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Review of the association between meat consumption and risk of colorectal cancer

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ABSTRACT

The incidence of colorectal cancer (CRC) is rapidly increasing in developing countries, especially among populations that are adopting Western-style diets. Several, but not all, epidemiological and experimental studies suggest that a high intake of meat, especially red and processed meat, is associated with increased CRC risk. Potential reasons for the association between high red and processed meat intake and CRC risk include the content of the meat (e.g. protein, heme) and compounds generated by the cooking process (e.g. *N*-nitroso compounds, heterocyclic amines). These factors can affect the large intestine mucosa with genotoxicity and metabolic disturbances. Increased bacterial fermentation (putrefaction) of undigested protein and production of bacterial metabolites derived from amino acids may affect colon epithelial homeostasis and renewal. This correlates with the fact that most colonic cancers are detected in the distal colon and rectum where protein fermentation actively occurs. However, there are still large controversies on the relationship between red meat consumption and CRC risk. Therefore, the purpose of this review is to enhance the current understanding on the association between high red and processed meat intakes with CRC risk. A principal focus of this review will be to discuss the meat-related components, such as proteins in the meat, heme, *N*-nitroso compounds, and heterocyclic amines, and the effects they have upon the large intestine mucosa and the intestinal gut microbiota.

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1. Colorectal cancer and diet

Colorectal cancer (CRC) is one of the leading global causes of cancer-related death. It is estimated that more than 1 million cases and 600,000 deaths occur every year [1]. CRC incidence has been rapidly increasing, especially in economically developed countries [2,3]. The etiology of CRC includes both genetic and environmental factors. Among CRC, only approximately 20% can be attributed to heritable gene variations [4], thus suggesting that the largest fraction of sporadic CRC cases is linked to environmental causes [5]. A subclass of CRC has emerged that is correlated to colitis and chronic inflammatory bowel diseases [6]. A classical model for the genetic changes that occur during colorectal tumorigenesis was proposed in

Abbreviations: ACF, aberrant crypt foci; BCFAs, branched chain fatty acids; CRC, Colorectal cancer; HAAs, heterocyclic aromatic amines; HP, high protein; NOCs, *N*-nitroso compounds; NP, normal protein; SCFAs, short chain fatty acids; SRB, sulfate-reducing bacteria; UC, ulcerative colitis.

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1990 by Fearon and Vogelstein [7]. These genetic and epigenetic alterations impact the normal process of large intestine epithelium renewal [8]. Briefly, these abnormalities interfere with the process of normal epithelial cell division, differentiation, migration, and apoptosis as well as with cell signaling in response to extracellular and intracellular regulators [9-12].

Among the environmental factors that are involved in the modulation of CRC risk, dietary parameters are presumed to play a significant role [13]. The variation of cancer incidence between countries appears largely associated with differences in dietary habits [14]. Several epidemiological and experimental studies have suggested that consumption of a typical Western-style diet that is high in fat and protein significantly increases CRC risk [15], but a high intake of fruits, vegetables, and whole grains is shown to be protective against CRC [16]. However, it is only fair to say that epidemiological studies are markedly dependent on the methodologies used to obtain and analyze dietary and lifestyle information and this can contribute to the variability of the results obtained. Furthermore, epidemiological studies cannot establish causal relationships between a given dietary parameter and the endpoint measured, eg, CRC [17]. Nevertheless, the occurrence of CRC is shown to be significantly increased in Japanese people who have migrated to the United States and adopted a more Western-style diet/lifestyle compared with those living in Japan [18]. This strongly suggests a significant role of the environment in the development of CRC. More recently though, the incidence of CRC has dramatically increased in Japan, which coincides with an overall increased consumption of the Westernized diet [19,20].

2. Meat consumption and CRC

Among all cancers, colon cancer appears to have the strongest association with meat consumption [14,21]. Meat is an important source of dietary protein and essential nutrients, including iron, zinc, and vitamin B_{12}, if consumed in non-excessive amounts. Some of these nutrients (such as iron) are deficient in the diets of many people in numerous countries [22].

Although there is heterogeneity between the different studies [13], several population studies suggest that a high meat intake, especially of red and processed meat, increases CRC risk in a dose-dependent manner [23-25]. Daniel et al found that CRC risk increased linearly with an increased consumption of red/processed meats at up to 140 g/d [26], and the frequency of red meat consumption was more strongly associated with a risk of colorectal carcinogenesis than the amount of meat consumed [27]. However, when the total meat intake was constant, a 10 g/1000 kcal increase in white meat consumption was associated with a significant reduction in the risk of cancer of the colon and rectum [28]. The World Cancer Research Fund and American Institute of Cancer Research report, which is based on an extensive review of the existing evidence by an international panel of experts, concluded that a high intake of red or processed meats is a convincing and probable cause of CRC. The risk of CRC was estimated to increase by 29% for every 100 g/day increase in red meat and by 21% for every 50 g/day increase in processed meat consumption [13]. In a recent prospective study of Danish men and women, the risk for rectal cancer was elevated with a higher intake of pork, while the risk for colon cancer was elevated with a higher intake of lamb. However, no overall association was found between intake of red meat, processed meat, or poultry for colon and rectal cancer [29].

It should be mentioned that the relationship between red meat consumption and CRC is still the subject of intense scientific debate. Recent reviews of prospective epidemiologic studies have concluded that the association between red meat consumption and CRC, when detected, is generally rather weak [30,31]. In a multiethnic cohort with 165,717 participants, it was concluded that there was no role for meat in the etiology of colorectal cancer [32]. Similarly, another study found no association between colorectal cancer risk and intake of meat [33]. Alexander et al also found a lack of a clear dose-response relationship between red meat intake and CRC in an analysis of more than 35 prospective studies [31]. Therefore, it appears that currently available epidemiologic evidence is still not sufficient enough to support an independent positive association between red meat consumption and CRC [34]. However, there is the possibility that an overall association may be modified or confounded by other dietary factors (eg, high intake of refined sugars and alcohol, or low intake of fruits and vegetables) and/or behavioral factors (eg, low physical activity and smoking). Abundant epidemiological evidence has shown a lower risk of colorectal cancer with higher overall levels of physical activity, although the effect on rectal cancer is less clear than the effect on colon cancer [13].

3. The mechanisms of action of meat-related components on CRC development

Several plausible mechanisms to explain the association between meat, and particularly red or processed meat, consumption and CRC risk have been suggested [35-38]. These mechanisms implicate proteins in the meat, heme iron, *N*-nitroso compounds (NOCs), and heterocyclic aromatic amines (HAAs) (Fig. 1). Notably, these different parameters

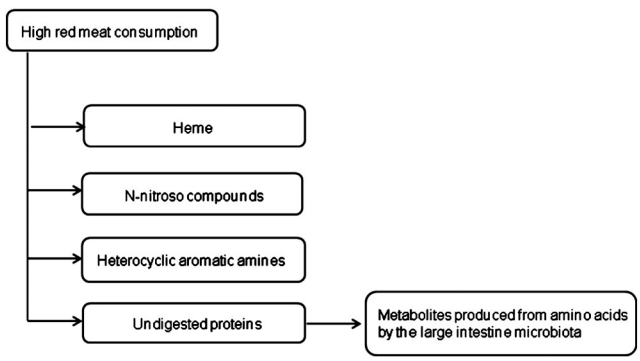

Fig. 1 – Schematic view of compounds associated with high meat consumption which may impact colonic epithelial cell physiology.

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