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RESEARCH ARTICLE

Young Adult Risk Factors for Cancer: Obesity, Inflammation, and Sociobehavioral Mechanisms

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Introduction: The paper assesses social disparities in the burdens of metabolic and inflammatory risks for cancer in the U.S. young adult population and examines psychosocial and behavioral mechanisms in such disparities.

Methods: Using data of 7,889 individuals aged 12–32 years from the National Longitudinal Study of Adolescent to Adult Health from 1994 to 2009, generalized linear models were used to assess the sex, race/ethnicity, and SES differences in the risks of obesity and inflammation, measured by C-reactive protein. Further tests examined the extent to which social isolation, smoking, physical inactivity, alcohol abuse, and illicit drug use explain social differentials in each biomarker outcome.

Results: Women, blacks, Hispanics, and socioeconomically disadvantaged groups had higher risks of obesity and elevated C-reactive protein, with the SES gradients being more pronounced in female participants. Health-related behaviors showed large variation across sex, race, and SES strata. After adjusting for these behavioral variables, sex, and race disparities in obesity and excess inflammation in blacks diminished, whereas the adolescent SES disparity in obesity remained. The associations of adolescent and young adult SES disadvantage and inflammation were also explained by behavioral mechanisms. Behavioral factors associated with higher risks of obesity and inflammation differed, with the exception of fast food consumption, a risk factor for both.

Conclusions: This study provides new knowledge of social distribution of early life exposures to physiologic precedents to cancer development later in life with implications for prevention and early intervention of modifiable risky behaviors in adolescents and young adults.

INTRODUCTION

Cancer is a major and increasing public health concern.[1,2] Prior research on cancer and its risk factors has focused on later adulthood. However, cancer is a chronic disease of aging that takes decades to develop and manifest. Research suggests that cancer onset is often preceded by a lengthy latency period, with clinically detectable levels of cellular dysfunction often not occurring until years after initial exposure to carcinogenic agents.[3,4] Adolescent and early adult circumstances may have enduring impacts on later life chronic disease outcomes, with implications for cancer in particular.[5,6] Although the specific etiology linking early life circumstances to later life cancer development remains unknown, previous studies indicate that young adulthood exposures to socioeconomic disadvantage, nutrition, physical activity, and risky behaviors, such as cigarette smoking, may all play a role.[7–10]

The links between adolescence and early adulthood and cancer in later life remain understudied. Previous research suggests that certain developmental time periods, particularly the transition from adolescence into

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early adulthood, may represent a "sensitive period" for health. As a developmental turning point, adolescence marks a transition where the environment becomes increasingly important for health as young people have more control over the environment and their behavioral choices.[6,11] A better understanding of the behavioral and psychosocial risk factors in adolescence and young adulthood linked to cancer development later in life would facilitate the development of early interventions to prevent cancer onset.

Given the low incidence rates of cancer in young adulthood, intermediary physiologic pathways involved in carcinogenesis signal the earliest, preclinical stage of the disease process. Obesity and inflammation are two prominent examples of pre-disease pathways amenable to early life course intervention for cancer prevention. Obesity has been linked to increased risks of multiple cancers, accounting for an estimated 20% of all cancer cases.[12–14] Even though the specific metabolic and hormonal mechanisms linking obesity to cancer are under investigation, the high likelihood of adolescent obesity status persisting into adulthood suggests the necessity of reducing obesity risk early on to curtail the development of cancers. Systemic inflammation can act synergistically with obesity to increase cancer risk.[15–17] Although obesity increases low-grade inflammation, the presence of inflammation as indicated by elevated acute-phase protein (e.g., C-reactive protein [CRP]) also plays a crucial role in tumorigenesis independent of obesity.[18,19]

The rate of obesity in adolescents has quadrupled over the past 30 years to 17%, and more-recent cohorts show increased risk of obesity than earlier cohorts, with the increase being particularly sharp for black women.[20–22] Previous studies have documented substantial social differentials in obesity as well as biomarkers of low-grade inflammation, with women, blacks, Hispanics, and those with lower SES at greater risk of obesity[23–25] and elevated CRP.[26–29] Much less is known about patterns of social disparities in the distributions of these biological risk factors for cancer in young adulthood.

Multiple behavioral and psychosocial factors have hypothesized links with inflammation, obesity, and cancer. Cigarette smoking is associated with elevated risks of CRP and a well-established cause of many leading cancers and related mortality.[30–32] Social isolation increases inflammation and the risk of cancer mortality.[33–38] A large body of research shows clear associations between nutritious diets and cancer.[39–41] In fact, obesity and the high-fat, low-vegetable western diet may represent the "largest avoidable cause of cancer in nonsmokers."[14] Physical inactivity may further heighten the risk of various cancers via its effects on adiposity and obesity, as well as immune activation and inflammation.[8] Alcohol consumption and illicit drug use (such as cocaine and opioids) have been linked to certain cancers, although the findings are mixed.[41–44] Individuals of lower social status are disproportionally exposed to adversities and higher levels of social stress that in turn increase disease susceptibilities through harmful behaviors and prolonged physiologic stress response.[45–48] Singular cross-sectional measures of SES are widely used, but fail to capture the dynamic and multidimensional nature of socioeconomic standing specific to each life period. The extent to which disadvantaged and poor adolescent and young adult population in the U.S. suffer from high risks of obesity and inflammation is unknown. The role of early life social behavioral factors in shaping social disparities in biological precursors to cancer is also unclear.

Despite the remaining uncertainty of the mechanisms linking obesity and CRP to cancer causation, research demonstrates clear associations between these biomarkers and tumorigenesis.[1,12–17] Given the rise of obesity and corresponding inflammation burdens in young adulthood, an examination of the population patterns of these biomarkers, as well as modifiable behavioral risk factors, could lead to a better understanding of possible strategies to reduce future disparities in cancer incidence. This study fills this gap using the largest population-based prospective cohort study of adolescents and young adults in the U.S. Also, the study examines the sex, race/ethnicity, and life course SES differences in obesity and CRP. Finally, the study further assesses six health-related behaviors—including social isolation, daily smoking, physical inactivity, consumption of fast food, alcohol abuse, and illicit drug use—as behavioral mechanisms underlying social disparities in obesity and inflammation.

METHODS

Data for the study come from the National Longitudinal Study of Adolescent and Adult Health (Add Health), a nationally representative study of adolescents in Grades 7–12 in the U.S. in 1994–1995 who were followed into adulthood. Add Health used a stratified school-based design and selected a nationally representative sample of all high schools and a feeder school in the U.S. The Add Health cohort was initially surveyed via in-school and in-home questionnaires in 1994–1995 (Wave I), and followed up in 1996 (Wave II); 2001–2002 (Wave III); and finally in 2008–2009 (Wave IV). The current study included 7,889 participants aged 12–19 years at Wave I (adolescence) and followed up when participants were aged 24–32 years in Wave IV (young adulthood) with valid responses on all covariates of interest. High-sensitivity CRP came from assays of dried blood spots collected at Wave IV. The measure of CRP indicates generalized inflammation in the analyses below. Height and weight measured at interviews at both Wave I and IV were used to calculate BMI.[49]

The independent variables and covariates for the present study are drawn from the in-school questionnaire and the in-home interviews at Wave I as well as the in-home interview at Wave IV. All psychosocial and behavioral covariates were collected via