using validated Diasony Infravascular devices. PP was defined as the difference between the systolic and diastolic ambulatory BP. Urinary caffeine, paraxanthine, theophylline, and theobromine excretions were measured in 24 h urine using ultra-high performance liquid chromatography tandem mass spectrometry. Urinary excretions were log-transformed to satisfy regression assumptions. We used linear mixed models to explore the associations of urinary caffeine and caffeine metabolite excretions with 24-hour, day- and night-time PP while adjusting for major confounders.

Results: The 836 participants (48.9 % men) included in this analysis had mean ±(SD) age of 47.8 (±17.5), and mean 24-hour systolic and diastolic BP of 120.1 ± (±13.9) and 78.0 (±8.6). Except theobromine, log transformed urinary caffeine and caffeine metabolite excretions were associated negatively with 24-hour, daytime and night-time ambulatory PP: 24-hour, daytime, and night-time ambulatory PP decreased by −0.804 mmHg (SE, 0.209), −0.749 (0.215), and −0.968 (0.243) (all P values <0.005), for each doubling excretion of caffeine. Strong negative associations with night-time ambulatory PP were observed for paraxanthine and theophylline.

Conclusions: The negative associations of PP with caffeine, paraxanthine, and theophylline excretions suggest that caffeine and its metabolites do lower BP, possibly by modifying arterial stiffness.

**1C.07**

**PRONEUROTENSIN INDEPENDENTLY PREDICTS CARDIOVASCULAR DISEASE. THE MALMÖ PREVENTIVE PROJECT**


Objective: Neotensin is released from the gut after fat intake and has a role in the negative associations with cardiovascular disease in middle aged participants of the Malmö Diet and Cancer Study. Here, we aimed at replicating the initial findings in an independent second cohort and to extend its validity to an older population.

Design and method: Malmö Preventive Project (MPP) is a Swedish population-based prospective study which comprised 18240 subjects for reexamination in 2002–2006. Fasting proneotensin was measured in plasma from a random sample of 4804 participants (Age 69 SD (6.2), 68% Male). Multivariate Cox proportional hazard models adjusted for age, sex, use of antihypertensive medications, systolic blood pressure, BMI, current smoking, high density lipoprotein cholesterol (HDL-C), LDL-C, history of diabetes were used to relate the log transformed levels of fasting proneotensin to the risk of first fatal or non-fatal cardiovascular event (myocardial infarction or stroke) in the mean follow up time of up to 6.5 years. Hazard ratios (HR) for CVD were expressed per 1 (SD) increment of log transformed proneotensin for cardiovascular disease in middle aged participants of the Malmö Diet and Cancer Study. Here, we aimed at replicating the initial findings in an independent second cohort and to extend its validity to an older population.

Results: There were 456 cardiovascular events observed in the study. Hazard ratios (HR) for CVD were expressed per 1 (SD) increment of log transformed proneotensin for cardiovascular disease as HR 1,102; 95% CI; 1,006–1,088; P = 0.037. For all-cause mortality HRs were 1.4 (1.2 to 1.8; 10 cohorts, 17709 participants; F = 62%) for IAD >=10mmHg and 1.4 (1.1 to 1.7, 12 cohorts, 18714 participants; F = 46%) for IAD >=15mmHg. Heterogeneity between studies could be accounted for by stratification according to underlying population cardiovascular risk, with higher HRs seen in populations at elevated risk; cardiovascular mortality with an IAD >=10mmHg: HR 1.4 (1.1 to 1.8; F = 0%) for community based cohorts compared to 3.8 (2.2 to 6.6; F = 0%) for those at elevated cardiovascular risk (p = 0.001; Figure).

Conclusions: New studies confirming the association of an IAD with increased cardiovascular and all-cause mortality are consistent with previously published findings. Risks associated with an IAD rise in association with the underlying vascular risk of the population studied.

**1C.08**

**THE INTER-ARM DIFFERENCE IN BLOOD PRESSURE AND MORTALITY: SYSTEMATIC REVIEW AND META-ANALYSIS**

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Objective: We previously reported the association of inter-arm differences in blood pressure measurements (IAD) with increased cardiovascular and all-cause mortality. Several new large cohorts have been reported since our 2012 meta-analysis. We have therefore updated our meta-analyses to take account of these new data.

Design and method: Systematic review and meta-analysis: Medline, Embase and CINAHL were searched for studies reporting survival data in association with IAD. Study level hazard ratios (HR) were extracted for systolic IADs >=10mmHg and >=15mmHg, and pooled using generic inverse variance in a random effects model. Statistical heterogeneity was assessed using the I² statistic.

Results: Searches to 12th November 2014 identified 3514 unique citations. Eighty full texts were assessed, and 13 studies (reporting data for 14 unique cohorts) contributed to the analyses, Median follow up ranged from 3 to 13 years. Five cohorts employed a simultaneous method of IAD measurement; the remainder used sequential measurements. Ten cohorts were recruited from community populations, including one hypertensive and one diabetic cohort. Four were selected hospital cohorts at increased vascular risk. Cardiovascular mortality was greater with an IAD >=10mmHg (HR 1.9 (95%CI 1.3 to 2.6; 7 cohorts, 13815 participants; F = 45%) and an IAD >=15mmHg (HR 1.7 (1.2 to 2.4; 9 cohorts; 18241 participants; F = 30%). For all-cause mortality HRs were 1.4 (1.2 to 1.8; 10 cohorts, 17709 participants; F = 62%) for IAD >=10mmHg and 1.4 (1.1 to 1.7, 12 cohorts, 18714 participants; F = 46%) for IAD >=15mmHg. Heterogeneity between studies could be accounted for by stratification according to underlying population cardiovascular risk, with higher HRs seen in populations at elevated risk; cardiovascular mortality with an IAD >=10mmHg: HR 1.4 (1.1 to 1.8; F = 0%) for community based cohorts compared to 3.8 (2.2 to 6.6; F = 0%) for those at elevated cardiovascular risk (p = 0.001; Figure).

Conclusions: Fasting proneotensin levels are independently associated with the risk of developing cardiovascular disease which replicates the findings in MDC study.

**1C.09**

**SERUM RESISTIN AS AN INDEPENDENT BIOMARKER WITH ALL-CAUSE AND CARDIOVASCULAR MORTALITY IN ELDERLY HYPERTENSIVE, NON-DIABETIC PATIENTS WITH CHRONIC KIDNEY DISEASE (CKD)**

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Objective: Hypertensive patients with CKD present an increased risk for cardiovascular mortality. Among the proteins synthesized and released from adipose tissue, resistin is a cytokine whose physiologic role has been the subject of much research and controversy. We and others have demonstrated that serum resistin levels are higher in patients with CKD and correlate directly with inflammatory markers, including TNF-a and hCRP. Since inflammation has been consistently linked to atherosclerosis, death, and cardiovascular (CV) events, our goal was to investigate

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