PR01   Estimating the current and future cancer burden attributable to obesity in Canada. Darren Brenner[1], Christine M. Friedenreich[2], Yibing Ruan[2], Abbey E. Poirier[2], Xin Grevers[2], Stephen Walter[3], Will King[4], Paul Demers[5], Paul Villeneuve[6], Eduardo Franco[7], Robert Nuttall[8], Leah Smith[9], Prithwish De[5]. [1]University of Calgary, Calgary, AB, Canada, [2]Alberta Health Services, Calgary, AB, Canada, [3]McMaster University, Hamilton, ON, Canada, [4]Queen's University, Kingston, ON, Canada, [5]Cancer Care Ontario, Toronto, ON, Canada, [6]Carleton University, Ottawa, ON, Canada, [7]McGill University, Montreal, QC, Canada, [8]Canadian Cancer Society, Toronto, ON, Canada, [9]Canadian Cancer Society, Toronto, ON, Canada.

Background: It is well recognized that excess body weight is strongly associated with several types of cancer. With an increasing prevalence of excess body weight in Canada, it is important to quantify the current and future cancer burden consequent to this risk factor.

Methods: As a collaborative group from multiple institutions across Canada, we are conducting the Canadian Population Attributable Risk of Cancer Project (ComPARe). The aim of the ComPARe project is to estimate the current and future cancer burden linked to modifiable lifestyle and environmental risk factors. We conducted extensive literature searches to identify the convincing and probable associations between excess body and the risk of specific types of cancer. We defined excess body weight as the combination of overweight (Body Mass Index [BMI] 25-30) and obesity (BMI >30). Reliable prevalence data were collected from Canadian population-based surveys, and age-sex-specific incidence data were obtained from the Canadian Cancer Registry. We also conducted sensitivity analyses to adjust for potential biases due to self-reporting. Future cancer burden attributable to excess body weight was estimated by combining projected overweight and obesity prevalence trends with projected cancer incidence trends (to 2042), based on historical data. To estimate the future potential for cancer prevention, we modeled the potential impact fraction of the associated cancers based on counterfactual interventions to reduce the prevalence of both overweight and obesity by 10%, 25%, and 50%.

Results: The population attributable risk for excess body weight was 9.4% among the 14 associated cancer sites in Canada in 2012. An estimated 3,886 cancer cases in men and 4,740 in women were attributable to excess body weight. Sensitivity analysis addressing possible self-report bias indicates that the attributable cases could be as high as 4,648 in men and 5,868 in women, or 11.5% of the associated cancer cases. From modeling the past prevalence in Canada from 1994 to 2011, we project that the prevalence of overweight and obesity will continue to rise over the next 15 years, leading to a higher cancer burden in the future. If we were able to intervene on excess body weight and reduce the prevalence of both overweight and obesity by 10%, 25%, and 50%, we would be able to prevent 1,660, 4,150, and 8,300 cancer cases in 2042, and a cumulative number of 11,966, 29,914, and 59,829 cancer cases by 2042, respectively.

Conclusions: Excess body weight contributes to a substantial and increasing number of cancers in Canada. Interventions impacting excess body weight have the potential to prevent tens of thousands of cancer cases in Canada over the next 30 years. Effective policies and public health interventions to reduce excess body weight are urgently needed.

This abstract is also being presented as Poster A02.

PR06   Body fat and risk of breast cancer in normal-size postmenopausal women. Neil Iyengar[1], Rhonda Arthur[2], JoAnn E. Manson[3], Candyce H. Kroenke[4], Lindsay Peterson[5], Ting-Yuan D. Cheng[6], Rowan T. Chlebowski[7], Elizabeth C. Feliciano[4], Dorothy Lane[8], Juhua Luo[9], Rami Nassir[10], Kathy Pan[11], Sylvia Wassertheil-Smoller[2], Thomas E. Rohan[2], Andrew J. Dannenberg[12]. [1]Memorial Sloan Kettering Cancer Center, New York, NY, [2]Albert Einstein College of Medicine, Bronx, NY, [3]Brigham and Women's Hospital, Harvard Medical School, Boston, MA, [4]Kaiser Permanente, Oakland, CA, [5]Washington University in St. Louis, St. Louis, MO, [6]University of Florida, Gainesville, FL, [7]City of Hope National Medical Center, Duarte, CA, [8]Stony Brook University School of Medicine, Stony Brook, NY, [9]Indiana University, Indianapolis, IN, [10]University of California Davis, Davis, CA, [11]Harbor-UCLA Medical Center, Torrance, CA, [12]Weill Cornell Medical College, New York, NY.

Background: Body mass index (BMI) is widely used to estimate body fat levels. Individual health recommendations are often based on BMI. Obesity, defined as BMI >30 kg/m^2, is associated with increased risk of estrogen receptor (ER)-positive breast cancer in postmenopausal women. However, BMI is an inexact measure of body fat. In individuals with normal BMI, it is unknown whether body fat levels contribute to breast cancer risk. Similarly, the potential role of systemic factors in the pathogenesis of breast cancer in normal BMI postmenopausal women is uncertain.

Methods: We examined the association between body fat levels, measured using dual energy X-ray absorptiometry (DXA), and breast cancer risk among women with normal BMI. The study, conducted in the Women's Health Initiative, included 3,460 postmenopausal women with BMI 18.5 to <25.0 kg/m^2 who had baseline DXA measurements and no history of breast cancer. By the median follow-up of 16 years, 182 incident invasive breast cancers had been ascertained, of which 146 were ER-positive. Multivariable adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) for the associations between body fat measures and risk of invasive breast cancer were estimated using Cox proportional hazards regression. Geometric mean concentrations of a variety of blood analytes, including insulin, C-reactive protein (CRP), interleukin-6 (IL-6), leptin, SHBG, HDL-cholesterol and triglycerides, were also determined.

Results: Multivariable-adjusted HRs for invasive breast cancer were 1.70 (95% CI 1.06 to 2.72) and 1.75 (95% CI 1.09 to 2.81) in the highest (versus lowest) quartile of whole body fat mass and trunk fat mass, respectively. Adjusted HRs for ER-positive breast cancer were 2.10 (95% CI 1.23 to 3.58) and 1.91 (95% CI 1.13 to 3.23) in the highest versus lowest quartiles of whole body fat mass and trunk fat mass, respectively. The associations remained statistically significant after additional adjustment for waist-hip ratio. Age and race/ethnicity-adjusted circulating levels of metabolic and inflammatory factors including insulin, CRP, IL-6, leptin, and triglycerides were higher in the upper versus lower quartiles of trunk fat mass (Ps<0.01). Levels of HDL-cholesterol and SHBG were lower in the upper versus lower quartiles of trunk fat mass (Ps<0.01).

Conclusions: In postmenopausal women with normal BMI, high body fat levels were associated with elevated risk of ER-positive breast cancer and altered levels of circulating metabolic and inflammatory factors. These results suggest that normal BMI is an inadequate proxy for the risk of breast cancer associated with body fatness in postmenopausal women.

This abstract is also being presented as Poster B22.

PR07   Obesity and metabolic syndrome correlate with poor oncologic outcome in prostate cancer patients who underwent radical prostatectomy. Arash Samiei[1], Ralph Miller[1], John Lyne[1], Shifeng Mao[2]. [1]Allegheny Health Network, Department of Urology, Pittsburgh, PA, [2]Allegheny Health Network, Department of Medical Oncology, Pittsburgh, PA.

Background: Prostate cancer is the most common cancer in males and the second leading cause of cancer death in the U.S.A. Studies investigating a link between obesity and metabolic syndrome with biochemical recurrence (BCR) after radical prostatectomy (RP) have yielded mixed results. Comparative studies with long-term follow-ups in different prostate cancer risk groups are still lacking.

Objective: To investigate the association of body mass index (BMI) and metabolic syndrome (MetS) with oncologic outcome in high- , intermediate- , and low-risk prostate cancer patients who underwent RP.

Material and Methods: This was a retrospective study of all RPs that were performed by two trained surgeons in a single center from 2003 to 2013. Preoperative and postoperative data (fasting glucose, triglycerides, and HDL, blood pressure, preoperation PSA, Gleason score (GS), pathologic stage, biochemical recurrence time, surgical margin status, preoperative BMI) were analyzed. Patients were categorized into three risk groups (high, intermediate, and low risk) according to the National Comprehensive Cancer Network (NCCN). MetS was defined according to the WHO classification, and BCR was defined as two consecutive PSA >0.2 ng/mL postoperatively.

Results: A total of 1100 prostatectomies were included in this study with a median (mean) follow-up time of 44 (48) months. The median (mean) age at the time of diagnosis was 61 (60) years old. We found more obese patients (BMI >30 kg/m^2) in the high-risk prostate cancer group compared to low/intermediate group (41.2% vs. 32.0%, p=0.006). There was no statistically significant difference between surgical margin status in each of the risk groups. Metabolic syndrome (p<0.001) and a BMI >30 (p=0.028) were associated with a higher GS. Data showed a significantly higher BCR in patients with a BMI >30 compared to patients with BMI <30 (32.4% vs. 16.9%, p<0.001). A multiple logistic regression model was also used to see whether metabolic syndrome increased the failure adjusted for different risk groups. We found patients with metabolic syndrome had a 4-fold higher risk of BCR compared to patients without metabolic syndrome (OR: 4.06, P <0.001).

Conclusion: High BMI correlates with higher risk of biochemical recurrence in prostate cancer patients following prostatectomy. In addition, metabolic syndrome significantly increases the BCR risk in prostate cancer patients who underwent radical prostatectomy. For this reason, obese individuals undergoing radical prostatectomy need more focused follow-up care.

This abstract is also being presented as Poster B27.

PR11   Sleep duration and circadian components of the sleep/wake cycle and their association with body mass index and eating behaviors in children. Bernard Fuemmeler[1], Jessica Lunsford-Avery[2], Bruno da Silva Brandao Goncalves[3], ShanShan Chen[1], Julia Schechter[2], Yaou Sheng[1], Rachel Maguire[4], Sierra Tolbert[2], Victoria Saba[2], Caroline Barry[5], Aleisha Majors[2], Elizabeth Do[1], Cathrine Hoyo[4], Susan Murphy[2], Scott Kollins[2]. [1]Virginia Commonwealth University, Richmond, VA, [2]Duke University, Durham, NC, [3]Instituto Federal Sudeste de Minas Gerais, Barbacena, Brazil, [4]North Carolina State University, Raleigh, NC.

Purpose: Overweight children are more likely to become overweight adults, increasing their risk for obesity-related cancers in adulthood. Understanding risk factors contributing to greater body weight in childhood may help inform obesity prevention strategies that could be implemented earlier in development. Increasing evidence suggests a connection between sleep patterns and obesity in adults; however, few studies have assessed these links in children. The primary purpose of this study was to examine sleep duration and circadian components of the sleep/wake cycle and their association with body mass index (BMI) and eating behaviors in children.

Methods: The study sample were children born of mothers from the Newborn Epigenetic Study, a prebirth cohort. The sample included a subset of children (n=92) who are being followed in a larger study examining prenatal factors contributing to childhood obesity. To be included in these analyses children had to have had weight/height data, eating behavior data, and data from hip-worn accelerometers worn continuously for 24 hours over at least a 5-day period. Children's weight and height were measured and they completed the eating in the absence of hunger (EAH) task—a lab-based assessment of how much children eat beyond satiety from an ad libitum meal. Accelerometer data were analyzed using established algorithms to derive several parameters assessing sleep and circadian components of the sleep/wake cycle. Regression analyses controlling for age, sex, and race were performed, evaluating the association between several components of the sleep/wake cycle and BMI z-score and calories consumed in the EAH task.

Results: Children were on average 8 years of age (SD=1.89), 62% African American, 38% White, and 51% boys. A shorter sleep duration (hours) was associated with a higher BMI z-score (ß = -0.17, p=0.03). With respect to circadian sleep/wake components, more fragmented circadian rhythms, and increased intradaily variability (a measure of rest-activity rhythm fragmentation) were associated with a higher BMI z score (ß = -1.87, p=0.03 and ß = 1.46, p=0.05, respectively). With respect to eating behaviors, a later onset of diurnal activity was associated with greater caloric intake in absence of hunger on the EAH task (ß = -0.001, p=0.01).

Conclusion: These data support links between childhood body weight, obesity-related eating behaviors, sleep duration, and circadian components of the sleep/wake cycle. Targeting only sleep duration as a childhood obesity prevention strategy may be sufficient, but it may be necessary to target other circadian components of the sleep/wake cycle.

This abstract is also being presented as Poster A01.