Late Breaking Abstracts

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108-LB

Alcohol Consumption, Plasma Fetuin-A and Risk of Type 2 Diabetes in Women

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Benefits of moderate alcohol consumption on type 2 diabetes have been well-documented and postulated to involve a mechanism of improved insulin sensitivity. Fetuin-A, a liver-derived protein that inhibits insulin signaling, has emerged as a biomarker associated with type 2 diabetes risk. Therefore, alcohol intake may influence circulating fetuin-A concentrations and subsequently diabetes risk through altering insulin signal. We hypothesized that moderate alcohol consumption would be associated with lower plasma fetuin-A and that fetuin-A would partly explain the association between alcohol consumption and type 2 diabetes in mid-aged and older women. Multiple linear regression was conducted among the Nurses' Health Study female participants with measures of plasma fetuin-A and alcohol consumption (n=1381). The proportion of alcohol consumption and type 2 diabetes association explained by fetuin-A was assessed within 470 matched incident diabetes case-control pairs from 2000 to 2006. Higher total alcohol intake was associated with lower plasma fetuin-A (p-trend=0.006): Least-squares means±SE 476.8±5.7μg/mL for abstainers, 469.0±5.1 μg/mL for 0.1-4.9 g/d consumers, 456.4±6.9 μg/mL for 5.0-14.9 g/d, and 449.3±9.2 μg/mL for ≥15 g/d. The association between alcohol consumption and diabetes explained by fetuin-A and fasting insulin were 18.3 % (95% CI 0.1-36.4) and 65.2 % (14.7-115.6) (both p-contribution<0.05), while liver enzymes were not a significant contributor of this association. Further, fasting insulin explained 61.7 % (25.7-97.8) of the association between fetuin-A and diabetes (p-contribution=0.0008). In conclusion, moderate total alcohol consumption is associated with lower plasma fetuin-A concentrations in women. Fetuin-A and insulin explain a significant proportion of the association between alcohol consumption and type 2 diabetes in this population. Further studies are needed to determine whether there are biological mechanisms underlying this association.

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109-LB

Prospective Study of Fast-Food Consumption and the Risk of Gestational Diabetes: The SUN Cohort

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Little is known about the influence of fast-food consumption on incident gestational diabetes mellitus (GDM). Therefore, our objective was to evaluate the association between fast-food consumption and GDM in a cohort of university graduates.

The prospective dynamic SUN cohort included data of 2903 women free of diabetes or previous GDM who reported at least one pregnancy between 1999 and 2010. Fast-food consumption was assessed through a validated semi-quantitative food frequency questionnaire. Fast-food was defined as the consumption of hamburgers, sausages, and pizza. Three categories of fast-food were established: low (0-3 servings/month), intermediate (>3 servings/month-2 servings/week) and high (>2 servings/week). Non-conditional logistic regression models were used to adjust for potential confounders.

We identified 169 incident cases of GDM during follow-up. After adjusting for age, baseline body mass index, smoking, physical activity, alcohol intake, fiber intake, Mediterranean dietary pattern, soft drinks consumption, family history of diabetes, cardiovascular disease and hypertension at baseline, and parity, regular fast-food consumption was significantly positively associated with incident GDM. Women in the intermediate category of consumption had an adjusted OR of 1.35 (95% CI 0.84-2.17) and those in the highest category had an adjusted OR of 1.77 (95% CI: 1.08-2.91) compared with the low consumption group; p for linear trend: 0.018.

Our results suggest that pre-pregnancy higher consumption of fast-food (defined as the consumption of hamburgers, sausages, and pizza) was a risk factor for GDM.

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EPIDEMIOLOGY—OTHER

110-LB

The Risk of Fractures after Initiating Oral Anti-Diabetic Drugs: Results from the National Claim Registry

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Thiazolidinedione (TZD) increases fracture risk. However, the effect of other oral anti-diabetic drugs (OADs) on fracture risk is not well known. We examined the risk of fractures after initiating OADs using the nationwide database of medical and pharmacy claims in South Korea. Among 2,886,555 subjects with antidiabetes prescriptions, 207,558 subjects aged 50 years and older, who initiated OADs from January 2008 to June 2011, were analyzed. Based on medication possession ratio data, subjects were classified as a non-user, metformin alone, sulfonylurea (SU) alone, alpha-glucosidase inhibitor alone, metformin+SU combination, metformin+TZD combination, metformin+DPP4 inhibitor combination and SU+TZD combination. The outcome measure was the first occurrence for a vertebral fracture or a non-vertebral fracture. The incidence of fracture was analyzed controlling for age, gender, comorbidity score, diagnosis of osteoporosis, osteoporosis treatment, and osteoporosis related diseases. Total of 5,996 fractures were observed among 207,558 subjects during the observation period. Fracture rate per 10,000 person-years varied significantly across type of OADs, with metformin+DPP4 inhibitor combination group having the lowest rate [124.9, 95% confidence interval (CI) 106.0-147.1] and SU+TZD combination group having the highest rate (269.6, 95% CI 222.1-327.4). Metformin+DPP4 inhibitor combination group had significantly reduced fracture risk compared with non-users [hazard ratio (HR)=0.83, 95% CI 0.70-0.98, P=0.025]. In models adjusting for all confounding factors, metformin+DPP4 inhibitor combination group showed a trend of lower non-vertebral fracture risk compared with metformin+SU combination group (HR=0.82, 95% CI 0.65-1.03, P=0.086). TZD was significantly associated with increased risk of fracture (HR=1.59, 95% CI 1.38-1.82), P<0.001). These findings suggest that DPP4 inhibitor may have a protective effect on bone metabolism.

111-LB

Dipeptidyl Peptidase 4 Inhibitors and Comparative Pancreatic Cancer Risk

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A recent study analyzing human pancreata described potentially detrimental effects of sitagliptin, a dipeptidyl peptidase 4 inhibitor (DPP4i), on human pancreas with implications for incident pancreatic cancer (PC). This adds to concerns raised by an analysis of the FDA Adverse Events Reporting System which reported increased PC rates with incretin-based drugs. Both studies are limited by many shortcomings. We compared PC risk after initiation of DPP4i versus sulfonylureas (SU) and thiazolidinediones (TZD) using a 20% sample of the 2006-10 Medicare claims. To address concerns about potential outcome detection bias, we compared the cumulative incidence of diagnostic work-up in the two cohorts before and after initiation (index date). This was a new user active comparator cohort study consisting of patients >65 years requiring a second prescription of the same drug within 180 days of initiation with follow-up starting at the second fill date. Using an as-treated approach, we used propensity score adjusted Cox models to estimate hazard ratios (HR) and 95% confidence intervals (CI). Diagnostic work-up pre and post index was compared using risk ratios (RR). There were 19294 DPP4i initiators with mean age 74. Over a 9 month median follow-up, 29 DPP4i initiators had a PC diagnosis. The hazard of PC with DPP4i was lower relative to SU (HR 0.5, CI 0.3 - 1.0) and similar to TZD (HR 1.1, CI 0.7 - 1.8). Excluding the first 9 months after drug initiation to reduce the potential for reverse causality did not alter results. In the 6 months post index, the cumulative incidence of diagnostic work-up among sitaglitpin initiators (79.4%) was similar to TZD (74.0%) (RR 1.07, CI 1.06 - 1.08) and SU (74.6%) (RR 1.06, CI 1.05 - 1.07). The probability of diagnostic workup pre index was similar for all groups (~80%). Though limited by sample size and real world duration of treatment, contrary to previous evidence, our data suggest no increased pancreatic cancer risk with DPP4i relative to SU or TZD and that diagnostic work-up is not affected by DPP4i use.