

## 4.4 PHARMACOEPIDEMOLOGY

Chair: Dr. John Frank, Canada

**04-4.1 NON-STEROIDAL ANTI-INFLAMMATORY DRUG AND ASPIRIN USE AND THE RISK OF HEAD AND NECK CANCER**

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**Introduction** The use of non-steroidal anti-inflammatory drugs (NSAIDs) has been associated with a reduced risk of several cancers. Evidence for NSAIDs preventing head and neck cancer (HNC) is inconclusive. We conducted a prospective cohort study to examine the association between NSAID use and HNC risk.

**Methods** Using data from the National Cancer Institute (NCI) Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer Screening Trial, we examined the association between aspirin / NSAID use and HNC incidence among 142 034 men and women aged 55–74 years. Information regarding regular use and frequency of use of aspirin and NSAIDs over the last 12 months was reported at enrolment. (1993–2001). Individuals were followed-up until 2006. HRs and 95% CIs were calculated using multivariable cox proportional hazards regression with adjustment for potential confounders including tobacco use, gender, body mass index and age.

**Results** Over the follow-up period 316 individuals were diagnosed with HNC. Regular aspirin use, compared to non-use, was associated with a significantly reduced incidence of HNC (Adjusted HR 0.78; 95% CI 0.62 to 0.98). No association was observed with regular NSAID use, compared to non-use, and HNC incidence (adjusted HR 0.99, 95% CI 0.76 to 1.28).

**Conclusions** Our study suggests that aspirin may have potential as a chemopreventative agent for HNC however further investigation is warranted.

**04-4.2 CHOLESTEROL-LOWERING DRUGS AND INCIDENT OPEN-ANGLE GLAUCOMA**

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**Purpose** To determine the association between the use of statins and non-statin cholesterol-lowering drugs and incident open-angle glaucoma.

**Methods** In a prospective population-based cohort study among 3939 participants aged 55 years and above, ophthalmic examinations including measurement of the intraocular pressure, assessment of the optic nerve head and perimetry were performed at baseline and after an average follow-up duration of 9.8 years. The use of statins and non-statin cholesterol-lowering drugs was monitored continuously during follow-up. Associations between incident glaucomatous visual field loss and the use of statins and non-statin cholesterol-lowering drugs were assessed using cox-regression models adjusted for age, gender, intraocular pressure lowering treatment and potential (mainly cardiovascular) confounders.

**Results** During follow-up, 108 participants (2.7%) developed glaucomatous visual field loss. The HR for statin use was 0.56 (95% CI 0.32 to 0.99;  $p=0.045$ ) and for non-statin cholesterol lowering drugs 1.82 (0.71 to 4.66;  $p=0.21$ ). There was a significant trend towards a reduced risk of developing OAG with prolonged statin use (HR 0.89,

95% CI 0.41 to 1.93 for use during 2 years or less; HR 0.44, 95% CI 0.22 to 0.89 for use during more than 2 years).

**Conclusions** Long-term use of statins seems to be associated with a reduced risk of open-angle glaucoma. This result is consistent with an earlier study and suggests that statins should be further explored as a new class of medications for the treatment of glaucoma, especially for those patients in whom disease progression continues despite an apparently sufficient intraocular pressure reduction.

**04-4.3 CANCER INCIDENCE AND INSULIN THERAPY IN A COHORT OF DIABETIC PATIENTS**

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The risk of incident cancer seems increased in patients with type 2 diabetes (T2D) and therapeutic regimens (metformin; insulin, analogues) may be involved. We investigated in a cohort of 26 742 T2D patients from a statewide disease management program the risk of incident cancer over a median follow-up time of 3.5 years. Data from all T2D patients in the age 40–79 years residing in the Muenster District were linked to cancer cases in the population-based regional cancer registry. Invasive cancer cases were identified using probabilistic record linkage procedures and pseudonymised personal identifiers, including only first cancers but no DCO cases. Censuring date was 31 December 2008. We computed standardised incidence ratios (SIR) and employed Cox regression models. We identified 759 first cancers among male T2D patients (18.7 per 1000 py) and 605 among females (12.7 per 1000 py). Relative to the general population, the risk of any incident cancer was raised (SIR 1.14; 95% CI [1.10 to 1.21]), it was particularly high for cancer of the liver (SIR 1.95 [1.18 to 2.99]) and pancreas (SIR 1.45 [1.07 to 1.92]). In Cox models, adjusting for diabetes duration, body mass index and sex, insulin therapy was related to higher cancer risk (HR 1.69 [1.55 to 1.84]). No effect was seen for metformin. Limitations relate to lack of numbers for analysing specific cancer types and lack of detail on medication type, duration and dosage. Our results seem to confirm previous reports of increased cancer risk with insulin therapy.

**04-4.4 EXPOSURE TO CYCLO-OXYGENASE-2 INHIBITORS AND RISK OF CANCER: NESTED CASE-CONTROL STUDIES**

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**Introduction** Selective cyclo-oxygenase-2 (COX2) inhibitors are a widely used analgesic for patients with intolerance to traditional non-steroidal anti-inflammatory drugs and it is unclear how long-term use affects cancer risk.

**Methods** A series of nested case-control studies were conducted using data from 574 UK general practices in the QResearch primary care database. All patients diagnosed with cancer between 1998 and 2008 were matched with up to 5 controls. Associations of COX2 inhibitors with risk of all cancers and 10 site-specific cancers (breast, prostate, lung, colorectal, haematological, bladder, melanoma, gastric, pancreatic and oesophageal) were estimated using conditional logistic regression adjusted for co-morbidities, smoking status, socio-economic status and use of non-steroidal anti-inflammatory drugs, aspirin and statins.

**Results** 88 125 cases with cancer and 362 254 matched controls with at least 6 years of records were analysed. Use of COX2 inhibitors for more than a year was associated with significantly increased overall risk of cancer (OR 1.06, 95% CI 1.03 to 1.09), particularly breast cancer (OR 1.24, 95% CI 1.08 to 1.42) and haematological