Obesity-Related Cancers Are Increasing in Young Adults

Rates for 6 of 12 obesity-related cancers diagnosed in the United States have been rising in successive generations of young adults, with the steepest increases occurring among the youngest cohorts, according to a study led by the American Cancer Society (ACS).¹

Investigators assessed incidence data for 30 cancers that occurred among individuals aged 25 to 84 years between 1995 and 2014. The data were derived from 25 states representing approximately 67% of the US population using a database provided by the North American Association of Central Cancer Registries. According to the researchers, the study is the first to their knowledge that systematically analyzes US incidence trends for young adults.

The findings demonstrated a significant increase in approximately one-half of the 12 obesity-related cancers, including colorectal cancer, endometrial cancer, gallbladder cancer, kidney cancer, multiple myeloma, and pancreatic cancer, among adults aged 25 to 49 years, with the largest increases noted to occur among the youngest adults. For example, among millennials (those born between 1981 and 1996), the risk of colorectal, endometrial, pancreatic, and gallbladder cancer is approximately double what it had been for baby boomers (those born between 1946 and 1964) when they were the same age. Researchers note that over the past several decades, the obesity epidemic has led to increases in adiposity earlier in life for younger generations. That adiposity, particularly during important developmental periods, may increase cancer risk, the authors say.

The researchers also assessed incidence rates for 18 cancers not connected to obesity, including those related to smoking and infection, among younger birth cohorts. However, they found that rates had either declined or stabilized in all but 2 cancers.

Ahmedin Jemal, DVM, PhD, senior vice president of the Surveillance and Health Services Research Program at ACS, who served as senior and corresponding author of the study, notes that although the absolute risk of obesity-related cancers is small in younger adults, the public health community still must consider and address the implications of growing obesity rates in young generations and their link to obesity-related cancers. As younger cohorts age, the rate of such cancers may rise, affecting progress that has been made in reducing cancer mortality, he adds.

Reference

DOI: 10.1002/cncr.32299

Protein May Be Linked to Racial Disparities in Prostate Cancer

A new study published in Cancer Research suggests that certain cellular mechanisms may help to explain why African American men with prostate cancer tend to develop greater therapeutic resistance and faster disease recurrence than their white counterparts.¹ Furthermore, according to investigators, the findings could create a pathway for new therapeutic targets.

The study was conducted by researchers from Roswell Park Comprehensive Cancer Center in Buffalo, New York, with support from the National Cancer Institute and the American Cancer Society. It found that prostate cancer cells from African American men tended to have higher expression of the cancer promoters c-Myc and NF-kB, which leads to the inhibition of a mitochondrial protein known as cytochrome c. Cytochrome c is needed for the formation of apoptosome, a large protein complex that helps an organism to eliminate cells; without it, tumor cells stand a better chance of surviving. Although cytochrome c deficiency can occur in individuals of any race, it is more common in those of African ancestry, according to lead author Dhyan Chandra, PhD, and his team.

Because standard prostate cancer therapies are often less effective in African American men, researchers say the study’s findings indicate the need for new therapies that inhibit c-Myc and NF-kB and can restore cytochrome c in prostate cancer cells that have the deficiency. They plan to develop clinical trials to test the effectiveness of drugs that are used for other applications but can inhibit c-Myc and NF-kB in the treatment of prostate cancer.

Reference

DOI: 10.1002/cncr.32300

Content in this section does not reflect any official policy or medical opinion of the American Cancer Society or of the publisher unless otherwise noted © American Cancer Society, 2019.