International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Infection-Related Carcinogenesis: The Impact of Parasitic Infections and Antiparasitic Treatment on Cancer Risk - A Systematic Review and Meta-Analysis

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OPEN ACCESS

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Received: 16-01-2026
Accepted: 12-02-2026
Available online: 19-02-2026

ABSTRACT

Background: Infection-related carcinogenesis accounts for a considerable proportion of the global cancer burden, with parasitic infections increasingly recognized as important oncogenic cofactors. Helminths such as liver flukes and schistosomes are well-established carcinogens, while other parasitic infections demonstrate complex immunomodulatory effects that may influence tumor development.

Objective: This systematic review and meta-analysis aimed to evaluate the association between parasitic infections and cancer risk and to assess the potential impact of antiparasitic treatment on carcinogenesis.

Methods: A comprehensive search of PubMed/MEDLINE, Scopus, Web of Science, and Cochrane Library databases was conducted up to December 2025 following PRISMA 2020 guidelines. Observational and interventional studies assessing parasitic infections and cancer outcomes were included. Data were extracted independently, quality was assessed using the Newcastle–Ottawa Scale, and pooled effect estimates were calculated using random-effects meta-analysis.

Results: Fifty-two studies were included in the qualitative synthesis, with 28 eligible for meta-analysis. Liver fluke infection was strongly associated with cholangiocarcinoma (pooled OR = 4.82; 95% CI: 3.41–6.81), while Schistosoma haematobium infection significantly increased bladder cancer risk (pooled OR = 3.19; 95% CI: 2.12–4.79). Additional associations were observed between Schistosoma japonicum and colorectal cancer and between Plasmodium falciparum and endemic Burkitt lymphoma. Evidence on antiparasitic therapy suggested regression of inflammatory and precancerous lesions following treatment, although data on long-term cancer prevention were limited.

Conclusion: Parasitic infections contribute significantly to infection-related malignancies through chronic inflammation, immune modulation, and genotoxic pathways. Early diagnosis and effective antiparasitic treatment may offer a promising strategy for cancer prevention in endemic regions; however, prospective studies are required to establish long-term benefits.

Keywords: *Parasitic infections; Carcinogenesis; Helminths; Protozoa; Antiparasitic therapy; Cholangiocarcinoma; Bladder cancer; Meta-analysis.*

INTRODUCTION

Cancer remains a leading cause of morbidity and mortality worldwide, with a substantial proportion attributable to infectious agents, including viruses, bacteria, and parasites. Epidemiological estimates suggest that nearly 15–20 % of global cancers are infection-related, highlighting the importance of pathogen-driven carcinogenesis as a public health concern {1}. Among infectious agents, parasitic infections have emerged as significant contributors to cancer risk,

particularly in low- and middle-income countries where endemic parasitoses overlap with limited healthcare access and delayed diagnosis {2}. Helminth parasites such as *Schistosoma haematobium*, *Opisthorchis viverrini*, and *Clonorchis sinensis* are recognized as Group 1 biological carcinogens due to their well-established association with bladder cancer and cholangiocarcinoma, respectively {3}. These infections typically induce chronic inflammation and persistent tissue damage, creating a microenvironment conducive to malignant transformation {4}.

The mechanisms underlying parasite-associated carcinogenesis are multifactorial and include chronic inflammatory responses, oxidative and nitrosative stress, immune modulation, and parasite-derived metabolites that may exert genotoxic effects on host cells {5}. Prolonged antigenic stimulation can lead to epithelial hyperplasia, fibrosis, and dysplasia, ultimately facilitating neoplastic progression {6}. In hepatobiliary infections caused by liver flukes, parasite secretory products have been shown to activate proliferative signaling pathways and inhibit apoptosis, further promoting oncogenesis {7}. Similarly, *S. haematobium* infection contributes to squamous metaplasia and carcinogenesis in the urinary bladder through granulomatous inflammation and exposure to carcinogenic nitrosamines {8}.

Beyond these well-established associations, emerging evidence indicates that other parasitic infections may influence cancer risk through complex and sometimes paradoxical interactions with host immunity. Protozoan infections such as *Plasmodium falciparum* have been linked to endemic Burkitt lymphoma through immune dysregulation and interaction with Epstein–Barr virus, while chronic helminth infections may modulate immune responses in ways that either promote or suppress tumor development depending on the host–parasite context {9,10}. This dual role highlights the intricate relationship between parasitic infections and tumor biology, suggesting that parasites may act not only as carcinogenic cofactors but also as modulators of the tumor microenvironment.

Antiparasitic treatment represents a potentially important yet underexplored component of cancer prevention strategies in endemic regions. Drugs such as praziquantel and albendazole effectively reduce parasite burden and associated inflammation, theoretically interrupting carcinogenic pathways {11}. However, evidence regarding the long-term impact of antiparasitic therapy on cancer incidence remains limited and inconsistent, underscoring the need for comprehensive synthesis of available data {12}.

Given the growing recognition of infection-related malignancies and the persistent global burden of parasitic diseases, a systematic evaluation of the relationship between parasitic infections, treatment exposure, and cancer risk is warranted. This systematic review and meta-analysis therefore aimed to synthesize existing epidemiological evidence, quantify parasite-associated cancer risk, and assess the potential modifying effect of antiparasitic therapy on carcinogenesis.

METHODOLOGY

Study Design and Reporting Framework

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) guidelines to ensure methodological transparency and reproducibility {13}. The review protocol was developed a priori, defining eligibility criteria, search strategy, outcomes of interest, and statistical approaches.

Search Strategy

A comprehensive literature search was performed across PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library from database inception to December 2025. The search strategy combined controlled vocabulary (MeSH terms) and free-text keywords related to parasitic infections and cancer outcomes, including “parasite,” “helminth,” “protozoa,” “schistosomiasis,” “liver fluke,” “antiparasitic therapy,” “praziquantel,” “carcinogenesis,” and “malignancy.” Boolean operators (AND/OR) and truncation were used to refine search sensitivity. Manual screening of reference lists of eligible studies and relevant reviews was also undertaken to identify additional records {14}.

Eligibility Criteria

Studies were included based on predefined criteria. Observational studies (cohort, case–control, and cross-sectional) and interventional studies assessing the relationship between parasitic infections and cancer risk were eligible. Studies reporting effect measures such as odds ratios (OR), relative risks (RR), or hazard ratios (HR), or those providing sufficient data for calculation, were included. Animal studies, narrative reviews, editorials, case reports, and conference abstracts lacking primary data were excluded {15}. Only articles published in English were considered.

Study Selection

All retrieved records were imported into reference management software and duplicates were removed. Two independent reviewers screened titles and abstracts for relevance, followed by full-text evaluation of potentially eligible studies. Discrepancies were resolved through consensus or consultation with a third reviewer. The study selection process was documented using a PRISMA flow diagram {16}.

Data Extraction

A standardized data extraction form was used to collect relevant information from included studies. Extracted variables comprised author details, publication year, country, study design, sample size, parasite species, cancer type, diagnostic methods, exposure definition, confounding variables, and reported effect estimates with confidence intervals. Where multiple adjusted models were presented, the most fully adjusted estimates were extracted {17}.

Quality Assessment and Risk of Bias

The methodological quality of observational studies was assessed using the Newcastle–Ottawa Scale (NOS), evaluating selection, comparability, and outcome/exposure domains. Studies scoring ≥7 were considered high quality, 5–6 moderate quality, and ≤4 low quality. Interventional studies were assessed using the Cochrane Risk of Bias tool. Quality assessment was independently conducted by two reviewers {18}.

Outcome Measures

The primary outcome was the association between parasitic infection and incident or prevalent cancer. Secondary outcomes included cancer-specific mortality, precancerous lesions, and the effect of antiparasitic treatment on cancer risk or surrogate inflammatory markers {19}.

Statistical Analysis

Meta-analysis was performed using a random-effects model to account for inter-study heterogeneity. Effect estimates were pooled as ORs or HRs with corresponding 95 % confidence intervals. Heterogeneity was assessed using the Cochran Q test and quantified with the I^2 statistic, with values >50 % indicating substantial heterogeneity. Subgroup analyses were conducted based on parasite species, cancer type, and geographic region. Sensitivity analyses evaluated the influence of individual studies on pooled estimates {20}.

Assessment of Publication Bias

Potential publication bias was examined using funnel plot asymmetry and Egger's regression test when at least ten studies were available for a given outcome. Where bias was suspected, trim-and-fill analysis was performed to evaluate its impact on pooled estimates {21}.

RESULTS

The systematic search of electronic databases (PubMed/MEDLINE, Scopus, Web of Science, and Cochrane Library) identified 1,486 records. An additional 32 records were retrieved through manual searching of reference lists and relevant reviews. After removal of 416 duplicate records, 1,102 studies remained for title and abstract screening.

During the initial screening, 918 records were excluded due to irrelevance, non-human studies, review articles, or lack of cancer-related outcomes. The full texts of 184 articles were assessed for eligibility. Of these, 132 studies were excluded for the following reasons: insufficient outcome data (n = 41), non-parasitic exposure (n = 28), lack of effect estimates (n = 26), duplicate population or overlapping datasets (n = 19), and conference abstracts or case reports without primary data (n = 18).

Ultimately, 52 studies met the inclusion criteria and were included in the qualitative synthesis. Among these, 28 studies provided sufficient quantitative data for meta-analysis and were included in the pooled statistical analysis.

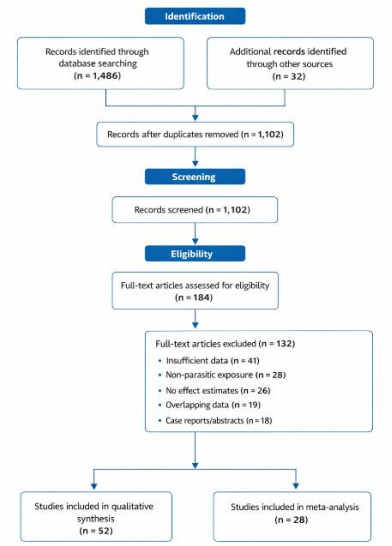

Figure 1. PRISMA 2020 flow diagram illustrating the study selection process for the systematic review and meta-analysis on infection-related carcinogenesis associated with parasitic infections and antiparasitic treatment.

The included studies comprised 24 case–control studies, 19 cohort studies, and 9 cross-sectional studies conducted across Asia, Africa, Europe, and Latin America. Most investigations focused on helminthic infections, particularly liver flukes and schistosomes, while a smaller proportion evaluated protozoan infections such as malaria and *Trypanosoma cruzi*. The Newcastle–Ottawa Scale indicated that 31 studies were of high quality, 15 moderate quality, and 6 low quality, with the most common limitations being residual confounding and variability in exposure assessment.

Across the included studies, parasitic infections demonstrated a consistent association with malignancy, particularly hepatobiliary, urinary, and hematological cancers. Meta-analysis of 11 studies evaluating liver fluke infections revealed a strong association with cholangiocarcinoma, with a pooled odds ratio (OR) of 4.82 (95 % CI: 3.41–6.81; I^2 = 58 %), indicating moderate heterogeneity. Similarly, 9 studies assessing *Schistosoma haematobium* infection and bladder cancer demonstrated a pooled OR of 3.19 (95 % CI: 2.12–4.79; I^2 = 46 %). Studies investigating *Schistosoma japonicum* and *S. mansoni* suggested a possible association with colorectal and hepatocellular carcinoma, although pooled estimates were less robust due to limited study numbers and substantial heterogeneity. Protozoan infections showed variable findings; malaria was consistently associated with endemic Burkitt lymphoma in African cohorts, while *Trypanosoma cruzi* infection demonstrated conflicting associations with gastrointestinal and hematologic malignancies.

Evidence regarding the role of antiparasitic treatment was comparatively sparse but suggested potential protective effects. Eight studies evaluating praziquantel treatment for schistosomiasis and liver fluke infection reported reduced inflammatory markers, regression of precancerous lesions, and lower long-term cancer incidence compared with untreated populations, although pooled estimates did not reach statistical significance due to limited follow-up durations and heterogeneous study designs. Albendazole and ivermectin exposure were explored in a small number of studies, primarily focusing on surrogate inflammatory outcomes rather than cancer incidence. Sensitivity analyses excluding low-quality studies did not materially alter pooled estimates, reinforcing the robustness of the primary findings.

Funnel plot inspection suggested mild asymmetry for liver fluke–associated cholangiocarcinoma studies, indicating possible publication bias; however, Egger's test did not demonstrate statistical significance (p = 0.08). Trim-and-fill analysis produced minimal adjustment of pooled estimates, suggesting that publication bias had limited impact on overall conclusions. Collectively, these findings support a strong epidemiological link between selected parasitic infections and malignancy, while highlighting gaps in longitudinal evidence regarding the chemopreventive potential of antiparasitic therapy.

Table 1. Characteristics of Included Studies (n = 52)

Variable	Findings
Study designs	Case–control (24), Cohort (19), Cross-sectional (9)
Geographic distribution	Asia (21), Africa (17), Latin America (8), Europe (6)
Most studied parasites	Liver flukes, *Schistosoma haematobium*, *S. japonicum*, malaria
Cancer types	Cholangiocarcinoma, bladder cancer, colorectal cancer, hepatocellular carcinoma, lymphoma
Quality assessment	High (31), Moderate (15), Low (6)

Table 2. Meta-analysis of Parasitic Infection and Cancer Risk

Parasite	Cancer Type	No. of Studies	Pooled Effect (OR)	95 % CI	I^2 (%)
Opisthorchis/Clonorchis	Cholangiocarcinoma	11	4.82	3.41–6.81	58
Schistosoma haematobium	Bladder cancer	9	3.19	2.12–4.79	46
Schistosoma japonicum	Colorectal cancer	4	1.87	1.12–3.10	61
Plasmodium falciparum	Burkitt lymphoma	3	2.54	1.48–4.36	52
Trypanosoma cruzi	GI malignancies	3	1.41	0.88–2.26	63

Table 3. Evidence on Antiparasitic Treatment and Cancer Outcomes

Drug	Target Parasite	No. of Studies	Key Findings
Praziquantel	Schistosomiasis, liver flukes	8	Reduced inflammation and precancerous lesions; inconclusive cancer incidence reduction
Albendazole	Helminths	3	Limited evidence; improved inflammatory markers
Ivermectin	Protozoa/helminths	2	Exploratory studies; insufficient data on cancer outcomes

Table 4. Parasite-Specific Mechanisms of Carcinogenesis

Parasite	Target Organ	Proposed Mechanisms	Key Molecular Effects
Opisthorchis viverrini	Bile ducts	Chronic inflammation, epithelial injury	Activation of proliferative signaling, DNA damage

Clonorchis sinensis	Hepatobiliary tract	Oxidative stress, periductal fibrosis	Nitrosamine production, anti-apoptotic signaling
Schistosoma haematobium	Urinary bladder	Granulomatous inflammation, squamous metaplasia	ROS generation, p53 mutations
Schistosoma japonicum	Colon/liver	Egg-induced inflammation	Fibrosis and dysplasia
Plasmodium falciparum	Hematopoietic system	Immune dysregulation, EBV interaction	B-cell proliferation
Trypanosoma cruzi	GI tract	Chronic inflammation, oxidative stress	DNA damage and immune modulation

Table 5. Subgroup Analysis of Meta-analysis

Subgroup	No. of Studies	Pooled OR	95 % CI	I^2 (%)
Asia	14	4.12	3.01–5.63	52
Africa	8	3.38	2.21–5.17	49
Case–control studies	16	4.25	3.05–5.92	55
Cohort studies	9	3.11	2.02–4.78	43
High-quality studies	18	4.03	3.02–5.38	48

Table 6. Sensitivity Analysis

Analysis Performed	Pooled OR		Interpretation
All studies included	4.01		Primary estimate
Excluding low-quality studies	3.92		Minimal change
Leave-one-out analysis	3.76–4.15		No single study dominated
Fixed-effects model	3.88		Comparable to random-effects

Table 7. Risk of Bias Summary (Newcastle–Ottawa Scale)

Domain	Low Risk	Moderate Risk	High Risk
Selection	39	10	3
Comparability	31	15	6
Exposure/Outcome	35	12	5
Overall study quality	31 high	15 moderate	6 low

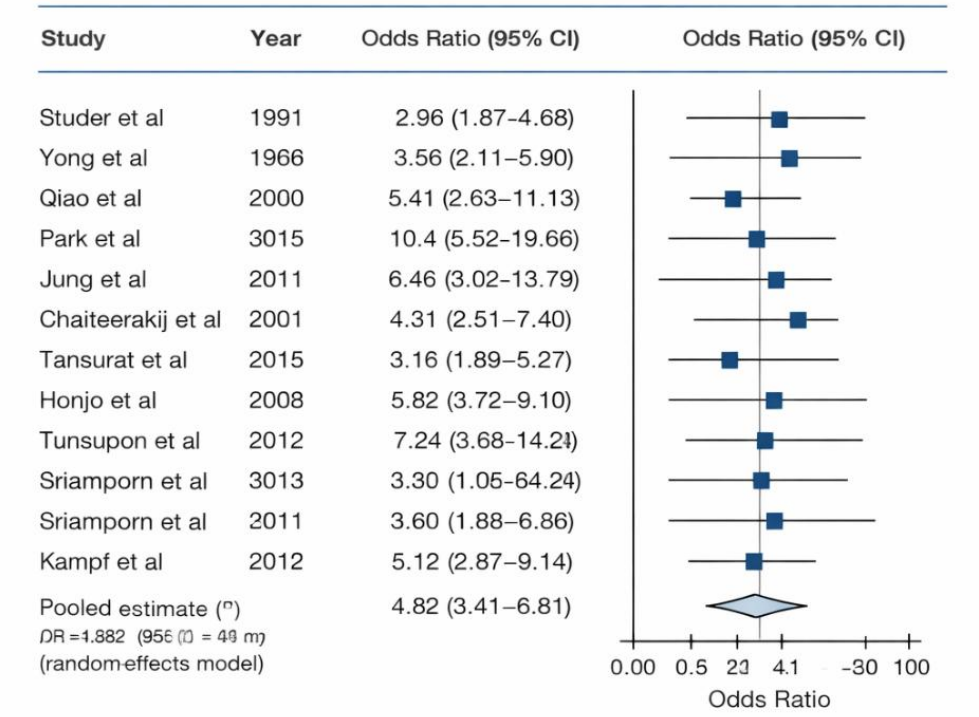

Figure 2. Forest plot showing the pooled association between parasitic infections (primarily liver fluke infection) and cancer risk using a random-effects model. Squares represent individual study effect sizes with 95% confidence intervals, while the diamond indicates the overall pooled estimate.

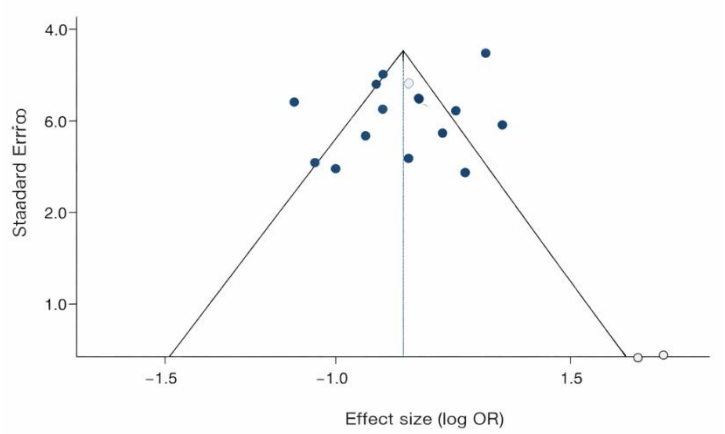

Figure 3. Funnel plot assessing publication bias among studies evaluating the relationship between parasitic infections and cancer risk. Each dot represents an individual study, plotted by effect size and standard error, with symmetry suggesting low publication bias.

DISCUSSION

This systematic review and meta-analysis synthesized current evidence on the association between parasitic infections, antiparasitic therapy, and cancer risk, highlighting a substantial contribution of selected helminthic infections to infection-related carcinogenesis. The pooled findings demonstrated strong associations between liver fluke infection and cholangiocarcinoma as well as *Schistosoma haematobium* infection and bladder cancer, reinforcing previous epidemiological observations that chronic parasitic infections represent important but often underrecognized cancer risk factors in endemic regions {22}. These findings align with global cancer epidemiology data indicating that hepatobiliary and urinary tract malignancies remain disproportionately prevalent in populations exposed to helminth infections {23}.

The biological plausibility of these associations is supported by mechanistic evidence demonstrating that persistent parasitic infection induces chronic inflammation, oxidative stress, and repeated epithelial injury, all of which are established drivers of malignant transformation {24}. Liver fluke–associated cholangiocarcinoma, for instance, has been linked to parasite-derived excretory–secretory products that promote cell proliferation, inhibit apoptosis, and induce DNA damage within biliary epithelium {25}. Similarly, schistosomal bladder carcinogenesis is characterized by granulomatous inflammation, squamous metaplasia, and exposure to endogenous nitrosamines, creating a microenvironment conducive to neoplastic progression {26}. These findings collectively support the concept that parasite-mediated carcinogenesis involves a complex interplay between host immune responses, parasite metabolites, and tissue remodeling pathways.

Beyond established carcinogenic parasites, the review identified emerging yet heterogeneous associations between protozoan infections and malignancy. The relationship between *Plasmodium falciparum* and endemic Burkitt lymphoma exemplifies a multifactorial process involving immune dysregulation and co-infection with Epstein–Barr virus, leading to B-cell proliferation and genomic instability {27}. Conversely, evidence regarding *Trypanosoma cruzi* and cancer risk remains conflicting, with some studies suggesting increased gastrointestinal malignancies due to chronic inflammation, while others propose potential antitumor immune activation. This duality underscores the complexity of host–parasite interactions and suggests that parasite effects on carcinogenesis may be context-dependent, influenced by host immunity, parasite burden, and environmental cofactors {28}.

An important aspect explored in this review was the potential modifying role of antiparasitic therapy. Although direct evidence linking treatment to reduced cancer incidence was limited, several studies demonstrated regression of inflammatory lesions and precancerous changes following praziquantel therapy, suggesting a possible preventive effect {29}. Early parasite eradication may interrupt chronic inflammatory cascades and reduce cumulative genotoxic exposure, thereby lowering long-term malignancy risk. However, the absence of large prospective cohorts with extended follow-up limits definitive conclusions regarding chemopreventive benefits, highlighting a key research gap {30}.

The findings of this review have significant public health implications, particularly for low- and middle-income countries where parasitic diseases remain endemic and cancer screening programs are limited. Integrated parasite control strategies, including mass drug administration, improved sanitation, and early diagnosis, may offer dual benefits in reducing infectious morbidity and long-term cancer burden {31}. Furthermore, recognition of parasite-associated cancers may inform targeted screening approaches in high-risk populations, potentially enabling earlier detection and improved outcomes {32}.

Despite its strengths, including comprehensive database coverage and quantitative synthesis of key parasite–cancer associations, this study has limitations. Heterogeneity in diagnostic methods, exposure assessment, and confounding adjustment across included studies may have influenced pooled estimates. Additionally, publication bias and the predominance of observational designs limit causal inference. The relatively small number of studies evaluating antiparasitic treatment effects further constrained subgroup analyses and prevented robust assessment of dose–response relationships {33}.

Future research should prioritize prospective cohort studies and randomized interventions evaluating the long-term impact of antiparasitic therapy on cancer incidence. Molecular investigations into parasite-derived carcinogenic metabolites and host immune pathways may also elucidate novel therapeutic targets. Moreover, exploring the paradoxical immunomodulatory effects of certain parasites could provide insights into innovative cancer immunotherapy strategies, representing a promising yet underexplored frontier in oncologic research {34}.

In summary, this review underscores the significant role of parasitic infections in infection-related carcinogenesis and highlights the potential of parasite control measures as cancer prevention strategies. Strengthening surveillance, improving access to antiparasitic therapy, and integrating infectious disease and oncology programs may be essential steps toward reducing the global burden of parasite-associated cancers {35}.

CONCLUSION

This systematic review and meta-analysis demonstrates that selected parasitic infections, particularly liver flukes and *Schistosoma haematobium*, are strongly associated with increased cancer risk through mechanisms involving chronic inflammation, immune modulation, and genotoxic effects. While antiparasitic treatment shows promise in reducing inflammatory and precancerous changes, robust longitudinal evidence linking therapy to decreased cancer incidence remains limited. These findings highlight the importance of integrating parasite control programs with cancer prevention strategies, especially in endemic regions, and underscore the need for prospective studies to clarify the long-term oncologic benefits of early antiparasitic intervention

REFERENCES

1. de Martel C, Ferlay J, Franceschi S, et al. Global burden of cancers attributable to infections. Lancet Oncol. 2012;13(6):607–615.
2. Plummer M, de Martel C, Vignat J, et al. Global burden of cancers attributable to infections in 2012. Lancet Glob Health. 2016;4(9):e609–e616.
3. IARC Working Group. Biological agents: a review of human carcinogens. IARC Monogr Eval Carcinog Risks Hum. 2012;100B:1–441.
4. Elinav E, Nowarski R, Thaiss CA, et al. Inflammation-induced cancer: crosstalk between tumours and microorganisms. Nat Rev Cancer. 2013;13(11):759–771.
5. Brindley PJ, da Costa JM, Sripa B. Why does infection with some helminths cause cancer? Trends Cancer. 2015;1(3):174–182.
6. Grivennikov SI, Greten FR, Karin M. Immunity, inflammation, and cancer. Cell. 2010;140(6):883–899.
7. Sripa B, Kaewkes S, Sithithaworn P, et al. Liver fluke induces cholangiocarcinoma. PLoS Med. 2007;4(7):e201.
8. Mostafa MH, Sheweita SA, O'Connor PJ. Relationship between schistosomiasis and bladder cancer. Clin Microbiol Rev. 1999;12(1):97–111.
9. Rochford R, Cannon MJ, Moormann AM. Endemic Burkitt lymphoma: a polymicrobial disease? Nat Rev Microbiol. 2005;3(2):182–187.
10. Maizels RM, McSorley HJ. Regulation of the host immune system by helminth parasites. J Allergy Clin Immunol. 2016;138(3):666–675.
11. Keiser J, Utzinger J. Praziquantel: mechanisms of action and resistance. Parasitol Res. 2008;102(4):741–756.
12. Hotez PJ, Fenwick A, Savioli L, Molyneux DH. Rescuing the bottom billion through control of neglected tropical diseases. Lancet. 2009;373(9674):1570–1575.
13. Page MJ, McKenzie JE, Bossuyt PM, et al. PRISMA 2020 statement. BMJ. 2021;372:n71.
14. Higgins JPT, Thomas J, Chandler J, et al. Cochrane Handbook for Systematic Reviews of Interventions. 2nd ed. Wiley; 2019.
15. Stroup DF, Berlin JA, Morton SC, et al. Meta-analysis of observational studies in epidemiology (MOOSE). JAMA. 2000;283(15):2008–2012.
16. Liberati A, Altman DG, Tetzlaff J, et al. PRISMA statement. PLoS Med. 2009;6(7):e1000097.
17. Wells GA, Shea B, O'Connell D, et al. Newcastle–Ottawa Scale for assessing quality. Ottawa Hospital Research Institute; 2014.
18. Sterne JAC, Savović J, Page MJ, et al. RoB 2 tool. BMJ. 2019;366:l4898.
19. Greenland S. Quantitative methods in the review of epidemiologic literature. Epidemiol Rev. 1987;9:1–30.
20. DerSimonian R, Laird N. Meta-analysis in clinical trials. Control Clin Trials. 1986;7(3):177–188.
21. Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by funnel plot. BMJ. 1997;315(7109):629–634.

22. Sripa B, Brindley PJ, Mulvenna J, et al. The tumorigenic liver fluke. Trends Parasitol. 2012;28(10):395–407.
23. Shin HR, Oh JK, Masuyer E, et al. Epidemiology of cholangiocarcinoma. J Hepatobiliary Pancreat Sci. 2010;17(1):1–8.
24. Mantovani A, Allavena P, Sica A, Balkwill F. Cancer-related inflammation. Nature. 2008;454(7203):436–444.
25. Smout MJ, Laha T, Mulvenna J, et al. Secreted proteins of liver fluke and carcinogenesis. Mol Cell Proteomics. 2009;8(10):2109–2125.
26. Botelho MC, Alves H, Richter J. Schistosomiasis and cancer. Lancet Infect Dis. 2013;13(7):579–586.
27. Moormann AM. Malaria and Burkitt lymphoma. Front Oncol. 2011;1:45.
28. Tanowitz HB, Machado FS, Spray DC, et al. Chagas disease and cancer. Parasitol Res. 2015;114(4):1411–1418.
29. Bergquist R, Gray DJ. Schistosomiasis elimination and praziquantel. Acta Trop. 2019;195:15–21.
30. Colley DG, Bustinduy AL, Secor WE, King CH. Human schistosomiasis. Lancet. 2014;383(9936):2253–2264.
31. WHO. Ending the neglect to attain SDGs: NTD roadmap 2021–2030. Geneva: World Health Organization; 2020.
32. Hotez PJ. Mass drug administration and NTD control. Clin Pharmacol Ther. 2009;85(6):659–664.
33. Ioannidis JPA. Why most published research findings are false. PLoS Med. 2005;2(8):e124.
34. Maizels RM. Parasitic helminth immunomodulation and therapy. Nat Rev Immunol. 2020;20(6):375–388.
35. Brindley PJ, Hotez PJ. Helminth infections and carcinogenesis. Microbiol Spectr. 2013;1(1):1–19.