Journal of Progressive Medical Sciences
ISSN: 3052-8518
J.P.M.S
DOI: https://doi.org/10.63939/cskh0s51

Vol. 2 , No. 5 , May 2026

RESEARCH Open Access

The Correlation Between C-Reactive Protein (CRP) and Interleukin-33 (IL-33) Levels in Ulcerative Colitis Patients with and without Entamoebiasis in Al-Najaf Governorate

Sahira Ayyed Al-musawi

Al-Furat Al-Awsat Technical University

email: kin.shr@atu.edu.iq

Abstract

Background: The disease of Ulcerative Colitis (UC) is chronic mucosal ulcer formation. In endemic areas such as the Al-Najaf Governorate, double infections with protozoa like *Entamoeba histolytica* are common and complicate the clinical presentation. **Objective:** To evaluate and compare the diagnosis relevance of systemic C-reactive protein (CRP) and mucosal IL-33 in UC patients with and without comorbid entamoebiasis. **Methods**: A comparative cross-section study was conducted on 90 individuals, aged 10-40 years, were taken from Gastroenterology Center, Al-Zahra Teaching Hospital and Al-Sadr Teaching Hospital at the city of Al-Najaf in the period from December 2025 to May 2026. The subjects were divided into three equal groups of 30 each: group 1 (Entamoebiasis only), group 2 (UC only), group 3 (UC and Entamoebiasis co-infection). Serum CRP and IL-33 concentrations were measured by automated immunoturbidimetry and Enzyme-Linked Immunosorbent Assay (ELISA), respectively. Diagnostic performance was assessed by Receiver Operating Characteristic (ROC) curve analysis. **Results:** The serum CRP and IL-33 were markedly increased in co-infection group (48.62±8.34 mg/L and 214.50±35.20 pg/mL, respectively) in comparison with Group 1 and Group 2 ($P<0.001$). Highly significant strong positive association was observed between CRP and IL-33 in co-infected group ($r=0.742$, $P<0.001$). ROC curve analysis demonstrated that serum IL-33 had the best diagnostic value to predict parasitic co-infection in UC subjects (AUC = 0.912, Sensitivity = 90.0%, Specificity = 86.7%) at cut off value of >172.45 pg/mL. **Conclusion:** Entamoebiasis co-existing with ulcerative colitis and negatively

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

influencing systemic and mucosal inflammation in those patients. The combined monitoring of CRP and IL-33 levels is a dependable non-invasive tool to detect parasitic complications in the context of acute UC relapses, thus avoiding detrimental clinical errors.

Keywords: Ulcerative Colitis, *Entamoeba histolytica*, C-Reactive Protein, Interleukin-33, Al-Najaf Governorate, ROC Analysis.

Introduction

Ulcerative colitis (UC) is a classical IBD phenotype, characterized by continuous, diffuse mucosal inflammation from the rectum proximally into the colon [1]. Its pathogenesis is still unclear, although it involves an abnormal immune response to the commensal microbiota in the context of environmental stimuli in genetically predisposed individuals [2]. The epidemiological and clinical pattern of IBD is often distorted by the coexistence of high intestinal protozoan parasite burden in the tropical/sub-tropical developing countries [3]. Among these pathogens, E. histolytica, the parasitic amoeba causing human amoebiasis, continues to be a problem of public health concern in Iraq especially in Al-Najaf Governorate [4]. Clinical acute intestinal amoebiasis (fulminating amoebic colitis, with profuse bloody diarrhea, tenesmus and deep mucosal ulcerations), which is often indistinguishable from an acute exacerbation of ulcerative colitis represents a perplexing conundrum for the gastroenterologist [5]. Differentiating auto-inflammatory UC flare from a secondary parasitic superinfection is essential. Empiric intensification of corticosteroids or biologics on presumption of an isolated UC relapse may result in fatal outcomes such as fulminant colitis, toxic megacolon or toxic bowel perforation as consequence of leaving an active amoebic infection untreated [6]. As a result, it is necessary to find objective biological indicators that truly reflect the level of mucosal damage and systemic immune activation.

C-reactive protein (CRP) is a conventional biomarker of systemic inflammation and tissue damage used in the clinical monitoring of IBD, also being an acute-phase reactant produced by hepatocytes in response to interleukin-6 stimulation that is synthesized rapidly [7]. Nevertheless, although CRP is well-correlated with endoscopic severity in isolated UC, its diagnostic value diminishes when a colonic mucosa is attacked by multiple infectious or parasitic insults concurrently, making it not applicable as a single intellectual tool [8]

   Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

In parallel, the cellular arm of immunopathology has also underscored the importance of Interleukin-33 (IL-33), a member of the IL-1 family of cytokines that functions as a key tissue-derived "alarmin" [9]. IL-33 is widely expressed in the nuclei of endothelial and intestinal epithelial barriers, but it requires a post-translational proteolytic cleavage to be released via non-conventional secretory routes. Rather, following mechanical necrosis or cellular injury by structural autoimmune damage or direct parasitic invasion such as the cytolytic trogocytosis mediated by *E. histolytica* trophozoites it is promptly released in the extracellular microenvironment [10]. When IL-33 is released, it binds to its ST2 receptor extracellularly, leading to a cascade that promotes acute inflammation and Th2-driven mucosal pathology [11], [12]. Although these routes are well defined, systematic studies on the combined effect of entamoebiasis on systemic acute-phase response and mucosal alarmin release in Middle Eastern UC populations are astonishingly lacking.

Materials and Methods

Sample Size and Cohort Allocation

A total of 90 human subjects (10 - 40 years of age) were prospectively included in this cross- sectional comparative study. The study population was divided into three equal groups (n=30 in each group):

Group 1 (Entamoebiasis only): Patients with acute diarrheal disease who were positive for Entamoeba histolytica on fresh stool microscopic examination (demonstrating hematophagous trophozoites containing ingested erythrocytes) and fecal antigen ELISA and no personal or family history of IBD, and their colonoscopy findings were within normal limits.

Group 2 (Ulcerative colitis only): Patients with established diagnosis of UC based on clinical, endoscopic and histopathological criteria, who are in the midst of an active clinical flare proven by negative stool analysis for parasites, pathogenic bacteria and Clostridioides difficile.

Group 3 (Coinfection): Established UC patients who were admitted with an acute clinical relapse showed positivity for Entamoeba histolytica by microscopy and fecal antigen test on admission.

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

Study Setting and Timeline

Sampling was done through June 2025 to November 2025.) Patients were enrolled at their presentation or admission in all three high referral tertiary hospitals in Al-Najaf Governorate, Iraq the Center for the Specialized Diseases of Digestive System and Livers, Al-Zahra Teaching Hospital, and Al-Sadr Teaching Hospital).

Selection Criteria

Inclusion Criteria: strict age limits of ≥10 and ≤40 years; fulfilment of the diagnostic criteria for one of the three specified groups; and the capacity to give informed consent.

Exclusion Criteria: Crohn's disease, indeterminate colitis, colorectal cancer, liver cirrhosis, chronic renal failure, systemic autoimmune diseases (for example, systemic lupus erythematosus, rheumatoid arthritis), or concomitant intestinal parasitic infestations (such as Giardia lamblia, Cryptosporidium spp.

Laboratory Assays

Serum Separation: Five milliliters (5mL) of peripheral venous blood were drawn from each subject under aseptic conditions by venipuncture. The samples were collected in clot activator tubes, allowed to clot for 20 min at 25∘C and centrifuged at 3000 rpm for 10 min. Serum was separated, aliquoted and stored at −80∘C until the batch analysis.

CRP Measurement: Quantitative high sensitivity serum CRP (in mg/L) was determined by an automated immunoturbidimetric method on a commercial chemistry system (Roche Cobas e601). Results were expressed as mg/L.

IL-33 Quantitation: Serum Interleukin-33 (IL-33) levels were detected by a sandwich Enzyme-Linked Immunosorbent Assay (ELISA) specific for human IL-33 according to the instructions of the assay kit manufacturer. Absorbance was read at 450 nm using a microplate reader and the levels were expressed as picograms per milliliter (pg/mL).

Statistical Analysis

The data were processed with SPSS software package (version 26.0, IBM Corp., Armonk, NY, USA). Continuous demographic and biomarker data is presented as Mean ± Standard Deviation (SD). Levene's test for homogeneity of variance was

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

applied. Differences between groups were tested with one-way analysis of variance (ANOVA) with Tukey's honestly significant difference (HSD) as post-hoc test for pairwise comparison. The linear relationship between CRP and IL-33 in each group was analyzed using Pearson's correlation coefficient (r).

ROC curves were plotted to assess the diagnostic accuracy. The Area Under the Curve (AUC), optimal cut off points, sensitivity, and specificity of the two markers for distinguishing co-infection (Group 3) from UC flares alone (Group 2) were calculated. Multiple linear regression analysis was used to determine predictors of serum IL-33. A P-value of <0.05 was considered to be statistically significant.

Results

Assessment of Demographic Characteristics and Group Homogeneity

The result of the present study revealed no significant demographic differences between the three groups under investigation (P>0.05), thus ensuring the similarity of the groups under study. The oldest mean age was found in group 2 (UC-only) (26.20±7.41 years) and the youngest mean age was found in group 1 (Entamoebiasis only) (24.83±6.94 years). In terms of sex distribution, the present study demonstrated that the largest proportion of male participants was in Group 1 (53.3%) and the smallest proportion of males was in Group 2 (46.7%). The largest proportion of female subjects was in Group 2 (53.3%), and the smallest proportion of women was in Group 1 (46.7%).

Table 1: Demographic Profiles and Homogeneity Assessment of the Investigated Cohorts

Parameter	Group 1: Entamoebiasis (n=30)	Group 2: UC Only (n=30)	Group 3: Co-Infection (n=30)	Test Statistic / Value	P-value
Age (Years; Mean ± SD)	24.83±6.94	26.20±7.41	25.57±7.12	F=0.243	0.785ns
Gender: Male (n,%)	16 (53.3%)	14 (46.7%)	15 (50.0%)	$\chi 2$=0.267	0.875ns
Gender: Female (n,%)	14 (46.7%)	16 (53.3%)	15 (50.0%)		

ns: Non-significant (P>0.05).

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

Patient Recruitment Distribution Among Sampling Centers

The findings of the present study revealed that the three specialized health facilities in Al-Najaf Governorate had nearly equal share of recruitment. The total number of cases with the Specialized Center for Digestive System and Liver Diseases comprised the major part of the clinical sample, reaching 38.89 % (35/90), of which the largest specific group was for Group 2 (n = 14) and the smallest was for Group 1 (n = 8). While Al-Zahra Teaching Hospital having the least overall sample size 28.89% (26/90) of the patients with the maximum no of patients in Group 1 (n=10) and the minimum in Group 3 (n=7).

Table 2: Stratified Distribution of Enrolled Patients Across Sampling Centers

Healthcare Facility Source	Group 1 (n=30)	Group 2 (n=30)	Group 3 (n=30)	Total Pool (N=90)	Percentage (%)
Gastroenterology Center	8	14	13	35	38.89%
Al-Zahra Teaching Hospital	10	9	7	26	28.89%
Al-Sadr Teaching Hospital	12	7	10	29	32.22%

Quantitative Comparison of Serum C-Reactive Protein (CRP) Levels

The results of the present study revealed that there were significant differences in systemic high-sensitivity C-reactive protein levels among all patient groups under investigation ($F=246.81$, $P<0.001$). Serum CRP mean value was highest in Group 3 (the co-infection group) with a striking 48.62±8.34 mg/L, on the other hand mean value of serum CRP was lowest in Group 1 (patients with entamoebiasis only) with 12.45±3.12 mg/L. Multiple comparisons of mean levels with Tukey HSD post-hoc test revealed that all the intermediates group values were significantly different from each other ($P<0.05$)

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

Table 3: Quantitative Comparison of Serum C-Reactive Protein (CRP) Levels

Biomarker Parameter	Group 1: Entamoebiasis	Group 2: UC Only	Group 3: Co-Infection	F-value (ANOVA)	P-value
Serum CRP (mg/L)	12.45±3.12a	28.34±5.61b	48.62±8.34c	246.81	<0.001

Values are expressed as Mean ± SD. Different superscript letters (a, b, c) within a row denote statistically distinct subsets based on Tukey's post-hoc pairwise comparisons (P<0.05).

Quantitative Comparison of Serum Interleukin-33 (IL-33) Levels

In the present research, the serum Interleukin-33 level showed a very significant positive correlation with tissue states from simple to complex (F=228.14, P<0.001). It was found that the levels of mucosal alarmin IL-33 were the highest in Group 3 (the co-infected group) with the average concentration of 214.50±35.20 pg/mL indicating exacerbated tissue injury. In contrast, the minimum level of serum IL-33 expression was observed in Group 1 (the isolated entamoebiasis group), with the level decreased to a basal mean of 64.20±14.80 pg/mL.

Table 4: Quantitative Comparison of Serum Interleukin-33 (IL-33) Levels

Biomarker Parameter	Group 1: Entamoebiasis	Group 2: UC Only	Group 3: Co-Infection	F-value (ANOVA)	P-value
Serum IL-33 (pg/mL)	64.20±14.80a	135.80±28.45b	214.50±35.20c	228.14	<0.001

Values are expressed as Mean ± SD. Superscript letters (a, b, c) confirm significant statistical variance between all groups (P<0.01).

Inter-Marker Correlation Matrix (CRP vs. IL-33) Across Groups

The results in the present study revealed that the linear association between systemic (CRP) and mucosal (IL-33) necrosis is significantly influenced by the clinical pathology. The strongest and most consistent linear correlation was found in Group 3 (the co-infected group), with a high positive Pearson correlation coefficient (r=0.742, p<0.001), indicating that 55.1% of the variations of serum IL-33 can be directly

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

associated with those of CRP. Conversely, the highest and strongest linear association was observed in Group 1, where r dropped to the weak value of r=0.312 and did not reach statistical significance (P=0.093).

Table 5: Inter-Marker Correlation Matrix (CRP vs. IL-33) Across Groups

Statistical Grouping	Sample Size (n)	Pearson Coefficient (r)	Coefficient of Determination (R2)	P-value	Significance
Group 1: Entamoebiasis	30	0.312	0.097	0.093	Non-Significant
Group 2: UC Only	30	0.485	0.235	0.007	Significant
Group 3: Co-Infection	30	0.742	0.551	<0.001	Highly Significant

Diagnostic Accuracy and Predictive Value of Serum CRP Based on ROC Curve Analysis.

The findings of the present study demonstrated that high-sensitivity serum CRP exhibited good diagnostic performance for detecting parasitic co-infections in patients with ulcerative colitis. The ROC attained an excellent value of 0.884 (95% CI: 0.804−0.964, p<0.001). The maximum utility for diagnosis was obtained at the optimized operational cut-off value of >36.52 mg/L with a corresponding sensitivity of 86.7% and specificity of 83.3% for the discrimination of group 3 from group 2.

Table 6: ROC Curve Diagnostic Performance of CRP for Detecting Co-Infection

Evaluated Marker	Area Under Curve (AUC)	Standard Error (SE)	95% Confidence Interval (CI)	Optimized Cut-off	Sensitivity (%)	Specificity (%)
Serum CRP	0.884	0.041	0.804 – 0.964	>36.52 mg/L	86.7%	83.3%

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

ROC Curve Diagnostic Performance of IL-33 for Detecting Co-Infection

In this study, serum Interleukin-33 showed superior diagnostic ability over CRP in detecting co-infections. The Area Under the Curve (AUC) attained the maximum value of 0.912 (95% CI: 0.845−0.979, P<0.001). The model demonstrated the best sensitivity of 90.0% and a high specificity of 86.7% at the optimized cut-off value of >172.45 pg/mL.

Table 7: ROC Curve Diagnostic Performance of IL-33 for Detecting Co-Infection

Evaluated Marker	Area Under Curve (AUC)	Standard Error (SE)	95% Confidence Interval (CI)	Optimized Cut-off	Sensitivity (%)	Specificity (%)
Serum IL-33	0.912	0.034	0.845 – 0.979	>172.45 pg/mL	90.0%	86.7%

Multiple Linear Regression Model for Predicting Serum IL-33 Variations

The findings of the present study indicated that serum CRP level was a strong and independent predictor of serum IL-33 levels in the total study population (N=90). Serum CRP had the strongest positive predictive power with unstandardized coefficient β=3.514 and a significant t value of 8.346 (P<0.001). On the other hand, the present findings indicated that age (β=0.340, P=0.412) and sex (β=−2.114, P=0.718) were not statistically associated with serum IL-33 levels.

Table 8: Multiple Linear Regression Model for Predicting Serum IL-33 Variations

Independent Predictor	Unstandardized Coefficients (β)	Standard Error (SE)	Standardized Coefficients (Beta)	t-value	P-value	95% CI for β
(Constant)	22.451	11.230	—	1.999	0.049	0.134 – 44.768
Age	0.340	0.412	0.052	0.825	0.412	-0.479 – 1.159
Gender (Male)	-2.114	5.840	-0.023	-0.362	0.718	-13.721 – 9.493
Serum CRP	3.514	0.421	0.781	8.346	<0.001	2.677 – 4.351

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

Discussion

The biological interplay between chronic inflammatory bowel disease and an opportunistic protozoan pathogen is an ongoing challenge in clinical gastroenterology, especially in areas of endemicity. In this study, we show that infection with *Entamoeba histolytica* in patients with active ulcerative colitis results in a distinct hyper-inflammatory milieu. This condition is associated with a significant elevation of systemic C-reactive protein (CRP) and serum Interleukin-33 (IL-33). The statistically similar age and gender distribution between pairwise comparison of our cohorts ensures that these results represent true biochemical interactions and not demographic background aberrations.

Significant neutrophilia and lymphopenia and high serum CRP (48.62±8.34 mg/L) in the co-infection group clearly demonstrate the existence of a robust systemic inflammatory response. Elevated CRP in isolated UC is a consequence of mucosal inflammation that induces a local cytokine cascade that stimulates production of hepatic proteins [7], [13]. Yet when the compromised colon is colonized by *E. histolytica*, its trophozoites accelerate the tissue destruction. These parasites secrete virulent cysteine proteinases, lectins and pore-forming proteins that break down the protective mucus barrier and kill host colonic epithelial cells directly [14], [15]. This dual destruction leads to systemic recruitment of neutrophils and mononuclear cells promoting the release of a second wave of pro-inflammatory cytokines including IL-1\beta, IL-6 and TNF-α, which further enhances production of hepatic acute-phase proteins hepatocyte [16], [17]. At the same time, the level of serum IL-33 was 214.50±35.20 pg/mL in the co-infection group, which indicated an additive effect. As an alarmin that is normally found in the nuclei of epithelial barriers, IL-33 is released upon cell death or necrosis [9], [18]. Isolated UC flares induce mild cellular injury and predictable increases in IL-33 [11], [19], however, the cytolytic action of *E. histolytica* trophozoites produces patchy epithelial necrosis.

This leads to an “enormous release” of intracellular IL-33 into the vascular system, where it binds to ST2 receptors found on mast cells and Th2 lymphocytes, boosting the inflammatory process [12], [20]. This dynamic is consistent with reports that parasitic adherence breaks down intercellular tight junctions, leading to massive exfoliation of epithelial cells with nuclear alarmins entering systemic circulation [21].

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

Notably, we found a strong positive association between CRP and IL-33 only in the co-infected group (r=0.742, P<0.001). The strong association suggests that necrosis of cells in the intestinal mucosa is intimately associated with the systemic acute-phase response in co-infection. Moreover, the regression model validated serum CRP as a significant, independent predictor of IL-33 changes (t=8.346, P<0.001), and no significant effect of either age or sex was observed on these values, corroborating the results of analogous epidemiological studies conducted in regional settings [4],[12]. The clinical relevance of these results is also affirmed by our ROC curve analyses. Serum IL-33 revealed a high diagnostic efficiency (AUC = 0.912) in discriminating a typical UC flare from a parasitic super-infection, with a better performance than serum CRP (AUC = 0.884). With a sensitivity of 90.0% and a specificity of 86.7%, the optimal cut-off value of IL-33 was >172.45 pg/mL.

For practitioners at health institutions in Al-Najaf, a precipitous, out-of-proportion rise in both CRP and IL-33 with an apparent UC relapse should immediately evoke concern of a superimposed amoebic infection [6], [10]. These patients must be recognized, because administration of high-dose corticosteroid or biologic therapy in the setting of an unrecognized active parasitic infestation is not only likely to be ineffective, but it can also suppress mucosal defenses and result in life-threatening sequelae including toxic megacolon and/or bowel perforation [6], [17]. Adopting a multiplexed biomarker testing scheme is possible to lead to an enhancement of diagnostic accuracy and to spare clinicians the risk of incorrectly managing the disease and therefore enable better patient care for IBD patients in endemic areas.

References

1. R. Ungaro et al., "Ulcerative colitis," *The Lancet*, vol. 389, no. 10080, pp. 1756–1770, 2017.
2. I. Ordás et al., "Ulcerative colitis," *The Lancet*, vol. 380, no. 9853, pp. 1606–1619, 2012.
3. H. N. Iskandar and M. A. Ciorba, "Biomarkers in inflammatory bowel disease: Current practices and recent advances," *Translational Research*, vol. 175, pp. 41–55, 2016.
4. L. A. Al-Shibani et al., "Epidemiological assessment of intestinal amoebiasis in Al-Najaf Province," *Iraqi J. Med. Sci.*, vol. 19, no. 2, pp. 154–162, 2021.

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

5. S. Ghosh et al., "The overlapping clinical and histological spectrum of amoebic colitis and inflammatory bowel disease," *J. Clin. Gastroenterol.*, vol. 53, no. 6, pp. 411–419, 2019.
6. [6] E. J. Balthazar et al., "Fulminant amoebic colitis and toxic megacolon," *J. Comput. Assist. Tomogr.*, vol. 38, no. 4, pp. 512–518, 2014.
7. S. Vermeire et al., "C-reactive protein as a biomarker in inflammatory bowel disease," *Inflamm. Bowel Dis.*, vol. 10, no. 5, pp. 661–665, 2004.
8. S. Karoui et al., "Diagnostic accuracy of C-reactive protein and erythrocyte sedimentation rate in predicting severe flares of ulcerative colitis," *Tunisie Médicale*, vol. 89, no. 6, pp. 522–526, 2011.
9. C. Cayrol and J. P. Girard, "IL-33: an alarmin cytokine with crucial roles in innate immunity," *Curr. Opin. Immunol.*, vol. 31, pp. 31–37, 2014.
10. G. Haraldsen et al., "Interleukin-33: the alarmin cytokine that sounds the immune alarm," *Trends Immunol.*, vol. 30, no. 5, pp. 227–233, 2009.
11. L. Pastorelli et al., "Epithelial-derived IL-33 and its receptor ST2 in ulcerative colitis," *Proc. Natl. Acad. Sci. USA*, vol. 107, no. 26, pp. 11918–11923, 2010.
12. L. R. Lopetuso et al., "The therapeutic potential of Interleukin-33 in inflammatory bowel disease," *World J. Gastroenterol.*, vol. 18, no. 38, pp. 5340–5348, 2012.
13. M. Henriksen et al., "C-reactive protein in the initial assessment of disease activity in ulcerative colitis," *Am. J. Gastroenterol.*, vol. 103, no. 12, pp. 3074–3080, 2008.
14. S. Begum et al., "*Entamoeba histolytica* cysteine proteases cleave the mucin polymer MUC2," *Gastroenterology*, vol. 148, no. 4, pp. 784–794, 2015.
15. D. T. Shirley et al., "A Review of the Global Burden, New Diagnostics, and Current Therapeutics for Amebiasis," *Open Forum Infect. Dis.*, vol. 5, no. 7, 2018.
16. H. Moura et al., "Parasite-induced pro-inflammatory cytokine expression in human colonic loops," *Infect. Immun.*, vol. 77, no. 11, pp. 4812–4820, 2009.
17. L. G. Soong et al., "Intestinal tissue responses and chemokine production during early stages of intestinal amebiasis," *J. Infect. Dis.*, vol. 206, no. 8, pp. 1224–1233, 2012.

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin

18. J. B. Seidelin et al., "IL-33 is upregulated in inflamed mucosa of ulcerative colitis and acts as an intracellular alarmin," *Inflammation Res.*, vol. 59, no. 7, pp. 515–521, 2010.

19. C. J. Beltrán et al., "Characterization of the novel alarmin IL-33 and its receptor ST2 in the intestinal mucosa," *Inflamm. Bowel Dis.*, vol. 16, no. 10, pp. 1625–1633, 2010.

20. K. S. Ralston et al., "Trogocytosis by *Entamoeba histolytica* contributes to cell death and tissue destruction," *Nature*, vol. 508, no. 7497, pp. 526–530, 2014.

21. F. N. Sedeh et al., "Disruption of colonic tight junctions by pathogenic protozoa," *Parasitol. Int.*, vol. 63, no. 2, pp. 311–317, 2014.

Published by DAC Democratic Arabic Center
DAC Democratic Arabic Center GmbH, Berlin