Link between Food Environment and Colorectal Cancer

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Food and diet are critical risk factors for colorectal cancer (CRC). Food environments (FEs) can contribute to disease risk, including CRC. CRC incidence was associated with the availability of specific foods such as red meat, meat, animal fats, energy from animal sources, and an unhealthy FE. Increased CRC mortality was linked with the availability of animal fat, red meat, alcoholic beverages, and calorie food availability, residence in food deserts, and lower FE index. There were a variety of associations between CRC and the FE. The availability of specific foods, unhealthy FE, and food desserts impact CRC incidence and mortality.

colorectal cancer food availability food environment

1. Introduction

Colorectal cancer (CRC) is the third most common cancer and accounts for more than 1.9 million new cases after breast and lung cancer [1]. According to GLOBOCAN 2018, CRC is the third most prevalent cancer in men and the second most common cancer in women. The age-standardised CRC incidence rate was 19.5%, which was higher than that of other cancers. The rapid rise in CRC incidence was associated with urbanisation and lifestyle westernisation, which caused a change in eating behaviour [2] that increased obesity prevalence [3][4][5]. Furthermore, dietary factors significantly contributed to CRC mortality risk [6]. Nevertheless, CRC is highly preventable [7]; therefore, an important CRC prevention measure is to improve the modifiable CRC risk factors in diet and nutrition.

The Committee on World Food Security stated that the food environment (FE) consists of the physical, economic, political, and sociocultural contexts in which consumers interact with the food system to attain, prepare, and consume food [8]. Diet patterns are strongly related to the FE based on availability, affordability, cost, and sustainability [2]. The FE and diet patterns were linked to increased risk of non-communicable diseases such as obesity, coronary heart disease, and diabetes [9]. The FE accessibility can be influenced by the population's physical, social, cultural, economic, and policy conditions and can impact the population's food selection, pattern, and quality. Many studies examined specific food items, such as red meat or fibre, and CRC incidence in diverse settings [10][11][12][13][14]. There must be interplay by the FE to affect CRC risks and outcomes [15]. Although numerous researchers used a population's FE to evaluate potential correlations with disease [16], there remains a lack of studies investigating the link between FEs and CRC.

2. Food Environment and Colorectal Cancer

Eight research on CRC and the FE were studied. Four studies [15][17][18][19] reported on the relationship between the FE and CRC. Broadly, FEs were divided into natural FEs (wild or cultivated) and built FEs (informal and formal markets) [20]. The FEs varied according to culture, tradition, habits, race/ethnicity, and setting (urban or rural) [21]. Varied results were attributed to the study population, FE attributes used, and variations in the FE and CRC measurements.

Mo et al. [15] described the association between poor FEs and CRC incidence. Poor (lower) FEI localities were characterised by limited access to healthy foods, lower annual income, farther distance from grocery shops, and unreliable food source. The CRC mechanism with underlying poor FE remains unknown. The availability of fast food outlets was associated with high body mass index (BMI), body fat, obesity, and frequent processed meat consumption, which were closely related to CRC risk factors [22]. Fewer studies investigated the relationship between built FEs (fast food restaurants and grocery shops) and CRC. An ecological study investigating diabetes prevalence reported that decreased diabetes prevalence was associated with grocery shops and full-service restaurants (seated and pay after eating) while high diabetes prevalence was associated with fast food restaurants [23]. Among Mexican adults, accessibility to grocery shops in food-insecure communities might have increased the likelihood of obesity [24] and led to a higher mean BMI [25]. Poor FEs affected the incidence of CRC and other non-communicable diseases, which emphasised the importance of recognising poor FEs and necessitating multidisciplinary perspectives and approaches.

Food deserts are characterised by low access to healthy food and the presence of low-income areas [26]. Food deserts and food accessibility are notably influenced by distance, race/ethnicity, income, and age [27]. Inaccessibility referred to barriers in the locality, such as accessibility to healthy food and personal barriers such as financial barriers, lack of transportation (public or personal), or below-average family income. Fong et al. [17] reported the association between food deserts and CRC mortality. Food deserts were also linked with heart disease [26] and increased risk of all-cause and cardiovascular hospitalisation [28]. Due to a lack of access to food markets that sold reasonably priced nutritious foods, poor communities were more likely to consume processed foods, refined grains, and fewer fresh vegetables [15]. Apart from food, poor communities also encountered difficulty accessing health facilities such as hospitals, clinics, and pharmacies, which could affect their health and disease outcomes [17]. Although socioeconomic inequalities were weakly associated with non-communicable diseases and risk factors [29], equal access to healthy foods in impoverished neighbourhoods must be highlighted [30]. An established healthy FE would support healthy eating and improve population health [31].

The availability of specific foods, such as red meat, animal fat, and energy from animal sources, was associated with CRC incidence [19] and mortality [18]. The findings were consistent with that of Hoang et al. [6], who reported a positive link between red meat and all-cause mortality among patients with CRC. Many studies reported a connection between CRC and red meat diet patterns [12][32][33][34] where fast food and westernised diets contained unhealthy combinations of red meat, processed meat, sugary drinks and desserts, and processed snacks, which have all been linked to gut inflammation [35]. The formation of mutagenic and carcinogenic agents in red meat was

linked to the disruptions in homeostasis and colonic epithelial cell renewal that lead to CRC [36]. Increased availability and consumption of animal-derived products and a concomitant reduction in the traditional plant-based diet may drive the rising incidence of CRC in many sub-Saharan African countries. Most African countries are transitioning rapidly from traditional foods to animal-sourced foods and highly processed diets, increasing diet-related non-communicable diseases and cancers [19].

Buamden [18] reported that high alcohol drink availability was associated with CRC mortality. Alcohol consumption may be influenced by its widespread availability. Many studies reported the association of alcohol with CRC [4][37][38][39], specifically, people who consumed at least four daily drinks were more likely to develop CRC than non-drinkers [40]. Alcohol intake may initiate carcinogenic processes by destroying folate when microbially converted into acetaldehyde in the colon. Subsequently, the folate deficiency results in chromosome breakage, uracil misappropriation, and other DNA precursor abnormalities, which initiates CRC [41]. Empowering consumers by providing health education and promoting healthy food choices may help reduce the impact of the high availability of unhealthy food. Moreover, a local framework can be proposed to facilitate FE monitoring [42].

FE may be linked to ethnicity, socioeconomic and environmental factors, resulting in CRC risk [15]. Mo et al. discovered stronger CRC associations in areas with a higher proportion of Black populations. According to Carethers [43], factors contributing to ethnic and racial disparities in CRC include genetic and environmental susceptibility (a high red meat, fat, or calorie diet, obesity, a low-fibre diet), and low screening utilisation. Previous research has limited evidence of the link between CRC, screening behaviour, and FE. Screening reduces incidence and mortality by 50% and 53%, respectively, whereas primary prevention can fill the remaining [44]. Because of its ability to completely visualise the colon, colonoscopy is diagnostic and therapeutic. The screening target in the United States is 80%; however, disparities in screening utilisation across US subpopulations may contribute to CRC disparities [45]. Black and Hispanic Americans have the lowest screening rates with a family history. Black Americans are less likely to be aware of their parent's cancer history than White Americans, and family members are less likely to report colonic polyps. Lack of provider recommendation for screening, fear of diagnosis, scheduling, implementation of screening, inability to pay for the colonoscopy due to economic difficulties, and loss of follow-up are likely barriers to colonoscopy utilisation [45]. CRC incidence and mortality could be effectively reduced through primary and secondary prevention. In addition to screening, adopted healthy behaviours (alcohol consumption, smoking, physical activity, BMI, and diet) [46] are associated with a lower risk of colorectal adenoma and higher adherence to a healthy lifestyle [47] associated with a lower risk of CRC. Previous research among Asians in California observed the strong relationships between CRC incidence, nativity and ethnic community, suggest a prominent role of acquired environmental variables such as FE [48]. The consequences of lifelong biological differences, as well as the effects of missed screening in populations, raise the risk of CRC [43].

FE may influence the pathogenesis and development of CRC. Cancer is not a single entity, and its causes are multifactorial. Significant advances in molecular carcinogenesis found diverse mutagenic events ranging from single-base substitutions to more extensive structural genetic alterations. Non-mutagenic environmental exposures interact with cellular processes and affect endogenous tumour mutations. Life-course events in the macro and micro-environments may leave genetic or epigenetic modifications expressed later [49].

Chronic inflammation has been linked to cancer. An unhealthy diet and alcohol consumption can contribute to chronic systemic inflammation and cancer cell growth. Early onset CRC is frequently associated with inflammatory bowel diseases such as Crohn's disease and ulcerative. Molecular studies also revealed that a poor diet produced pro-inflammatory cytokines and a slew of free radicals at the cellular level, with potential gene-environment interactions in the colorectum [50][51]. Foods with a high dietary inflammatory index score were linked to an increased risk of CRC [52].

There were shreds of evidence implicating a robust effect of a diet that may put young, non-obese and healthy people at risk of CRC [35][53]. Another CRC risk factor is age, with CRCs increasing after age 50, but recent trends indicate more early-onset CRC [54]. In the absence of traditional hereditary factors, genetic abnormalities conferring increased susceptibility and environmental factors are likely to play a role in young-onset CRC [55]. Fast food consumption with obesity, type 2 diabetes, metabolic syndrome, and the smoking trend has increased among young people, reduces the age at which CRC develops [55]. The gut microbiota may likely occur at the intersection of these risk factors and young CRC [51][56]. Breastfeeding, diet, and obesity affect microbiome composition, increasing CRC risk in younger adults [57]. The westernisation of diets characteristically includes a high intake of red and processed meats, high-fructose corn syrup, unhealthy cooking methods, stress, antibiotics, synthetic food dyes, and monosodium glutamate are key risk factors [53]. Studies suggested that carcinogenic chemicals such as heterocyclic amines (HCAs), polycyclic aromatic hydrocarbons (PAHs), and acrylamides produced during food preparation nowadays increase CRC risk [58][59][60]. Examining foods and their environment that account for the interaction of several nutrients may shed light on the role of diet in colorectal carcinogenesis among young people.

According to a systematic research by Puzzono et al. [53], the interaction of genetic and environmental factors is still unknown. A study supported Japanese high CRC risk relative to Caucasians by genetic susceptibilities [61]. However, studies have proposed that diet partially explains the degree of variation; for example, the risk of CRC is not distributed evenly among the four Lynch syndrome genes. Thus, diet may explain an increased or decreased risk of CRC [53]. The risk of CRC is modifiable and predominantly environmental according to temporal trends and migrant studies [62]. A research by Maskarinec and Noh [63] analysed the cancer incidence trends among Japanese in Japan and Japanese and Caucasians in Hawaii between 1960 and 1997, highlighting that migration's impact on cancer risk was substantial for CRC. The wide range of time migrants adopts the host population's cancer risk demonstrates that risk factors have organ-specific effects or work at different times in life. The disparity in cancer incidence across generations suggests that staying in the host country is insufficient to increase cancer risk to the host population. Although known etiologic factors can partly explain migration, much of the variable risk remains unknown. A study found that comparing Okinawan ancestors in Hawaii to Japanese migrants from all other provinces increased exposure to cancer risks unique to a specific environment and discovered that Okinawan food environment risk factors, such as flour usage from cycad nuts, caused bowel tumours [64].

Literature on the link between FE and CRC is less consistent, as several studies reported no connections. Besson's research found no association between the availability of food and the occurrence of colon cancer. Still, the availability of sugar, sweetened animal products, milk, meat, and fat are positively related to colorectal polyp incidence (precursors to CRC) [65]. Shvetsov et al. found a strong association between the change in the local

obesogenic environment to the CRC risk. The high-density communities had low socioeconomic and resource inequality; thus, changes in behaviour or obesity-related stress may increase CRC risk. However, the FE attributes have no significant association with CRC risk. Significant relationships were only observed in one race/ethnicity, highlighting the need to study the impacts of neighbourhood change by race/ethnicity [66]. Gibson et al. [67] proposed possible explanations for the lack of an association between the food environment and CRC incidence: the availability of unhealthy foods may be influenced by factors such as transportation and dietary preferences. Another possible explanation for mixed findings is that the direction and strength of the association between diet and cancer incidence vary according to lag time [22]. Individual-level behavioural factors such as obesity and physical activity may play a role in the link between the environment and cancer risk [68]. Besson stated that the diverging trends for cancer incidence and mortality were due to improvements in screening diagnostic capacity and treatment [65]. More accurate approaches by measuring CRC incidence instead of mortality rate are suggested by Buamden [18]. Compared to the incidence rate, the mortality rate can be influenced by timely diagnosis and treatment, which varies by count and is not entirely influenced by diet or FE. Furthermore, it is concerned that there is a disparity in data quality reported across African countries, which may not reflect true heterogeneity [19]. The small sample size, lack of data on possible confounding factors such as sedentary behaviour, and lifestyle and environmental factors are other drawbacks [18].

There is a need for a reliable and validated standard to assess the FE in a specific area. The literature contains many FE indicators from interviews, questionnaires, checklists, and inventories [21]. Nevertheless, the usage of these indicators is not standardised, possibly because FE categorisation covers a broad area, such as food shop environments (grocery shops, supermarkets, farmers' markets), restaurants (fast food and full-service), schools (cafeterias, vending machines, snack shops), and the workplace [21]. The FEI is a measure from the County Health Rankings produced from the University of Wisconsin Population Health Institute and is determined by socioeconomic conditions and considers the proximity to healthy foods and income. The elements include the distance an individual lives from a grocery shop or supermarket, the locations for healthy food purchases in most communities, and the inability to access healthy food due to cost barriers. Many studies utilised the FEI to determine the association between FEs and chronic diseases such as obesity and hepatocellular carcinoma [69]. In the US, the FEI aided investigations of increased cardiovascular disease (CVD) mortality associated with FE [70]. The US Department of Agriculture Economic Research Service initiated a food research atlas to assist and guide stakeholders in illustrating the impact of the FE [71] and developing standardised FE assessments according to region.

It is currently unknown as to whether changing FE will affect cancer outcomes. For example, providing access or establishing a new supermarket in a low-income neighbourhood may benefit residents' economic well-being and health in terms of less diagnosed dyslipidaemia, arthritis and diabetes; however, it does not impact much on dietary habits [72]. Therefore, more research is needed to determine whether changing FE affects cancer outcomes.

Significant findings could help policymakers and program managers gain knowledge and lay the groundwork for future city planning to integrate a healthy FE. Policymakers could consider instituting an FE-linked policy for improving the community diet. The policy must address other FE-related issues such as nutritional composition,

labelling, promotion, pricing, provision, retail, and investment [73]. In New Zealand, FE policy implementation led to excellent recommendations, such as the implementation of a national action plan for preventing non-communicable diseases, establishing priorities for reducing childhood and adolescent obesity, doubling funding for population nutrition promotion, and reducing the marketing of unhealthy foods [73]. In India, various nutrition interventions were formulated to enhance the FE through systematic planning and embracing differences in the problem for decoding and dividing to simplify resolutions to be addressed by policymakers and nutritionists in the future [74].

A nutritious diet supported by a healthy FE potentially protects against CRC development [75]. A previous research examined the benefit of a specific diet for reducing CRC incidence via food intake with a high omega 3 polyunsaturated fatty acid (PUFA) to omega 6 PUFA ratio and rich in fibre; vitamins B6, C, D, E; folic acid; selenium; and magnesium [76] and the Mediterranean diet, which is high in antioxidant properties. Nevertheless, obtaining and sustaining meals for such diet plans may be difficult [77] and largely depend on the FE. Therefore, the FE is critical in the food system as consumers can decide based on the best available options to support sustainable diets and enhance successful nutrition intervention [20].

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