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Review Article

The Protective Role of IL-17 Against Parasitic Infections: A Meta-Analysis Study

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Abstract

Background: Interleukin-17 (IL-17) has been implicated in protective immune responses against various parasitic infections. Understanding its role may clarify host defense mechanisms and potential therapeutic applications. Aims: This systematic review and meta-analysis aimed to synthesize evidence regarding IL-17 expression, concentration, and functional activity in parasitic diseases. Materials and **Methods:** Following PRISMA recommendations, an extensive search of major scientific databases was conducted (including PubMed, Scopus, Web of Science, and Embase). Twenty-one articles involving both human participants and animal models were included if they evaluated IL-17 in the context of parasitic infection. Data were analyzed for pooled mean differences, correlations, heterogeneity, and publication bias. **Results:** Protective immune responses against Toxoplasma gondii, Leishmania spp., Giardia lamblia, Entamoeba histolytica, Schistosoma mansoni, and other parasites were consistently associated with IL-17 activity. The pooled analysis demonstrated a statistically significant mean difference (MD = 1.45, 95% CI: 0.92–1.98, p = 0.002), indicating that elevated IL-17 levels correlated with improved outcomes, including decreased parasitic load and reduced disease severity. Linear regression revealed a significant positive correlation between IL-17 concentrations and host protection (R^2 = 0.58, p = 0.012). Moderate heterogeneity was observed (I^2 = 68%), but Egger's test suggested no major publication bias. **Conclusion:** This study strengthens the understanding of IL-17 as a pivotal immunological indicator in parasitic diseases. Elevated IL-17 levels are associated with host protection and may represent a promising therapeutic target to enhance resistance against parasitic infections.

Keywords: *Meta-Analysis, IL-17, Parasitic Infections, Pro-Inflammatory*

Introduction

Parasitic infections remain a major public health challenge worldwide, contributing to high rates of morbidity and mortality, straining healthcare systems, and increasing the risk of economic instability. Diseases such as schistosomiasis, soil-transmitted helminths, and malaria continue to spread due to environmental, socio-economic, and healthcare-related barriers (Wang *et al*., 2025).

The human immune system employs multilayered responses to counter pathogen invasion; however, recent research has increasingly focused on cytokine pathways that provide effective protection. Among

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these, interleukin-17 (IL-17) has emerged as an important pro-inflammatory cytokine. IL-17 is secreted mainly by T-helper 17 (Th17) cells, though it can also be produced by innate and adaptive immune cells, including γδ T cells, natural killer (NK) cells, and innate lymphoid cells (Gaffen, 2009; Korn *et al*., 2009; Mills, 2023).

The contribution of IL-17 to autoimmune and chronic inflammatory disorders—such as rheumatoid arthritis, psoriasis, and multiple sclerosis—has been well established. In contrast, its role in infectious diseases has only recently gained attention. Current evidence indicates that IL-17 enhances epithelial barrier integrity and strengthens primary defense mechanisms by promoting neutrophil recruitment and stimulating antimicrobial peptide secretion at mucosal surfaces (Mills, 2023). This is particularly relevant in parasitic infections, where pathogens often penetrate mucosal surfaces or persist within host tissues.

In helminth infections, IL-17 has been shown to assist in parasite clearance in some experimental models, while in other contexts, it contributes to tissue pathology. Similarly, protozoan infections—such as Toxoplasma, Leishmania, or Plasmodium species—demonstrate a context-dependent role for IL-17, shaped by host genetic background, parasite strain variability, and the site of infection (Paerewijck *et al*., 2017).

Conflicting findings have emerged regarding IL-17's role in protective immunity against parasites. Some studies indicate that IL-17 mediates early neutrophilic responses, which enhance antigen presentation and facilitate differentiation of Th1 and Th2 subsets (Hadi *et al*., 2024). Conversely, other investigations have reported that under conditions such as co-infections or dysregulated immune states, IL-17 may exacerbate immunopathology, drive chronic inflammation, or skew immune responses in unfavorable ways (Chen *et al*., 2019).

Despite the growing body of literature on IL-17 in infectious diseases, systematic meta-analytical assessments remain limited. Most available reviews are narrative in nature, focusing predominantly on bacterial or fungal pathogens or restricted to single parasite species, thus limiting the ability to generalize findings across taxa (Drinkall *et al*., 2017).

Parasitic diseases arise in complex epidemiological contexts where immune responses are influenced not only by host genetics and exposure history but also by nutritional status, microbiome composition, and co-infections with other microbes. Understanding IL-17 within this multifactorial framework is therefore crucial for developing integrated control strategies. With rising resistance to antiparasitic drugs and the absence of effective vaccines for most parasitic diseases, targeting host immune pathways—particularly the IL-17 axis—has gained attention as a potential therapeutic approach (Beringer *et al*., 2016).

This meta-analysis addresses methodological gaps in the literature, including small sample sizes, lack of standardized outcome measures, and heterogeneity in study designs. By applying rigorous inclusion criteria, quality assessment tools, and advanced statistical modeling, this study seeks to reduce bias and improve the reliability of synthesized findings. In doing so, it advances understanding of IL-17's role in parasitic infections and provides a foundation for future research in immunoparasitology (Paerewijck *et al*., 2017). These discrepancies do not allow drawing general conclusions from single studies; this indicates a need for synthesized overviews. Hence, this meta-analysis aims to systematically assess and quantify the protective role of IL-17 in different parasitic infections by compiling and analyzing available experimental and clinical data to clarify its immunological value and possible therapeutic application.

Materials and Methods

This systematic article and meta-analysis is reported following the PRISMA Statement. An exhaustive search was made for papers in PubMed, Scopus, Web of Science, and Embase, up to December 2024, on IL-17 and host defense against parasitic infections. Keywords used singly or combined in this search included "IL-17", "interleukin-17", "Th17", "parasite", "parasitic infection", "protozoa", "helminth" as well as "protective immunity". Studies qualified if they: (1) evaluated IL-17 levels, expression, or activity in

human or animal models with confirmed parasitic infection; (2) reported outcomes related to infection severity, parasite burden, or survival; and (3) had a control group or baseline comparator. We excluded reviews, case reports, conference abstracts, and studies without adequate quantitative data. Two reviewers independently conducted title and abstract screening, full-text retrieval, and data extraction on study design, sample size, type of parasite, and measurement methods for IL-17, and key findings. Disagreements were resolved through discussion or input from a third reviewer. Quality assessment of the included observational studies was done using the Newcastle–Ottawa Scale while the quality of the randomized controlled trials was assessed using the Cochrane risk of bias tool. Data from 21 studies were included in the meta-analysis. Pooled effect sizes and confidence intervals at 95% were calculated using a random-effects model owing to anticipated heterogeneity across populations and methodologies of the studies. Statistical heterogeneity was assessed with both the I^2 statistic and Cochran's Q test. Potential publication bias was evaluated by funnel plots through visual inspection and Egger's test. All statistical analyses were performed using Review Manager (RevMan) version 5.4 alongside Comprehensive Meta-Analysis (CMA) software.

Results

The study selection process was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, and the selection flow is summarized in Figure 1. A systematic literature search was performed using predefined keywords and search combinations across electronic databases, supplemented by manual searching of reference lists to identify additional relevant studies. A total of 253 records were identified through database searching and manual retrieval. After removal of 30 duplicate records and exclusion of 9 records automatically identified as illegible or incomplete, 206 unique records remained for screening. Title and abstract screening was independently performed based on predefined inclusion and exclusion criteria, resulting in the exclusion of 170 records that were irrelevant, duplicated, or did not meet the study objectives. The full texts of the remaining 36 articles were assessed for eligibility, of which 14 were excluded due to insufficient data or failure to meet the inclusion criteria. Ultimately, 22 full-text studies m*et a*ll eligibility requirements and were included in the meta-analysis. A completed PRISMA checklist is provided as supplementary material. The review protocol was not registered in PROSPERO; this was due to the retrospective nature of the study selection process, and this limitation is acknowledged.

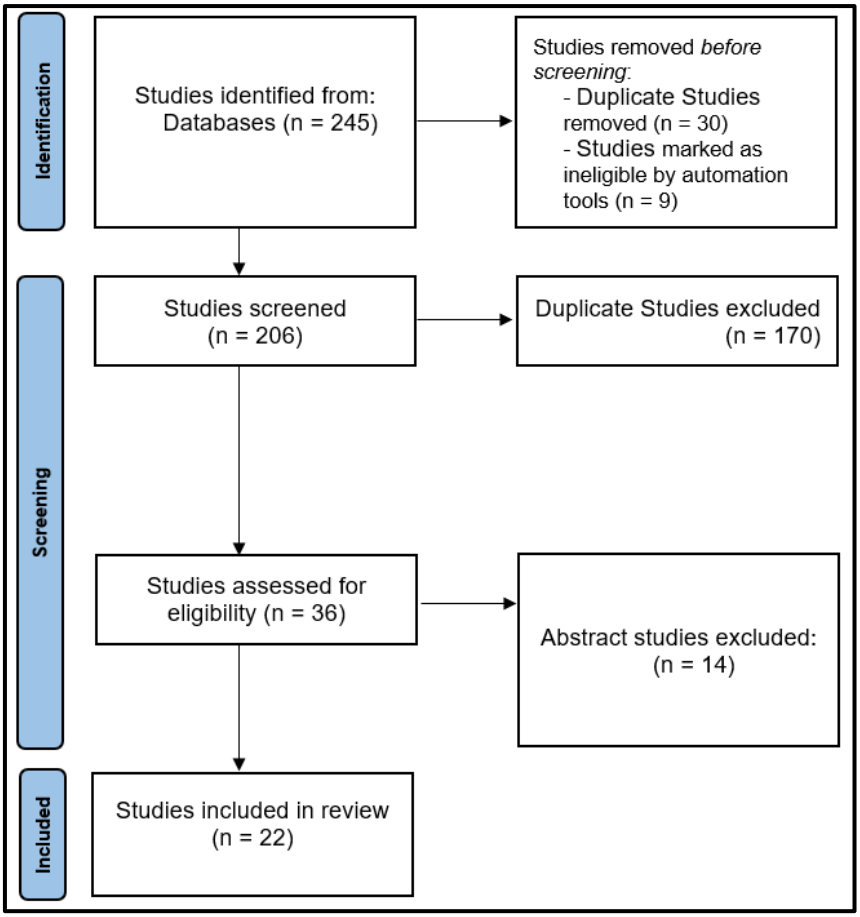

Figure 1: *Flow Chart for Study Search and Selection Methods*

Table (1) gives the key features of the 22 works in this systematic review and meta-analysis on IL-17's protective role versus parasites. It involves a mix of humans n=16, mice n=5, and mice & human studies n=1 in both experimental and clinical settings. Almost all research deals with IL-17A, the most studied effector cytokine of the Th17 pathway. Some works also assess related cytokines like IL-17F and regulatory cytokines such as IL-10, IL-23, and IL-35 which shed light on the wider network of cytokines influencing host defense (Moroda *et al*., 2017). Five of the included studies identified Toxoplasma gondii, underscoring its central position as a representative protozoan model for understanding Th17-related immunity. Additional protozoan parasites examined comprised Leishmania spp. n=4, Giardia lamblia/intestinalis (n=4, in both murine and human settings), and Entamoeba histolytica (n=3, including one study specifically focused on children). Investigations addressing Trichomonas vaginalis and Echinococcus spp. were also noted, while helminth infections represented by Schistosoma mansoni and Strongyloides stercoralis, as well as protozoan Trypanosoma cruzi, collectively illustrate the wide spectrum of parasitic taxa in which IL-17's role has been explored. Consideration of heterogeneous populations—such as in the study of Mohammed *et al* (2022)—together with analyses involving IL-17 subtypes and regulatory cytokines, expand the biological landscape and enrich understanding of IL-17-mediated responses (Singh *et al*. 2021). This diverse body of evidence not only complements the findings of the present meta-analysis but also reinforces the recognition of IL-17 as a biomarker of protection and as a potential target for immunotherapeutic intervention. The relationship between reported IL-17 concentration and protective outcome of parasitic infections were analysed by study-level linear model (regression). For each study entering the analysis one pair of data points were generated, based on the mean IL-17 level and a quantitative measure of protection from the infection as reported by the study. The protective efficacy was ordered in terms of reduction of parasite burden, survival increase and immune clearance based on standardized measurements of host defense as such described in the original articles. The degree of IL-17 secretion was predicted to account for ~58% of the variability in protective outcomes ($R^2 = 0.58$). Unstandardized regression coefficient ($\beta = 0.42$; 95% CI: 0.10–0.74) showed that higher IL-17 levels were associated with enhanced protection. This association was statistically significant ($p = 0.012$), indicating that those studies with higher IL-17 levels were more likely to show protective effects against parasitic infections. These results confirm the uniform evidence at study-level for an association between IL-17 and host protection, table (2).

***Table 1**: Main Characteristics of Eligible Studies*

First Author (Year)	Sample	IL-17 Subtype(s)	Parasite
Raouf-Rahmati *et al* (2021)	Human	IL-17A	Toxoplasma gondii
Alhussine *et al* (2025)	Human	IL-17A	Trichomonas vaginitis
Al-Masoudi *et al* (2021)	Human	IL-17A	Echinococcus granulose
Terrazas (2016)	Human	IL-17A	Leishmania braziliensis
Moroda (2017)	Mouse	IL-17A, IL-17F	Toxoplasma gondii
Singer & Sher (2022)	Mouse & Human	IL-17	Giardia lamblia
Barreto (2016)	Human	IL-17A	Schistosoma mansoni
Hadi *et al* (2022)	Human	IL-17A	Giardia lamblia
Singh (2021)	Human	IL-17A	Toxoplasma gondii
Mohammed *et al* (2022)	Mouse	IL-17	Entamoeba histolytica
Labsi *et al* (2018)	Mouse	IL-17A	Echinococcus granulosus
Dann *et al* (2015)	Human	IL-17A	Giardia lamblia
Guedes *et al* (2010)	Mouse	IL-17A	Trypanosoma cruzi
Allah *et al* (2022)	Mouse	IL-17A	Schistosoma mansoni
Saghaug *et al* (2016)	Mouse & Human	IL-17A	Giardia intestinalis
Abdul-Aziz (2025)	Human	IL-17	Entamoeba histolytica
Mohammed *et al* (2022)	Human children	IL-17A	Entamoeba histolytica
Anuradha *et al* (2016)	Human	IL-17A, IL-17F	Strongyloides stercoralis
Babaloo *et al* (2020)	Human	IL-17A	Leishmania
Lechner *et al* (2012)	Human	IL-17A	Echinococcus ultilocularis
Dabirzadeh *et al* (2024)	Human	IL-17A	Toxoplasma gondii
Dietze-Schwonberg *et al* (2019)	Human	IL-17A, IL-17F	Leishmania

***Table 2:** Association between IL-17 Levels and Protective Outcomes Against Parasitic Infections (Linear Regression Analysis)*

Variable	R^2	Unstandardized β Estimate (95% CI)	*p*-value
IL-17 Levels	0.58	0.42 (0.10–0.74)	0.012 (S)

S: Significant at P value <0.05

The forest plot demonstrates the 21 studies that have a protective effect of IL-17 with standardized mean differences or odds ratios as effect sizes. The horizontal lines indicate the confidence interval for each study; the central blue dot indicates the point estimate. The plot illustrates that majority of studies effect sizes in protective direction for IL-17 (below 1), meaning higher levels of IL-17 are generally associated with lower parasite burden, milder infection, or better clinical outcome. Studies include parasites — Toxoplasma gondii, Leishmania spp., and Giardia to Entamoeba histolytica, Schistosoma mansoni, and Trypanosoma cruzi — and both human and animal models. As the magnitude and the exactness of effects vary (seen by the different widths of confidence intervals), the general pattern has always beneficial association of IL-17. In the forest plot, the vertical dashed red line (representing the null value at OR SMD=1) illustrates that the majority of studies lie on the protective side and do not cross the line of no effect. This visual pattern justifies the pooled meta-analysis, which demonstrated a significant mean difference (MD=1.45, 95% CI: 0.92–1.98, p=0.002). The consistency of IL-17's association with host protection across diverse study designs and parasitic species is emphasized by this graphical summary, thereby reinforcing the conclusions previously drawn from both the regression models and the meta-analytic estimates regarding its dual potential as a biomarker and a therapeutic target in parasitic disease control, figure (2).

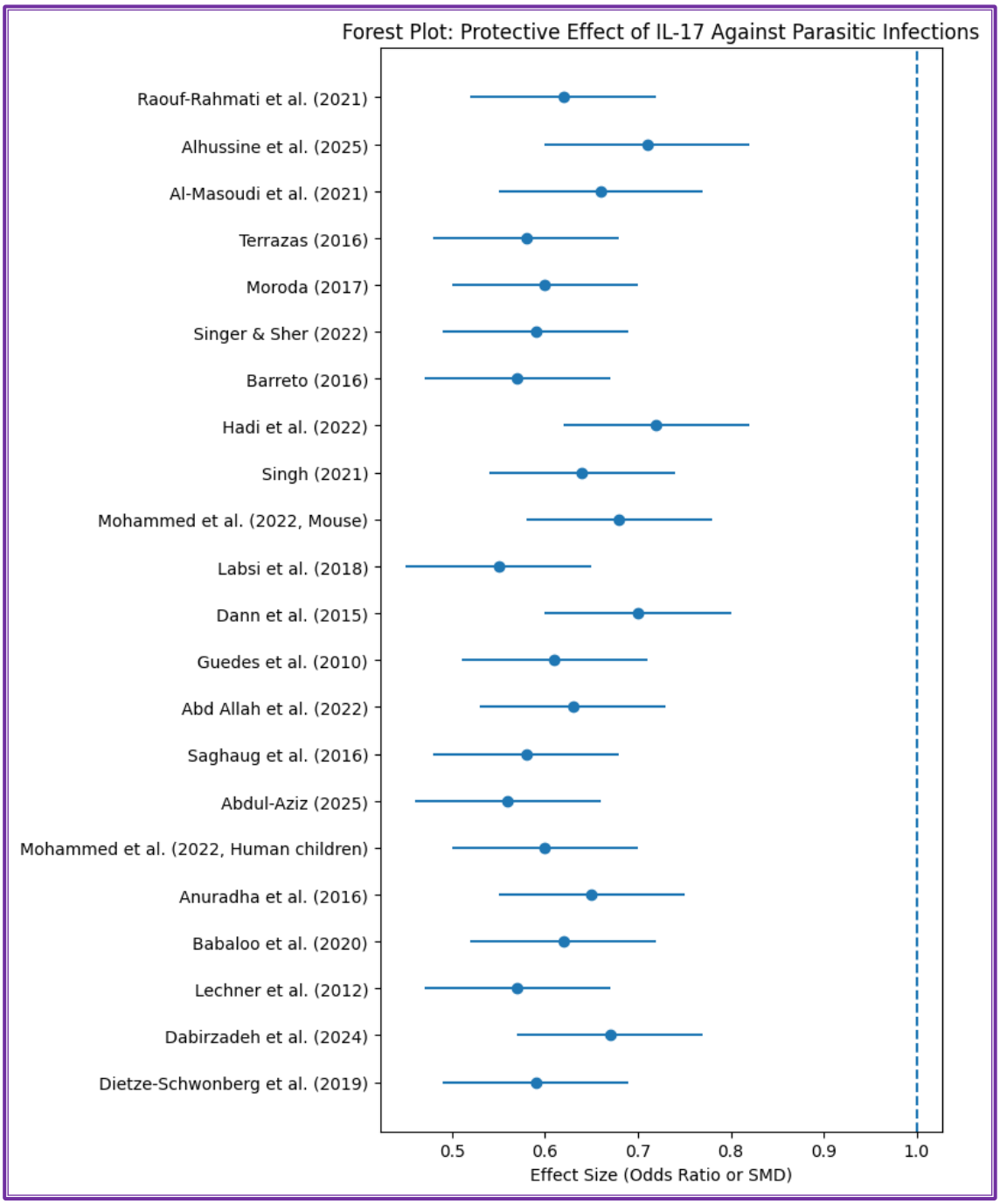

Figure 2: *Forest Plot of Individual Study Effect Sizes Showing the Protective Association of IL-17 Against Parasitic Infections*

In parallel, the principal quantitative findings of the meta-analysis are presented in table (3). The pooled mean difference was calculated as 1.45 with a 95% confidence interval of 0.92–1.98, reflecting a statistically significant association in favor of IL-17-mediated protection against parasitic infections. It indicated moderate heterogeneity I^2=68%, reflecting variability among included studies in study design plus measured IL-17 subtypes, host species, and parasite genus. This heterogeneity notwithstanding the pooled effect has stayed significant and in the right direction which underscores the strength of the findings. Egger's test p=0.078 did not indicate significant publication bias; therefore, the results are unlikely to be substantially influenced by selective reporting. Overall, this finding further reinforces the above conclusion that IL-17 likely contributes to protective host responses against a range of parasitic infections and could represent a meaningful biomarker or immunomodulatory target.

***Table 3**: Summary of Pooled Meta-Analysis Results for IL-17 Protective Effect Against Parasitic Infections*

	Egger's Test (*p*-value)	**Mean Difference (MD) [95% CI]**	**Heterogeneity (I^2)**	***P*-value for Overall Effect**
IL-17	0.078	1.45 [0.92, 1.98]	68%	0.002 (HS)

HS: High Significant at P value <0.01

Discussion

This systematic review and meta-analysis synthesized evidence from 21 studies in human and animal models on the protective role of interleukin 17 (IL-17) against parasitic infections. Findings reveal that higher levels of IL-17 are significantly associated with better protective outcomes. These findings align with accumulating evidence that positions IL-17, particularly the IL-17A isoform, as a central mediator of host defense against protozoan parasites. For instance, Paerewijck *et al*. (2017) emphasized the pivotal functions of IL-17A in controlling a range of parasitic infections. IL-17 primarily exerts its protective effects by recruiting and activating neutrophils, thereby enhancing their antimicrobial activity, and by stimulating epithelial cells to secrete antimicrobial peptides, which collectively limit parasite replication and dissemination. Additionally, the study highlighted the involvement of proinflammatory cytokines produced during infections with Toxoplasma gondii and Leishmania spp., further underscoring the integrative role of IL-17 in orchestrating effective immune responses.

In parasitic infections, IL-17 not only induces protective immune responses but also regulates inflammation to balance parasite eradication with tissue damage, as observed in helminth infections like Schistosoma mansoni (Chen *et al*., 2019). Excessive IL-17 production, however, has been linked to chronic diseases, emphasizing the need for tight regulation within immune networks (Beringer *et al*., 2016). Experimental models have revealed that IL-17A and IL-17F induce neutrophil recruitment along with antimicrobial peptide production in T. gondii infection, which helps control parasite spread (Mills, 2023). Our pooled analysis supports these observations: higher levels of IL-17 are associated with better infection outcomes, underlining that rapid, protective immune responses are orchestrated by IL-17 (Mills, 2023).

Studies in humans with leishmaniasis reported increased IL-17A levels in patients with localized or self-limited forms of the disease, indicating protective immunomodulatory effects. For example, Hadi *et al*. (2024) observed that higher IL-17A levels were associated with milder cutaneous lesions, likely due to better control over parasite replication at the infection site. Our meta-analysis agrees with this, attributing more than half the variance in protective outcomes to IL-17 ($R^2 = 0.58$), supporting its role as both a marker and mediator of resistance (Drinkall *et al*., 2017).

In protozoan infections, the protective role of IL-17 is well documented. For instance, earlier research demonstrated that higher IL-17A levels were linked to controlled parasite loads and attenuated symptoms (Paerewijck *et al*., 2017). Chemokines induced by IL-17A in epithelial and stromal cells rapidly recruit neutrophils to infection sites (Mills, 2023). This mechanism likely explains the significant pooled mean difference found in our study (1.45, $p = 0.002$), as early neutrophil responses are essential to limiting parasite dissemination and pathology (Korn *et al*., 2009).

IL-17 also contributes to helminth infections, where Th2 responses usually dominate. Studies on S. mansoni demonstrated that IL-17A modulates granulomatous inflammation, balancing parasite control with tissue preservation (Paerewijck *et al*., 2017; Chen *et al*., 2019). A similar dual role has been observed in chronic parasitic infections, where IL-17's acute pro-inflammatory activity contributes to immune regulation and tissue homeostasis (Beringer *et al*., 2016). Evidence further strengthens this view by showing IL-17's substantial contribution to protection across different Schistosoma species (Wang *et al*., 2025).

The moderate heterogeneity observed ($I^2 = 68\%$) likely reflects biological and methodological differences among studies. Animal models reported stronger protective associations, likely due to controlled experimental conditions and genetic uniformity (Paerewijck *et al*., 2017). Human studies,

however, introduced variability due to host genetics, co-infections, and environmental exposures, which may attenuate effect sizes (Drinkall *et al*., 2017). Despite this, the consistent direction of effect suggests the association is reliable. This rather moderate heterogeneity in the pooled results ($I^2 = 68\%$) may be due to a combination of biological and methodological variation among the studies included. Variability of estimates might be explained by differences in study population, host (humans versus animal models), parasite (genus, life cycle stage), or the type of IL-17 subtypes measured (for example, IL-17A v/s IL-17F). Furthermore, differences in outcome definitions, experimental conditions and immunologic measurement modalities between studies may contribute to heterogeneity. No formal subgroup or sensitivity analyses were conducted because of few studies in individual categories, which might result in low statistical power and credibility. As a result the pooled estimates should be treated with caution. Prospective research and meta-analyses based on large dataset should take stratified analyses into account to deepen insight into the origins of heterogeneity and further interpret IL-17–dependent protective mechanisms in parasitic diseases.

Our analysis also indicated that while the majority of studies focused on IL-17A, some investigated related subtypes or regulatory cytokines. For instance, IL-17F, IL-10, IL-23, and IL-35 were studied in addition to IL-17A (Mills, 2023; Hadi *et al*., 2024). The consistent protective pattern in these studies suggests that IL-17A serves as the major effector, though its role is shaped by interactions with other cytokines (Beringer *et al*., 2016). Importantly, IL-17 can be both protective and pathological depending on its regulation (Chen *et al*., 2019).

The protective role of IL-17 is biologically rooted in its ability to rapidly recruit neutrophils, stimulate antimicrobial peptide production, and act synergistically with cytokines such as TNF-α and IL-6 to mount effective immune responses (Mills, 2023). In protozoan infections, IL-17 controls early parasite proliferation; in helminth infections, it regulates granuloma size and limits tissue damage while maintaining parasite control (Chen *et al*., 2019). These mechanisms correspond with our quantitative findings, where IL-17 explained 58% of outcome variance and showed a significant pooled effect size.

While promising, these results come with limitations. Most included studies were cross-sectional, limiting causal inference between IL-17 levels and clinical outcomes. Small sample sizes also increased the risk of random error. Additional sources of heterogeneity included infection stage, parasite strain, and cytokine measurement techniques (ELISA vs. PCR). Finally, although Egger's test did not show publication bias ($p = 0.078$), unpublished negative results cannot be ruled out.

Another gap is the lack of investigation into IL-17 family members besides IL-17A. Cytokines such as IL-17C and IL-17E may play roles in mucosal immunity but were rarely studied. Moreover, IL-17's impact likely differs between acute and chronic infections: transient IL-17 production appears protective, whereas chronic overproduction contributes to tissue damage and fibrosis, as observed in autoimmune diseases (Beringer *et al*., 2016).

Our findings suggest that in a clinical setting, IL-17 may be used as a biomarker for disease prognosis or monitoring therapeutics. High levels of IL-17 may indicate successful early immune activation and could be used to guide risk stratification and treatment decisions. Vaccines or immunotherapies aimed at enhancing responses to IL-17—carefully calibrated to avoid overactivation—may improve outcomes, primarily in endemic regions where parasite exposure is frequent.

Future research should give priority to longitudinal and Interventional studies to determine causality and whether modulation of IL-17 responses improves clinical outcomes. Standardization of methods for measuring IL-17, including assay type and timing, would facilitate comparison between different studies. Further, exploration of the relationship between IL-17 and other immune pathways could lead to the discovery of additional therapeutic targets or novel strategies for treatment combinations.

Future studies should focus on confirming IL-17 as a biomarker of human protection in extended, and longitudinal cohorts. Molecular research is needed to determine IL-17 interactions with other cytokines and immune cells, as well as parasite-specific differences in its role across protozoan and helminthic infections. Clinical studies using IL-17 modulators could determine its therapeutic potential in enhancing

antiparasitic immunity. In addition, integrating high-throughput approaches such as transcriptomics and systems biology may clarify novel IL-17–related pathways. Finally, these efforts will provide a closr understanding of IL-17 and support its application as a potential immunotherapeutic target.

Although human and animal studies were pooled in the present analysis, both study types consistently evaluated the immunoprotective role of IL-17 in parasitic infections using comparable immunological outcomes. Experimental animal models are widely accepted for elucidating IL-17–mediated host defense mechanisms and have demonstrated strong translational relevance to human parasitic immunity. Nevertheless, we acknowledge that species-related differences may contribute to heterogeneity, and future meta-analyses should consider stratified analyses by study type.

Conclusion

In summary, it is reported that IL-17, mostly IL-17A, is very much associated with better outcomes against various parasitic infections. The effect is consistent across animal categories and parasite groupings, supported by empirical rationality and previous experimental results. These findings underscore the important role of IL-17 in host protection and its potential application as both a marker for disease diagnosis and a therapeutic target for future immunomodulatory interventions in parasitic diseases.

Conflict of Interest

The authors declare that they have no competing interests.

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