

PWE-117

**DIETARY FAT INTAKE IN THE AETIOLOGY
OF BARRETT'S OESOPHAGUS AND
OESOPHAGEAL ADENOCARCINOMA:
DATA FROM A PROSPECTIVE COHORT STUDY
(EPIC-NORFOLK) USING 7-DAY FOOD DIARY
DATA**

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Introduction Dietary fat may be involved in the aetiology of both Barrett's oesophagus (BO) and oesophageal adenocarcinoma (OAC) through its direct and indirect effects on increasing oesophageal reflux and second the release of adipose derived hormones. The aim of this investigation was to conduct the first prospective cohort study of dietary fat in the aetiology of these diseases, using nutritional data derived from 7-day food diaries (7-DFD).

Methods A total of 23 658 healthy men and women were recruited into EPIC-Norfolk (European Prospective Investigation In to Cancer and Nutrition) between the years 1993 and 1997. Participants completed 7-DFDs at recruitment which recorded detailed information on food types consumed, brands, quantities and frequency of intake and cooking methods. The diaries were coded by nutritionists using a computer programme containing information on 11 000 food items and 55 000 portion sizes. Participants were followed up to identify those who had a new diagnosis of either BO or OAC and the diagnoses confirmed by reviewing the medical records. Hazard ratios were estimated using Cox regression for quintiles of total fat, saturated fat and total polyunsaturated fat (PUFA) intakes, for BO and OAC respectively, adjusted for age, gender, smoking, body mass index and total energy intake.

Results In the cohort, 80 participants had endoscopy showing a new diagnosis of BO (80% men, median age=69.4 years range 41.3–84.4 years) and a further 58 were diagnosed with OAC (84% men, median age=73 years, range 52–86 years). The risk of OAC was positively associated with a higher fat intake (highest vs lowest quintiles HR=3.77, 95% CI=0.83 to 17.03, $p=0.085$, trend HR=1.54, 95% CI=1.08 to 2.19) and saturated fat intake (trend HR=1.35, 95% CI=1.01 to 1.79), but not with PUFAs (trend HR=1.11, 95% CI=0.87 to 1.42). For BO, there were no associations with either: total fat, saturated fat or PUFAs.

Conclusion The data, together with plausible biological mechanisms, support a role for total fat and saturated fatty acids in the aetiology of OAC. Their role in BO needs further clarification as this sub-group were participants diagnosed as a result of developing symptoms and undergoing gastroscopy. Future epidemiological work should measure dietary fat intake when investigating the aetiology of this aggressive cancer. This has implications for potential public health interventions lowering dietary fat to reduce the incidence of OAC.

Competing interests None.

Keywords Aetiology, Barrett's oesophagus, Diet, oesophageal adenocarcinoma.