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Review

Diet and colorectal cancer

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ABSTRACT

Background: Colorectal cancer (CRC) is the third most common diagnosed cancer in men and the second in women. Dietary factors and lifestyle may contribute to the increasing CRC incidence, making these issues interesting for both the general population and the medical oncology community.
Objective: The aim of this report is to present a review of the published epidemiologic research to date reflecting the most current scientific evidence related to diet and CRC risk.
Design: EMBASE and PubMed-NCBI were searched for relevant articles up to November 2014 that identified potential interactions between foods or dietary patterns and CRC risk.
Results: Obesity increases the risk of CRC by 19%. Regular physical activity reduces this risk by 24%. CRC risk derived from red meat intake is influenced by both total intake and its frequency. Fish consumption may decrease CRC risk by 12% whereas garlic intake is not significantly associated with reduced CRC risk. Intakes of more than 20 g/day of fiber are associated with a 25% reduction of CRC risk and 525 mL/day of milk reduces colon cancer risk by 26% in men. Moderate amounts of alcohol (25–30 g/day) increase CRC risk.
Conclusions: CRC is a preventable disease through the modification of associated risk factors, including physical inactivity, obesity and overweight, excessive meat intake, smoking and alcoholic beverage consumption. Nonetheless, epidemiological evidence in this regard is not conclusive so further research is warranted.

Contents

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1. Introduction

Colorectal cancer (CRC) is the third most common diagnosed cancer in men (746,000 new cases per year) and the second in women (614,000 new cases per year) worldwide. It is rare in people younger than 50 years, and its incidence increases with age. Interestingly, CRC incidence is 18% higher in developed regions in comparison with lower-income areas. There is high geography-dependent variability in CRC rates. The highest incidence is in European countries, North America and Oceania, where the incidence rate reaches 44.8 cases per 100,000 population in men and 32.2 cases per 100,000 population in women. On the contrary, Western Africa has the lowest rate with 4.5 cases and 3.8 per 100,000 population in men and women, respectively [1]. This variability highlights the principal role of environmental factors in cancer development in general [2] and CRC in particular, and diet is the most important risk factor apart from age, male sex and hereditary factors, which are responsible for 35% of CRC [3].

Since the last report about CRC published in 2011 by the *World Cancer Research Fund* (WCRF) and the *American Institute for Cancer Research* (AICR), several epidemiological studies have been conducted and provided new findings and working hypotheses regarding diet-associated risk factors for CRC. The aim of this work is to examine the latest information available about the diet–CRC interaction. This review is mainly focused on epidemiological results from studies conducted in the last five years, in order to identify potential CRC risk factors and their level of evidence.

2. Methods

An extensive search of scientific literature was conducted in EMBASE and PubMed Central (PMC)-NCBI to identify human studies written in the English or Spanish language published up to November 2014. The search included the following keywords or phrases: diet, colorectal cancer, epidemiology, risk factors and lifestyle. Studies that reported risk estimates (hazards ratios, odds ratios [ORs], and relative risk) of cancer and measures of variability (SEs or 95% CIs from which these could be derived) were selected preferably.

3. Obesity prevention and healthy weight maintenance

Changing of dietary patterns and lifestyle in most developed countries have led to an increase in overweigh and obesity prevalence. Obesity, defined as a body mass index (BMI, kg/m^2) greater than $30\ kg/m^2$, and overweight, defined as a BMI between 25 and $29.9\ kg/m^2$, are associated with increased mortality in CRC [4]. Individuals with a BMI > 30 show a 19% increased risk of CRC when compared to those with a BMI between 20 and $25\ kg/m^2$ [5]. Epidemiological evidence shows some discrepancies related to the association between high fat diets and increased risk of tumors [6]. A lack of effect has been reported on reduction of CRC risk after the adoption of a healthy dietary pattern, low in fat and high in fiber, fruits, and vegetables [7]. However, recent studies that analyzed dietary patterns indicate that adoption of a "Western dietary pattern" (high intake of red meat and/or processed meat, high-fat dairy products, fast food, refined grains, and sweet foods and drinks) increases CRC risk. Contrariwise, adoption of a "healthy dietary pattern" (high intake of fruits and vegetables, whole grain cereals, fish, white meats and soy derivatives) decreases CRC risk [8].

Recent meta-analyses which examined the impact of diets high in sugars, with a high glycemic index (GI) and a high glycemic load (GL), on CRC risk have not been able to establish a direct association between them [9,10].

In the obesity–cancer relationship, multiple biologic processes participate and there is implication of certain factors such as insulin, insulin-like growth factor (IGF)-1, insulin resistance, sexual hormones (estrogens), pro-inflammatory cytokines (tumor necrosis factor alpha (TNF), interleukine-6 (IL-6)) and C-reactive protein (a marker of chronic vascular inflammation). The balance and/or interactions between all these factors determine a low or high increase of cancer risk.

One hypothesized mechanism of the influence of body fat on CRC risk is based on its direct effect on certain hormone levels, such as insulin, estrogens and IGF-1, that produces a favorable environment for carcinogenesis and decreases cellular apoptosis [4].

Chronic hyperinsulinemia (due to higher insulin resistance) leads to an increase of IGF-1 bioavailability. Insulin also stimulates ovarian androgen synthesis and growth hormone receptor expression, and inhibits liver production of binding proteins such as sex hormone binding globulins (SHBG) and IGF-1 binding proteins, leading to a greater bioavailability of circulating estrogens and IGF-1 [11,12]. In particular, abdominal fat increases insulin resistance and subsequent hyperinsulinemia, which increases risk of CRC [13].

Together with possible cell microenvironment variations caused by present inflammatory molecules (TNF alpha, IL-6, CPR), this contributes to malignant cell proliferation, increased angiogenesis and metastases [14].

4. Physical activity levels

Regular and moderate physical activity elevates basal metabolism and improves tissue oxygenation, leading to better metabolic efficiency and capacity and finally reducing body fat, insulin levels, insulin resistance and adipose tissue volume. Consequently, physical activity reduces risk of CRC. Recent studies have shown that more physically active subjects have a 24% decreased risk of CRC in comparison to people with more sedentary lifestyle [15], and individuals who take regular exercise decrease CRC risk by 40%, regardless of their BMI [5]. Thirty minutes of daily moderate exercise result in an 11% reduction of CRC [4]. Current guidelines for adults recommend at least 150 min/week of moderate-intensity or 75 min/week of vigorous-intensity aerobic activity for overall health [16]. In contrast, physical inactivity is associated with a status of low grade chronic inflammation or latent inflammation, and higher estrogen, androgen and insulin levels.

5. Red and processed meat consumption

CRC is the main cancer type that has been associated with high meat consumption, based on a considerable number of studies [13]. During the last three decades, diversely located epidemiological studies have shown a population change in dietary habits, with an increased intake of red meat and the associated risk of CRC [17].

For the analysis of this causal relationship, different products' intake can be considered, such us total fresh meat, red or white