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EDITORIAL

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REVIEW

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ORIGINAL ARTICLE**Case Control Study**

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Retrospective Cohort Study

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Retrospective Study

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Obesity and colorectal cancer risk: A systematic review and meta-analysis

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Abstract

BACKGROUND

An observed correlation between increased colorectal cancer (CRC) incidence in patients with obesity [body mass index (BMI) ≥ 30 kg/m²] has been identified in past literature. However, there has been limited data in recent decades. This, along with a dramatic global increase in obesity rates, exposure to environmental and lifestyle risk factors for CRC development, and large updates to the proposed biological mechanisms underpinning this relationship, warrants an updated review of recent data between CRC and obesity.

AIM

To determine if an updated correlation exists between obesity and the risk of CRC development.

METHODS

We evaluated recent data, synthesising pooled estimate effects to determine if an updated correlation exists between obesity and CRC. Observational studies were identified from a range of databases (PubMed, EMBASE, Scopus and the Cochrane database). From the studies identified, sex-stratified meta-analyses were conducted. Additionally, studies included in this review that were unfit for meta-analysis underwent qualitative analysis.

RESULTS

In a pooled sample size of 83506 male participants obtained from six observational studies, a significant positive correlation between obesity and CRC incidence was identified with a hazard ratio (HR) of 1.71 [95% confidence interval

(CI): 1.44-2.02]. A pooled sample size of 152043 female participants from six observational studies also revealed a positive correlation with an HR effect of 1.26 (95%CI: 1.03-1.53). Qualitative analysis of studies not included in the meta-analysis consistently supported this relationship for both sexes.

CONCLUSION

Obesity, diagnosed by a BMI ≥ 30 kg/m², significantly increases the risk of CRC incidence compared to those of a healthy BMI underscoring the importance of focused strategies to prevent obesity as a modifiable risk factor to reduce CRC incidence.

Key Words: Body mass index; Colorectal cancer; Meta-analysis; Obesity; Systematic review

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Core Tip: This systematic review and meta-analysis identify obesity as a modifiable risk factor for colorectal cancer (CRC) in both men and women. The risk is further heightened by associated lifestyle factors such as poor diet, physical inactivity, and the presence of comorbidities, including diabetes. These elements contribute synergistically to the development of CRC, highlighting the importance of recognising obesity not just as a health concern but as a cancer-promoting condition. Implementing earlier and targeted screening strategies for CRC in obese individuals can play a key role in reducing incidence and improving outcomes through earlier detection and intervention.

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INTRODUCTION

The obesity epidemic remains one of the most significant public health concerns of this century. Being a complex condition with significant social and psychological dimensions, obesity impacts all ages and socioeconomic groups, affecting almost 1 billion individuals globally[1]. Since 1975, rates of obesity have increased disproportionately to population growth[2]. This past decade, despite an increased recognition of obesity's global health and burden[3], obesity rates continue to grow dramatically, extending to even the most already obese-prevalent societies[4].

Obesity is closely associated with a plethora of comorbidities, exacerbating its impact on health and quality of life. Comorbidities include, but are not limited to, coronary heart disease, type 2 diabetes mellitus (T2DM), chronic kidney disease, metabolic syndrome, and cancer[4,5]. Of particular concern, obesity has been observed as a leading risk factor for various cancers in the colon, rectum, breast, and oesophagus[6,7].

Colorectal cancer (CRC) has the third highest incidence rate and second highest mortality rate out of all known cancers [6]. The aetiology of CRC is multifaceted, with extensive research suggesting cellular mechanisms linking obesity to CRC, including inflammatory dysregulation, altered gut microbiome, and insulin and insulin-like growth factor 1 (IGF-1) hypersecretion[8-10]. Obesity, a well-documented risk factor, is closely associated with pro-inflammatory diets and sedentary lifestyles, further exacerbating these pathways[8,11,12].

Previous reviews investigating the relationship between obesity and CRC provide strong corroborative evidence. Ning and colleagues found that from 56 observational studies, obese individuals, defined as a body mass index (BMI) of ≥ 30 kg/m², had a 41% increased risk of developing CRC compared to those within the healthy BMI range[13]. Moghaddam *et al's* study[14] also suggested that, from 31 studies, obese males (≥ 30 kg/m²) possessed a 1.41 increased risk of CRC [95% confidence interval (CI): 1.30-1.51] compared to healthy weight males, while obese females displayed no differences. With global obesity rates exceeding 16% among adults and 8% among children and adolescents, affecting approximately 878 million and 128 million individuals, respectively, the relationship between obesity and CRC raises serious concerns, suggesting a potential surge in CRC cases in the coming years[15].

Of the previously identified systematic reviews, studies published up to February 2008 were used to evaluate the relationship between obesity and CRC[13]. However, global obesity rates since 2008 have increased exponentially, particularly in low- and middle-income countries[16]. This, along with a global increased exposure to environmental and lifestyle risk factors for CRC development, indicates that further research is required to explore the association between obesity and CRC. Additionally, substantial advances have been made in understanding the biological mechanisms linking obesity to CRC. These developments highlight the need for updated research to reassess this association in the context of current epidemiological trends and mechanistic insights.

Therefore, this study aims to evaluate recent data to determine if an updated correlation exists between obesity and CRC, considering the rising prevalence of obesity and evolving risk factors while exploring the underlying molecular mechanisms driving these associations.

MATERIALS AND METHODS

This systematic review and meta-analysis are reported according to the PRISMA guidelines[17].

Search strategy

Studies were sourced from PubMed, EMBASE, Scopus and the Cochrane database by two independent investigators (Leung LJC and Sharma RS), using key terms including: Obesity, BMI, CRC, colorectal carcinoma, bowel cancer and registry. Additional studies were also obtained *via* snowballing, identifying papers of interest from the reference list of both included papers. Investigated studies were published between March 2008 and August 2024. The search terms used for each database can be found in the [Supplementary Table 1](#).

Eligibility criteria

The inclusion criteria: (1) Observational studies (cross-sectional, cohort, and case-control) investigating the relationship between obesity and CRC or colon cancer; (2) Obesity patient categorisation follows the WHO obesity diagnostic criteria (BMI ≥ 30 kg/m² internationally (17), BMI ≥ 25 kg/m² for individuals of Asian-Pacific descent[18]); (3) Cases of CRC or colon cancer are received from registry data; (4) odds ratios (OR), hazard ratios (HR) or relative risks (RR) with 95%CI were presented; (5) Includes a reference group who possessed a healthy BMI (internationally, 18.5-24.9 kg/m²; Asian-pacific descent, 18.5-22.9 kg/m²)[18,19]; and (6) Studies published in English.

The exclusion criteria: (1) Case-reports, review-articles, opinion pieces, non-English studies, grey literature and unpublished papers; (2) Studies assessing risk of obesity within a defined CRC patient cohort; (3) Studies where obesity is not clearly defined or do not match the WHO guidelines of obesity; and (4) Studies published prior to March 2008.

Data extraction

Following full-text review, the remaining studies underwent data extraction by two independent reviewers (Leung LJC and Sharma RS), which included: Author names, publication date, data registry source, obesity definition utilised, sample size, sex proportion, age range (including average), follow-up duration (years) and adjusted covariates. For the topic of this review, the term 'sex' was defined as the biological attributes associated with the chromosomal genotype of the patient and was recorded based on self-report of the patient. The incidence ratios, such as HR, OR and RR were also obtained along with the 95%CI.

Quality assessment

The Newcastle-Ottawa Scale (NOS) is a quality assessment tool used for non-randomised studies in meta-analyses[20]. The NOS was chosen as it is considered an easy and convenient quality-assessment tool with established content validity and inter-rater reliability. As all included studies were cohort and case-control studies, two modified criteria of the NOS were used for each study type. Using this tool, two independent reviewers evaluated each study, awarding stars out of a possible nine. Studies were classified as either high quality (≥ 7 stars) or low quality (< 7 stars)[21]. Stars were assigned based on three categories: (1) The quality of cohort selection; (2) The comparability of obese and non-obese cohorts; and (3) The methods used for assessment of CRC. Any discrepancies in quality assessment were resolved by consensus between the two reviewers.

Statistical analysis

All statistical data analysis was conducted on RStudio version 2024.4.2.764[21] using the meta-analysis package 'metafor'[22]. Between-study heterogeneity was assessed using τ^2 , I^2 and the Cochran Q test, which quantifies the variability of results across studies, and publication bias was evaluated using funnel plots, which are included in the [Supplementary Figures 1 and 2](#). To minimise heterogeneity, two separate meta-analyses were performed to compare the association between obesity and CRC development in males and females. A random-effects model was applied when significant heterogeneity was detected, whereas a fixed-effects model was used when no heterogeneity was present. Studies not included in the meta-analysis were reviewed and synthesised qualitatively.

RESULTS

An initial database search yielded 368 papers, of which 107 were removed as duplicates. An additional 243 records were excluded during title and abstract screening. Among the 18 studies selected for full-text review, 3 were excluded: 2 for not directly investigating the association between obesity and CRC, and 1 for not utilising a cancer registry to identify CRC cases. Overall, 15 studies satisfied the inclusion criteria and were included in the review. The full details of this screening process can be found in the [Supplementary Figure 3](#).

Study characteristics

The included studies had a combined sample size of 12871700 participants. Among these, two were case-control studies [23,24] and 13 were cohort studies[25-37] ([Supplementary Table 2](#)). Sample sizes ranged from 929 to 1368 participants for the case-control studies and from 708 to 9959605 participants for the cohort studies. Additional study details are presented in the [Supplementary Table 3](#). Of the 15 studies, 14 were assessed as high quality[24-29,31-37], while one study

was determined to be of low quality[30]. The results for each study can be found in the [Supplementary Tables 3 and 4](#).

This systematic review identified seven observational studies (all cohort studies) with comparable incidence ratios and study design to include in the meta-analysis to quantitatively assess the relationship between obesity and CRC[27,29-32,34,35]. This analysis was stratified by sex and presented graphically as forest plots ([Figure 1](#)). Ultimately, six studies were included in the male meta-analysis[27,29-32,35] ([Figure 1A](#)) and six were included in the female meta-analysis[27,29,30,32,34,35] ([Figure 1B](#)), encompassing 83506 male and 152043 female participants.

The meta-analysis revealed a significant positive association between obesity and CRC for both sexes. Obese men demonstrated a 1.71-fold increased risk of CRC (HR 1.71, 95% CI: 1.44-2.02), while obese women showed a 1.26-fold increased risk (HR 1.26, 95% CI: 1.03-1.53), compared to their normal BMI counterparts. The 95% CI for both analyses excluded 1, confirming statistical significance. Both the male and female meta-analyses possess a τ^2 of close to, or equal to 0 (0 and 0.0298, respectively) as well as non-significant Cochrane Q P values (0.64 and 0.07, respectively), which indicates non-significant heterogeneity for both demographics. Furthermore, the male meta-analysis reveals an I^2 value of 0, indicating low to no heterogeneity ([Figure 1A](#)), while the female meta-analysis indicates potential moderate heterogeneity with an I^2 value of 50% ([Figure 1B](#)). These findings suggest a consistent relationship between obesity and CRC risk, with a more pronounced effect observed in males. The low heterogeneity in the male analysis strengthens the reliability of this finding, whereas the moderate heterogeneity in the female analysis warrants further exploration of potential sources of variation.

Findings from meta-analyses

Among males, five of the six included studies reported a positive correlation between obesity and CRC incidence, with HRs ranging from 1.6 to 2.5[27,29,31,32,35] ([Figure 1A](#)). Notably, one study by Han *et al*[30], which contributed the lowest weight within the male meta-analysis, found a non-statistically significant association between obesity and CRC ([Figure 1A](#)). Among females, three of the six studies revealed a positive association, with HR ranging from 1.4 HR to 1.6 HR[30,32,34,35], while the remaining three reported no measurable effect[27,29,30] ([Figure 1B](#)).

Qualitative analysis

A graphical summary of the studies excluded from the meta-analysis is presented in [Figure 2](#), adapted from Boon and Thompson[38]. Exclusion criteria included: (1) Use of different measures of association, such as OR and RR[23-25,28,33]; (2) Use of alternative obesity measures in addition to the WHO obesity diagnostic criteria[26,36]; and (3) Methodological limitations that prevented accurate comparison with studies included in the meta-analysis[37].

Obesity at varying age demographics on CRC development

This systematic review identified one case-control study exploring the relationship between obesity and CRC across various age demographics[23]; However, this study was unable to find a statistically significant 95% CI for any of these demographics. This may be attributed to methodological limitations, including the use of self-reported weight and height to estimate BMI retrospectively for each age decade, potentially introducing recall bias and data misrepresentation. Additionally, restricting controls to individuals aged 40 years or older may have further skewed the results.

One cohort study was also identified that investigated CRC risk for obese individuals at various ages[24]. This study found that, at ages 20 and 30, statistically significant positive correlations were identified with incidence ratios of (OR 1.44, 95% CI: 1.18-1.75) and (RR 2.06, 95% CI: 1.25-3.40), respectively. While this study possesses too few age demographics to accurately determine the link between obesity, age and CRC, they have reported a potential increased susceptibility to obesity-induced CRC in older ages.

Ultimately, no overall conclusion about obesity, age and CRC incidence can be made from these two studies, and more research will be required to properly investigate this relationship.

Severity of obesity on CRC development

This systematic review identified three cohort studies that investigated and compared the association between different severities of obesity and CRC[25,26,28]. Wang reported a positive link between obesity and CRC for all levels of obesity for both men and women, observing a greater increased risk for CRC for those categorised in the severe obesity BMI range (BMI \geq 35 kg/m²)[25]. This was reflected by Oxtenko *et al*[28], which found an increasingly positive link between obesity and CRC as the obesity BMI reached the severe category. Song found a similar trend, identifying the correlation between obesity and CRC to be greater in those in the severe obesity category than those at baseline obesity; however, the incidence ratio of middle severity and CRC was found to be statistically insignificant[26].

Another cohort study was identified in this systematic review that investigated two separate forms of obesity and their link to CRC (general obesity with abdominal obesity and general obesity without abdominal obesity)[36]. Nam *et al*[36] reported no link between general obesity without abdominal obesity for men, women and combined. However, a statistically significant positive correlation was identified between general obesity with abdominal obesity and CRC for all demographics.

This systematic review identified one cohort study by Bao *et al*[33] that investigated the link between obesity and both colon cancer and rectal cancer independently. However, for both colon cancer and rectal cancer, no statistically significant ORs were found.

Weight change on CRC development

This systematic review identified one retrospective cohort study by Seo *et al*[37] that investigated obesity status in patients at a four-year interval (from 2005 to 2009) and how it affected their CRC development risk. Seo's study found

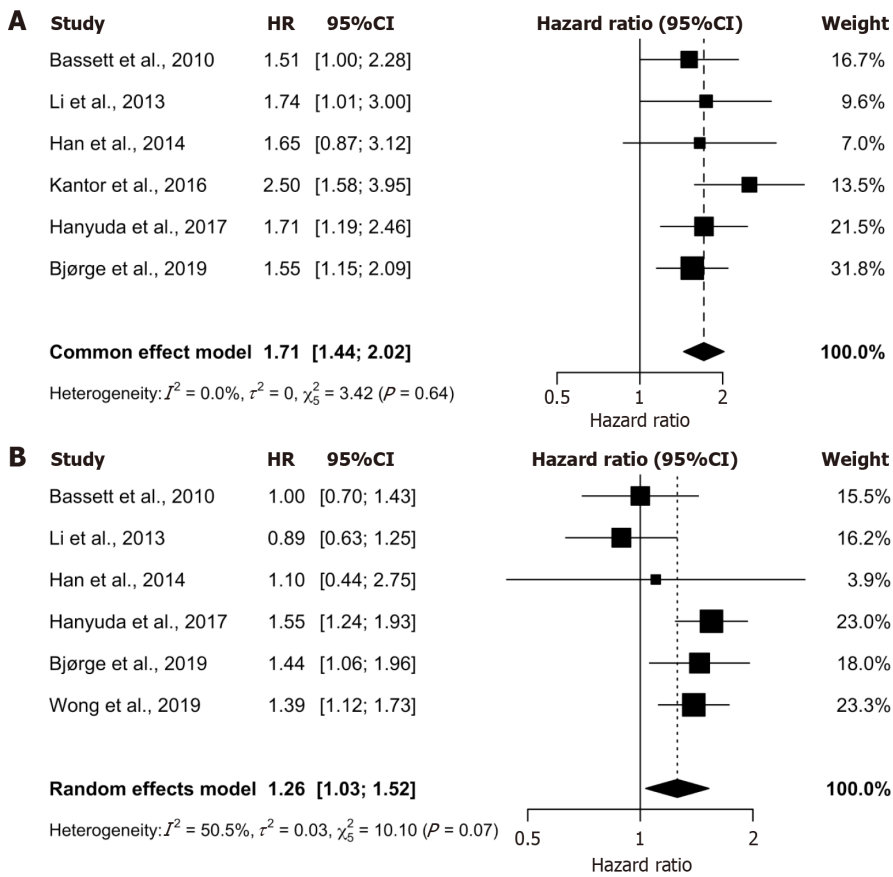


Figure 1 Forest plots of the association between obesity and colorectal cancer risk stratified by sex. A: Pooled analysis for men shows a strong, statistically significant positive association (HR 1.71, 95%CI: 1.44-2.02) with no observed heterogeneity ($I^2 = 0.0\%$); B: Pooled analysis for women indicates a significant but attenuated positive association (HR 1.26, 95%CI: 1.03-1.52) with moderate heterogeneity ($I^2 = 50.5\%$).

Study	Study design	CRC incidence (male)	CRC incidence (female)	CRC incidence (total)
Nock 2008 [23]	CC	◀ ▶	◀ ▶	◀ ▶
Wang 2008 [25]	PC	▲	▲	
Song 2008 [26]	PC		▲	
Oxentenko 2011 [28]	PC		▲	
Bao 2018 [33]	PC			◀ ▶
Nam 2020 [36]	RC	▲	▲	
Li 2022 [24]	CC			▲
Seo 2023 [37]	RC	▲	◀ ▶	▲

Figure 2 Effect direction plot for studies not included in meta-analyses. Effect direction: ▲ = Positive health impact, ▼ = Negative health impact, ◀▶ = No change/mixed effects/conflicting findings. Sample size: Final sample size (individuals) in intervention group large arrow ▲ > 300; medium arrow ▲ 50-300; small arrow ▲ < 50. Study quality: Denoted by row colour: Green = Low risk of bias; amber = Some concerns; red = High risk of bias. CC: Case-control; PC: Prospective cohort; RC: Retrospective cohort.

that those who were consistently obese during this four-year interval possessed an increased risk of CRC development of 1.08 HR (95%CI: 1.06-1.11) compared to those who were consistently non-obese[37]. Interestingly, this study observed that participants whose weight changed from obese to non-obese and non-obese to obese saw no statistically significant increase in CRC risk; however, these findings were later justified by the authors to be due to an insufficient follow-up period.

Overall, this study was unable to make a definitive conclusion whether weight gain or loss affected an individual's risk for CRC development, highlighting the need for further research with longer follow-up durations.

Summary of proposed mechanisms in the obesity-CRC association

The specific biological mechanism describing the linkage between obesity and CRC remains unclear; however, it is believed to be multifactorial in nature. An overview of these mechanisms is shown in [Figure 3](#). Although there are many obesity-linked factors contributing to colorectal carcinogenesis, specific attention has been raised towards the significance of obesity-induced inflammatory dysregulation and dysregulation of insulin and IGF-1 secretion. [Figures 4](#) and [5](#) have also been used to summarise the current literature of these proposed mechanisms.

DISCUSSION

The aim of this study was to explore whether recent data and literature confirmed a positive association between obesity and CRC, which was achieved. As mentioned earlier in this review, the current proposed mechanisms between obesity and CRC are multifaceted but can be broadly classified into two categories: Physiological changes brought on by obesity (inflammatory dysregulation, altered gut microbiome, dysregulation of insulin and IGF-1 secretion, T2DM)[[8,10](#)], and complications of obesity-linked behaviours (pro-inflammatory diets and sedentary behaviour)[[9,12,13](#)]. This review seeks to investigate each of these risk factors and describe the current scientific understanding of how these factors may predispose to CRC ([Figure 3](#)).

Inflammatory dysregulation

Adipocytes, primarily white adipose tissue (WAT), are heavily involved in the initiation and regulation of the inflammatory process[[39](#)]. Obesity induces a phenotypic change to WAT, resulting in adipocyte dysfunction and inflammatory cell infiltration. This may ultimately result in inflammatory dysregulation, inducing the uncontrolled release of potent pro-inflammatory substances, such as inflammatory cytokines [*e.g.* tumour necrosis factor alpha (TNF- α), interleukin-1 (IL-1), interleukin-6, monocyte chemoattractant protein-1, interleukin-1 beta], and systemic adipokine release (*e.g.* leptin) [[8,40](#)]. Additionally, it also results in downregulation of the release of anti-inflammatory adipokines such as adiponectin [[41](#)]. This inflammatory dysregulation induces a chronic low-grade inflammatory state[[8,41-43](#)], which *via* a variety of pathways, NF- κ B signalling, leptin hypersecretion and reduced adiponectin levels, can lead to CRC development. A summary of these processes is presented in [Figure 4](#).

NF- κ B signalling

The NF- κ B signalling pathway, which is made up of five transcription factors that regulate the expression of a number of genes, is activated *via* cytokines such as IL-1 and TNF- α [[42-44](#)]. *Via* a detailed cascading pathway, activation of NF- κ B leads to the formation of a pro-inflammatory tumour microenvironment, the activation of oncogenes and subsequent CRC development[[8,42](#)].

Leptin hypersecretion

Leptin exerts its action *via* binding to a variety of receptors, including a long isoform obesity receptor (OB-Rb), four short isoforms (OB-Ra, OB-Rc, OB-Rd, OB-Re) and a circulating, soluble form (OB-Re)[[44](#)]. Studies reveal that activation of OB-Rb results in the downstream activation of a variety of signalling pathways (JAK/STAT3, PI3K/AKT, MAPK/ERK) that contribute to CRC formation. As such, obesity-induced leptin hypersecretion may substantially increase the risk of CRC. A study by Aleksandrova *et al*[[45](#)] has also proposed that obesity reduces concentrations of the OB-Re receptor. Studies indicate that OB-Re exerts CRC protective effects at non-obese, normal plasma concentrations (RR 0.42, 95%CI: 0.40-0.76), which may provide an additional link between obesity and CRC.

Adiponectin

The anti-inflammatory, anti-diabetic and anti-atherogenic effects of adiponectin have been well-established in academic literature[[46](#)]. These actions are thought to occur from adiponectin activating multiple CRC protective intracellular signalling pathways, the main of which interacts with AMPK[[46-48](#)]. Obesity's downregulation of adiponectin may strip individuals of these CRC protective mechanisms, ultimately increasing their risk of CRC development.

Altered gut microbiome

Risk factors for obesity, such as poor diet and low physical activity, as well as obesity in itself, are all features that can alter an individual's gut microbiome[[10](#)]. Patients with CRC possess different compositions of gut microbiota compared to healthy individuals, with *Fusobacterium nucleatum*, *Bacteroides fragilis*, *Streptococcus gallolyticus* and *Escherichia coli* being some of the most common bacteria related to CRC development[[10,49](#)]. Interestingly, *Fusobacterium nucleatum* is elevated in the saliva and colon of individuals with obesity, and thus may act as a potential link between obesity, gut microbiome changes and CRC. *Fusobacterium*, as well as other high-risk CRC-inducing microbiotas, are hypothesised to cause chronic inflammation, dysregulation of immune responses and altered dietary metabolism[[49](#)]. This ultimately may result in the formation of harmful metabolites (*e.g.* secondary bile acids, nitrosamines and formate) that, over long periods of exposure, may result in the formation of an oncogenic environment. Despite this, no scientific evidence currently directly links obesity-related gut microbiome changes to CRC[[49](#)].

Dysregulation of insulin and IGF-1 secretion

Plasma insulin concentration is often raised in obese individuals, regardless of their diabetic status[[8,50](#)]. This is believed

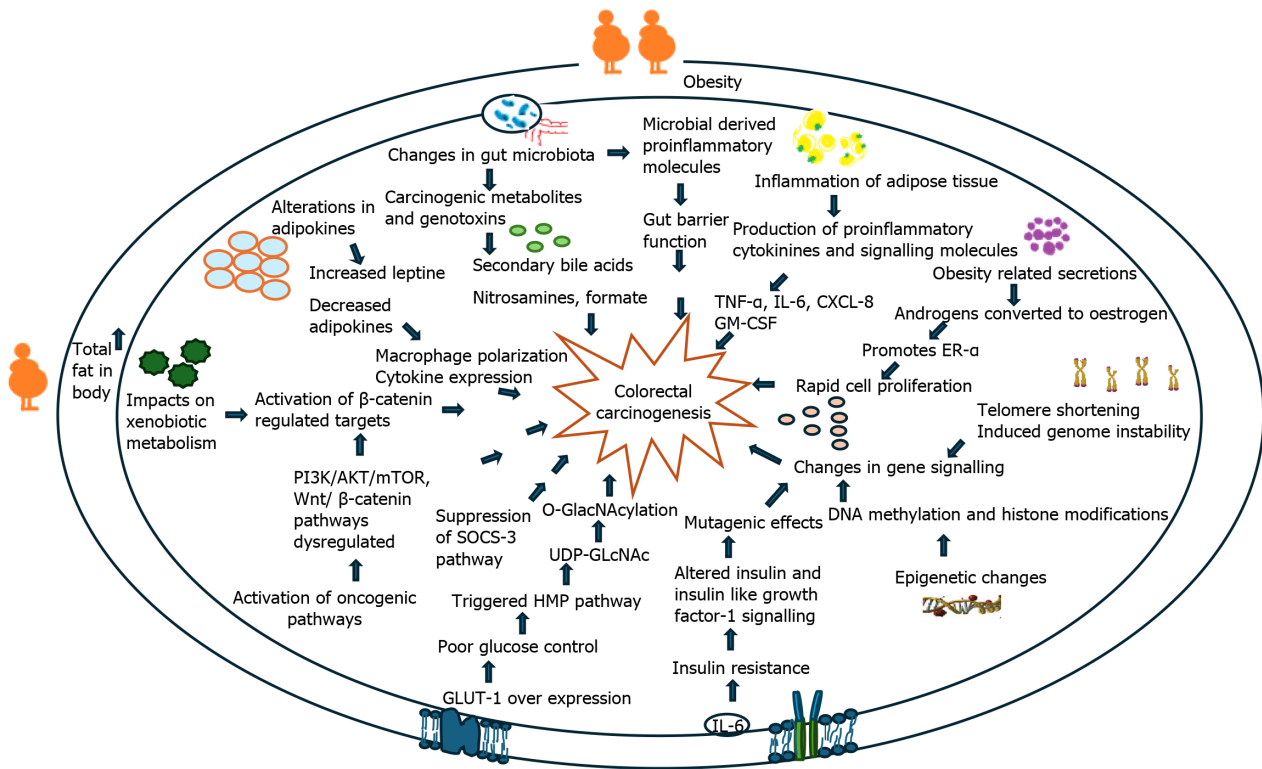


Figure 3 Summary diagram of major molecular mechanisms linking obesity and obesity-linked behaviours with colorectal cancer.

to potentially result from dysfunction of normal energy utilisation in obese individuals, as well as more energy and insulin being required to carry out normal metabolic processes compared to those of lean BMI. Insulin is a direct stimulus for IGF-1 production, resulting in a proportional hypersecretion of both IGF-1 and insulin in obese people[8].

IGF-1 receptors have been demonstrated to be present on both healthy colonic epithelium and CRC cells. Upon binding to these receptors, IGF-1 prevents apoptosis of the cell and stimulates cellular proliferation[8]. As CRC development, much like any other cancer, is formed from specific genetic and molecular alterations, an over induced cellular proliferation (a result of obesity-mediated IGF-1 hypersecretion) may increase the risk of these alterations developing within the colonic epithelium, potentially leading to CRC formation.

Additionally, it is proposed that together, insulin and IGF-1 promote rat sarcoma activation, a well-established proto-oncogene related to CRC, responsible for the transformation of colonic adenomas into CRC[8,51]. A summary of these processes is presented in Figure 5.

T2DM

T2DM is defined as a chronic medical condition describing a dysfunctional secretion of insulin and/or an improper biologic response to insulin, resulting in a consistently hyperglycaemic state[52,53]. Obesity is considered one of the strongest risk factors for T2DM, obese men and women possessing a 4.6 and 3.5 times greater risk of developing T2DM, respectively, compared to non-obese individuals[53]. Siddiqui *et al*[54] identified a significant link between elevated glycated haemoglobin levels and the early onset of CRC. Additionally, due to mechanisms mentioned previously, the insulin resistance and associated hyperinsulinemia that is synonymous with T2DM greatly increase the risk of CRC[53].

Obesity-linked behaviours

Diet: For many individuals, obesity can result from the adoption of a typical Western diet, which is, characterised by excessive consumption of red meat, processed meat, sugar, saturated fatty acids, cholesterol and trans fats[55,56]. Shivappa *et al*[11] found that this type of diet contains food of high inflammatory potential, which when taken in excess, can place an individual under a chronic inflammatory state, playing a significant role in colon oncogenesis as discussed earlier in this review. Their study revealed that a high inflammatory potential diet resulted in a 1.40 times increased risk of CRC development (95%CI: 1.25-1.55 RR)[11]. Additionally, overconsumption of red and processed meats increases the concentration of haem iron, N-nitroso compounds, polycyclic aromatic hydrocarbons and heterocyclic aromatic amines, which also have oncogenic properties[56]. To reduce CRC risk, individuals should shift away from the Western diet and instead opt for a diet rich in high-fibre foods, fruits, vegetables, dairy products and folate.

Sedentary lifestyle: A sedentary lifestyle, which is often the case for those with obesity, can also be a significant factor for CRC development. A systematic review found that higher levels of sedentary time greatly increased the risk of CRC (RR 1.54, 95%CI: 1.19-1.98) compared to those of an active lifestyle[12]. The mechanisms underpinning this increased risk for CRC development involve sedentary behaviour's tendency to increase blood glucose, decrease insulin-sensitivity and

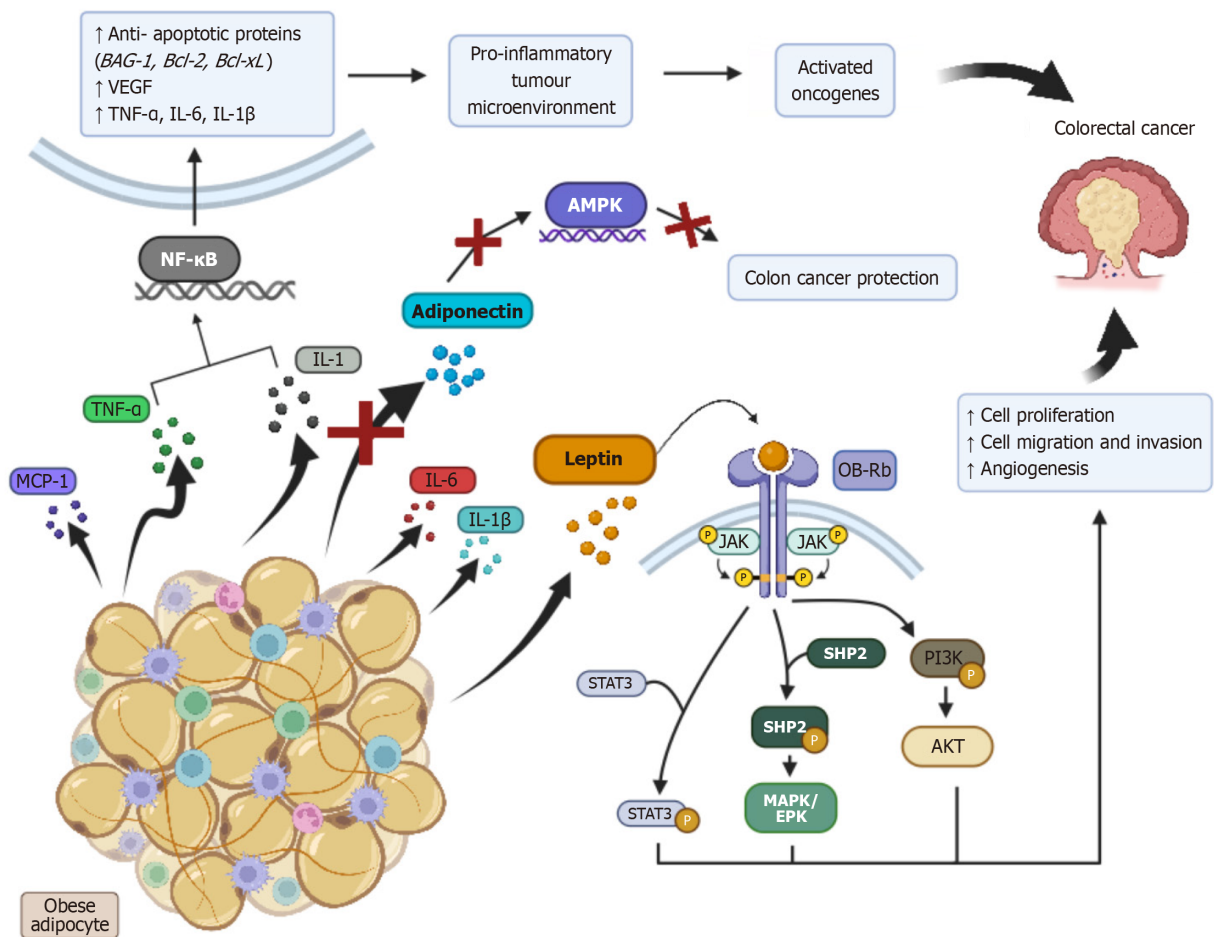


Figure 4 Diagram showing biological mechanisms of obesity-induced inflammatory dysregulation and colorectal cancer development. TNF-α: Tumour necrosis factor alpha; IL: Interleukin.

place an individual under a chronic-inflammatory state by upregulating proinflammatory factors and decreasing anti-inflammatory markers[57,58].

Implications

This systematic review examined 15 studies that investigated the link between obesity and CRC, including 13 cohort studies and two case-control studies. Our findings confirm a positive relationship between these two variables, which has reflected the results of previously conducted meta-analyses mentioned earlier in this review[13,14]. From these results, as well as the detailed literature of the biological mechanisms linking obesity and CRC, a definitive association can be made, where obese individuals are more likely to develop CRC. By considering this increased risk of CRC development, we recommend screening for CRC at younger ages for obese individuals to mitigate the potential of early-onset CRC in these groups.

This systematic review also uncovered a potential trend in obesity-induced CRC risk, where a higher severity of obesity induces a greater increased risk for CRC development[25,26,28], which will require further research. Another identified study investigated the association of both general obesity and central obesity with CRC, finding only a positive correlation when both were present in an individual. This reveals another area for further study to investigate if central obesity is an independent risk factor for CRC development, regardless if generalised obesity is present[36].

Limitations

This review is affected by a few limitations. Much like all meta-analyses, particularly those investigating observational studies, this review may suffer from the presence of publication bias, potentially resulting in overstated HR measures between obesity and CRC. However, the funnel plots for both male and female meta-analyses revealed no gross publication bias. Additionally, as there was considerable variability in adjusted confounders between all studies included in this review, there exists the potential for increased heterogeneity. This is evident in Figure 1B, where an I^2 value of 50% was observed, indicating moderate heterogeneity between these studies. Despite the female meta-analysis revealing a non-significant Cochrane-Q test ($P > 0.05$) (Figure 1B), a non-significant Q-test is not an indicator of an absence of heterogeneity, and thus, this I^2 value cannot be disregarded[59]. These sources of heterogeneity may be due to a variety of factors, such as dietary differences (e.g. western vs eastern diet), lifestyle factors (e.g. smoking, exercise) and genetic

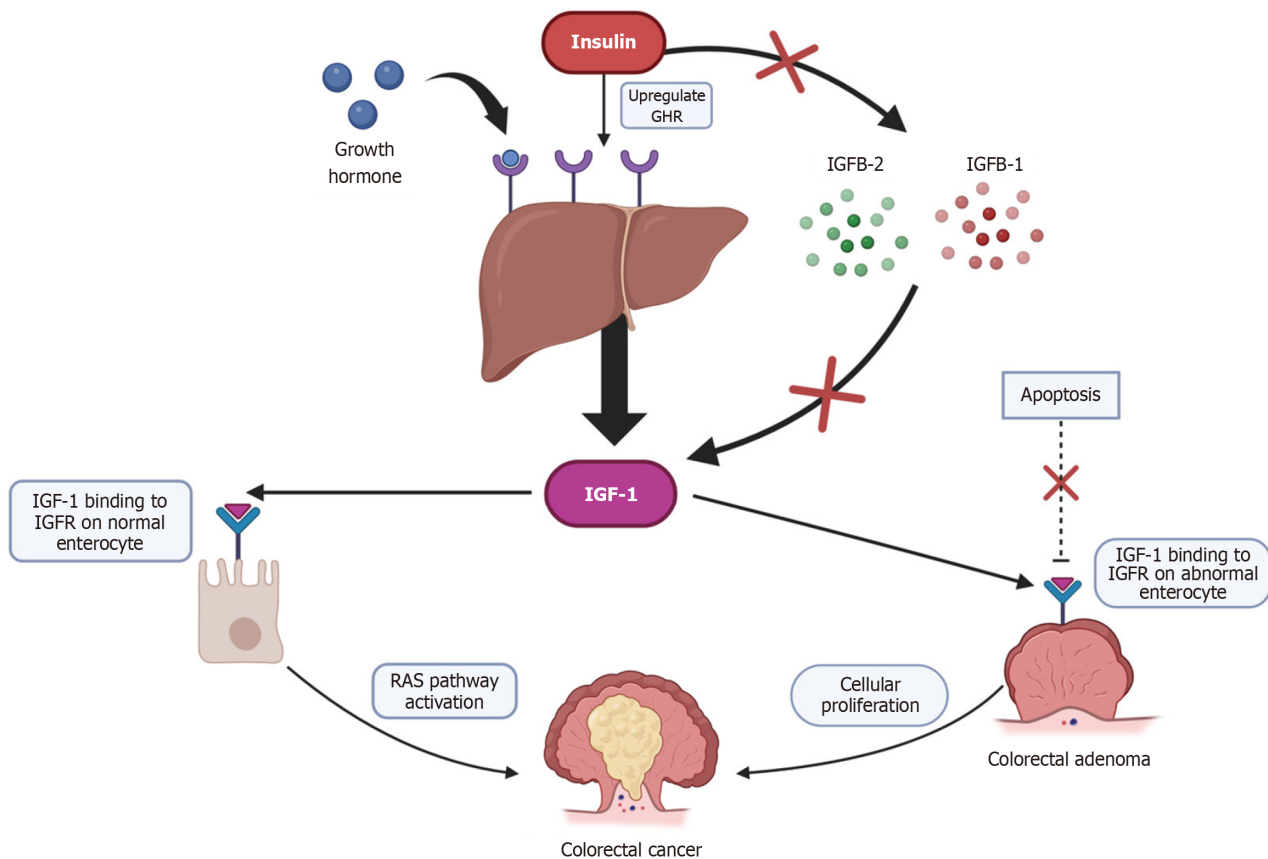


Figure 5 Diagram showing biological mechanisms of proposed insulin and insulin-like growth factor 1 induced carcinogenesis. IGF-1: Insulin-like growth factor 1.

predisposition.

Additionally, BMI has been described as a non-perfect measure of adiposity, and thus introduces the possibility for stronger associations to be present between CRC and other measurements of overall fatness than what was observed in this meta-analysis[14]. As such, to further investigate the association between obesity and CRC, other measures of adiposity, including waist circumference, waist-hip ratio, or body fat percentage should be used as an alternative or complementary obesity indicator to BMI in future research.

It was also observed that many of the included studies only used a single measure of BMI, which has a tendency to fluctuate over time. As a result, future studies should incorporate multiple repeated measures of an individual's adiposity to allow for more accurate associations between obesity and CRC to be made.

Despite using a random-effects model to help reduce potential variances in the study designs, they may still have been affected by potential regional and dietary confounding factors, which may explain the moderate heterogeneity of the female meta-analysis (Figure 1B). Lastly, while all studies used in the male and female meta-analyses were weighed according to the amount of precision in their estimated effects, the significant range in sample sizes for the included studies may have potentially been a limitation to this review.

CONCLUSION

This review explored the association between obesity and CRC, revealing that obesity significantly increases the risk of CRC in both males and females. These findings are supported by established biological mechanisms, including inflammatory dysregulation, insulin resistance, and gut microbiome alterations. Although this review was affected by some limitations, a few strengths were also identified. By including only studies from data registries, the review ensured a standardised case identification process, enhancing the reliability of the findings despite the exclusion of smaller-scale research. Furthermore, the use of large sample sizes for all studies included in this review further enhanced its reliability and reduced the chances of small-study bias. While the review provides strong evidence for this association, the included studies varied in methodology and population characteristics, which may limit the generalisability of the findings.

Given the strong evidence linking obesity to CRC, we recommend commencing screening for CRC at younger ages in obese individuals to mitigate the potential risk of obesity-induced early-onset CRC. Further research is needed to optimise screening guidelines, implications and explore targeted interventions to address this growing public health concern. Additional research should also be conducted to explore genetic predispositions to CRC and its interaction with obesity.

FOOTNOTES

Author contributions: Leung LJCL contributed substantially to the conception and design of the study, acquiring, analysing and interpreting the data, and was a major contributor in the writing of the manuscript; Sharma RS and Cheng B greatly assisted with the analysis and interpretation of the data as well as contributing to the manuscript drafting; Gopalan V assisted with the conception of the study as well as provided critical revisions of the intellectual content of the manuscript draft; Akalanka HMK was also another major contributor of providing critical revisions of the manuscript drafting; Leung LJCL and Gopalan V confirm the authenticity of all the raw data. All authors read and approved the final manuscript. Both authors listed as co-corresponding authors were heavily involved in the concept creation of this paper. They were also involved in the supervision of other authors, data analysis and coordination of writing. As such, it was deemed that both authors should be listed as corresponding authors for this paper due to their contributions to this paper.

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