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ENVIRONMENTAL FACTORS AND COLORECTAL CANCER

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The roles of both chemoprevention and diet are being evaluated as CRC remains the third most common malignancy and second most common cause of cancer-related deaths in the United States.¹ Screening colonoscopy is the most effective form of secondary prevention of CRC due to the early identification of early colorectal cancers and the removal of premalignant polyps.² Colorectal cancer screening guidelines have been published with agreement by multiple medical societies,^{3,4} and yet, less than half of the individuals who meet criteria for screening at age 50 underwent screening in 2005.⁵

Furthermore, certain minority groups are at greater risk for developing CRC when compared to Caucasians. For example, African Americans carry the highest risk for the proximal and aggressive neoplastic compared to all other racial groups. African Americans are more likely to present at earlier ages, later stage cancers, and cancers with more advanced histology.⁶ Hispanics are less likely to be screened for CRC.⁷ In spite of advances in cancer treatment, multi-society guidelines, and improved health care coverage for CRC screening, the racial divide relative to cancer diagnosis and therapy has had little improvement from 1992 to 2002 for certain minority groups.⁸ Other factors that negatively impact CRC screening include fewer years of education, lower socioeconomic status, lack of availability of providers, provider recommendation, decreased patient awareness, increased cost to patients, a higher BMI, and limited or a lack of insurance coverage for screening.^{2,5,9} Therefore, primary prevention is an area of intense investigation. The role of chemoprevention is reviewed in Chapter 5. This chapter, a limited overview of recent and emerging data, will specifically address the role of diet in CRC prevention.

Deciphering the vast body of literature regarding diet and CRC can be overwhelming, unless the studies are reviewed in the context of the adenoma-carcinoma sequence and the time frame required for the adenoma to become a carcinoma for the majority of colon and rectal cancers. In the case of the majority of sporadic polyps (sessile and pedunculated), there is a multistep process that begins with a mutation of the APC gene, is associated with the p53 oncogene and other mutations resulting in the transition from adenoma to carcinoma, occurring over a 10-year period in most cases. In contrast to sessile and