Hypothyroidism and Risk of Inflammatory Bowel Disease: Influence of Age and Race in a Global Cohort

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ABSTRACT

BACKGROUND: Thyroid and gastrointestinal autoimmune conditions may share overlapping immunological, genetic, and microbial pathways, but the relationship between hypothyroidism and inflammatory bowel disease (IBD) remains unclear.

METHODS: We conducted a retrospective cohort study using electronic health record data from 125 healthcare organizations within a global research network to examine the association between hypothyroidism and IBD in a large, racially diverse, multinational population, stratified by age and race. Adults aged 18 to 100 years with or without hypothyroidism were included. Those with IBD risk factors or prior diagnoses were excluded. The primary outcome was a new diagnosis of Crohn's disease or ulcerative colitis within five years after the hypothyroidism or control index date. Propensity score matching was performed 1:1 based on age and sex. Odds ratios were calculated to assess the association, stratified by race and age.

RESULTS: Among nearly 30 million individuals, patients with hypothyroidism had a significantly increased risk of developing IBD in African American and Asian populations, with odds ratios of 1.329 and 1.662, respectively. In Caucasians, risk increased only in individuals over age 50, while those under 50 showed a slight reduction. The strength and direction of association varied by age and race.

CONCLUSIONS: Hypothyroidism is associated with increased IBD risk primarily among older adults in African American, Asian, and Caucasian populations, while younger Caucasians showed a modest reduction in risk. These findings suggest a complex autoimmune relationship influenced by both age and race.

KEYWORDS: hypothyroidism; inflammatory bowel disease; autoimmune disease; electronic health records; race

INTRODUCTION

Inflammatory bowel disease (IBD) refers to chronic inflammatory conditions of the gastrointestinal (GI) tract, primarily Crohn's disease (CD) and ulcerative colitis (UC). CD is characterized by transmural inflammation and skip lesions that can affect any part of the GI tract, typically presenting with colicky abdominal pain, weight loss, and watery diarrhea. Complications include strictures and fistulas. In contrast, UC involves continuous mucosal inflammation limited to the colon and rectum, often causing bloody diarrhea, tenesmus, and systemic symptoms such as fever and weight loss.[1,2]

Globally, IBD prevalence increased from 3.7 million in 1990 to nearly 7 million in 2017, with age-standardized rates rising from 79.5 to 84.3 per 100,000.[3] Given its typical onset between ages 15 and 30 and chronic nature, IBD imposes substantial costs—estimated at $9,000–12,000 per person in high-income countries and approximately $50 billion annually in the United States (U.S.).[4]

Given the rising global burden of IBD, numerous studies have explored its pathogenesis and risk factors. Although its exact etiology remains unclear, IBD is thought to result from a complex interplay of environmental, genetic, immunological, and microbial factors. However, few studies have specifically examined the link between hypothyroidism and IBD.

Hypothyroidism is a chronic thyroid disorder marked by deficient production of thyroxine (T4) and triiodothyronine (T3). Its prevalence in the U.S. is estimated at 3–7%, with similar upper-range estimates in Europe and parts of Asia. An additional 5% may have undiagnosed overt hypothyroidism. Globally, iodine deficiency is the leading cause of thyroid dysfunction, while in iodine-sufficient regions, Hashimoto's thyroiditis is most common.[5] Hypothyroidism is also associated with gastrointestinal symptoms such as constipation, dyspepsia, and delayed gastric emptying due to reduced GI motility.[6]

Given the importance of thyroid hormones on the gut and incompletely understood etiology of IBD, this paper examines the associated risks of developing IBD in patients with a history of hypothyroidism stratified by race and age. Here, we utilize the TriNetX Global Collaborative Network database, one of the most comprehensive global databases, to perform a retrospective cohort study to investigate this association.

MATERIALS AND METHODS

Data Source

We utilized a comprehensive, de-identified global federated health research network with data sourced from the TriNetX Global Collaborative Network, encompassing 125 healthcare organizations (HCOs) (data accessed in July, 2024). TriNetX continuously aggregates clinical data directly from participating HCOs, ensuring rigorous data quality and accuracy assessment. TriNetX does not provide identifiers for participating HCOs; typically, these organizations include large academic health centers offering inpatient, outpatient, and specialty care services.

Patient and Data Selection

We included all adult patients (aged 18–100 years) from the 125 HCOs globally in the TriNetX Global Collaborative Network, recording their age, sex, and race. Due to the large sample size exceeding the platform's computational limits, analyses were stratified by race—Caucasians (Whites), African Americans or Blacks (Blacks), and Asians (Asians). [**Figure 1**].

To mitigate confounding, we excluded patients with certain known or suspected risk factors for IBD, which could not be adequately adjusted for using multivariable regression or high-dimensional propensity score matching, as these were not supported on the platform at the time of analysis. Specifically, patients were excluded if they had a history of: mood disorders, anxiety, stress-related and other nonpsychotic mental disorders (ICD-10-CM codes F30–F39, F40–F48), vitamin D deficiency (E55), nicotine dependence (F17), or gastrointestinal cancers (ICD-10-CM: Z85.01–Z85.04, Z85.06; ICD-9-CM: V10.0, V10.09).

These conditions were selected based on their established or hypothesized associations with IBD in the literature and were identified using ICD-10/ICD-9 codes that are routinely mapped in the TriNetX platform. While TriNetX does not independently validate individual coding accuracy across its federated network, these diagnostic codes are derived from electronic health records of contributing institutions and have been used in numerous peer-reviewed studies leveraging this database.

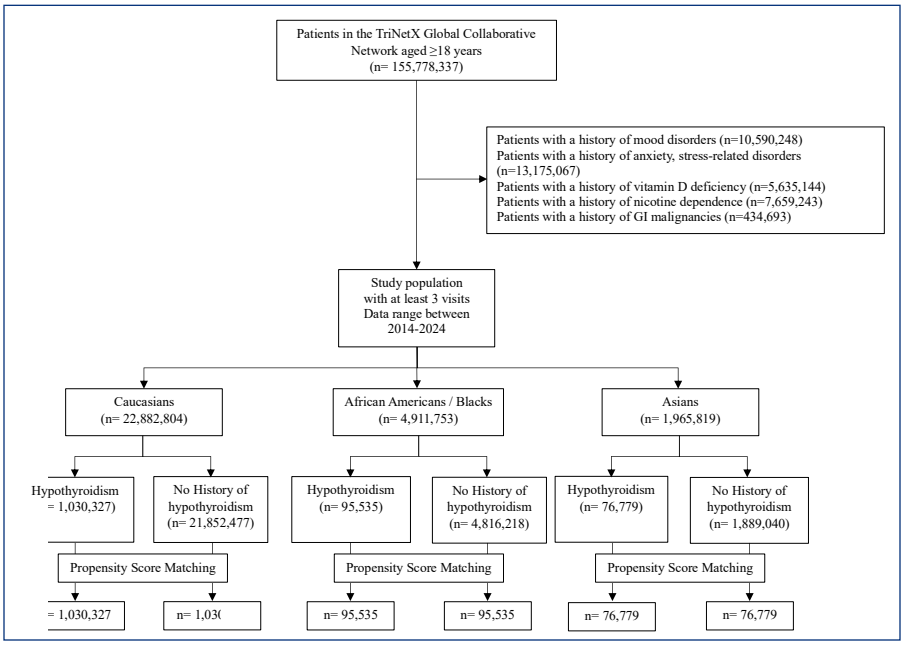
Hypothyroidism

Patients with a diagnosis of hypothyroidism (ICD 10-CM codes: E01-E03, E06.3) between July 1, 2014, and July 1, 2024, documented in three or more visits, were identified. Only patients aged 18 years and older at the time of their first hypothyroidism diagnosis were included in the analysis. Patients meeting the selection criteria but without a hypothyroidism diagnosis served as the control group.

Inflammatory Bowel Disease

Patients diagnosed with Crohn's disease (ICD 10-CM code: K50) or ulcerative colitis (ICD 10-CM code: K51) within five years after the diagnosis of hypothyroidism or the index date in the control group were identified as the outcome of interest.

Figure 1. Flowchart of cohort selection and matching. Adult patients aged 18 years or older with at least three clinical visits between 2014 and 2024 were identified from the TriNetX Global Collaborative Network. Exclusions included patients with prior mood or anxiety disorders, vitamin D deficiency, nicotine dependence, or gastrointestinal malignancies. Eligible patients were stratified by race (Caucasians, African Americans or Blacks, and Asians) and by hypothyroidism status. Propensity score matching was performed 1:1 by age and sex within each racial group.



STATISTICAL ANALYSES

Given the substantial sample size differences between cohorts (control: Whites, n=21,852,477, Blacks, n= 4,816,218, Asians, n=1,889,040; hypothyroidism: Whites, n=1,030,327, Blacks, n=95,535, Asians, n=76,779) and to account for potential confounders, we performed 1:1 propensity score matching without replacement based on age at index and sex. This method generates matched samples, balancing the distribution of observed characteristics between the hypothyroidism and control groups, thereby reducing the confounding effect of these factors and isolating the true effect of hypothyroidism on the study outcomes. Patients with missing data for any variables used in propensity score matching (e.g., age, sex, race in stratified analyses)

were automatically excluded from matching and subsequent analyses by the TriNetX platform. This ensures that only complete cases were included in the matched cohorts.

We examined differences in baseline characteristics before and after matching using standardized mean differences (SMDs), with an SMD less than 0.1 considered indicative of adequate covariate balance between groups. For descriptive purposes, continuous variables were summarized as means with standard deviations and categorical variables as frequencies with percentages. Outcome differences were evaluated using odds ratios (ORs) and 95% confidence intervals (CIs). All statistical tests were two-tailed, and a p-value of less than 0.05 was considered statistically significant. All analyses were performed within the TriNetX integrated analytics environment, which utilizes Java 11.0.16, R version 4.0.2, and Python 3.7 with relevant statistical libraries. The platform's analytic engine is proprietary and its underlying code is not publicly available.

To evaluate the robustness of our findings against potential unmeasured confounding, we calculated E-values for the observed associations between hypothyroidism and IBD. The E-value quantifies the minimum strength of association that an unmeasured confounder would need to have with both the exposure and the outcome to fully explain away the observed association. Additionally, we assessed consistency of the association across multiple racial and age-stratified subgroups to strengthen causal inference through triangulation of evidence.

Ethical Considerations

Institutional Review Board (IRB) approval was obtained (Taichung Veterans General Hospital IRB# CE24249A). This retrospective study is exempt from informed consent. The data reviewed is a secondary analysis of existing data, does not involve intervention or interaction with human subjects, and is de-identified per the de-identification standard defined in Section §164.514(a) of the HIPAA Privacy Rule.

RESULTS

A total of 29,760,376 patients were included for analysis: 22,882,804 Caucasians (Whites), 4,911,753 African Americans or Blacks (Blacks), and 1,965,819 Asians (Asians). Prior to propensity score matching (PSM), the mean age for hypothyroidism and control groups was 59.5 and 47.8 years (p<0.001) in Whites, 56.9 and 43.0 years (p<0.001) in Blacks, and 54.1 and 45.3 years (p<0.001) in Asians. Among Whites, 71.4% vs. 53.5% were female, 76.3% vs. 58.5% in Blacks, and 75.8% vs. 56.5% in Asians (all p<0.001) in the hypothyroidism and control groups, respectively [**Table 1**].

After PSM, the mean age was 59.5 years in Whites, 56.9 years in Blacks, and 54.1 years in Asians for both groups, with 71.4% female in Whites, 76.3% female in Blacks, and 75.8% female in Asians. The mean follow-up durations post-matching were 877.9 days for the hypothyroidism group and 866.7 days in the control group in Whites; 817.5 days and 836.4 days in Blacks; 845.9 days and 871.0 days in Asians [**Table 1**].

Table 1. Baseline demographics of hypothyroidism and control groups before and after propensity score matching, stratified by race

Caucasians	**Pre-matching**			**Post-matching**		
	Hypothyroidism (n=1,030,327)	Control (n=21,852,477)	SMD	Hypothyroidism (n=1,030,327)	Control (n=1,030,327)	SMD
Age, mean (SD), yr	59.5 (17.0)	47.8 (18.4)	0.661	59.5 (17.0)	59.5 (17.0)	<0.001
Sex, n (%)						
Female	736,140 (71.4)	11,073,454 (53.5)	0.378	736,140 (71.4)	736,140 (71.4)	<0.001
Male	293,020 (28.4)	9,614,097 (46.4)	0.378	293,020 (28.4)	293,020 (28.4)	<0.001
African Americans/Blacks	**Pre-matching**			**Post-matching**		
	Hypothyroidism (n=95,535)	Control (n=4,816,218)	SMD	Hypothyroidism (n=95,535)	Control (n=95,535)	SMD
Age, mean (SD), yr	56.9 (16.7)	43.0 (17.4)	0.813	56.9 (16.7)	56.9 (16.7)	<0.001
Sex, n (%)						
Female	72,892 (76.3)	2,690,578 (58.5)	0.387	72,892 (76.3)	72,892 (76.3)	<0.001
Male	22,577 (23.6)	1,904,245 (41.4)	0.386	22,577 (23.6)	22,577 (23.6)	<0.001
Asians	**Pre-matching**			**Post-matching**		
	Hypothyroidism (n=76,779)	Control (n=1,889,040)	SMD	Hypothyroidism (n=76,779)	Control (n=76,779)	SMD
Age, mean (SD), yr	54.1 (17.9)	45.3 (17.6)	0.496	54.1 (17.9)	54.1 (17.9)	<0.001
Sex, n (%)						
Female	58,229 (75.8)	998,341 (56.5)	0.418	58,229 (75.8)	58,229 (75.8)	<0.001
Male	18,536 (24.1)	767,992 (43.5)	0.417	18,536 (24.1)	18,536 (24.1)	<0.001

SD: standard deviation; SMD: standardized mean difference

Table 2. Associations between Hypothyroidism and Inflammatory Bowel Disease in Different Racial Groups

Caucasians	Hypothyroidism	Control	OR (95% CI)	*p* value
IBD, n (%)	3,682 (0.3)	4,185 (0.4)	0.886 (0.847, 0.926)	<0.001
Ulcerative colitis, n (%)	2,467 (0.2)	2,326 (0.2)	1.065 (1.007, 1.127)	0.029
Crohn's disease, n (%)	1,309 (0.1)	1,796 (0.2)	0.731 (0.681, 0.785)	<0.001
African Americans/Blacks	**Hypothyroidism**	**Control**	**OR (95% CI)**	***p* value**
IBD, n (%)	271 (0.3)	205 (0.2)	1.329 (1.108, 1.593)	0.002
Ulcerative colitis, n (%)	190 (0.2)	101 (0.1)	1.887 (1.482, 2.403)	<0.001
Crohn's disease, n (%)	109 (0.1)	78 (0.1)	1.400 (1.047, 1.873)	0.023
Asians	**Hypothyroidism**	**Control**	**OR (95% CI)**	***p* value**
IBD, n (%)	205 (0.3)	124 (0.2)	1.662 (1.330, 2.078)	<0.001
Ulcerative colitis, n (%)	145 (0.2)	79 (0.1)	1.843 (1.401, 2.425)	<0.001
Crohn's disease, n (%)	58 (0.1)	46 (0.1)	1.263 (0.858, 1.860)	0.236

All results were calculated after propensity score matching. CI: confidence interval; SD: standard deviation

In Whites, 3,682 (0.3%) in the hypothyroidism group and 4,185 (0.4%) in the control group had IBD, with an OR of 0.886 (p<0.001). In Blacks, 271 (0.3%) in the hypothyroidism group and 205 (0.2%) in the control group had IBD, yielding an OR of 1.329 (p=0.002). In Asians, 205 (0.3%) in the hypothyroidism group and 124 (0.2%) in the control group had IBD, with an OR of 1.662 (p<0.001) [**Table 2**].

Table 3. Associations between Hypothyroidism and Subtypes of Inflammatory Bowel Disease in Different Age Groups

	Hypothyroidism vs control	OR (95% CI)	*p* value
Caucasians			
IBD	18 ≤ Age < 50 (yr)	0.891 (0.809, 0.980)	0.018
	50 ≤ Age ≤ 100 (yr)	1.229 (1.161, 1.301)	<0.001
UC	18 ≤ Age < 50 (yr)	0.886 (0.783, 1.002)	0.054
	50 ≤ Age ≤ 100 (yr)	1.178 (1.106, 1.255)	<0.001
CD	18 ≤ Age < 50 (yr)	0.607 (0.529, 0.696)	<0.001
	50 ≤ Age ≤ 100 (yr)	0.769 (0.708, 0.835)	<0.001
African Americans/Blacks			
IBD	18 ≤ Age < 50 (yr)	0.962 (0.644, 1.435)	0.848
	50 ≤ Age ≤ 100 (yr)	1.646 (1.291, 2.099)	<0.001
UC	18 ≤ Age < 50 (yr)	1.622 (1.057, 2.488)	0.025
	50 ≤ Age ≤ 100 (yr)	2.317 (1.731, 3.100)	<0.001
CD	18 ≤ Age < 50 (yr)	0.893 (0.558, 1.428)	0.636
	50 ≤ Age ≤ 100 (yr)	1.692 (1.184, 2.418)	0.004
Asians			
IBD	18 ≤ Age < 50 (yr)	0.886 (0.596, 1.319)	0.552
	50 ≤ Age ≤ 100 (yr)	1.642 (1.251, 2.155)	<0.001
UC	18 ≤ Age < 50 (yr)	0.801 (0.503, 1.276)	0.349
	50 ≤ Age ≤ 100 (yr)	1.549 (1.179, 2.036)	0.002
CD	18 ≤ Age < 50 (yr)	0.731 (0.445, 1.200)	0.213
	50 ≤ Age ≤ 100 (yr)	1.364 (0.887, 2.098)	0.156

A subgroup analysis was conducted to examine the association across different age groups, using 50 years as the cut-off. Among White patients, those diagnosed with hypothyroidism before the age of 50 had a lower likelihood of developing inflammatory bowel disease (IBD) (OR 0.891, p=0.018). Conversely, for those aged 50 years and older with hypothyroidism, there was a significantly increased risk of IBD in Whites (OR 1.229, p<0.001), Blacks (OR 1.646, p<0.001), and Asians (OR 1.642, p<0.001) [**Table 3**].

To assess the robustness of our findings against potential unmeasured confounding, we calculated E-values for the observed associations between hypothyroidism and IBD. Among Asians, the observed odds ratio (OR=1.662) corresponded to an E-value of 2.71, indicating that an unmeasured confounder would need to be associated with both hypothyroidism and IBD by a risk ratio of at least 2.71 each, above and beyond the measured covariates, to explain away the observed association. The lower bound of the 95% confidence interval (CI=1.330) yielded an E-value of 1.99. Similarly, for Blacks (OR=1.329), the E-value was 1.99 (lower CI bound=1.454), and for Whites aged ≥50 years (OR=1.229), the E-value was 1.76 (lower CI bound=1.593). These E-values suggest that only unmeasured confounders with relatively strong associations would be capable of fully accounting for the observed effects.

DISCUSSION

In this large, multinational cohort study of nearly 30 million patients, hypothyroidism was significantly associated with increased risk of IBD among African American and Asian individuals, and among Caucasians aged 50 years and older. In contrast, younger Caucasians with hypothyroidism showed a modest reduction in IBD risk. These findings highlight a complex, age- and race-dependent relationship between hypothyroidism and IBD.

Hypothyroidism is linked to alterations in gut microbiome, including reduced bacterial diversity, increased in pro-inflammatory species, and slowed gut motility. Growing evidence that gut microbiota influences thyroid hormone synthesis, conjugate hydrolysis, and immune modulation, potentially impacting autoimmune thyroid diseases, particularly Hashimoto's thyroiditis.[7,8] Additionally, short-chain fatty acids produced by gut bacteria can influence the conversion of T4 to T3.[9] Microbial metabolites may impact thyroid malignancies by affecting DNA damage, apoptosis, and chronic inflammation.[8] Conversely, studies have also shown that thyroid function can affect intestinal microbial

population, where overt hypothyroidism is associated with a greater chance of bacterial overgrowth development.[10] The complex bidirectional relationship of the thyroid-gut axis highlights the need for further investigation into their interaction and its potential therapeutic implications for thyroid-related disorders.

Hashimoto's thyroiditis, the most common type of hypothyroidism, and IBD are autoimmune diseases, suggesting a common underlying immune dysregulation. Many complications of IBD are extra-intestinal and affect other organ systems, such as dermatologic, musculoskeletal, ocular, renal, and pulmonary systems.[11] Therefore, it seems reasonable to conclude a link between thyroid disorders and IBD. However, previous literature had inconclusive results regarding the relationship between thyroid disorders and IBD. A 2016 review of 46 total cases of thyroid disorders and IBD found no difference in the prevalence of the diseases.[12] One study suggests an inverse relationship between IBD and thyroid disorders, while another suggests an increased risk of developing Graves' disease (GD) with Crohn's disease, but UC might reduce the likelihood of developing GD.[13,14] A 2018 study concluded that patients with congenital hypothyroidism have an increased risk of developing IBD, and another suggested a significant association between UC and thyroid disorders, particularly simple goiter and hyperthyroidism.[15,16] These conflicting findings highlight the complexity of the relationship between IBD and thyroid disorders, suggesting that different mechanisms may be at play depending on the specific subtype of IBD and the type of thyroid dysfunction

Hypothyroidism leads to systemic low-grade inflammation, which could exacerbate gut inflammation and contribute to IBD. Both hyperthyroidism and hypothyroidism are associated with oxidative stress and dual oxidases (DUOX), which are essential for thyroid peroxidase-catalyzed hormone synthesis.[17,18] Loss-of-function mutations in DUOX2 are common genetic sources of congenital hypothyroidism that has been linked to an increased risk of developing IBD.[16] DUOX2 also plays a crucial role in the barrier epithelial cells of the gastrointestinal tract, helping to defend against potential microbial threats, such as limiting *Helicobacter* colonization.[19] This dual role of DUOX2 in both thyroid hormone synthesis and gastrointestinal epithelial defense suggests a potential link between thyroid disorders and inflammatory bowel diseases.

Additionally, Grasberger et al have shown an association between loss-of-function DUOX2 variant mice and increased plasma levels of interleukin-17C, a proinflammatory cytokine released by activated T cells, which has been associated with exacerbating intestinal inflammation and IBD.[20,21]

Other genes play a crucial role in the development of both thyroid disorders and IBD, with several being implicated in the pathogenesis of both conditions. For example, cytotoxic T-lymphocyte-associated protein 4 (CTLA-4) has been associated with increased susceptibility to thyroid autoimmunity.[22] CTLA-4 is an immune checkpoint regulator that influences T-cell activation, and its polymorphisms have been linked to several autoimmune diseases, including hypothyroidism and IBD.[23] Genetic variations can influence immune system regulation, making individuals more susceptible to developing autoimmune diseases, such as hypothyroidism and IBD. They can help explain the overlap between thyroid disorders and IBD. One study by Wu et al utilized genome-wide association studies to determine that elevated levels of TSH found in hypothyroidism were associated with reduced risk of CD, with the interferon-inducible protein-10 (IP-10) being linked as one of the main suspected causal factors.[24] This is consistent with the decreased odds ratio for Caucasians for all age groups in our dataset.

Subgroup analysis showed that age at hypothyroidism diagnosis significantly influenced IBD risk. Among Caucasians, those diagnosed before age 50 had a slightly reduced risk, while those diagnosed at 50 or older had a significantly increased risk. Similarly, older African American and Asian patients exhibited markedly elevated IBD risk. These findings suggest that later-onset hypothyroidism may reflect greater immune dysregulation or cumulative inflammation, while earlier-onset cases may involve different mechanisms or benefit from earlier management. This age-dependent pattern highlights the complex interplay between thyroid function, immunity, and gut inflammation.

To further evaluate the potential impact of unmeasured confounding on our findings, we performed E-value analysis. The results indicated that the observed associations, particularly those in Asians and older African Americans, were relatively robust to unmeasured confounding. For example, the association between hypothyroidism and IBD in Asians (OR=1.662) had an E-value of 2.71, and even the lower limit of the confidence interval (1.330) corresponded to an E-value of 1.99. These thresholds imply that an unmeasured confounder would need to have a strong, dual association with both exposure and outcome to nullify the observed relationship. Given the rigor of our inclusion criteria and exclusion of known IBD risk factors, it is unlikely that residual confounding alone could explain these associations. The use of E-value analysis adds quantitative support to the robustness of our findings.

Strengths and limitations

This study benefits from a large, ethnically diverse population drawn from the TriNetX Global Collaborative Network, supporting the generalizability of findings across multiple healthcare settings. The application of rigorous propensity score matching enhanced internal validity by reducing confounding related to demographic characteristics, and the requirement for hypothyroidism to be documented at three or more separate clinical visits likely improved diagnostic specificity.

Despite these strengths, several limitations should be acknowledged. First, as an observational study, causality between hypothyroidism and IBD cannot be definitively

inferred. Although statistical matching and exclusion criteria were applied, residual confounding may persist due to unmeasured variables such as family history of IBD, medication use (including levothyroxine), and socioeconomic status, which were not available on the TriNetX platform for matching at the time of analysis. For medication use specifically, we were unable to ensure compliance, account for dose adjustments, or determine the adequacy of disease management; therefore, these variables were not included. To address the possibility of unmeasured confounding, we performed E-value analysis, which suggested that only confounders with relatively strong associations with both hypothyroidism and IBD would be capable of fully explaining away the observed associations. Second, the reliance on ICD-10-CM codes for disease identification introduces potential for misclassification bias. While our definition of hypothyroidism required multiple diagnostic codes to increase specificity, the same robustness could not be applied to IBD as biopsy confirmation was not available. However, prior validation work in U.S. Veterans Health Administration datasets found that using ICD-10-CM codes for Crohn's disease (K50) or ulcerative colitis (K51), with at least one outpatient encounter, achieved a positive predictive value of approximately 89% for overall IBD, with especially high concordance for ulcerative colitis (~94%) and moderate concordance for Crohn's disease (~80%).[25] Third, missing data were handled using a complete-case approach inherent to the TriNetX platform: patients with missing values for matching or stratification variables (e.g., age, sex, race) were automatically excluded from propensity score matching and subsequent analyses. This ensures that all included cases had complete information, but may reduce sample size and could introduce selection bias if data were not missing at random. Finally, the actual duration of hypothyroidism prior to diagnosis could not be reliably determined. Many patients may have had subclinical or undiagnosed hypothyroidism for years before formal documentation in the EHR, which could affect the timing and magnitude of observed associations.

CONCLUSION

In this large, multinational cohort study, hypothyroidism was associated with an increased risk of IBD, particularly among older adults in African American, Asian, and Caucasian populations. In contrast, younger Caucasians with hypothyroidism showed a modestly reduced risk. These findings suggest an age- and race-dependent relationship between thyroid dysfunction and IBD, potentially driven by shared autoimmune, genetic, or microbial mechanisms. Further research is needed to clarify causality and explore whether early management of hypothyroidism could influence IBD risk.

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Disclosures

Funding: None

Conflict of Interest Disclosures (includes financial disclosures): The authors have no conflicts of interest relevant to this article to disclose.

Ethics approval: Institutional Review Board (IRB) approval was obtained (Taichung Veterans General Hospital IRB# CE24249A). This retrospective study is exempt from informed consent. The data reviewed is a secondary analysis of existing data, does not involve intervention or interaction with human subjects, and is de-identified per the de-identification standard defined in Section §164.514(a) of the HIPAA Privacy Rule.

Prior presentations: None.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author, C-H W. The data are not publicly available due to their containing information that could compromise the privacy of research participants.

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