Systematic Review/Meta-analysis

INTERNATIONAL JOURNAL OF SURGERY

OPEN

Incidence and relative risk of colorectal cancer in autoimmune diseases: a global pooled-analysis with more than 91 million participants

Ji Cheng, MD*, Ming Cai, MD, Guobin Wang, MD

Background: Dysregulation of immune system could be a vital stimulant of colorectal cancer development. We aimed to provide the most comprehensive meta-analysis on this topic.

Methods: Observational cohort studies reporting incidence or risk ratio of colorectal cancer among patients with autoimmune disorders as exposures were eligible. Relative risk was the primary endpoint and adjusted risk ratio were effect sizes. Incident cases per 100 000 person-years at risk were used for incidence analysis as secondary endpoint.

Results: 523 studies were included, containing 91 265 886 participants. Overall pooled risk ratio was 1.244 ($p < 0.001$). The results were consistent irrespective of sub-localization (colon: 1.308, $p < 0.001$; rectum: 1.173, $p < 0.001$), sex (male: 1.229, $p < 0.001$; female: 1.209, $p < 0.001$) and country (Nordic: 1.301, $p < 0.001$; Western: 1.213, $p < 0.001$; East Asian: 1.213, $p < 0.001$). Specifically, primary sclerosing cholangitis, idiopathic inflammatory myopathies, inflammatory bowel disease, autoimmune hepatitis, ANCA-associated vasculitis, sarcoidosis, scleroderma, type 1 diabetes, psoriasis, membranous nephropathy, hidradenitis suppurativa and idiopathic thrombocytopenic purpura were associated with higherrisk of colorectal cancer. Interestingly, several autoimmune diseases might help to lower colorectal cancer risk, especially rheumatoidarthritis.

Conclusions: Patients with autoimmune diseases were associated with higher risk of colorectal cancer under generally or specific settings. Unlike ourgastric and small bowel cancer pooled results, this risk-increasing impact on colorectal cancer was consistent across Nordic, Western and East Asian countries. Both digestive organ-specific or systemic autoimmune diseases could significantly increase risk of colorectal cancer. However, several autoimmune diseases might act as protective factors against colorectal cancer, especially rheumatoid arthritis, while not on gastric or small bowel carcinogenesis.

Keywords: autoimmune disease, colorectal cancer, incidence, meta-analysis, risk

Introduction

Colorectal cancer was one of the most prevalent types of malignancies worldwide[1]. According to GLOBOCAN 2022[2], colorectal cancer ranked third in incidence ranking, while second in mortality ranking, respectively, suggesting its heavy burden on global healthcare systems. Therefore, early surveillance, diagnosis as well as treatment, especially among high-risk group of population could effectively improve disease control and survival outcomes.

Dysregulation of the immune system might be a vital trigger for cancer development[3,4]. Several studies reported potential carcinogenic roles of immune disturbances in colorectal cancer progression[5–7]. Nevertheless, regarding roles of different autoimmune diseases in colorectal carcinogenesis, current evidence failed to reach a consensus, even though for the same type of autoimmune disease[8–11]. Furthermore, despite previous meta-analyses with certain conclusions on this topic, those meta-analyses were either antiquated or not methodologically and

HIGHLIGHTS

- This was the most updated and comprehensive meta-analysis based on global population.
- Patients with autoimmune disorders had a higher risk of colorectal cancer.
- The risk-increasing impact was quite consistent across regions.
- Both organ-specific or systemic diseases could significantly increase risk.
- Several diseases acted as protective factors, especially rheumatoid arthritis.

Department of Gastrointestinal Surgery, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China

**Corresponding author. Address: Department of Gastrointestinal Surgery, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, 1277 Jiefang Avenue, Wuhan 430022, China. Tel.: +86 15871761705. E-mail: jicheng1@hust.edu.cn (J. Cheng).*

International Journal of Surgery (2025) 111:7236–7242

Received 6 May 2025; Accepted 8 June 2025

Supplemental Digital Content is available for this article. Direct URL citations are provided in the HTML and PDF versions of this article on the journal's website, www.lww.com/international-journal-of-surgery.

Published online 20 June 2025

http://dx.doi.org/10.1097/JS9.0000000000002837

statistically credible and comprehensive, which indicated the necessity of performing an updated and comprehensive meta-analysis.

Hence, through a thorough literature retrieval and statistical analysis, we performed an updated and comprehensive meta-analysis based on global population, which helped investigate the association of different autoimmune diseases with colorectal cancer in terms of incidence and risk ratio.

Methods

Search strategy and selection criteria

The work has been reported in line with PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) and AMSTAR (Assessing the methodological quality of systematic reviews) Guidelines[12–14]. Literature retrieval, methodological assessment, data extraction, and statistical analysis of our meta-analysis conformed to requirements in Cochrane Handbook. Each procedure was performed by two members in our research group. The third researcher helped settle unsolved disputes if happened. Our meta-analysis was registered in PROSPERO. Moreover, our study complied with the TITAN Guidelines 2025[15]. No AI was used in research and manuscript development.

We thoroughly searched the databases PubMed, Web of Science, and Embase. Since each study needed comprehensive assessment on methodological quality and we only included observational studies, Cochrane Central and ASCO/ESMO meeting libraries were not searched. Moreover, reference lists of previous relevant meta-analyses were also scrutinized. The preliminary literature retrieval started from 2 January 2024 to 30 April 2024, while we made an update search on 10 January 2025, hence covering all records from inception to January 2025. Before full-text assessment, the titles and summaries of the retrieved records were first scrutinized. Details of our search strategy could be found in Supplementary Materials, http://links.lww.com/JS9/E478

Studies meeting all inclusion criteria were eligible to be included (based on the PICOS framework):

1. Participant: Real-world unselected participants from global sources were included, without restrictions on races, social status, etc. Among adult participants with pre-specified autoimmune diseases, we excluded those with co-morbidities such as concomitant autoimmune disorders besides exposure or precancerous lesions. Here, we considered specific medications for exposure as potential heterogeneous elements but still eligible for inclusion.

2. Intervention: Adult-onset pre-specified autoimmune diseases were exposure. The definitions and types of autoimmune diseases were confirmed according to the American Autoimmune Related Diseases Association (AARDA). If studies contained a small number of adolescents besides adult participants, we considered the proportion<20% to be excellent. Studies with larger proportions of adolescents were still included and however were further removed by sensitivity analysis.

3. Comparator: General population or external control without certain autoimmune diseases were non-exposure.

4. Outcome: Incidence as well as the risk ratio of colorectal cancer were main outcomes. We only included primary cancer cases diagnosed with pathological evidences. High-grade intraepithelial neoplasia and recurrent cases were not included. Studies reporting separate colorectal cancer cases were included. Those with combined gastrointestinal cancer cases or studies featuring melanoma, lymphoma, or anal malignancies were ineligible. In terms of incidence, participants with autoimmune diseases must have data on incidence, and only studies providing person-years at risk data (direct or calculative) could be included. Concerning the risk ratio, only adjusted RR/HR or stratified SIR based on general population was eligible, and crude effect sizes were not qualified.

5. Study design: We aimed to investigate the incidence and relative risk of colorectal cancer in autoimmune diseases; therefore, only cohort studies were eligible compared to other observational study types or interventional studies. We only included studies from inception to January 2025. Study language should be English.

Studies with one of following criteria were excluded:

1. Participants had a history of transplantation or surgical treatments.

2. Cohort studies without details for methodological assessments.

Data analysis

We used the Newcastle-Ottawa Scale for methodological assessments. The adaptive standards of the scale for our meta-analysis are demonstrated in Supplemental Digital Content, Table S1 http://links.lww.com/JS9/E478. Studies with at least six points were considered of high-quality, four or five points were of medium-quality and three points or lower were of low-quality in methodology.

We used electronic sheets to help collect raw data from eligible studies. Baseline characteristics, incidence, and risk ratio data were extracted from the main text or supplement of studies. The mean follow-up and person-years at risk had mutual conversion relations if needed. The risk ratio was the primary endpoint of our meta-analysis, while incidence served as the secondary endpoint.

We used STATA 14.2 to perform statistical calculation. Adjusted RR/HR or stratified SIR with a 95% confidence interval was utilized as effect sizes for pooled risk analysis, while incident cases as well as total person-years at risk were used for pooled incidence calculation. Classical and proportion meta-analysis was applied for pooling results of risk and incidence, respectively[16]. As for risk analysis, if the confidence interval of pooled estimates did not cover 1.0 and *P*-value was less than 0.05, the results were regarded as significantly different. However, for incidence analysis, since the pooled results of incidence were proportional, they served more like descriptive instead of comparative outcomes. Cochran's Q test and the I^2 statistic were used to evaluate internal heterogeneity. The *P*-value of the Q test less than 0.05 or $I^2 > 50\%$ indicated significant heterogeneity within pooled studies. The random-effects model was utilized for pooled calculation under circumstances of significant heterogeneity. Funnel plots and Egger's test were used to evaluate publication bias in each category,

where a generally symmetrical funnel plot and $P > 0.05$ of Egger's test usually suggested a low risk of publication bias. Sensitivity analysis was conducted by a leave-one-out method, or excluding studies with heterogeneous population groups, specific medications, heterogeneous follow-up definitions, specific effect-size definitions, or small sample sizes (participants with autoimmune diseases <500). Details of exclusion reasons are listed in Supplemental Digital Content, Table S3 http://links.lww.com/JS9/E478. Due to various baseline features of the included studies, we also performed multiple subgroup analyses according to sub-localization, sex, publication era, case count, type of effect size, population source, nationality, and disease type, which helped provide more specific interpretations of pooled outcomes.

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Baseline features and methodological quality

Via literature retrieval, 145 030 records were preliminarily obtained from main databases, in which 523 studies were ultimately included (Fig. 1). Reasons for exclusion during full-text assessment are listed in Supplemental Digital Content, Table S2 http://links.lww.com/JS9/E478. The overall sample size was 91 265 886. The majority of studies featured mean/median age older than 30 years old at diagnosis, with female predominance as well. Overall, data of more than 50 types of autoimmune diseases were available for pooling analysis in our study, derived from over 100 types listed in AARDA. Details of baseline characteristics are further listed in Supplemental Digital Content, Table S3 http://links.lww.com/JS9/E478.

A total of 327 studies were of high quality in methodological assessment, while 193 were of medium quality, and only 3 studies were of low quality according to the Newcastle-Ottawa Scale (Supplemental Digital Content, Table S4 http://links.lww.com/JS9/E478). The major reasons for studies categorized as

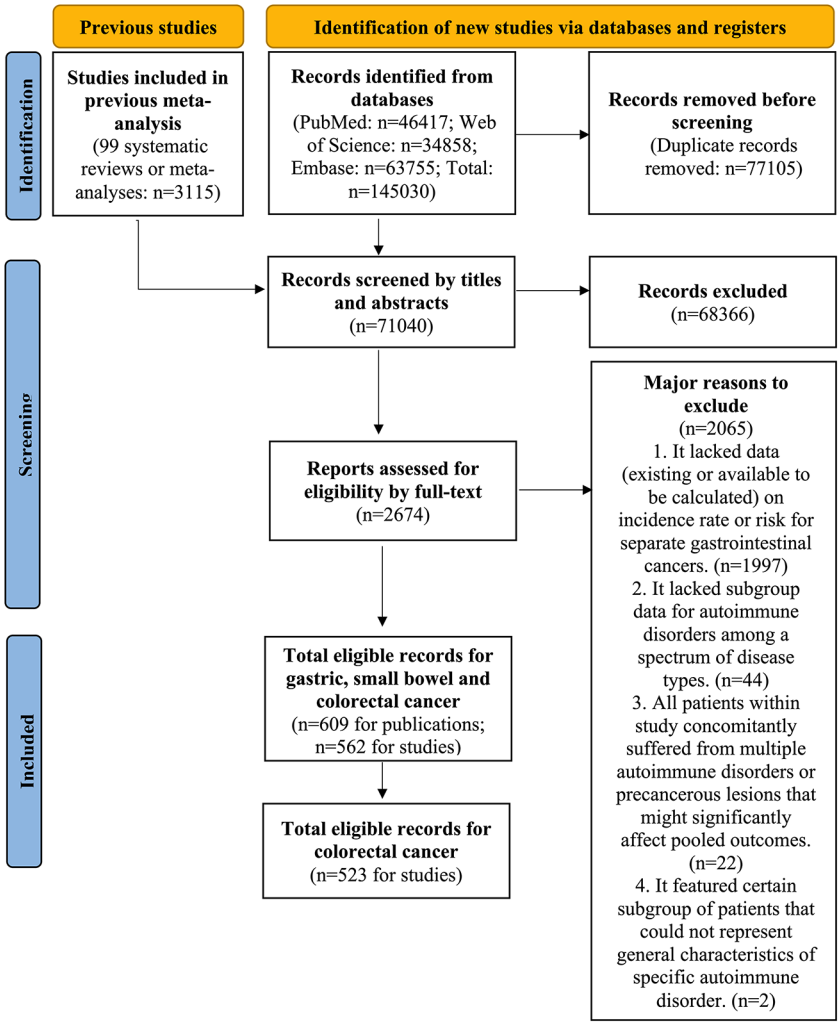

Figure 1. Flow chart of literature retrieval. Note: We concomitantly searched literatures for gastric cancer, small bowel cancer, and colorectal cancer, while for this specific study, colorectal cancer was eligible.

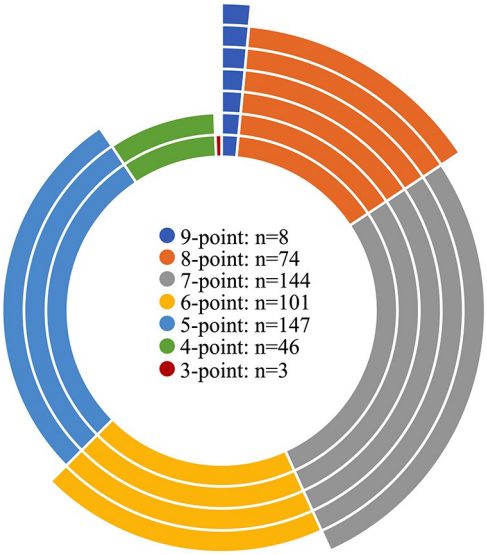

Figure 2. Quality assessment of eligible studies by the Newcastle-Ottawa Scale.

medium or low-quality studies were their single-arm design, or they only had data on the exposure group to be included. Therefore, the overall quality of the included studies was considerably reliable (Fig. 2).

Pooled incidence of colorectal cancer

In terms of colorectal cancer incidence, data from 590 cohorts were eligible for meta-analysis. The pooled incidence of overall cohorts was 96.622 (95% CI: 92.173–101.070) per 100 000 person-years, and sensitivity analyses did not find instability within the pooled results. Colon cancer (54.771, 95% CI: 50.509–59.033) nearly doubled rectal cancer (27.616, 95% CI: 24.938–30.294) in terms of incidence among participants with autoimmune diseases. Those autoimmune patients displayed relatively comparable incidence of colorectal cancer across the majority of included nations, except Finland (58.790, 95% CI: 45.565–72.014), Italy (53.150, 95% CI: 35.099–71.201), and Turkey (40.802, 95% CI: 6.004–75.600), which were considerably lower than overall estimates. Meanwhile, participants with autoimmune disorders from Australia (156.874, 95% CI: 114.321–199.427) and Israel (163.112, 95% CI: 112.650–213.573) displayed relatively high colorectal cancer incidence compared to overall estimates. Regarding specific autoimmune disease types (n>5), patients with primary sclerosing cholangitis (373.515, 95% CI: 265.178–481.852), autoimmune pancreatitis (251.424, 95% CI: 156.372–346.476), IgG4-related disease (211.443, 95% CI: 129.694–293.191), autoimmune gastritis (202.932, 95% CI: 149.811–256.053), inflammatory bowel disease (122.611, 95% CI: 114.015–131.206), or ulcerative colitis alone (149.043, 95% CI: 134.702–163.384) had relatively high incidence compared to overall results. On the other hand, patients with type 1 diabetes (20.485, 95% CI: 12.587–28.382), celiac disease (47.057, 95% CI: 25.756–68.357), Graves' disease (49.292, 95% CI: 28.892–69.691), lupus (56.499, 95% CI: 44.888–68.111), systemic lupus erythematosus alone (55.971, 95% CI: 44.008–67.933), multiple sclerosis (57.306, 95% CI: 44.565–70.048), and Behçet's disease (57.906, 95% CI: 43.232–72.580) had lower incidence of colorectal cancer than overall results (Fig. 3).

Pooled risk ratio of colorectal cancer

In terms of colorectal cancer risk, data from 436 cohorts were eligible. Overall, the pooled risk ratio was 1.244 (95% CI: 1.190–1.300, P < 0.001), suggesting significantly the increased risk of colorectal cancer among autoimmune disease patients than disease-free counterparts. Significant heterogeneity was validated by Cochran's Q test (P < 0.001) and the I^2 statistic (93.5%). Publication bias was suspected according to the funnel plot (Figure S1) and Egger's test (P < 0.001). The stability of the pooled estimates was proved by sensitivity analyses. Regarding subgroup analyses, irrespective of sex, sub-localization, or major geographical regions, patients with autoimmune diseases had a significantly increased risk of colorectal cancer than the control population. Specifically, participants with autoimmune disorders from New Zealand (2.976, 95% CI: 1.725–5.133, P < 0.001), Germany (2.186, 95% CI: 1.080–4.424, P = 0.030), China (1.421, 95% CI: 1.157–1.747, P < 0.001), and Sweden (1.409, 95% CI: 1.237–1.605, P < 0.001) were even more susceptible to colorectal cancer compared to disease-free participants. As for specific autoimmune disease types (n>5), primary sclerosing cholangitis (6.717, 95% CI: 4.103–10.997, P < 0.001), idiopathic inflammatory myopathies (2.468, 95% CI: 1.889–3.225, P < 0.001), dermatomyositis alone (3.366, 95% CI: 1.897–5.971, P < 0.001), inflammatory bowel disease (1.778, 95% CI: 1.591–1.988, P < 0.001), Crohn's disease alone (1.714, 95% CI: 1.501–1.956, P < 0.001), ulcerative colitis alone (1.720, 95% CI: 1.459–2.028, P < 0.001), autoimmune hepatitis (1.571, 95% CI: 1.138–2.167, P = 0.006), ANCA-associated vasculitis (1.384, 95% CI: 1.071–1.790, P = 0.013), sarcoidosis (1.317, 95% CI 1.166–1.486, P < 0.001), scleroderma (1.222, 95% CI: 1.016–1.469, P = 0.033), type 1 diabetes (1.174, 95% CI: 1.022–1.348, P = 0.024), and psoriasis (1.147, 95% CI: 1.059–1.243, P = 0.001) were confirmed to increase the risk of colorectal cancer among autoimmune participants, while Sjögren syndrome (0.774, 95% CI: 0.607–0.988, P = 0.040) and rheumatoid arthritis (0.840, 95% CI: 0.788–0.895, P < 0.001) were linked to risk-decreasing effects by the pooled risk ratio. Meanwhile, despite fewer studies, several types of autoimmune disease were also potentially associated with an elevated or decreased risk of colorectal cancer among autoimmune participants in the pooled risk ratio, including membranous nephropathy (3.499, 95% CI: 1.115–10.979, P = 0.032), hidradenitis suppurativa (1.426, 95% CI: 1.210–1.680, P < 0.001), idiopathic thrombocytopenic purpura (1.371, 95% CI: 1.176–1.598, P < 0.001), alopecia areata (0.940, 95% CI: 0.895–0.987, P = 0.013), and lichen sclerosus (0.887, 95% CI: 0.798–0.985, P = 0.025) (Fig. 3).

Discussion

Currently, associations between autoimmune diseases and colorectal cancer were unclear[17,18]. There were few updated and comprehensive pooled results on the associative role of overall

Category			Incidence					Risk				
			Total cohorts	Incidence (per 100000 person-years; 95%CI, random-effects)	Cochran's Q	I^2	Egger's test	Total cohorts	Risk ratio (95%CI, random-effects)	Cochran's Q	I^2	Egger's test
Total			n=590	96.622 (92.173-101.070), p<0.001	22691.81 (df=589), p<0.001	97.4%	p<0.001	n=436	1.244 (1.190-1.300), p<0.001	6689.99 (df=435), p<0.001	93.5%	p<0.001
Sensitivity analysis	Leave-one-out		n=590	Stable	-	-	-	n=436	Stable	-	-	-
	Specific population excluded		n=552	95.583 (90.946-100.219), p<0.001	16904.95 (df=551), p<0.001	96.7%	p<0.001	n=380	1.275 (1.213-1.341), p<0.001	6354.70 (df=379), p<0.001	94.0%	p<0.001
	Specific medication excluded		n=487	95.863 (91.102-100.624), p<0.001	21204.27 (df=486), p<0.001	97.7%	p<0.001	n=403	1.237 (1.200-1.316), p<0.001	6513.84 (df=402), p<0.001	93.8%	p<0.001
	Specific follow-up excluded		n=565	96.650 (92.135-101.165), p<0.001	22529.92 (df=564), p<0.001	97.5%	p<0.001	n=433	1.248 (1.194-1.305), p<0.001	6659.65 (df=432), p<0.001	93.5%	p<0.001
	Specific effect size excluded		-	-	-	-	-	n=434	1.245 (1.191-1.302), p<0.001	6687.27 (df=433), p<0.001	93.5%	p<0.001
	Small sample-size excluded		n=421	95.855 (91.152-100.559), p<0.001	22294.30 (df=420), p<0.001	98.1%	p<0.001	n=364	1.198 (1.144-1.254), p<0.001	6356.57 (df=363), p<0.001	94.3%	p<0.001
Subgroup analysis	Location	Colon	n=225	54.771 (50.509-59.033), p<0.001	3526.88 (df=224), p<0.001	93.6%	p<0.001	n=153	1.308 (1.205-1.420), p<0.001	1446.37 (df=152), p<0.001	89.5%	p=0.734
		Rectum	n=135	27.616 (24.938-30.294), p<0.001	1552.66 (df=134), p<0.001	91.4%	p<0.001	n=131	1.173 (1.090-1.263), p<0.001	475.08 (df=130), p<0.001	72.6%	p=0.649
	Sex	Male	n=64	98.395 (86.522-110.269), p<0.001	1687.05 (df=63), p<0.001	96.3%	p<0.001	n=124	1.229 (1.132-1.333), p<0.001	1081.36 (df=123), p<0.001	88.6%	p=0.044
		Female	n=65	79.299 (69.152-89.445), p<0.001	2139.07 (df=64), p<0.001	97.0%	p<0.001	n=105	1.209 (1.112-1.315), p<0.001	784.20 (df=104), p<0.001	86.7%	p=0.004
	Era	2020s	n=143	106.779 (96.826-116.732), p<0.001	6692.34 (df=142), p<0.001	97.9%	p<0.001	n=110	1.157 (1.081-1.237), p<0.001	904.86 (df=109), p<0.001	88.0%	p=0.082
		2010s	n=265	89.707 (83.667-95.748), p<0.001	9484.21 (df=264), p<0.001	97.2%	p<0.001	n=213	1.175 (1.120-1.233), p<0.001	1738.46 (df=212), p<0.001	87.8%	p=0.001
		2000s	n=93	91.307 (81.221-101.394), p<0.001	2528.83 (df=92), p<0.001	96.4%	p<0.001	n=72	1.475 (1.198-1.815), p<0.001	3333.44 (df=71), p<0.001	97.9%	p=0.353
		<2000s	n=89	110.624 (94.788-126.460), p<0.001	778.62 (df=88), p<0.001	88.7%	p<0.001	n=41	1.433 (1.131-1.815), p=0.003	448.51 (df=40), p<0.001	91.1%	p=0.180
	Count	All	n=493	99.906 (94.491-105.321), p<0.001	17654.31 (df=492), p<0.001	97.2%	p<0.001	n=330	1.263 (1.199-1.331), p<0.001	3769.51 (df=329), p<0.001	91.3%	p<0.001
		First	n=97	86.577 (78.289-94.864), p<0.001	3958.28 (df=96), p<0.001	97.6%	p<0.001	n=106	1.197 (1.088-1.318), p<0.001	2861.05 (df=105), p<0.001	96.3%	p=0.028
	Effect-size	Adjusted HR/RR	-	-	-	-	-	n=147	1.113 (1.058-1.172), p<0.001	1219.69 (df=146), p<0.001	88.0%	p=0.124
		Stratified SIR	-	-	-	-	-	n=293	1.354 (1.266-1.448), p<0.001	5475.84 (df=292), p<0.001	94.7%	p<0.001
	Population	National	n=291	96.856 (91.143-102.569), p<0.001	19965.42 (df=290), p<0.001	98.5%	p<0.001	n=254	1.148 (1.095-1.202), p<0.001	2708.43 (df=253), p<0.001	90.7%	p=0.010
		Regional	n=299	98.154 (90.592-105.715), p<0.001	2506.52 (df=298), p<0.001	88.1%	p<0.001	n=182	1.475 (1.334-1.632), p<0.001	3979.76 (df=181), p<0.001	95.5%	p<0.001
	Country	Nordic	n=171	94.055 (87.124-100.986), p<0.001	10010.13 (df=170), p<0.001	98.3%	p<0.001	n=158	1.301 (1.200-1.411), p<0.001	2126.52 (df=157), p<0.001	92.6%	p=0.177
		~Denmark	n=55	98.130 (84.315-111.944), p<0.001	997.78 (df=54), p<0.001	94.6%	p=0.107	n=43	1.140 (1.050-1.238), p=0.002	105.62 (df=42), p<0.001	60.2%	p=0.082
		~Finland	n=18	58.790 (45.565-72.014), p<0.001	103.32 (df=17), p<0.001	83.5%	p=0.236	n=16	1.194 (0.913-1.562), p=0.196	88.19 (df=15), p<0.001	83.0%	p=0.991
		~Norway	n=5	115.712 (68.235-163.189), p<0.001	3.22 (df=4), p=0.521	0.0%	p=0.277	n=4	1.471 (0.908-2.383), p=0.117	0.83 (df=3), p=0.842	0.0%	p=0.557
		~Sweden	n=81	97.273 (87.545-107.002), p<0.001	5259.93 (df=80), p<0.001	98.5%	p<0.001	n=84	1.409 (1.237-1.605), p<0.001	1727.92 (df=83), p<0.001	95.2%	p=0.052
		Western	n=259	104.837 (97.224-112.439), p<0.001	5295.88 (df=258), p<0.001	95.1%	p<0.001	n=170	1.213 (1.135-1.296), p<0.001	1449.83 (df=169), p<0.001	88.3%	p=0.013
		~Australia	n=18	156.874 (114.321-199.427), p<0.001	50.19 (df=17), p<0.001	66.1%	p<0.001	n=10	1.163 (0.865-1.565), p=0.318	28.57 (df=9), p=0.001	68.5%	p=0.978
		~Belgium	n=2	156.874 (114.321-199.427), p<0.001	0.00 (df=1), p=0.977	0.0%	-	-	-	-	-	-
		~Canada	n=11	109.030 (68.169-149.892), p<0.001	385.60 (df=10), p<0.001	97.4%	p=0.159	n=11	1.288 (1.080-1.537), p=0.005	76.69 (df=10), p<0.001	87.0%	p=0.213
		~France	n=14	92.747 (74.234-111.261), p<0.001	212.13 (df=13), p<0.001	93.9%	p=0.718	n=7	1.080 (0.926-1.259), p=0.328	46.06 (df=6), p<0.001	87.0%	p=0.382
		~Germany	n=7	117.795 (45.675-189.914), p<0.001	25.48 (df=6), p<0.001	76.5%	p=0.661	n=2	2.186 (1.000-4.424), p=0.030	1.24 (df=1), p=0.265	19.6%	-
		~Greece	n=5	108.291 (39.070-177.511), p=0.002	3.63 (df=4), p=0.459	0.0%	p=0.009	-	-	-	-	-
		~Israel	n=11	163.112 (112.650-213.573), p<0.001	260.04 (df=10), p<0.001	96.2%	p=0.040	n=5	1.067 (0.838-1.360), p=0.598	18.78 (df=4), p<0.001	78.7%	p=0.423
		~Italy	n=22	53.150 (35.099-71.201), p<0.001	50.37 (df=21), p<0.001	58.3%	p=0.001	n=9	0.879 (0.622-1.241), p=0.464	13.55 (df=8), p=0.094	41.0%	p=0.665
		~New Zealand	n=8	132.813 (76.414-189.212), p<0.001	12.24 (df=7), p=0.093	42.8%	p=0.015	n=6	2.976 (1.725-5.133), p<0.001	10.79 (df=5), p=0.056	53.6%	p=0.971
		~Portugal	n=2	202.922 (90.594-315.249), p<0.001	0.49 (df=1), p=0.483	0.0%	-	-	-	-	-	-
		~Spain	n=11	70.395 (34.208-106.581), p<0.001	36.54 (df=10), p<0.001	72.6%	p=0.018	n=9	0.744 (0.415-1.335), p=0.322	30.06 (df=8), p<0.001	73.4%	p=0.847
		~The Netherlands	n=11	103.819 (66.457-141.181), p<0.001	44.18 (df=10), p<0.001	77.4%	p<0.001	n=5	1.728 (0.883-3.382), p=0.110	14.98 (df=4), p=0.005	73.3%	p=0.498
		~UK	n=47	107.374 (91.128-123.619), p<0.001	1828.53 (df=46), p<0.001	97.5%	p<0.001	n=43	1.180 (1.038-1.341), p=0.011	269.11 (df=42), p<0.001	84.4%	p=0.067
		~USA	n=70	105.876 (91.265-120.486), p<0.001	570.48 (df=69), p<0.001	87.9%	p=0.012	n=54	1.230 (1.076-1.407), p=0.002	575.76 (df=53), p<0.001	90.8%	p=0.037
		East Asian	n=143	86.414 (78.053-94.775), p<0.001	3569.98 (df=142), p<0.001	96.0%	p=0.007	n=103	1.213 (1.101-1.335), p<0.001	2845.95 (df=102), p<0.001	96.4%	p=0.063
		~China	n=48	72.230 (60.838-83.623), p<0.001	495.15 (df=47), p<0.001	90.5%	p<0.001	n=47	1.421 (1.157-1.747), p<0.001	2485.40 (df=46), p<0.001	98.1%	p=0.113
		~Japan	n=38	102.311 (83.613-121.008), p<0.001	80.66 (df=37), p<0.001	54.1%	p=0.004	n=11	0.873 (0.606-1.258), p=0.466	55.31 (df=10), p<0.001	81.9%	p=0.085
		~Singapore	n=4	218.618 (-94.326-531.562), p=0.171	5.36 (df=3), p=0.147	44.1%	p=0.032	-	-	-	-	-
		~South Korea	n=53	89.082 (77.025-101.140), p<0.001	2149.52 (df=52), p<0.001	97.6%	p=0.350	n=44	1.116 (1.024-1.216), p=0.012	264.30 (df=43), p<0.001	83.7%	p=0.056
		Other	n=17	108.077 (65.121-151.034), p<0.001	109.87 (df=16), p<0.001	85.4%	p=0.986	n=5	0.986 (0.581-1.675), p=0.959	3.14 (df=4), p=0.534	0.0%	p=0.462
		~Brazil	n=2	165.663 (145.641-185.685), p<0.001	0.45 (df=1), p=0.505	0.0%	-	-	-	-	-	-
		~Hungary	n=5	96.907 (28.868-164.946), p=0.005	21.31 (df=4), p<0.001	81.2%	p=0.328	n=4	0.945 (0.535-1.669), p=0.845	2.98 (df=3), p=0.395	0.0%	p=0.606
		~India	n=4	175.992 (32.654-319.330), p=0.016	25.22 (df=3), p<0.001	88.1%	p=0.032	-	-	-	-	-
		~Turkey	n=5	40.802 (6.004-75.600), p=0.022	2.73 (df=4), p=0.605	0.0%	p=0.014	-	-	-	-	-
	Type	AAV	n=14	118.624 (77.909-159.339), p<0.001	21.67 (df=13), p=0.061	40.0%	p=0.001	n=15	1.304 (1.071-1.790), p=0.013	8.69 (df=14), p=0.851	0.0%	p=0.124
		~GPA	n=4	135.466 (62.636-208.295), p<0.001	5.99 (df=3), p=0.112	49.9%	p=0.213	n=5	1.510 (1.014-2.248), p=0.043	2.92 (df=4), p=0.571	0.0%	p=0.467
		AD	-	-	-	-	-	n=2	0.850 (0.518-1.395), p=0.520	0.09 (df=1), p=0.763	0.0%	-
		AIG	n=7	202.932 (149.811-256.053), p<0.001	21.60 (df=6), p=0.001	72.2%	p=0.116	n=6	1.033 (0.906-1.183), p=0.612	7.30 (df=5), p=0.199	31.5%	p=0.270
		AIH	n=9	106.462 (77.190-135.734), p<0.001	15.29 (df=8), p=0.054	47.7%	p=0.757	n=7	1.571 (1.138-2.167), p=0.006	14.04 (df=6), p=0.029	57.3%	p=0.069
		AIHA	-	-	-	-	-	n=2	1.241 (0.868-1.774), p=0.236	0.31 (df=1), p=0.577	0.0%	-
		AIP	n=7	251.424 (156.372-346.476), p<0.001	2.21 (df=6), p=0.900	0.0%	p=0.333	n=2	0.883 (0.568-1.370), p=0.578	0.40 (df=1), p=0.526	0.0%	-
		AIA	n=2	39.864 (9.813-69.915), p=0.009	231.58 (df=1), p<0.001	99.6%	-	n=2	0.940 (0.895-0.987), p=0.013	0.90 (df=1), p=0.342	0.0%	-
		ALS	-	-	-	-	-	n=2	0.968 (0.578-1.621), p=0.901	2.77 (df=1), p=0.096	63.9%	-
		BD	n=6	57.906 (43.232-72.580), p<0.001	5.60 (df=5), p=0.347	10.8%	p=0.594	n=5	1.056 (0.763-1.462), p=0.740	5.56 (df=4), p=0.235	28.1%	p=0.025
		BP	n=2	232.913 (159.632-306.194), p<0.001	0.29 (df=1), p=0.592	0.0%	-	n=2	0.937 (0.765-1.148), p=0.531	0.81 (df=1), p=0.369	0.0%	-
		CeD	n=9	47.057 (25.756-68.357), p<0.001	152.82 (df=8), p<0.001	94.8%	p=0.671	n=11	1.067 (0.966-1.179), p=0.200	8.88 (df=10), p=0.544	0.0%	p=0.611
		CSHD	-	-	-	-	-	n=2	1.167 (0.954-1.427), p=0.134	2.27 (df=1), p=0.132	56.0%	-
		DH	n=6	65.467 (35.786-95.147), p<0.001	4.33 (df=5), p=0.503	0.0%	p=0.221	n=4	1.027 (0.575-1.833), p=0.928	3.37 (df=3), p=0.338	11.0%	p=0.849
		GBS	-	-	-	-	-	n=2	1.075 (0.911-1.268), p=0.391	0.21 (df=1), p=0.643	0.0%	-
		GCA	n=4	209.962 (33.897-386.027), p=0.019	27.26 (df=3), p<0.001	89.0%	p=0.907	n=4	1.128 (0.950-1.340), p=0.168	2.91 (df=3), p=0.406	0.0%	p=0.526
		GD	n=7	49.292 (28.892-69.691), p<0.001	115.95 (df=6), p<0.001	94.80%	p=0.740	n=8	0.925 (0.816-1.048), p=0.221	17.97 (df=7), p=0.012	61.1%	p=0.031
		HS	n=3	47.201 (21.946-72.456), p<0.001	18.57 (df=2), p<0.001	89.2%	p=0.014	n=3	1.426 (1.210-1.680), p<0.001	0.39 (df=2), p=0.822	0.0%	p=0.262
		HT	n=2	75.238 (58.997-91.480), p<0.001	0.63 (df=1), p=0.428	0.0%	-	n=3	1.345 (0.543-3.329), p=0.522	6.28 (df=2), p=0.043	68.2%	p=0.055
		IBD	n=173	122.611 (114.015-131.206), p<0.001	2403.50 (df=172), p<0.001	92.8%	p<0.001	n=107	1.778 (1.591-1.988), p<0.001	1584.36 (df=106), p<0.001	93.3%	p=0.143
		~CrD	n=59	85.225 (75.052-95.397), p<0.001	348.01 (df=58), p<0.001	83.3%	p=0.287	n=55	1.174 (1.501-1.956), p<0.001	281.83 (df=54), p<0.001	80.8%	p=0.061
		~UC	n=84	149.043 (134.702-163.384), p<0.001	1171.66 (df=83), p<0.001	92.9%	p<0.001	n=53	1.720 (1.459-2.028), p<0.001	1215.42 (df=52), p<0.001	95.7%	p=0.523
		IgG4-RD	n=8	211.463 (129.694-293.191), p<0.001	8.53 (df=7), p=0.288	18.0%	p=0.079	n=2	0.911 (0.550-1.430), p=0.685	1.01 (df=1), p=0.315	1.1%	-
		~AIP-I	n=4	267.121 (161.722-372.520), p<0.001	0.40 (df=3), p=0.941	0.0%	p=0.305	n=2	0.883 (0.568-1.370), p=0.578	0.40 (df=1), p=0.526	0.0%	-
		~IgG4-SC	n=2	259.041 (10.737-507.346), p=0.041	1.46 (df=1), p=0.227	31.4%	-	-	-	-	-	-
		IIMs	n=22	126.741 (89.751-163.731), p<0.001	18.17 (df=21), p=0.638	0.0%	p=0.001	n=16	2.468 (1.889-3.225), p<0.001	95.43 (df=15), p<0.001	84.3%	p=0.009
		~DM	n=8	152.995 (58.997-246.994), p=0.001	7.20 (df=7), p=0.408	2.8%	p=0.001	n=5	3.366 (1.897-5.971), p<0.001	17.72 (df=4), p=0.001	77.4%	p=0.594
		~PM	n=4	163.411 (71.202-255.619), p<0.001	2.97 (df=3), p=0.396	0.0%	p=0.285	n=5	1.742 (0.724-4.190), p=0.215	36.77 (df=4), p<0.001	89.1%	p=0.027
		ITP	n=2	132.949 (-18.457-284.356), p=0.085	36.56 (df=1), p<0.001	97.3%	-	n=4	1.371 (1.176-1.598), p<0.001	1.68 (df=3), p=0.642	0.0%	p=0.342
		LP	n=3	77.442 (31.466-123.419), p=0.001	4.43 (df=2), p=0.109	54.8%	p=0.091	n=4	0.856 (0.690-1.061), p=0.155	1.69 (df=3), p=0.639	0.0%	p=0.803
		LS	n=3	95.823 (11.066-180.580), p=0.027	55.38 (df=2), p<0.001	96.4%	p=0.952	n=2	0.887 (0.798-0.985), p=0.025	0.58 (df=1), p=0.444	0.0%	-
		Lu	n=29	56.499 (44.888-68.111), p<0.001	155.35 (df=28), p<0.001	82.0%	p=0.070	n=29	1.059 (0.927-1.210), p=0.396	82.47 (df=28), p<0.001	66.0%	p=0.005
		~SLE	n=27	55.971 (44.008-67.933), p<0.001	153.81 (df=26), p<0.001	83.1%	p<0.001	n=27	1.083 (0.938-1.250), p=0.279	81.67 (df=26), p<0.001	68.2%	p=0.005
		MG	n=5	109.126 (66.125-152.127), p<0.001	13.67 (df=4), p=0.008	70.7%	p=0.782	n=3	1.246 (0.973-1.596), p=0.081	3.44 (df=2), p=0.179	41.8%	p=0.316
		MN	n=3	248.968 (84.040-413.896), p=0.003	1.28 (df=2), p=0.528	0.0%	p=0.086	n=3	3.499 (1.115-10.979), p=0.032	0.83 (df=2), p=0.661	0.0%	p=0.159
		MS	n=22	57.306 (44.565-70.048), p<0.001	336.72 (df=21), p<0.001	93.8%	p=0.350	n=12	0.923 (0.798-1.068), p=0.281	40.93 (df=11), p<0.001	73.1%	p=0.957
		PBC	n=11	131.464 (94.290-168.639), p<0.001	11.20 (df=10), p=0.342	10.7%	p=0.760	n=7	1.168 (0.953-1.430), p=0.135	2.18 (df=6), p=0.902	0.0%	p=0.392
		PMR	n=2	222.060 (-50.351-494.471), p=0.110	8.75 (df=1), p=0.003	88.6%	-	n=4	0.940 (0.860-1.027), p=0.172	0.43 (df=3), p=0.934	0.0%	p=0.685
		Ps	n=22	85.141 (64.066-106.215), p<0.001	1573.80 (df=21), p<0.001	98.7%	p=0.946	n=22	1.147 (1.059-1.243), p<0.001	115.69 (df=21), p<0.001	81.8%	p=0.028
		PSC	n=20	373.515 (265.178-481.852), p<0.001	68.54 (df=19), p<0.001	72.3%	p<0.001	n=9	6.717 (4.103-10.997), p<0.001	32.68 (df=8), p<0.001	75.5%	p=0.507
		RA	n=101	100.577 (91.014-110.139), p<0.001	1329.91 (df=100), p<0.001	92.5%	p=0.246	n=54	0.840 (0.788-0.895), p<0.001	350.14 (df=53), p<0.001	84.9%	p=0.054
		RF	-	-	-	-	-	n=3	1.088 (0.848-1.397), p=0.507	0.42 (df=2), p=0.809	0.0%	p=0.193
		SpA	n=20	70.471 (54.600-86.342), p<0.001	229.78 (df=19), p<0.001	91.7%	p=0.034	n=21	0.918 (0.826-1.020), p=0.111	28.09 (df=20), p=0.107	28.8%	p=0.964
		~AS	n=10	81.853 (55.402-108.304), p<0.001	164.37 (df=9), p<0.001	94.5%	p=0.017	n=14	0.869 (0.744-1.014), p=0.074	28.71 (df=13), p=0.007	54.7%	p=0.318
		~PsA	n=7	71.223 (51.382-91.263), p<0.001	17.90 (df=6), p=0.006	66.5%	p=0.012	n=6	0.976 (0.804-1.184), p=0.802	3.91 (df=5), p=0.563	0.0%	p=0.481
		Sa	n=8	61.922 (41.608-82.236), p<0.001	32.14 (df=7), p<0.001	78.2%	p=0.796	n=11	1.317 (1.166-1.486), p<0.001	14.24 (df=10), p=0.162	29.8%	p=0.637
		Sc	n=18	69.513 (43.701-95.324), p<0.001	76.62 (df=17), p<0.001	77.8%	p=0.020	n=10	1.222 (1.016-1.469), p=0.033	11.18 (df=9), p=0.263	19.5%	p=0.160
		~SSc	n=16	72.532 (41.569-103.494), p<0.001	71.94 (df=15), p<0.001	79.1%	p=0.005	n=9	1.207 (0.969-1.504), p=0.093	10.99 (df=8), p=0.202	27.2%	p=0.198
		SS	n=11	74.818 (49.854-99.782), p<0.001	49.70 (df=10), p<0.001	79.9%	p=0.458	n=11	0.774 (0.607-0.988), p=0.040	22.87 (df=10), p=0.011	56.3%	p=0.572
		T1DM	n=8	20.485 (12.587-28.382), p<0.001	497.63 (df=7), p<0.001	98.6%	p=0.374	n=10	1.174 (1.022-1.348), p=0.024	24.68 (df=9), p=0.003	63.5%	p=0.829
		VI	n=4	69.996 (56.530-83.463), p<0.001	12.36 (df=3), p=0.006	75.7%	p=0.801	n=4	0.873 (0.646-1.180), p=0.376	24.45 (df=3), p<0.001	87.7%	p=0.024

Figure 3. Pooled estimates for colorectal cancer incidence and risk. Note: Acronyms of different types of autoimmune diseases are listed in Table S3, http://links.lww.com/JS9/E478.

or specific autoimmune diseases in colorectal cancer risk, especially when individual studies reported inconsistent outcomes.

Compared to baseline data from GLOBOCAN 2022[2], patients with autoimmune diseases in our analysis had relatively higher incidence of colorectal cancer, irrespective of sub-localization (colon or rectum), sex, or region. Similarly, all risk ratios of overall colorectum, colon, or rectum alone and sex-specific subgroups demonstrated significantly a higher risk of colorectal carcinogenesis among autoimmune patients. Moreover, unlike similar pooled analyses on gastric and small bowel cancer samples performed by our team (data not published yet), this risk-increasing impact on colorectal cancer was quite consistent across Nordic, Western, and East Asian countries, suggesting its universal role irrespective of racial and socioeconomical backgrounds. In terms of specific disease types, both digestive organ-specific or systemic autoimmune diseases could significantly increase the risk of colorectal cancer compared to disease-free participants, among which patients with organ-specific diseases were actually more susceptible, such as primary sclerosing cholangitis and inflammatory bowel disease. The potential carcinogenic mechanisms of digestive organ-specific autoimmune diseases were easy to expect, based on its direct inflammatory stimulants on colorectum, which also explained why New Zealand and Sweden had the highest risk compared to other countries. As for systemic diseases such as idiopathic inflammatory myopathies and ANCA-associated vasculitis, autoimmune disturbances among patients could result in escape of tumor cells from immune surveillance, causing an elevated risk of colorectal cancer. Meanwhile, systemic immune dysregulation could also change microenvironment around colorectal tissue, falsely stimulate proinflammatory and carcinogenic pathways, and thus result in colorectal tumorigenesis. Moreover, autoimmune diseases and carcinogenesis could be induced by similar genetic background, such as *TP53*, a tumor-suppressive gene whose mutation leads to susceptibility of both inflammatory bowel disease and colorectal cancer. Besides, therapeutic factors should not be ignored since steroids and other immune-suppressive treatments against autoimmune diseases could also lead to lowered immune surveillance and thus higher colorectal cancer risk. Interestingly, we found that several autoimmune diseases might help to lower colorectal cancer risk, especially rheumatoid arthritis. This risk-decreasing impact seemed quite convincible due to large study amounts and significant effect size; however, specific mechanisms on how rheumatoid arthritis helped to reduce colorectal cancer risk remained unclear, especially when rheumatoid arthritis had no such effects on gastric cancer or small bowel cancer. Concerning potential mechanisms of these protective impacts, therapeutic usage of nonsteroidal anti-inflammatory drugs, which were already believed to lower colorectal cancer risk, lifestyle adjustments, increased colonic examination, and early treatment of precancerous lesions could all contribute to its protective effects. Besides, rheumatoid arthritis or other selected types might have different immune microenvironments and impacts on gut microbiota and cross antigenicity, which eventually contributed to their tumor-suppressive effects. All these results suggested that besides the traditional risk factors of colorectal cancer such as unhealthy diet habits and genetic susceptibility, autoimmune disease could potentially be another vital risk factor, despite that different autoantibodies and specific immune microenvironments might have contradictory effects.

Our meta-analysis was an updated and most comprehensive meta-analysis based on global population on this topic. We only included English-language articles since English was predominantly applied in medical researches even though in non-English speaking countries. Although there might be some non-English articles on this topic, their methodological quality, research details, and authenticity could not be fully checked and trusted not only by us but also by readers of our analysis, since not all readers were multilinguals. Therefore, we believed that this strategy could still guarantee the integrity and quality of literature retrieval. Total sample sizes and numbers of included studies were adequate for credible results. Meanwhile, we conducted several sensitivity analyses and various types of subgroup analyses to confirm the stability as well as provide extensive applicability of overall estimates. Although publication bias was detected, we further used a "trim-and-fill" method to prove that this bias had insignificant impacts on pooled estimates. However, there were still several limitations in our meta-analysis. First, since this was not an individual-participant-data meta-analysis, the impacts of internal heterogeneity on pooled estimates could not be fully eliminated despite the random-effects model with adjusted effect sizes. Different population sources and regions hinted diversified racial and genetic backgrounds, various lifestyles, and economic statuses, as well as different disease subtypes. All these could contribute to significant heterogeneity in our pooled analysis. Besides, differences in diagnostic criteria for the same disease type could also lead to different compositions of patients. Second, different studies based on the same source of the database might partially overlap in terms of included participants, which resulted in repetitive data and could not be fully recognized. Thirdly, due to a limited amount of subgroup data, we could not evaluate impacts of other confounding factors such as medication, diseases course, social status, smoking, and so on.

In conclusion, patients with autoimmune diseases were associated with significantly a higher risk of colorectal cancer in general or specific settings. Unlike our gastric and small bowel cancer pooled results, this risk-increasing impact on colorectal cancer was quite consistent across Nordic, Western, and East Asian countries. Both digestive organ-specific or systemic autoimmune diseases could significantly increase the risk of colorectal cancer, including primary sclerosing cholangitis, idiopathic inflammatory myopathies, inflammatory bowel disease, autoimmune hepatitis, ANCA-associated vasculitis, sarcoidosis, scleroderma, type 1 diabetes, psoriasis, membranous nephropathy, hidradenitis suppurativa, and idiopathic thrombocytopenic purpura. Interestingly, several autoimmune diseases might help to lower colorectal cancer risk, especially rheumatoid arthritis, while it had no such effects on gastric or small bowel carcinogenesis.

Ethical approval

Not applicable.

Sources of funding

This meta-analysis was funded by the National Natural Science Foundation of China (81902487) and the Natural Science Foundation of Hubei Province (2024AFB673) to Ji Cheng.

Author contributions

Study design: Ji Cheng; Manuscript writing and revision: Ji Cheng and GuobinWang; Literature retrieval: Ji Cheng and Ming Cai; Discretion of eligibility: Ji Cheng and Ming Cai; Quality assessment: Ji Cheng and Ming Cai; Data extraction: Ji Cheng and Guobin Wang; Statistical analysis: Ji Cheng and Ming Cai.

Conflicts of interest disclosure

We declare no competing interests.

Research registration unique identifying number (UIN)

PROSPERO (CRD42024547332).

Guarantor

Ji Cheng.

Provenance and peer review

Not commissioned, externally peer-reviewed.

Data availability statement

Not applicable.

References

[1] Eng C, Yoshino T, Ruiz-Garcia E, *et al*. Colorectal cancer. Lancet 2024;404:294–310.
[2] Bray F, Laversanne M, Sung H, *et al*. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2024;74:229–63.
[3] Masetti R, Tiri A, Tignanelli A, *et al*. Autoimmunity and cancer. Autoimmun Rev 2021;20:102882.
[4] Zitvogel L, Perreault C, Finn OJ, Kroemer G. Beneficial autoimmunity improves cancer prognosis. Nat Rev Clin Oncol 2021;18:591–602.
[5] Bruni D, Angell HK, Galon J. The immune contexture and Immunoscore in cancer prognosis and therapeutic efficacy. Nat Rev Cancer 2020;20:662–80.
[6] Schurch CM, Bhate SS, Barlow GL, *et al*. Coordinated cellular neighborhoods orchestrate antitumoral immunity at the colorectal cancer invasive front. Cell 2020;182:1341–59e19.
[7] Li J, Wu C, Hu H, *et al*. Remodeling of the immune and stromal cell compartment by PD-1 blockade in mismatch repair-deficient colorectal cancer. Cancer Cell 2023;41:1152–69e7.
[8] Pierret C, Mulliez A, Le Bihan-Benjamin C, Moisset X, Bousquet PJ, Leray E. Cancer risk among patients with multiple sclerosis: a 10-year nationwide retrospective cohort study. Neurology 2024;103:e209885.
[9] Wewer MD, Letnar G, Andersen KK, *et al*. Thiopurines and the risk of cancer in patients with inflammatory bowel disease and reference individuals without inflammatory bowel disease – a Danish nationwide cohort study (1996-2018). Clin Gastroenterol Hepatol 2024;23:1030–38.
[10] Finstad A, Glockler-Lauf SD, Almuhanna N, Saskin R, Alhusayen R. Investigating the risk of malignancy in elderly patients with psoriasis: a population-based retrospective matched cohort study. J Am Acad Dermatol 2023;88:1129–31.
[11] Wu S, Xie S, Yuan C, *et al*. Inflammatory bowel disease and long-term risk of cancer: a prospective cohort study among half a million adults in UK biobank. Inflamm Bowel Dis 2023;29:384–95.

[12] Page MJ, McKenzie JE, Bossuyt PM, *et al*. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. Int J Surg 2021;88:105906.
[13] Kelly SE, Moher D, Clifford TJ. Quality of conduct and reporting in rapid reviews: an exploration of compliance with PRISMA and AMSTAR guidelines. Syst Rev 2016;5:79.
[14] Shea BJ, Reeves BC, Wells G, *et al*. AMSTAR 2: a critical appraisal tool for systematic reviews that include randomised or non-randomised studies of healthcare interventions, or both. BMJ 2017;358:j4008.
[15] Agha RA, Mathew G, Rashid R, *et al*. Transparency In The reporting of Artificial INtelligence – the TITAN guideline. Prem J Sci 2025;10:100082.
[16] Vaengebjerg S, Skov L, Egeberg A, Prevalence LND. Incidence, and risk of cancer in patients with psoriasis and psoriatic arthritis: a systematic review and meta-analysis. JAMA Dermatol 2020;156:421–29.
[17] Bai X, Guo Y, Zhu X, Dai D. Autoimmune diseases and risk of gastrointestinal cancer: an umbrella review of meta-analyses of observational studies. Int J Surg 2025;111:2273–82.
[18] He MM, Lo CH, Wang K, *et al*. Immune-mediated diseases associated with cancer risks. JAMA Oncol 2022;8:209–19.