)				
AIS = 2	116 (8.5)	114 (6.2)		1 (0.4)	1 (0.4)	
AIS = 3	5 (0.4)	2 (0.1)				

(Continues)

TABLE 2 (Continued)

	Before match			After match		
N	SI > 47.74 n = 1366	SI ≤ 47.74 n = 1831	p-value	SHRI > 47.74 n = 249	SHRI ≤ 47.74 n = 249	p-value
AIS (thorax), n (%)			<0.001			-
AIS = 0	1130 (82.7)	1617 (88.3)		249 (100.0)	249 (100.0)	
AIS = 1	30 (2.2)	36 (2.0)				
AIS = 2	51 (3.7)	56 (3.1)				
AIS = 3	97 (7.1)	83 (4.5)				
AIS = 4	50 (3.7)	35 (1.9)				
AIS = 5	8 (0.6)	4 (0.2)				
AIS (abdomen), n (%)			<0.001			-
AIS = 0	1292 (94.6)	1777 (97.1)		249 (100.0)	249 (100.0)	
AIS = 1	0 (0.0)	5 (0.3)				
AIS = 2	48 (3.5)	25 (1.4)				
AIS = 3	18 (1.3)	17 (0.9)				
AIS = 4	7 (0.5)	7 (0.4)				
AIS = 5	1 (0.1)	0 (0.0)				
AIS (extremity), n (%)			0.002			>0.999
AIS = 0	566 (41.4)	656 (35.8)		58 (23.3)	58 (23.3)	
AIS = 1	22 (1.6)	49 (2.7)				
AIS = 2	352 (25.8)	552 (30.1)		67 (26.9)	67 (26.9)	
AIS = 3	424 (31.0)	573 (31.3)		124 (49.8)	124 (49.8)	
AIS = 4	2 (0.1)	1 (0.1)				
AIS (external), n (%)			0.027			>0.999
AIS = 0	1282 (93.9)	1732 (94.6)		248 (99.6)	248 (99.6)	
AIS = 1	55 (4.0)	83 (4.5)				
AIS = 2	16 (1.2)	12 (0.7)		1 (0.4)	1 (0.4)	
AIS = 3	8 (0.6)	1 (0.1)				
AIS = 4	0 (0.0)	0 (0.0)				
AIS = 5	4 (0.3)	2 (0.1)				
AIS = 6	1 (0.1)	1 (0.1)				
ICU stay, days	0.00 [0.00, 5.00]	0.00 [0.00, 3.00]	<0.001	0.00 [0.00, 0.00]	0.00 [0.00, 0.00]	0.858
Inhospital stay, days	9.80 [5.73, 18.40]	7.80 [4.80, 14.45]	<0.001	8.00 [4.80, 12.50]	7.00 [4.60, 12.00]	0.346
Mortality, n (%)	86 (6.3)	58 (3.2)	<0.001	8 (3.2)	6 (2.4)	0.786

Note: This table presents clinical variables and outcomes comparing high- and low-SI groups, with statistical results for both unmatched and matched cohorts (*n* = 1366 vs. 1831 pre-match; 249 vs. 249 post-match).

Abbreviations: AIS, Abbreviated Injury Scale; BMI, body mass index; CAD, coronary artery disease; CHF, congestive heart failure; CVA, cerebrovascular accident; DBP, diastolic blood pressure; DM, diabetes mellitus; ESRD, end-stage renal disease; GCS, Glasgow Coma Scale; HbA1c, hemoglobin A1c; HTN, hypertension; ICU, intensive care unit; ISS, Injury Severity Score; SBP, systolic blood pressure; SHR, stress hyperglycemia ratio; SHRI, stress hyperglycemia ratio index; SI, stress index; TRISS, Trauma and Injury Severity Score.

not distinguish diabetic from nondiabetic patients. More recent investigations have emphasized that it is acute hyperglycemia relative to baseline—that is, true stress hyperglycemia—that most strongly signals risk.[17–19] In critically ill nondiabetic patients, stress hyperglycemia above certain thresholds confers markedly higher mortality.[3] This threshold phenomenon is consistent with our trauma data and our chosen SHRI cutoff

TABLE 3 Comparison of trauma patients stratified by SHRI >0.41 versus ≤0.41 before and after propensity score matching.

	Before match			After match		
n	**SHRI > 0.41** ***n* = 675**	**SHRI ≤ 0.41** ***n* = 2522**	***p*-value**	**SHRI > 0.41** ***n* = 360**	**SHRI ≤ 0.41** ***n* = 360**	***p*-value**
SHRI	0.48 [0.44, 0.56]	0.28 [0.23, 0.33]	<0.001	0.47 [0.43, 0.54]	0.27 [0.23, 0.33]	<0.001
SI	70.86 [57.79, 86.30]	39.47 [31.32, 50.29]	<0.001	69.19 [57.21, 83.90]	39.26 [30.37, 49.71]	<0.001
SHR	1.75 [1.55, 2.01]	1.11 [0.94, 1.30]	<0.001	1.74 [1.52, 1.98]	1.10 [0.93, 1.28]	<0.001
Sugar, mg/dL	247.00 [193.00, 317.50]	154.00 [123.00, 202.00]	<0.001	257.50 [195.75, 320.00]	153.00 [123.00, 200.50]	<0.001
HbA1c, %	6.30 [5.70, 7.40]	6.50 [5.80, 7.60]	<0.001	6.50 [5.70, 7.43]	6.60 [5.90, 7.53]	0.102
K, mEq/L	3.60 [3.20, 3.90]	3.90 [3.60, 4.30]	<0.001	3.60 [3.30, 4.00]	4.00 [3.60, 4.40]	<0.001
Age, years	67.00 [56.00, 76.00]	69.00 [60.00, 77.00]	<0.001	70.00 [61.00, 79.00]	70.00 [61.00, 77.00]	0.407
Male, *n* (%)	350 (51.9)	1289 (51.1)	0.765	188 (52.2)	190 (52.8)	0.941
BMI	24.11 [21.60, 27.40]	24.49 [21.80, 27.39]	0.451	23.90 [21.40, 26.84]	24.40 [21.84, 27.12]	0.337
GCS	15.00 [13.00, 15.00]	15.00 [15.00, 15.00]	<0.001	15.00 [15.00, 15.00]	15.00 [15.00, 15.00]	0.066
ISS	10.00 [9.00, 20.00]	9.00 [4.00, 16.00]	<0.001	9.00 [9.00, 16.00]	9.00 [9.00, 16.00]	>0.999
TRISS	0.97 [0.89, 0.98]	0.97 [0.94, 0.98]	<0.001	0.97 [0.96, 0.98]	0.97 [0.94, 0.98]	0.552
CVA, *n* (%)	70 (10.4)	298 (11.8)	0.328	46 (12.8)	47 (13.1)	>0.999
HTN, *n* (%)	395 (58.5)	1530 (60.7)	0.333	241 (66.9)	229 (63.6)	0.389
CAD, *n* (%)	68 (10.1)	323 (12.8)	0.063	41 (11.4)	48 (13.3)	0.497
CHF, *n* (%)	21 (3.1)	56 (2.2)	0.23	4 (1.1)	7 (1.9)	0.543
ESRD, *n* (%)	31 (4.6)	136 (5.4)	0.464	17 (4.7)	15 (4.2)	0.856
DM, *n* (%)	377 (55.9)	1458 (57.8)	0.384	229 (63.6)	217 (60.3)	0.398
Temperature, °C	36.50 [36.20, 37.00]	36.50 [36.20, 37.00]	0.581	36.60 [36.20, 37.10]	36.50 [36.20, 37.00]	0.129
Heart rate, times/min	91.00 [78.00, 104.00]	85.00 [75.00, 97.00]	<0.001	88.00 [76.75, 100.00]	82.00 [74.00, 94.00]	<0.001
SBP, mmHg	155.00 [129.00, 181.00]	156.00 [137.00, 181.00]	0.05	159.00 [137.00, 185.50]	157.50 [138.00, 180.00]	0.69
DBP, mmHg	85.00 [73.00, 99.00]	87.00 [76.00, 99.00]	0.024	87.00 [75.00, 100.00]	87.00 [75.00, 98.00]	0.713
Resp rate, times/min	18.00 [18.00, 20.00]	18.00 [18.00, 20.00]	0.005	18.00 [18.00, 20.00]	18.00 [18.00, 20.00]	0.084
AIS (head/neck), *n* (%)			<0.001			>0.999
AIS = 0	355 (52.6)	1527 (60.5)		236 (65.6)	236 (65.6)	
AIS = 1	16 (2.4)	61 (2.4)		1 (0.3)	1 (0.3)	
AIS = 2	14 (2.1)	60 (2.4)		1 (0.3)	1 (0.3)	
AIS = 3	61 (9.0)	225 (8.9)		22 (6.1)	22 (6.1)	
AIS = 4	144 (21.3)	558 (22.1)		79 (21.9)	79 (21.9)	
AIS = 5	84 (12.4)	89 (3.5)		21 (5.8)	21 (5.8)	
AIS = 6	1 (0.1)	2 (0.1)				
AIS (face), *n* (%)			<0.001			>0.999
AIS = 0	593 (87.9)	2297 (91.1)		355 (98.6)	355 (98.6)	
AIS = 1	13 (1.9)	57 (2.3)		1 (0.3)	1 (0.3)	
AIS = 2	64 (9.5)	166 (6.6)		4 (1.1)	4 (1.1)	
AIS = 3	5 (0.7)	2 (0.1)				

(Continues)

TABLE 3 (Continued)

	Before match			After match		
n	SHRI > 0.41 *n* = 675	SHRI ≤ 0.41 *n* = 2522	*p*-value	SHRI > 0.41 *n* = 360	SHRI ≤ 0.41 *n* = 360	*p*-value
AIS (thorax), *n* (%)			<0.001			>0.999
AIS = 0	544 (80.6)	2203 (87.4)		354 (98.3)	354 (98.3)	
AIS = 1	8 (1.2)	58 (2.3)				
AIS = 2	29 (4.3)	78 (3.1)		2 (0.6)	2 (0.6)	
AIS = 3	59 (8.7)	121 (4.8)		2 (0.6)	2 (0.6)	
AIS = 4	29 (4.3)	56 (2.2)		2 (0.6)	2 (0.6)	
AIS = 5	6 (0.9)	6 (0.2)				
AIS (abdomen), *n* (%)			<0.001			-
AIS = 0	629 (93.2)	2440 (96.7)		360 (100.0)	360 (100.0)	
AIS = 1	0 (0.0)	5 (0.2)				
AIS = 2	30 (4.4)	43 (1.7)				
AIS = 3	9 (1.3)	26 (1.0)				
AIS = 4	6 (0.9)	8 (0.3)				
AIS = 5	1 (0.1)	0 (0.0)				
AIS (extremity), *n* (%)			0.001			>0.999
AIS = 0	283 (41.9)	939 (37.2)		120 (33.3)	120 (33.3)	
AIS = 1	10 (1.5)	61 (2.4)		1 (0.3)	1 (0.3)	
AIS = 2	155 (23.0)	749 (29.7)		78 (21.7)	78 (21.7)	
AIS = 3	225 (33.3)	772 (30.6)		161 (44.7)	161 (44.7)	
AIS = 4	2 (0.3)	1 (0.0)				
AIS (external), *n* (%)			0.003			0.801
AIS = 0	628 (93.0)	2386 (94.6)		358 (99.4)	357 (99.2)	
AIS = 1	28 (4.1)	110 (4.4)		1 (0.3)	1 (0.3)	
AIS = 2	10 (1.5)	18 (0.7)		0 (0.0)	1 (0.3)	
AIS = 3	6 (0.9)	3 (0.1)		1 (0.3)	1 (0.3)	
AIS = 4	1 (0.1)	0 (0.0)				
AIS = 5	2 (0.3)	4 (0.2)				
AIS = 6	0 (0.0)	1 (0.0)				
ICU stay, days	0.00 [0.00, 7.00]	0.00 [0.00, 3.00]	<0.001	0.00 [0.00, 3.00]	0.00 [0.00, 2.00]	0.144
Inhospital stay, days	10.80 [6.00, 20.65]	8.00 [4.90, 14.80]	<0.001	8.70 [5.50, 14.55]	8.25 [5.10, 14.67]	0.753
Mortality, *n* (%)	69 (10.2)	75 (3.0)	<0.001	24 (6.7)	10 (2.8)	0.022

Note: This table contrasts demographic and clinical outcomes between high- and low-SHRI groups, both before and after propensity score matching ($n = 675$ vs. 2522 pre-match; 360 vs. 360 post-match), illustrating SHRI's stronger discriminatory power.

Abbreviations: AIS, Abbreviated Injury Scale; BMI, body mass index; CAD, coronary artery disease; CHF, congestive heart failure; CVA, cerebrovascular accident; DBP, diastolic blood pressure; DM, diabetes mellitus; ESRD, end-stage renal disease; GCS, Glasgow Coma Scale; HbA1c, hemoglobin A1c; HTN, hypertension; ICU, intensive care unit; ISS, Injury Severity Score; SBP, systolic blood pressure; SHR, stress hyperglycemia ratio; SHRI, stress hyperglycemia ratio index; SI, stress index; TRISS, Trauma and Injury Severity Score.

(~0.41, roughly corresponding to an admission glucose ~1.4 times baseline).

The concept of combining glucose and potassium in a SI also deserves discussion. In trauma and emergency surgery, hyperglycemia concurrent with hypokalemia may indicate catecholamine surges and insulin effects from shock.[16,18,20] Our findings corroborate that SI does differentiate survivors versus nonsurvivors (we saw a ~21% higher median SI in fatalities). However, the SI's performance was limited by the influence of chronic

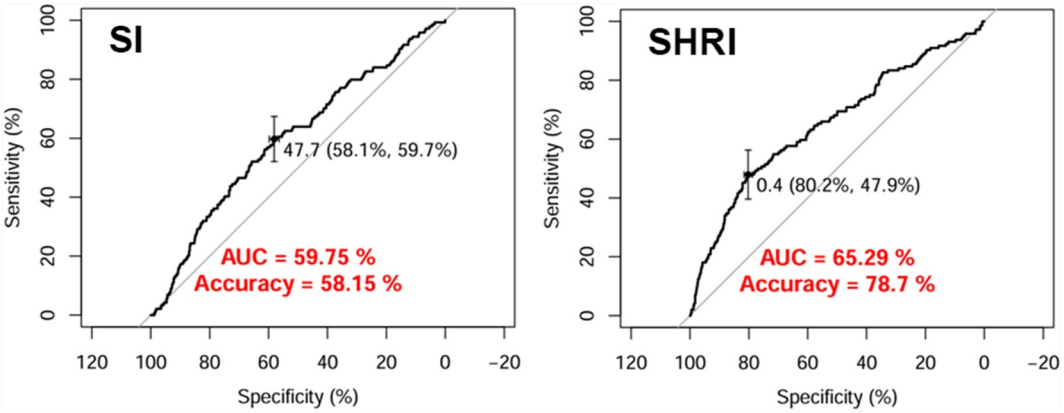

FIGURE 2 Receiver operating characteristic curves comparing SHRI and SI for predicting inhospital mortality. Receiver operating characteristic curves comparing the predictive accuracy of SI and SHRI for inhospital mortality. SHRI demonstrated superior discrimination with a higher area under the curve. AUC, area under the curve; ROC, receiver operating characteristic; SHRI, stress hyperglycemia ratio-integrated index; SI, stress index.

hyperglycemia—many diabetic patients had high SI primarily due to elevated baseline glucose rather than an extreme stress response. By replacing glucose with SHR, the SHRI effectively "corrects" the SI for diabetic status. In our data, the SHRI-high group included both diabetic and nondiabetic patients who truly had an acute hyperglycemic surge, whereas the SI-high group was dominated by diabetics. Consequently, SHRI was more tightly linked to actual stress physiology and outcome.

Clinically, the implications of these findings are significant for trauma care. First, SHRI could be used as an early triage tool in the emergency department or ICU. A patient with an unusually high SHRI on admission might be identified as extremely high risk even if other clinical scores seem moderate. In our cohort, SHRI $\leq$ 0.2 was almost uniformly associated with survival, whereas SHRI >0.5 often portended critical illness. Second, our results raise considerations for glycemic control strategies. Our data suggest that patients with high SHRI are those who might benefit most from careful glucose management, as they have the most excess glycemic exposure relative to their usual state. Some authors have proposed individualized glycemic targets in the ICU based on the degree of stress hyperglycemia versus chronic hyperglycemia.[3] Our study supports this concept: SHRI could help tailor glycemic control, identifying truly stress-induced hyperglycemia that might merit intervention.

It is also noteworthy that the SHRI maintained a prognostic value in both diabetic and nondiabetic patients in our cohort (though the relative impact was greater in nondiabetics), indicating that stress hyperglycemia confers incremental risk regardless of baseline diabetic status. Stress hyperglycemia thus appears to confer incremental risk regardless of baseline diabetic status. For trauma clinicians, this means an elevated SHRI should not be dismissed simply because a patient has known diabetes; it still indicates that the patient's glucose is disproportionately high relative to their chronic levels, which could reflect uncontrolled stress or impending physiological collapse. Our findings align with recent evidence that the predictive power of stress hyperglycemia indices is not diminished by the presence of diabetes. For example, a 2024 study of ICU trauma patients found that an elevated SHR was associated with higher mortality risk in both diabetics and nondiabetics, that is, the relationship between SHR and outcomes is not affected by the presence of diabetes.[15] In addition, a recent 2025 study by Parmer et al. showed that trauma patients who had acute stress hyperglycemia with elevated SHR had mortality rates comparable to trauma patients with known diabetes.[14]

Our study has several limitations. First, this was a retrospective analysis from a single center with an older population. In the future, it would be beneficial to conduct a prospective investigation that includes all trauma patients. Second, not all trauma patients routinely have HbA1c measured on admission; in our center, this was done broadly for clinical purposes, but in other settings, SHR might not be readily available without a baseline A1c. Third, we focused on inhospital mortality as the primary outcome rather than complications or functional outcomes. Fourth, we did not adjust for all possible confounders in multivariable models; instead, we used matching on major known confounders.

Despite these limitations, our study provides novel evidence that a SHR-based SI is superior to a glucose-based index for risk stratification in trauma. Our chosen SHRI cutoff of 0.41 corresponds to an SHR of ~1.2, which remarkably aligns with thresholds observed in other studies of critical illness.[21] This convergence suggests SHR ~1.2 may be an inflection point beyond which risk accelerates in various populations. In our trauma cohort, median SHR in nonsurvivors was 1.45, well beyond 1.2, highlighting how severe stress can

push patients into a high-risk hyperglycemic range even without diabetes. Notably, implementing routine HbA1c screening in a trauma cohort enabled the identification of previously unrecognized diabetic and pre-diabetic patients.[14] Recent advances have made point-of-care HbA1c devices commercially available for rapid results in acute care, which we note as a potential facilitator of using SHRI in practice.[22]

5 | CONCLUSION

This study introduces the SHRI as an improved prognostic tool in trauma, demonstrating its superior performance over the conventional SI in predicting mortality among 3197 trauma patients. By incorporating baseline glycemic context through HbA1c, SHRI better identifies patients with true stress hyperglycemia who are at highest risk of poor outcomes. The findings support integrating HbA1c-derived metrics into trauma assessment for enhanced risk stratification and potential personalization of care, representing a significant advancement toward precision medicine in emergency and critical care settings.

AUTHOR CONTRIBUTIONS

Cheng-Shyuan Rau: Writing—review and editing; writing—original draft. **Shao-Chun Wu**: Writing—original draft; writing—review and editing. **Chun-Ying Huang**: Resources. **Peng-Chen Chien**: Formal analysis. **Ching-Hua Hsieh**: Conceptualization; funding acquisition; supervision.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ETHICS STATEMENT

This study was approved by the Institute Review Board with approved number of 202500605B0. The patient consent is waived due the retrospective design.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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PEER REVIEW

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