

Preface

As the worldwide pandemic of overweight and obesity continues to expand, with over 1 billion overweight and 315 million obese adults estimated worldwide [1–3], obesity is increasingly recognized as a significant risk factor for cancer. In the USA, approximately 20% of all cancer deaths in women and 14% in men have been attributed to overweight and obesity [4]. Epidemiological studies indicate an association between obesity and specific malignancies in multiple organ systems including colon, postmenopausal breast, endometrial, esophageal adenocarcinoma and renal cell cancer [5–7]. In addition to colon and esophageal adenocarcinoma, more recent evidence supports an association of obesity with other gastrointestinal malignancies including pancreatic, gallbladder, and hepatocellular cancer [4, 7, 8]. In 2009, the American Cancer Society estimated that the combined deaths in the USA from gastrointestinal cancers, including pancreas, colon, and rectum, esophageal, liver, and bile ducts exceeded 135,000 [9]. During the same year, there were 100,000 new cases and 50,000 deaths from colon cancer [9]. The relative risk of mortality from colon cancer according to body mass index in a prospective population study of more than 900,000 US adults [4] was found to be 1.20 for overweight and 1.47–1.84 for obese men, and 1.1 for overweight and 1.3–1.46 for obese women. In contrast, a recent study showed that in men with nonmetastatic colorectal cancer at diagnosis, increased physical activity was associated with improved colorectal cancer mortality and overall mortality [10]. Thus, gastrointestinal cancer in general and colorectal cancer more specifically causes an enormous burden of morbidity and mortality in the USA with significant impact from obesity and benefit associated with physical activity.

Persistence of this vexing problem both at the personal and public health levels can be attributed, in part, to the multicomponent and complex nature of the relation between energy balance and cancer in which many of the cytokines, hormones, and other obesity-associated factors may act in combination with environmental and lifestyle factors as both mutagens and cancer promoters. Persistence of the problem is associated also with the difficulty in implementing effective and sustainable biobehavioral interventions to control obesity and associated mediators and comorbidities.



<http://www.springer.com/978-1-4614-2366-9>

Energy Balance and Gastrointestinal Cancer

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2012, XII, 184 p., Hardcover

ISBN: 978-1-4614-2366-9