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Childhood obesity 2020: Evaluating the expression of known pro-inflammatory, obesity and cancer markers in South Carolinian children- Kaya Stokes- South Carolina State University

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In the US, childhood obesity has been a growing epidemic with, 1/3 folks children considered overweight or obese. The increased number of overweight and obese children can be linked to several factors including nutrition and social economic status. Households that do not have access to healthy, nutritious foods are significantly more likely to be obese earlier in life than other children. Obesity in children can lead to numerous health complications such as diabetes, high blood pressure, chronic inflammation and carcinogenesis. African American minorities are more likely to be diagnosed and die from one of the various forms of cancer. Therefore, eliminating or reducing preventable risk factors like unhealthy nutrition and childhood obesity could have important implications for reducing clinical manifestations of adult cancer outcomes. In order to understand the implication of inflammation in the participants, we first analyzed the expression of the inflammation biomarkers in prostate cancer cells, used as our baseline data. The pro-inflammatory markers and obesity related genes investigated include adiponectin, leptin, CRP, VEGF, SAA1 /2, Interleukin 1 and 6. The transcriptional levels of pro-inflammatory genes was measured by quantitative Real-time polymerase chain reaction. The results indicated that the expression of chronic inflammation markers were increased in cancer DNA as compared normal DNA. Overall, reducing childhood obesity and pro-inflammatory diets, while increasing physical activity and access to healthy foods are beneficial in the reduction of cancer risk and will serve as preventive measures for early-stage onset of adult cancers. Work funded by USDA/NIFA Grant Number SCX-311-20-16.Worldwide there are 1.1 billion overweight people with a BMI between 25 kg/m2 and 30 kg/m2 and 312 million with a BMI > 30 kg/m2 .The American Cancer Society calculates that currently new cancer cases are in the order of 1.5 million with 0.5 million cancer deaths per year, nearly 1 in 5 due to obesity. A large number of epidemiological studies link obesity/metabolic syndrome/diabetes-associated diseases to an increased risk for the event of several sorts of cancer, particularly gastrointestinal, glandular, and reproductive tract cancers. In addition, obesity can cause poorer treatment outcomes, worsened prognosis, and mortality. A comprehensive systematic review of the evidence by the planet Cancer Research Fund (ECRF) and therefore the American Institute for Cancer Research (AICR) concluded that obesity is a longtime risk factor for several cancers. In a standardized meta-analysis of prospective observational studies by Renehan et al., 2008 (1966–2007: 221 datasets; 282, 137 incident cases; 20 cancer types), quantifying associations between a 5 kg/m2 increase in BMI and risk of incident cancer, showed that, in men, the increase was associated with (a) oesophageal adenocarcinoma (RR 1.52,); (b) thyroid (1.33,); (c) colon (1.24,); and (d) renal (1.24,) cancers. In women, the association was found for (a) endometrial (1.59,); (b) gallbladder (1.59,); (c) oesophageal adenocarcinoma (1.51,); and (d) renal (1.34,) cancers. Weaker positive associations () were found in men with rectal cancer and malignant melanoma and in women with postmenopausal breast, pancreatic, thyroid, and colon cancers. They also found an association for both sexes with leukemia, multiple myeloma, and non-Hodgkin lymphoma. The associations were generally similar in studies from North America, Europe, and Australia, as well as the Asia-Pacific region. Studies with long-term followup of patients undergoing bariatric surgery for morbid obesity showed a discount in cancer incidence in women related to sustained weight loss supporting a causal association between obesity and cancer risk.One of the major challenges in the association of obesity and cancer has been linking the epidemiology with the biological basis. Biological mechanisms underlying the connection between obesity and cancer are poorly understood. Of the most studied candidates for this association are the energy balance-associated factors (adipokines, growth factors, hormones, and their cell signaling pathways) and other emerging candidates include obesity-associated hypoxia, genetic susceptibility, adipose stromal cells, and inflammatory processes. Several energy balance-related factors are known to influence tumor progression and these are implicated as contributors to the consequences of obesity on cancer outcome. These factors include leptin, adiponectin, steroid hormones, insulin, insulin-like growth factor-1, and sirtuins. The links between obesity and inflammation and between chronic inflammation and cancer suggest that inflammation might be important in the obesity-cancer link. Changes in the adipose tissue during the process of going from lean to obese, including modulation of adipokine levels, hypoxia, ROS, FFA, and ER stress, might lead to a chronic state of inflammation in the obese individual. The increased risk of obesity-related cancers could be mediated in part by these changes in the adipose tissue. Some of the most important elements of this association are, among others, insulin resistance; overexpression of leptin, inflammatory cytokines, sex hormones, transcriptions factors like NF-κB, AP-1, STAT3, and oxidative stress; and downregulation of the expression of anti-inflammatory factors like adiponectin and PPARγ, which disrupt the balance between cell proliferation and apoptosis. Accumulating evidence indicates that chronic inflammatory states in the obese might be associated with esophageal, liver, colon, postmenopausal breast, and endometrial cancers. The association between obesity, inflammation, and other cancers

This work is partly presented at 13th International Conference on Childhood Obesity and Nutrition, March 16-17, 2020 at London, UK

like prostate, renal, gastric, pancreatic, and gallbladder has been addressed in other papers. As more data accumulates and the molecular mechanisms between some of these factors and carcinogenesis start to unravel, the prospect of anti-inflammatory cancer prevention becomes an important goal in research.

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