more features of the metabolic syndrome than those with hsCRP less than 2 mg/L (table 1).

The MESA JUPITER population closely resembled the placebo group in the JUPITER trial (webappendix p 2). Median age of patients in the JUPITER placebo group was 66 years (IQR 60–71), mean calculated 10-year Framingham risk of 10%, and median hsCRP was 4·3 mg/L (IQR 3·0–7·8). The MESA JUPITER population had more women than the JUPITER population (51% *vs* 38%) because of its population-based recruitment with similar initial enrolment by gender, coupled with the higher concentrations of hsCRP in women.

444 (47%) patients in the MESA JUPITER population had a CAC score of 0. Of those with CAC, 267 (28%) had scores 1–100, and 239 (25%) had scores more than 100. The number needed to scan to identify one individual with a CAC score of 0 was 2, and to identify one individual with a CAC score more than 100 was 4. The frequency of increased CAC burden was similar in the low hsCRP group (p=0·09, webappendix pp 3–6). Prevalence of CAC differed according to sex. 259 (53%) women had a CAC score of 0 compared with 185 (40%) men, and 97 (20%) of women had a score of more than 100 compared with 142 (31%) men.

Table 2 shows the frequency of cardiovascular disease events and coronary heart disease events, the corresponding event rates per 1000 person-years, and the multivariable-adjusted HRs associated with prevalence and burden of CAC in MESA JUPITER participants. Event rates for coronary heart disease and cardiovascular disease were low when CAC scores were 0 and high when CAC scores were more than 100 (table 2). Only 6% of all coronary heart disease events and 17% of all cardiovascular disease events were in the individuals with scores of 0 (table 2). Almost 75% of all coronary heart disease events, and about 60% of all events of cardiovascular disease, were in the 25% of participants with scores more than 100 (table 2).

The presence of CAC was associated with an HR of 11·0 (95% CI 2·51–48·5) for coronary heart disease, and 3·20 (1·41–7·24) for cardiovascular disease in the MESA JUPITER population in the fully adjusted model. We noted a graded increase in events for both diseases with increasing burden of CAC (table 2).

Figure 2 shows Kaplan-Meier estimates of event-free survival for coronary heart disease and cardiovascular disease for the MESA JUPITER population by CAC burden. Table 3 shows the Kaplan-Meier failure (event) function. From these estimates, the 5-year NNT to prevent an event of coronary heart disease was 549 for CAC score 0, 94 for scores 1–100, and 24 for scores more than 100. The corresponding 5-year NNT to prevent an event of cardiovascular disease was 124, 54, and 19, respectively (table 3). Webappendix p 1 shows the results of the sensitivity analysis.

In the total study population, overall event rates were similar in the low (<2 mg/L) and high (≥2 mg/L) hsCRP groups for coronary heart disease (7·6 *vs* 6·4 per 1000 person-years, p=0·47) as were event rates for cardiovascular disease (10·1 *vs* 10·4 per 1000 person-years, p=0·87). Figure 3 shows Kaplan-Meier plots stratified by hsCRP status. hsCRP status did not predict coronary heart disease events (HR 0·98, 95% CI 0·62–1·57) or cardiovascular disease events (1·15, 0·78–1·68) after adjustments for age, sex, and race. By contrast, presence of CAC was a strong predictor of both coronary heart disease (6·65, 2·99–14·78) and cardiovascular disease (3·06, 1·82–5·13) in similarly adjusted models. CAC prevalence and increased CAC burden were significant predictors of events after full multivariable adjustment (table 4).

See Online for webappendix

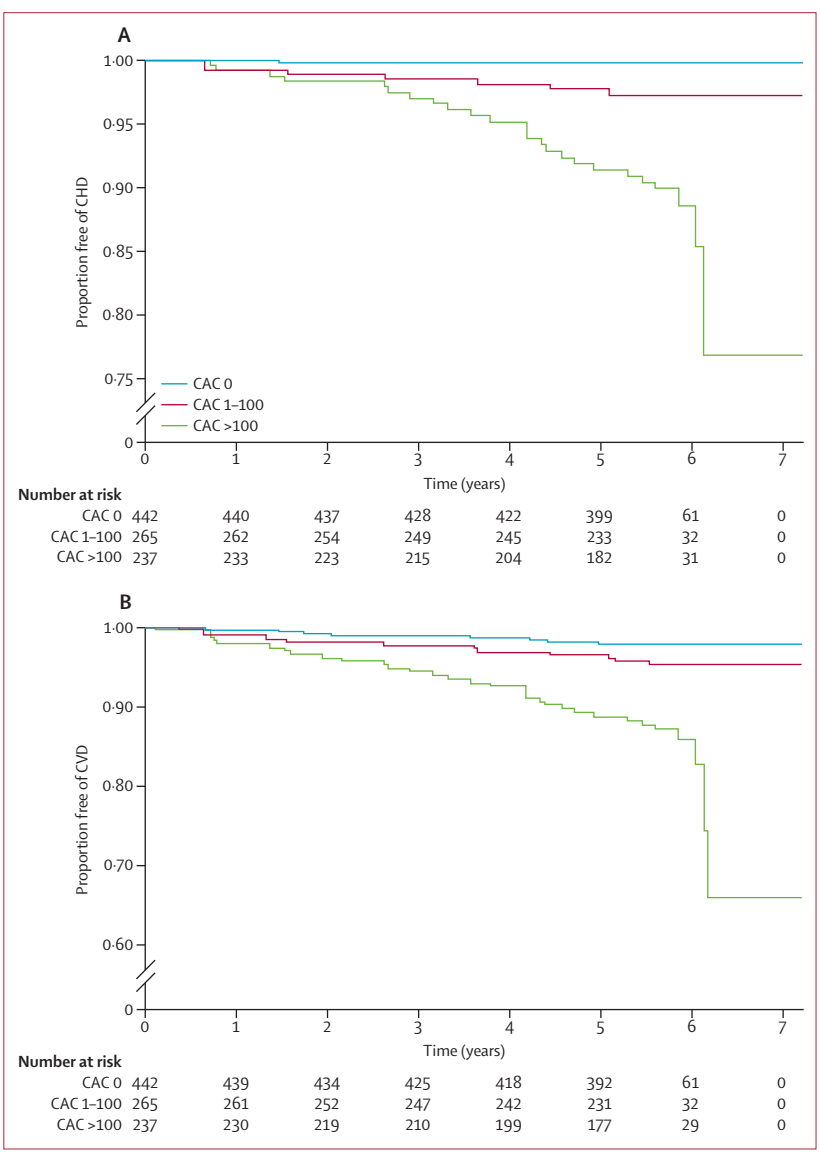

***Figure 2*: Kaplan-Meier estimates of CHD (A) and CVD (B) event-free survival by CAC burden in the MESA JUPITER population**

CHD=coronary heart disease. CAC=coronary artery calcium. CVD=cardiovascular disease.