SESSION 9

The Future of Tumor Prevention: Consens and Controversy

S26. Overweight, Physical Activity and Cancer Prevention

I. Thune

Norwegian Cancer Society Faculty of Medicine, Oncology, Tromso, Norway

Introduction: A positive energy balance characterised by overweight and physical inactivity may influence complex and varying biological mechanisms (altered endogenous hormonal levels, immune system functioning, storing of carcinogenic agents etc) of importance in cancer prevention.

Methods: In large epidemiological studies body mass index (BMI) is the measurement most used when studying the association between overweight and site-specific cancer risk. More limited data exist on weight change, waist and hip circumferences and skinfold thickness as indicators of body fat distribution. Questionnaires including work and/or leisure time activity are the methods most often used when studying the association between physical activity and site-specific cancer risk. Various components of physical activity such as type, frequency, intensity and lifetime physical activity have been recorded. Recently, methodological improvements in assessing physical activity, combined with inclusion of physiological markers (heart rate, hormonal levels, body composition) have been developed.

Results – Overweight: Data from meta-analyses, numerous case-control and cohort studies support that both overweight (BMI > 25kg/m^2) and obesity (BMI > 30kg/m^2) are associated with increased site-specific cancer risk. Effect modifiers and confounding effects such as diet, physical activity and obesity are often included as covariated in the analyses. Obesity are associated with a two-fold increased risk of colon cancer among men, somewhat weaker for women, an increase in postmenopausal breast cancer, two-fold increased risk of cancers of oesophagus and gastric cardia, a 84% increase in renal cell cancer and a three-fold increase in endometrial cancer. In contrast, an inverse association has been observed between obesity and premenopausal breast cancer risk. No consistent association has been observed between obesity and cancer of the rectal, ovarian, prostate, lung and head and neck. Physical inactivity: The evidence from observational studies support that both leisure time and occupational physical activity protect against site-specific cancer risk, with a dose-response association suggested in both sexes, but varying by cancer site. The observed protective effect of physical activity on colon cancer risk in men (20-60%) and women (20-30%) is supported by hypothesised and identified biological mechanisms as well as a dose-response association observed in most observational studies. In contrast, no clear association was observed for rectal cancer for either sex. The observed association between physical activity and breast cancer (pre- and post menopausal) (2-50%) and endometrial cancer risk (10-30%) is weaker than that for colon cancer, possibly dependent on age at exposure, age at diagnosis, menopausal status and other effect modifiers, e.g., body mass index. However, hypothesised and identified biological mechanisms confirm the protective effect of physical activity on breast and endometrial cancer risk. Further data concerning carcinoma of other cancers (prostate, lung, ovary and renal cells, testicular cancers) are required. The optimal permutation of type, intensity, duration and frequency of physical activity across the lifespan is gender-, age-and site-specific.

Conclusion: At present, a consistent association has been observed between overweight/obesity and cancers of the colon, renal cells, oesophagus, gastric cardia, postmenopausal breast, endometrium and thyroid (women) supported by identified biological mechanisms. Is has been estimated that population attributable risk (PAR) due to overweight range from 9%; postmenopausal breast cancer up to 39%; endometrial cancer. Furthermore the protective effect of physical activity on colon, breast and endometrial cancer risk is supported by identified biological mechanisms. Discrepancies between studies elaborating on the association between physical activity and site-specific cancer risk may be explained by real differences or lack of information on the various components of physical activity (type, intensity, duration, frequency) or incomplete information about the cancer type studied (localisation, histology).