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THE ROLE OF IgE-MEDIATED ALLERGIC RESPONSES IN COLORECTAL CANCER IMMUNOSURVEILLANCE: A LITERATURE REVIEW

Ștefania-Eugenia BĂLĂȘOIU[1] (ORCID: 0009-0002-8265-4026); Alexandru MURARU[1]; Florin PRUNAR[1]; Silvia PRUNAR[1]

University of Life Sciences "King Mihai I" from Timișoara, Faculty of Agriculture, Department of Biology

Corresponding author: balasoiu.stefania-eugenia.fa@usvt.ro

Abstract. *The relationship between allergic diseases and cancer development has attracted increasing scientific interest, particularly regarding the potential interaction between allergic immune responses and antitumor immunit). This literature review examines the possible association between pollen allergy and colorectal cancer (CRC) risk, focusing on the immunological mechanisms that may contribute to this relationship. The main objective is to evaluate epidemiological findings alongside current knowledge of immune surveillance, IgE-mediated hypersensitivity, and the colorectal tumor microenvironment. The review discusses the cellular and molecular pathways involved in pollen-induced allergic responses, including T helper 2 (Th2) polarization, immunoglobulin E (IgE), mast cell activation, and eosinophil recruitment. These mechanisms are considered in relation to colorectal carcinogenesis, chronic inflammation, and the immune system's capacity to recognize and control malignant cells. Epidemiological cohort studies and meta-analyses reporting associations between allergic conditions and CRC risk are compared, with particular attention to differences between allergic rhinitis, asthma, and other allergic diseases. The available evidence suggests that certain allergic conditions, particularly hay fever and allergic rhinitis, may be associated with a modestly lower risk of colorectal cancer, and recent Mendelian randomization data indicate that eosinophils may partly mediate this association. However, findings remain inconsistent, and evidence concerning pollen allergy specifically is limited and largely indirect. Differences in study design, allergy classification, population characteristics, and potential confounding factors limit the interpretation of current results. Overall, this review highlights the need for further research integrating epidemiological evidence with mechanistic immunology to clarify whether, and through which pathways, pollen-associated immune responses may influence colorectal cancer development.*

Keywords: *pollen allergy; colorectal cancer; IgE; eosinophils; immune surveillance; tumor microenvironment.*

INTRODUCTION

The immune system functions as a complex network of cells and molecules involved in protecting the body. Its role is to identify and eliminate both pathogens and abnormal cells. Beyond its classical role in anti-infectious defense, the immune system also helps maintain tissue homeostasis by recognizing and eliminating cells with malignant potential. This process is known as immune surveillance, reflecting the body's ability to limit tumor development at an early stage. Allergic diseases represent a particular type of immune response, characterized by exaggerated reactions to antigens that are normally harmless, found in the environment, such as pollen, dust mites, or certain foods. Allergic reactions are mainly driven by IgE (immunoglobulin E) antibodies and mast cell activation, which lead to histamine release (COLAS et al., 2022; PAWANKAR et al., 2011).

General background: Immunologically, allergic manifestations are predominantly driven by a T helper 2 (Th2) immune profile, which differs markedly from the T helper 1 (Th1) polarization typical of cell-mediated anti-infectious defense and classical antitumor surveillance

mechanisms (PALM et al., 2012). Numerous epidemiological investigations have evaluated the complex relationship between allergic diseases and cancer incidence, yielding divergent results across different anatomical sites. Inverse associations suggesting a protective effect against colorectal cancer have been repeatedly reported for atopic allergic conditions in general (MA et al., 2017; TAMBE et al., 2015), whereas asthma has shown little evidence of an inverse colorectal cancer association and, in some analyses, a possible increase in risk (ALDUHAYH et al., 2025; YE et al., 2017). A meta-analysis of prospective studies reported a pooled relative risk (RR) of 0.88 (95% CI 0.83-0.92) for colorectal cancer among individuals with allergic conditions (MA et al., 2017), while another meta-analysis of observational studies found no significant association, with a pooled estimate of RR = 1.01 (95% CI 0.88-1.17) (YE et al., 2017). These conflicting observational patterns have generated contrasting immunological hypotheses, debating whether allergic hyper-reactivity enhances systemic tumor surveillance or whether persistent allergic tissue inflammation fuels carcinogenesis (LIU et al., 2023; PALM et al., 2012). To clarify these discrepancies, this review focuses on pollen allergy and its relationship with colorectal cancer risk, where inverse associations appear most frequently in the epidemiological literature and where systemic Th2-driven cellular pathways warrant detailed evaluation.

Aim and objectives: The primary objective of this review is to evaluate and integrate current literature data on the link between pollen allergy and colorectal cancer, analyzing both population-level epidemiological evidence and the underlying cellular mechanisms. Furthermore, this study aims to delineate the current degree of immunological understanding while highlighting methodological and theoretical limitations in the field. The specific objectives are: (i) outlining the core components and functions of immune surveillance relevant to colorectal malignancy; (ii) describing the IgE-mediated cellular cascade triggered by pollen exposure; (iii) analyzing colorectal tumor progression and its interactions with host immunity; (iv) comparing literature findings on allergic conditions and CRC risk; and (v) examining the specific roles of inflammatory mediators, mast cells, and eosinophils in colorectal tumor microenvironments.

Immune protection operates through two specialized branches: innate immunity and adaptive immunity. Innate immunity functions as an immediate, generalist defense mechanism without antigen-specific memory, relying on macrophages, neutrophils, dendritic cells, and natural killer (NK) cells, which recognize conserved molecular patterns to trigger phagocytosis and cytotoxicity. Adaptive immunity provides precise, antigen-specific memory mediated by B and T lymphocytes: B cells generate specific immunoglobulins, while CD4+ helper T cells coordinate responses through Th1, Th2, and regulatory T (Treg) subsets, and CD8+ cytotoxic T lymphocytes (CTLs) directly destroy infected or malignant cells via perforin and granzyme secretion (COLAS et al., 2022; PALM et al., 2012). Cytokines mediate communication between these branches: Th1-derived interferon-gamma (IFN-γ) drives antiviral and antitumor cellular immunity, whereas Th2-derived interleukin-4 (IL-4) and interleukin-13 (IL-13) mediate anti-parasitic responses and allergic hypersensitivity. While acute inflammation promotes tissue repair, persistent chronic inflammation can drive pathological transformations, including carcinogenesis.

IgE-mediated hypersensitivity: Allergic hypersensitivity represents an exaggerated adaptive response to non-pathogenic environmental antigens. During initial sensitization, dendritic cells in the respiratory mucosa capture pollen proteins and present allergen-derived peptides to naive CD4+ T cells, favoring Th2 differentiation. The resulting Th2 microenvironment induces B-cell class switching to IgE production. Antigen-specific IgE

antibodies circulate and bind high-affinity FcεRI receptors on mucosal mast cells and basophils, sensitizing these target effectors (COLAS et al., 2022; PAWANKAR et al., 2011).

Mast cell activation and mediator release: Subsequent allergen exposure leads to multivalent pollen binding and cross-linking of FcεRI-bound IgE molecules on sensitized mast cells. This cross-linking triggers rapid degranulation within minutes, releasing preformed mediators including histamine, tryptase, and heparin (PAWANKAR et al., 2011). Histamine induces classic allergic symptoms such as vasodilation, tissue edema, and rhinorrhea. Concurrently, activated mast cells synthesize lipid mediators (prostaglandins and leukotrienes) and secrete cytokines (IL-4, IL-5, IL-13) that sustain late-phase inflammation. Specifically, IL-5 drives the recruitment and activation of eosinophils in allergic inflammation (PAWANKAR et al., 2011; ROTHENBERG and HOGAN, 2006). Whether pollen-induced airway responses alter eosinophil activity in gastrointestinal tissues remains unresolved.

Carcinogenesis in the colon: Colorectal cancer typically develops through a multi-step adenoma-carcinoma sequence initiated by somatic mutations in key oncogenes and tumor suppressor genes, such as APC, KRAS, and TP53. The colorectal mucosa represents a dynamic tissue in continuous contact with gut microbiota, dietary antigens, and patrolling immune cells, making local tumor development highly dependent on host mucosal immunity (ZHANG et al., 2025). Antitumor immunosurveillance in colorectal tissue relies on infiltration by CD8+ CTLs, NK cells, and Th1 lymphocytes; tumors characterized by dense cytotoxic T-cell infiltration correlate with superior patient survival (GRISARU-TAL et al., 2020). Malignant tumors frequently escape immune elimination by downregulating surface MHC class I expression, upregulating PD-L1 immune checkpoint ligands, and recruiting immunosuppressive populations such as Tregs and myeloid-derived suppressor cells (GRISARU-TAL et al., 2020; ZHANG et al., 2025).

Acute vs. chronic inflammation: Inflammatory responses exhibit dual roles in oncology. Acute inflammation is a transient, self-limiting process that clears tissue injury and initiates resolution. In contrast, chronic unresolved inflammation creates persistent low-grade immune infiltration where ongoing tissue destruction and compensatory cellular proliferation promote tumorigenic mutations. Activated inflammatory macrophages and neutrophils release reactive oxygen and nitrogen species (ROS/RNS) capable of causing direct oxidative DNA damage in surrounding colonic epithelial cells, while pro-inflammatory cytokines such as TNF-α and IL-6 foster epithelial survival and proliferation, as demonstrated in inflammatory bowel disease-associated carcinogenesis (LIU et al., 2023; ZHANG et al., 2025). The net effect of mucosal inflammation depends heavily on its cellular and cytokine composition: Th1/IFN-γ-dominated responses lean towards effective tumor destruction, whereas persistent TNF-α/IL-6-driven pathways facilitate tumor development. Systemic Th2/eosinophil polarization associated with pollen hypersensitivity represents a distinct immunologic profile that differs from both classical pro-inflammatory bowel inflammation and pure Th1 cytotoxicity, necessitating a detailed synthesis of epidemiological and mechanistic literature.

MATERIAL AND METHODS

This article presents a non-exhaustive narrative synthesis of selected publications on allergic disease, eosinophils, and colorectal cancer. The original reference list was supplemented during revision through targeted searches and consultation of PubMed/PMC records, publisher pages, and DOI metadata, and publications were selected for topical relevance. No prespecified systematic-search protocol, formal risk-of-bias assessment, or new meta-analysis was undertaken.

References were included in the synthesis if they met at least one of the following relevance criteria: (i) epidemiological studies evaluating statistical associations between atopic conditions (hay fever, allergic rhinitis, asthma) and CRC risk, including prospective cohorts (TAMBE et al., 2015), meta-analyses (MA et al., 2017; YE et al., 2017), and nationwide registry studies (HEMMINKI et al., 2014); (ii) Mendelian randomization studies examining genetic liability to allergic diseases or eosinophil traits in relation to CRC risk (ALDUHAYH et al., 2025; WANG et al., 2024); (iii) clinical case-control studies of allergy and cancer (ARYAIE et al., 2024); and (iv) mechanistic studies and reviews of mucosal mast cells, Th2 cytokines, and tumor-infiltrating eosinophils in the colorectal microenvironment (GRISARU-TAL et al., 2020; LIU et al., 2023; MOLFETTA and PAOLINI, 2023). No systematic review protocol was applied, and no formal quality-appraisal or meta-analytic procedure was used; the reported estimates of each included study were compiled and interpreted narratively. The conclusions of this synthesis should therefore be regarded as a structured interpretation of the available literature rather than as a systematic evaluation.

RESULTS AND DISCUSSION

Epidemiological evidence: Large prospective investigations provide substantial evidence of an inverse relationship. The Multiethnic Cohort Study, which ascertained 4,834 incident colorectal cancer cases between 1993 and 2010, reported that atopic allergic conditions (asthma, hay fever, or allergy) were associated with a reduced risk of CRC incidence among both men and women (RR = 0.86, 95% CI 0.80-0.92); the reduction was statistically significant in whites, African Americans, Native Hawaiians, and Japanese Americans, but not in Latinos (TAMBE et al., 2015). A meta-analysis of 12 prospective studies including 515,379 participants and 10,345 CRC cases confirmed a similar inverse association for both colon cancer (pooled RR = 0.83, 95% CI 0.72-0.97) and rectal cancer (pooled RR = 0.83, 95% CI 0.74-0.93) (MA et al., 2017). Table 1 summarizes the key studies and their estimates.

Table 1

Summary of epidemiological and Mendelian randomization studies on allergic conditions and colorectal cancer risk

Study	Design	Exposure	Outcome	Main estimate (95% CI)
TAMBE et al., 2015	Prospective cohort (Multiethnic Cohort, USA; 4,834 cases)	Atopic allergic conditions (self-reported)	CRC incidence	RR 0.86 (0.80-0.92)
MA et al., 2017	Meta-analysis of 12 prospective studies (515,379 participants)	Any allergic condition	CRC incidence	RR 0.88 (0.83-0.92)
YE et al., 2017	Meta-analysis of 9 observational studies (775,178 individuals)	History of allergy	CRC incidence	RR 1.01 (0.88-1.17), no association
ALDUHAYH et al., 2025	Two-sample Mendelian randomization (52,775 CRC cases)	Genetic liability to allergic diseases (per log-odds unit)	Overall and early-onset CRC	OR 0.90 (0.85-0.96) overall; OR 0.83 (0.73-0.95) early-onset
WANG et al., 2024	Mendelian randomization	Genetically predicted eosinophil count	CRC risk	Inverse association; eosinophils mediate part of asthma effect
ARYAIE et al., 2024	Case-control study (115 CRC cases among 556 cancer patients, 1:1 ratio)	Environmental itching / urticaria	CRC	AOR 0.40 (0.17-0.94)

Conversely, a systematic review and meta-analysis of nine observational studies including 775,178 individuals observed no overall association between a general history of allergy and CRC risk (pooled adjusted RR = 1.01, 95% CI 0.88-1.17) (YE et al., 2017). A nationwide Swedish registry cohort of 138,723 patients with medically diagnosed hay fever or allergic rhinitis found that overall cancer risk was essentially unchanged (SIR = 1.03); this estimate covers all cancer sites and does not by itself indicate the presence or absence of a colorectal-specific association (HEMMINKI et al., 2014).

Mendelian randomization evidence: MR studies provide a complementary, genetically informed perspective. A recent two-sample MR analysis found that genetic liability to allergic diseases was inversely associated with overall CRC (OR = 0.90, 95% CI 0.85-0.96) and early-onset CRC (OR = 0.83, 95% CI 0.73-0.95, per unit increase in the log-odds of genetic liability); the inverse association was restricted to hay fever/allergic rhinitis and eczema, whereas asthma-related phenotypes showed no association (ALDUHAYH et al., 2025). Importantly, multivariable MR adjustment for eosinophil counts attenuated these associations (overall CRC: OR = 0.96, 95% CI 0.89-1.03; early-onset CRC: OR = 0.86, 95% CI 0.73-1.01), suggesting that eosinophils may partially mediate the relationship (ALDUHAYH et al., 2025). A separate MR analysis reported a significant inverse association between genetically predicted eosinophil count and CRC risk and indicated that eosinophils mediate part of the effect of asthma on CRC (WANG et al., 2024).

Regional and design differences have also been documented. Protective associations have been reported predominantly in North American cohorts, whereas European estimates have been closer to null (ALDUHAYH et al., 2025; HEMMINKI et al., 2014). A case-control study including 115 colorectal cancer patients among 556 cancer cases found a protective association

between environmental itching/urticaria and CRC (AOR = 0.40, 95% CI 0.17-0.94) and indicated that the timing of allergy onset may modulate risk estimates (ARYAIE et al., 2024).

Separately from incidence studies, histological investigations consistently show that tumor-infiltrating eosinophils represent a favorable prognostic biomarker in patients with established CRC, associated with improved overall survival and reduced recurrence (HARBAUM et al., 2015; NIELSEN et al., 1999; PRIZMENT et al., 2016). These prognostic associations are distinct from the question of cancer prevention: a better outcome among patients who already have CRC does not by itself demonstrate that allergy prevents tumor development.

Interpretation of findings: Findings for clinically defined allergic conditions remain heterogeneous. MA et al. (2017) reported an inverse association for allergic conditions overall (pooled RR = 0.88, 95% CI 0.83-0.92), but not a statistically significant hay-fever-specific association (pooled RR = 0.94, 95% CI 0.85-1.04). ALDUHAYH et al. (2025) reported inverse associations for genetic liability to hay fever/allergic rhinitis or eczema, whereas asthma-related phenotypes showed no association; these findings do not establish protection from clinically confirmed pollen allergy. This differentiation indicates that generic Th2 activation alone does not necessarily confer a lower risk. The MR attenuation after adjustment for eosinophil counts suggests, but does not prove, that eosinophils act as effector cells mediating part of the reduced CRC risk (ALDUHAYH et al., 2025; WANG et al., 2024).

Biological hypotheses: Biologically, allergic rhinitis involves IgE-associated type 2 responses, mast-cell activation, and blood eosinophilia (COLAS et al., 2022; PALM et al., 2012; PAWANKAR et al., 2011). Eosinophils are normally abundant in the gastrointestinal mucosa (GRISARU-TAL et al., 2020; ROTHENBERG and HOGAN, 2006), but whether pollen-induced airway responses alter eosinophil activity in colorectal tissue remains unresolved. In the tumor microenvironment, eosinophils are equipped to exert direct cytotoxic antitumor activity through the release of granule proteins, including major basic protein and eosinophil cationic protein (GRISARU-TAL et al., 2020), consistent with clinical data linking dense eosinophilic infiltration to favorable survival outcomes (HARBAUM et al., 2015; PRIZMENT et al., 2016). Mast cells, by contrast, appear to play a dual role in CRC, with both pro-tumor (angiogenic and lymphangiogenic) and anti-tumor activities described (LIU et al., 2023; MOLFETTA and PAOLINI, 2023). Alternative non-causal explanations, such as surveillance bias or reverse causation, remain plausible and cannot be excluded by observational data (ALDUHAYH et al., 2025; YE et al., 2017).

Limitations: Main study limitations include inconsistent allergy definitions across cohorts, heavy reliance on self-reported data, unexplained geographical differences between continents, and limited studies isolating pollen allergy specifically from other atopic diseases (TAMBE et al., 2015; YE et al., 2017). Indeed, allergic rhinitis or hay fever is not equivalent to biochemically confirmed pollen allergy, because these diagnoses comprise multiple aeroallergens. Furthermore, genetic liability to allergic disease reflects lifelong predisposition and is not identical to clinically manifest pollen hypersensitivity.

Future research directions: Future research should prioritize prospective cohorts directly measuring pollen-specific IgE titers and absolute eosinophil counts alongside screening colonoscopy findings. Mechanistic studies tracking sensitized eosinophil homing and local cytotoxic action within dysplastic gut tissue will be essential to establish direct causality.

CONCLUSIONS

Epidemiological cohort data suggest a modestly lower risk of colorectal cancer among individuals with atopic allergic conditions in general (MA et al., 2017; TAMBE et al., 2015), and

Mendelian randomization analyses indicate inverse associations for genetic liability to hay fever/allergic rhinitis or eczema, with stronger estimates for early-onset disease (ALDUHAYH et al., 2025). Findings for clinically defined allergic conditions remain heterogeneous, and none of these studies establishes protection from clinically confirmed pollen allergy; the evidence concerning pollen allergy specifically remains limited and largely indirect.

Mechanistically, Th2-driven eosinophil activation is a plausible mediator of this association: eosinophils are abundant in the gastrointestinal mucosa, exert cytotoxic antitumor effects in the colorectal microenvironment, and appear to mediate part of the genetic association between allergic disease and CRC risk (ALDUHAYH et al., 2025; GRISARU-TAL et al., 2020). However, the available data do not establish that pollen allergy directly protects against colorectal cancer.

Future prospective and mechanistic investigations isolating pollen hypersensitivity are necessary to validate these cellular pathways and to clarify whether the observed associations can be translated into targeted diagnostic or therapeutic strategies.

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