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Research Article

Colorectal Carcinoma and Parasitic Infections in the USA

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Abstract

We establish the relationship between Colorectal cancer (CRC) and intestinal and extra-intestinal parasites associated with pathologies such as inflammatory bowel disease (IBD). Periodic and long-term cases of cancer influence the changes into a number of neoplastic diseases. We have produced a number of epidemiologic parameters like inflammation, Crohn's disease, ulcerative colitis (UC), gut leakage, diarrhea, constipation, and inflammatory bowel disease (IBD) as well as fatigue, skin rash, and allergies as byproducts of intestinal parasite infections predisposing chronic diseases. We document the relationship between these parameters and neoplastic diseases such as colorectal cancer (CRC). More recent research demonstrated the chemical and molecular parameters relationship to the generation of CRC. The analysis of intestinal and extraintestinal parasites were performed upon the recovery of intestinal and tissue samples following routine parasitological examination. For CRC research, a thorough publication search was performed using key words such as CRC, inflammation, ulcerative colitis, and inflammatory bowel disease (IBD). Of the many ways by which humans can acquire parasites, water borne and food borne heavy parasite infections and pathologies appear to be causal agents of chronic diseases such as CRC. These are associated with nucleotide-binding oligomerization domain-containing protein 2 (NOD2) and toll like receptor (TLR) signaling pathways are clearly involved in the inflammatory process and are therefore implicated in the transformation of normal colonic mucosa to premalignant and malignant disease. The disturbance of intestinal flora might be involved in this process. Genetic and environmental factors appear to influence IBD and CRC. We conclude that a causal relationship was established between parasitic infections and metastasis causing CRC especially in heavy parasitic infections associated with such pathologies as IBS and ulcerative colitis (UC).

Keywords: *Parasites; Colorectal Cancer; Irritable Bowel Syndrome; Ulcerative Colitis*

Introduction

In 2020 there were 1.8M people diagnosed with cancer and 606K deaths in the USA. Colorectal cancer (CRC) was responsible for 53,200 deaths in 2020 and Irritable Bowel Disease (IBD) was responsible for 12.5% (6650) of those deaths according to Hnatyszyn., *et al*. [1].

Research on parasites and number of deaths they cause in Colorectal Cancer based on 2020-21 PCI research are 1. Prostate, 2. lung, 3. CRC for men and 1. breast, 2. lung, 3. CRC for women; see below.

USA Population	Diagnoses	Deaths	Cause (Men Top 1)	Cause (Men Top 2)	Cause (Men Top 3)	Cause (Women Top 1)	Cause (Women Top 2)	Cause (Women Top 3)	CRC deaths*
331M	1.8M	606K	Diet-hormonal	Smoking	IBD + other	Genetic, alcohol, other	Smoking	IBD + other	6650

*6650 CRC deaths from IBD; 40% of IBD caused by parasites; 2660 CRC deaths from parasites.

Table 1: *Cancer statistics and top causes in the USA in 2020.*

When looking at the impact of parasites on those numbers, we at the Parasitology Center, Inc, Scottsdale, Arizona (PCI) have analyzed the 3rd most common form of cancer (CRC) which has a direct correlation with parasites, Colorectal cancer (CRC).

Amin [2] has identified 8 major pathways by which humans can acquire parasitic infections. These include (1) Drinking water contaminated with fecal material from infected persons. This simple cycle occurs in water from running streams as well as from tap water in homes. Parasites transmitted in this manner include *Cryptosporidium* and *Giardia*. (2) Skin contact with contaminated water which is the only method of infection available to certain parasites such as the schistosomes, some of the deadliest fluke (trematode) parasites of mankind in Africa, China, Mexico, and Puerto Rico. (3) Food intake is a common way of transmission of parasitic infections caused by microscopic (Protozoa) and macroscopic (worm, helminth) parasites alike. Examples in the US include *Blastocystis* and the cysts of the amoebas (both are protozoans) are infective when swallowed with contaminated food via the fecal-oral route. Similarly, the ingestion of the eggs of *Ascaris*, readily occurs when fresh vegetables, e.g. lettuce, grown in farms fertilized with infected human waste, are eaten without proper washing. (4) Blood sucking insects are capable of transmitting infectious agents via their bite as they feed on human blood. In the US, ticks transmit Lyme disease, Rocky Mountain spotted fever, relapsing fever, Colorado tick fever, babesiosis, and rabbit fever; fleas transmit plague and endemic typhus, mosquitoes transmit malaria and dog heartworm, Triatoma (kissing) bugs transmit Chagas disease, and head lice can transmit epidemic typhus. (5) Air: Air-borne viruses, bacteria, and fungi are usually eliminated with the feces (occasionally orally) of a natural reservoir (usually wild life) host but infect humans upon accidental inhalation. Examples in North America include histoplasmosis, Valley fever, and Hanta virus. (6) Pets: Dogs carry an intestinal tapeworm, *Echinococcus*, swine carry tapeworms, *Taenia*. (7) People: Close human-to-human contact is conducive to transmission of quite an assortment of sexually transmitted diseases including AIDS and herpes as well as other viruses causing cold and flu. (8) Soil: Certain roundworm (nematode) parasites spend their transitional stages as immature larvae in warm moist soil and larvae of hookworms or *Strongyloides* will penetrate the exposed skin and migrate in the body to finally become adults in the intestinal tract where the damage is done.

Heavy infections of all the above-mentioned forms of parasites have shown a causal relationship with CRC. Compromised human tissues are forced to uncontrollably divide to compensate for damaged cells thus inducing metastasis. Water and food transmitted infections are notable in the initiation of this process.

Colorectal cancer (CRC) is the third most common malignancy and fourth leading cause of cancer-related mortality worldwide [1]. It is currently the leading cause of cancer mortality in men and the second leading cause in women under 50 in the US. Developed countries account for greater than 55% of all CRC cases [3,4]. Differences in the occurrence of CRC are over 10-fold: from the highest rates observed in Australia, New Zealand, Western Europe and the United States to the lowest in Africa and Central Asia [5,6]. There is a number of genomic, environmental and physiological factors that influence the development of CRC including chronic inflammation of the gastrointestinal tract [7-10] that we at PCI have documented based on intensity of tested parasitic infections and symptomology. In our PCI patient population, intensity of infection and symptomology varied with geography, patient susceptibility, and virulence of parasite species. For instance, patients from the north Indian subcontinent suffered almost incurable cases of virulent Blastocystis hominis infections making them "chronic cases" requiring a much longer course of treatment extending for months. Generally, patients with IBD and UC had an elevated risk of CRC with the incidence of CRC increasing by 60% [1,11].

Materials and Methods

The identification of intestinal and extraintestinal parasites were performed upon the recovery of intestinal and tissue samples following routine parasitological examination as explained in Amin [12,13]. For CRC research, a thorough publication search was performed using key words such as CRC, inflammation, ulcerative colitis, and inflammatory bowel disease (IBD).

Results and Discussion

Parasitological findings

Our PCI patient-based research has determined the % of IBD that was caused by parasites was 40%. Due to the lack of research published on parasites, we made the following assumptions.

Assumption #1: The most common 3 parasites (*Blastocystis hominis, Entamoeba histolytica*, *Cryptosporidium parvum*) make up 86% of all parasites in the USA. We are assuming that this 86% is representative of all parasites and therefore are using the symptoms of these 3 parasites for the purposes of this study.

Most common parasites	Total	Symptoms	None
Blastocystis	581	400	181
Cryptosporidium	90	63	27
E. histolytica	40	30	10
Total	711	493	218
All others	115	91	34
Reference	2000 Seasonal Prevalence article by Amin [12,13]		

Table 2: *Common parasites in the USA in 2000.*

Assumption #2: Since the IBD symptoms are the same as those produced by parasitic and bacterial infections, we assume that most IBD cases are caused by parasites and bacteria and to a lesser extent viruses. Comparability of symptoms suggest comparability of causes.

Most notable symptoms present	Diarrhea	Fatigue	Abdominal pain	Abdominal cramping	Blood in stool	Weight loss
IBD symptoms	x	x	x	x	x	x
Parasite symptoms	x	x	x	x	x	x
Virus symptoms	x		x	x		
Bacteria symptoms	x	x	x	x	x	x

Table 3: *Distribution of symptoms by causative agents.*

Assumption #3: Based on the similarity of symptoms between IBD and the 3 pathogens mentioned above, we suggest a weight of 40% of IBD cause to parasites and bacteria each and 20% to viruses.

Weight of the different pathogens affecting IBD are indicated	Parasites	Viruses	Bacteria
% IBD from:	40	20	40

Table 4: *Comparative distribution of IBD symptoms by causative agents.*

IBD-related CRC

A collection of multi-faceted environmental exposures complicated by genetic susceptibilities of unknown etiologies constitute the gastro-intestinal pathology of IBD and its concomitant CRC pathogenesis. IBD includes Crohn's disease (CD) and ulcerative colitis (UC) together bringing higher risk of developing CRC. Patients with chronic UC and CD are at a higher risk of developing CRC compared to the general population [11]. Although IBD-associated CRC accounts for approximately 2% of all CRC, the rate of death resulting from CRC in IBD patients ranged from 10 to 15% [14]. The risk of CRC is increased at least 2-fold in IBD patients with a positive family history of CRC compared to patients without a family history of Nuako., *et al.* [15] and Cremer., *et al*. [16]. Chronic inflammation has been documented in about 20% of all human cancers [1] with this relationship being most apparent in cases of CRC [17].

Cytokines

Disequilibrium in the production of Th1, Th2, and Th17 cytokines controls the immune response of the IBD chronic inflammation which promotes disease recurrence [1]. Extra-intestinal symptoms (carefully monitored in our PCI patient population) that accompany IBD usually reflect disorderly serum cytokine environment [18,19]. Cytokines secreted in response to infection and inflammation in the course of IBD may influence cancer initiation and progression. A pro-inflammatory cytokine is IL-6, which promotes tumor growth in human CRC cells [1]. Cytokines have a dual role during cancer development and may transform from an inhibitor to a promoter of disease [20].

Familial relationships

Patients with a first-degree relative diagnosed with CRC before 50 years of age had a higher relative risk (9.2, C.I. = 3.7 - 23.0) and the highest absolute risk (29%) of disease [21]. Patients with IBD have a 3- to 5-fold increased risk of CRC, and those with CRC in a first-degree relative had an almost 8-fold increase in disease risk. Apparently, family history may help identify individuals with IBD at highest risk for CRC and Samadder [22] recommended enhanced surveillance in this group of patients. Further research into families is needed where IBD patients originate, especially those who carry polymorphic variants in genes involved in the inflammatory response.

Microbiota

The intestinal microbiota, first line of defense against invading infections, is important in the pathogenesis of CRC by altering intestinal bacterial biofilms, homeostasis, and immune reaction. Microbiota imbalance promotes the development of IBD-associated CRC by initiating

and maintaining colon mucosal inflammation [23,24]. Disruption of homeostasis will promote inflammation in the large intestine, resulting in damage to the mucous membrane and subsequently, the development of neoplastic lesions. Pathogenic microorganisms, as we have demonstrated in the many PCI publications, will contribute to intestinal inflammation through toxin secretion, invasion, feeding activities, migration, or adherence [25-39]. Dysbiosis especially involving *E. coli* and *D. fragilis* [39] will promote CAC development predisposed to with tissue damage. Ensuing chronic inflammation is not always recognizable at the clinical level but it is definitely a major factor in the generation and development of CRC as it regulates inflammatory reaction.

Conclusion

We have studied intestinal and extra-intestinal parasitic infections nationally and internationally since 1994 and monitored their relationship with colorectal cancer (CRC), particularly in cases associated with chronic inflammatory conditions such as inflammatory bowel disease (IBD), ulcerative colitis (UC), and irritable bowel syndrome (IBS). The relationship between these pathologies and CRC appears to be of causal nature. Heavy parasitic loads, especially from prevalent protozoans like *Blastocystis hominis*, *Entamoeba histolytica*, and *Cryptosporidium parvum*, appear to contribute significantly to chronic gut inflammation, microbiota dysbiosis, and tissue damage that promote chronic inflammatory responses manifesting into cases of CRC, among others. The development of CRC is associated with neoplastic transformation through pathways involving NOD2 and TLR signaling, cytokine imbalance (e.g. elevated IL-6), and disruption of intestinal homeostasis.

Our analysis, drawing from long-term patient data at the Parasitology Center, Inc. (PCI) since 1994 and supported by literature on inflammation-driven carcinogenesis, indicates that approximately 40% of IBD cases may be attributable to parasitic infections. Since IBD contributes to about 12.5% of CRC deaths (approximately 6,650 in the USA in 2020), and applying the 40% attribution, parasites are linked to roughly 2,660 CRC deaths annually in the USA. Some epidemiological studies overlook reports of related genetic, dietary, or lifestyle factors.

The clinical symptoms of acute or chronic parasitic infections are comparable to those of IBD/CRC-related pathologies (e.g. diarrhea, abdominal pain, fatigue, blood in stool). These similarities suggest that parasites serve as underrecognized environmental triggers for chronic inflammation predisposing to malignancy. Waterborne and foodborne transmission remain primary routes of parasite acquisition in the USA. Improved diagnostics, prevention, and targeted antiparasitic interventions in high-risk populations are needed.

Our findings highlight the fact that parasites are modifiable risk factors in CRC pathogenesis irrespective of the role played by genetic and environmental cofactors. Further large-scale, prospective studies are warranted to validate these associations beyond clinic-based observations and assumptions regarding pathogen contributions. We have performed such long-term, large-scale studies in the Los Angeles area and across the continental USA. Enhanced surveillance for parasitic infections in patients with IBD or CRC could qualify preventive strategies and reduce the impact of CRC. Recognizing parasitic infections as a contributing etiology may shift paradigms in CRC prevention and management toward integrated parasitological screening and treatment.

Postscript

In confirmation of our findings while in press, the American cancer society's flagship journal CA: A cancer journal for Clinicians published the "Colorectal cancer statistics, 2026" by Siegel RL., *et al*. (March 02, 2026 https://doi.org/10.3322/caac.70067). Siegel., *et al.* (2026) declared that CRC is currently the leading cause of cancer mortality in people under 50 and that incidence and mortality have been increasing since 2023 and 2004 in the USA with the disease being diagnosed at more advanced stages. Data were drawn from the National Cancer Institute and the Centers for Disease Control and Prevention where the corresponding author used to work. Siegel., *et al*. (2026) estimated that there "will be 158,850 new cases of bowel cancer with over 55,000 aged 50-64 projected to die in 2026". The incidence was 32% higher in men than in women but declined from the peak rate of 1985. The authors established a variety of risk factors

for CRC including family history and inflammatory bowel disease as we have already proposed but also included alcohol consumption, obesity, diet, and smoking. Siegel., *et al.* (2026) and our research demonstrated that CRC screening will reduce mortality by preventing cancer through the identification and removal of precancerous factors including lesions and IBD-related parasites and pathogens for more effective preventive treatment. Siegel., *et al.* (2026) concluded that "many cases and deaths could be averted through increased efforts to achieve broad initiation of screening at ages 45 years (or earlier in individuals with a family history that increases risk), regular screening, and timely workup of individuals with positive non-colonoscopy test results. Risk can also be mitigated through… increased awareness of red flag symptoms for the disease (rectal bleeding, abdominal pain, diarrhea, iron-deficiency anemia) among clinicians and the general public to facilitate earlier detection and timely treatment".

Availability of Data and Material

Reference samples of parasites are available from PCI upon request.

Ethical Approval

All applicable institutional, national, and international guidelines for the care and potential use of animals were followed.

Disclaimer (Artificial Intelligence)

Authors hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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Authors' Contributions

This work was carried out in collaboration among all authors. All authors contributed significantly to the writing and research and approved the final version of the work.

Competing Interests

Authors have declared that no competing interests exist.

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