International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

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ARTICLE TITLE	SELECTED BIOMARKERS IN SEPSIS: IL-6, sTREM-1 AND NEUTROPHIL CD64 - A LITERATURE REVIEW
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5632
RECEIVED	26 February 2026
ACCEPTED	16 April 2026
PUBLISHED	04 May 2026
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SELECTED BIOMARKERS IN SEPSIS: IL-6, sTREM-1 AND NEUTROPHIL CD64 - A LITERATURE REVIEW

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ABSTRACT

Background: Sepsis is a life-threatening condition characterised by organ dysfunction caused by a dysregulated host response to infection and remains a major cause of morbidity and mortality worldwide. Early recognition is clinically challenging because initial symptoms are non-specific and commonly used inflammatory markers have limited diagnostic performance. Consequently, there is ongoing interest in biomarkers that enable faster and more accurate identification of sepsis.
Aim: To discuss selected sepsis biomarkers: interleukin-6 (IL-6), soluble TREM-1 (sTREM-1), and neutrophil CD64 expression, with particular emphasis on biological mechanisms, clinical utility, and diagnostic limitations.
Materials and methods: A narrative review of the literature was undertaken. Publications from 2016 to 2025 indexed in PubMed/MEDLINE, Scopus, and Google Scholar were analysed, including clinical studies, reviews, and meta-analyses. Evidence was assessed with respect to biological mechanisms, diagnostic and prognostic performance, and limitations of the biomarkers.
Results: Neutrophil CD64 expression shows high specificity for bacterial infection and sepsis; however, routine use is constrained by limited assay standardisation and the absence of validated cut-off values. IL-6 rises very early and has prognostic value, correlating with the severity of organ dysfunction and mortality risk, but it has low specificity. sTREM-1 reflects activation of the TREM-1 axis and the intensity of the inflammatory response; however, its ability to distinguish sepsis from non-infectious SIRS is limited.
Conclusions: None of the reviewed biomarkers should be used as a stand-alone diagnostic test. The greatest clinical value is likely to be achieved with a multiparametric approach that combines clinical assessment and organ dysfunction scores with biomarkers representing different stages of the immune response. This strategy may improve early recognition, risk stratification, and monitoring of sepsis.

KEYWORDS

Sepsis, Biomarkers, Interleukin-6, sTREM-1, Neutrophil CD64

CITATION

Aleksander Midera, Kinga Popielarska, Klaudia Elżbieta Niwińska, Natalia Maria Leśniak, Anna Maria Zakrzewska, Julia Aleksandra Leśniak, Michał Borowski, Julia Agnieszka Michalak, Monika Augustyn. (2026) Selected Biomarkers in Sepsis: IL - 6, sTREM-1 and Neutrophil CD64 - A Literature Review. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5632

1. Introduction

This article discusses selected biomarkers that may support the detection of sepsis. Before addressing these biomarkers, it is necessary to outline the concepts of sepsis and biomarkers. According to the current and widely adopted definition established by the Third International Consensus (Sepsis-3), sepsis is a life-threatening condition caused by organ dysfunction resulting from a dysregulated host response to infection. In clinical practice, organ dysfunction is identified as an acute increase of at least 2 points in the SOFA (Sequential Organ Failure Assessment) score attributable to infection, which is associated with a markedly increased risk of death. In pre-hospital care and in emergency departments, where full SOFA assessment is not always feasible, the simplified screening tool qSOFA (quick Sequential Organ Failure Assessment) is used. qSOFA is based on three clinical variables: respiratory rate ≥22/min, systolic blood pressure ≤100 mmHg, and altered mental status. It allows rapid identification of patients with suspected sepsis who are at increased risk of adverse outcomes [1, 2]. It should be emphasised that qSOFA is a screening tool and does not replace full assessment of organ dysfunction using SOFA.

A biomarker is an objective, measurable biological characteristic, such as a substance, structure, or process, whose measurement can indicate normal or pathological biological processes, or responses to therapeutic interventions [3].

Sepsis remains one of the most serious global health problems. According to recent analyses, in 2017 there were approximately 48.9 million incident cases of sepsis worldwide, with 11 million deaths [4]. A later

estimate for 2021 suggested 166 million cases and 21.4 million sepsis-associated deaths [5], indicating that sepsis contributes substantially to global mortality. In a meta-analysis including hospital- and ICU-treated sepsis across multiple centres, in-hospital mortality among patients treated in hospital was estimated at ~26.7% (95% CI 22.9–30.7%), while among those treated in intensive care the mortality before hospital discharge was ~41.9% (95% CI 36.2–47.7%) [6].

The most used inflammatory markers are C-reactive protein (CRP) and procalcitonin (PCT). Despite their widespread use, both have important limitations in early sepsis recognition. CRP is sensitive, but specificity is low because concentrations also rise in many non-infectious conditions, including trauma, surgery, autoimmune disease, and malignancy [7, 8]. In addition, CRP has delayed kinetics, typically increasing 12-24 hours after the onset of inflammation, which limits its value as an early sepsis marker [9]. Procalcitonin is more specific for bacterial infection and generally offers better diagnostic accuracy than CRP, but it also has limitations [7]. PCT may increase after major trauma, burns, or extensive surgery, and its rise often occurs several hours after symptom onset, which may be insufficient in rapidly evolving sepsis [8, 10]. These limitations mean that CRP and PCT may not provide adequate sensitivity and specificity in the earliest phase of disease, supporting the search for more precise biomarkers.

Although analyses of data from 1990-2019 suggested a 37% reduction in age-standardised sepsis incidence and a 52.8% reduction in sepsis-related mortality, data from 2021–2022 indicate an increase in incidence by 230% compared with 1990 and an increase in mortality by 23.6%. This rise may be partly attributable to the COVID-19 pandemic; nevertheless, it highlights the need for robust, well-validated biomarkers to improve diagnosis and monitoring of sepsis across a range of clinical settings [5].

This review focuses on three biomarkers: IL-6, sTREM-1, and neutrophil CD64 expression, which represent different stages of inflammatory activation and have differing clinical utility in sepsis.

2. Methodology

This study is a narrative review aimed at synthesising current evidence on selected biomarkers used in the diagnosis of sepsis.

The literature search was conducted using PubMed/MEDLINE, Scopus, and Google Scholar. Publications dated predominantly from 2016 to 2025 were considered, with particular emphasis on recent clinical studies, reviews, and meta-analyses.

Search terms were combined in English and included: *sepsis, biomarkers, interleukin-6, IL-6, sTREM-1, soluble TREM-1, neutrophil CD64, FcγRI, SOFA score, C-reactive protein, procalcitonin*.

Eligible publications included original clinical studies, narrative and systematic reviews, meta-analyses, and consensus statements addressing biological mechanisms, diagnostic and prognostic performance, and limitations of these biomarkers in sepsis. Case reports, publications without accessible full text, and articles not directly relevant to sepsis diagnostics were excluded.

A qualitative synthesis was performed, focusing on activation and regulation mechanisms, temporal kinetics during sepsis, clinical utility, and interpretative limitations. Particular attention was paid to comparisons with conventional inflammatory markers and to the rationale for multiparametric strategies integrating biomarkers with clinical assessment and organ dysfunction scoring.

3. Results

3.1 Neutrophil CD64 expression

CD64, also known as FcγRI, is a high-affinity receptor for the Fc portion of IgG. Under physiological conditions, it is strongly expressed on cells of the mononuclear phagocyte system (e.g., monocytes and macrophages), whereas expression on neutrophils is minimal or undetectable. During bacterial infection, innate immune mechanisms trigger rapid release of inflammatory mediators, particularly interferon-γ (IFN-γ), tumour necrosis factor-α (TNF-α), and interleukin-6 (IL-6). This leads to marked induction of CD64 on the neutrophil surface via upregulation of *FCGR1A* transcription. The response develops within hours of pathogen exposure and reflects neutrophil activation in bacterial infection [11].

Multiple studies indicate that increased neutrophil CD64 expression has high specificity for bacterial infection and sepsis. In a meta-analysis in adults, neutrophil CD64 demonstrated strong diagnostic performance (AUC 0.94), with pooled sensitivity and specificity of approximately 0.88 [12].

However, key limitations remain. There is no universally adopted assay standardisation; laboratories use different antibodies and reporting approaches, and cut-off values vary across studies [11]. In a 2018 study, a cut-off of 1.45 yielded a specificity of 75%, indicating a clinically meaningful false-positive rate [13].

3.2 Interleukin-6

Interleukin-6 (IL-6) is a pleiotropic cytokine with pro-inflammatory and immunoregulatory functions. Its production is triggered by pathogen recognition through pattern recognition receptors (PRRs), including Toll-like receptors (TLRs), particularly TLR4 activated by lipopolysaccharide (LPS) from Gram-negative bacteria. IL-6 is produced by myeloid cells (e.g., macrophages and dendritic cells), as well as T lymphocytes and endothelial cells. Activation of these receptors initiates intracellular signalling pathways, mainly NF-κB, MAP kinases, and JAK–STAT, resulting in IL-6 gene transcription [14, 15]. IL-6 drives downstream effects including induction of acute-phase protein synthesis in hepatocytes, activation of B and T cells, effects on haematopoiesis, and modulation of immune responses such as phagocytosis and inflammatory signalling [16, 17].

In a prospective observational study of patients admitted to the emergency department or intensive care, IL-6 was measured over the first 24 hours in patients with sepsis. IL-6 correlated strongly with disease severity (SOFA score) and mortality risk. Regarding SOFA subcomponents, IL-6 was significantly associated with cardiovascular dysfunction ($r \approx 0.61$), renal dysfunction ($r \approx 0.64$), and neurological status ($r \approx 0.60$). IL-6 was an independent predictor of mortality, and higher IL-6, together with SOFA, and a greater need for antimicrobial therapy was associated with increased risk of adverse outcomes. The authors concluded that IL-6 should be interpreted alongside SOFA and clinical assessment rather than used in isolation [18, 19].

Although IL-6 rises early and is sensitive, its specificity is limited. IL-6 may also increase in acute non-infectious inflammatory states such as major trauma, postoperative inflammation, and autoimmune disease, which reduces diagnostic specificity for sepsis. Elevated IL-6 has also been reported in burns, malignancy, and intense physical exertion. Many studies show that IL-6 can rise markedly after trauma and surgery within 60-90 minutes of skin incision or injury, and that peak levels correlate with the extent of tissue damage. Consequently, IL-6 is best interpreted in clinical context and in combination with other biomarkers and severity assessment [20, 21].

3.3 sTREM-1

STREM-1 is the soluble form of a receptor from the TREM family. Its plasma concentration increases during activation of myeloid cells (especially neutrophils and monocytes) in severe inflammatory responses, typically associated with infection. sTREM-1 is regarded primarily as a biomarker of TREM-1 axis activation rather than a membrane receptor directly mediating signalling [22]. Two mechanisms for sTREM-1 generation have been described: proteolytic cleavage (shedding) of the extracellular domain of membrane-bound TREM-1 and alternative splicing [23]. In sepsis, innate immune activation (e.g., via TLRs) triggers inflammatory cascades in neutrophils and monocytes. TREM-1 amplifies this response, and greater pathway activation is associated with higher circulating sTREM-1 concentrations [22].

In one study, sTREM-1 was evaluated in 120 patients divided into three groups: uninfected (n=31), sepsis (n=37), and septic shock (n=52). sTREM-1 was significantly higher in both sepsis and septic shock than in the uninfected group ($p < 0.001$). Levels were also higher in septic shock than in sepsis. Higher sTREM-1 may therefore reflect greater severity and may have prognostic value, although further research is required [23].

Another study found no significant difference in sTREM-1 between sepsis and non-infectious SIRS (NI-SIRS), suggesting that sTREM-1 alone does not reliably distinguish infectious from non-infectious SIRS. In ROC analyses, sTREM-1 had a lower AUC than CRP and IL-6 for differentiating severe sepsis/septic shock from NI-SIRS [24].

A practical advantage is that sTREM-1 can be measured using immunoassays such as ELISA, supporting potential clinical use. However, the absence of widely accepted cut-off values remains a major limitation for routine decision-making [25, 26].

4. Discussion

Sepsis is a complex clinical syndrome in which disease trajectory and prognosis are largely determined by the extent of dysregulation of the host immune response. Under Sepsis-3, diagnosis hinges on the presence of organ dysfunction assessed using SOFA [1]. Despite advances in diagnostics and treatment, sepsis continues to carry high mortality, particularly among patients treated in intensive care [6]. Epidemiological observations of increasing incidence further emphasise the importance of timely diagnosis and monitoring [5].

Clinical tools such as SOFA and qSOFA are central to severity assessment, but they do not fully capture the immunological dynamics of sepsis [1, 2]. This has driven interest in biomarkers as adjuncts to clinical evaluation [3, 26]. Conventional markers such as CRP and PCT have limited specificity and relatively delayed kinetics, restricting their usefulness for very early recognition of sepsis [7, 8, 9, 10].

Neutrophil CD64 reflects early innate immune activation in bacterial infection and has shown strong diagnostic performance, with reported sensitivity and specificity around 0.88 [11, 12]. However, lack of assay standardisation and consistent cut-off values limits its role as a stand-alone diagnostic marker [11, 13].

IL-6 is a key mediator with very early kinetics and established prognostic value, correlating with SOFA-assessed organ dysfunction and mortality risk [18, 19]. Its principal limitation is low specificity, as it also rises in trauma, postoperative inflammation, and non-infectious inflammatory disease [20, 21]. Therefore, IL-6 should be interpreted strictly within clinical context.

sTREM-1 reflects activation of the TREM-1 axis and overall inflammatory intensity. Higher concentrations have been reported in sepsis and septic shock and may correlate with severity [22, 24]. Nevertheless, its ability to distinguish sepsis from NI-SIRS is limited, and the lack of validated cut-off values remains a barrier to wider implementation [25, 26].

Overall, current evidence suggests that the greatest diagnostic and prognostic value is likely to be achieved with multiparametric strategies combining clinical assessment and organ dysfunction scores with biomarkers reflecting distinct components of the immune response. This is particularly relevant in emergency and pre-hospital contexts, where early risk stratification may guide immediate management [6, 18, 26, 27].

5. Conclusions

The reviewed evidence suggests that the most clinically useful biomarkers are those reflecting early and direct immune activation in response to infection. Neutrophil CD64 demonstrates high specificity for bacterial infection and promising diagnostic performance for early sepsis. IL-6 is notable for very rapid kinetics and prognostic value, particularly in relation to organ dysfunction severity and mortality risk. sTREM-1 provides information on inflammatory amplification and may support assessment of disease severity, although its diagnostic value for distinguishing sepsis from NI-SIRS is limited. Each biomarker offers clinical utility but also has limitations that preclude its use as a definitive stand-alone test.

The literature supports combining biomarkers representing different mechanisms and stages of the inflammatory response. A multi-marker approach incorporating neutrophil activation (CD64), cytokine response (IL-6), and receptor-axis activation (sTREM-1) may reflect sepsis pathophysiology more comprehensively than any single marker. Integrating biomarker results with clinical evaluation and organ dysfunction scoring (e.g., SOFA) may improve diagnostic accuracy, enhance risk stratification, and support monitoring of disease course and treatment response.

In clinical practice, biomarkers should be regarded as adjuncts rather than substitutes for clinical judgement. Their use may be particularly valuable in emergency and pre-hospital settings to identify high-risk patients and support early diagnostic and therapeutic decisions. Further research is required to standardise assays, establish validated cut-off values, and evaluate cost-effectiveness and accessibility. These steps are essential for broader and safe implementation in routine clinical practice.

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