**SHORT REPORT**

Height, body mass index, and survival in men with coronary disease: follow up of the diet and reinfarction trial (DART)

A R Ness, D Gunnell, J Hughes, P C Elwood, G Davey Smith, M L Burr

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Taller people have a lower risk of coronary death and death from all causes, but there are few data on the association between height and survival in those with coronary disease. Similarly being either underweight or obese is associated with increased coronary and all cause mortality but the association of body mass index (BMI) and survival in people with symptomatic coronary heart disease has not been so widely reported. We examined these associations in the Diet and Reinfarction Trial.

**METHODS AND RESULTS**

Between 1983 and 1987 2033 men who had recently suffered a myocardial infarction were enrolled from 4371 potentially eligible men identified. The men were enrolled shortly after discharge from hospital with a mean interval from myocardial infarction to enrolment of 41 days. The main reason for exclusion was that the men intended to eat one of the study diets (n=1044). In addition, 237 men died before they could be randomised. Past history of disease, smoking habits, and drug treatment were noted. Height and weight were recorded and BMI (weight/(height)^2) calculated as a measure of adiposity. Participants were randomly allocated to one of the eight dietary regimens—fat advice, fish advice, fibre advice, fat and fish, fat and fibre, fish and fibre, all three or no advice. All smokers were advised to stop and those with a BMI over 30 were given weight reducing advice.

All men in the study were flagged with the NHS central register. By the end of February 2000, 1083 (53%) men enrolled in the study had died after a total of 21 147 person years of observation. Of these deaths 738 (68%) were attributed to coronary heart disease. Cox’s proportional hazards regression was used to examine survival in relation to height and BMI and to adjust for confounding factors.

The associations between quartiles of height and BMI and coronary and all cause mortality are shown in table 1. There was no association between height and survival or height and coronary mortality. The age adjusted hazards for height were essentially unaltered after adjustment for the baseline variables used in the original report, smoking, BMI, and randomisation group. There was a reverse J shaped association between BMI and CHD and all cause mortality. Those with the lowest BMI were at highest risk of coronary and all cause mortality. Those in the middle quartiles (BMI 24 to 28 kg/m^2) were at lowest risk.

To eliminate confounding by smoking we repeated these analyses in non-smokers (n=1242). Compared with those with the lowest BMI the adjusted hazard for all cause mortality in non-smokers was 0.64 (95% CI 0.48 to 0.86) in quartile 2, 0.65 (95% CI 0.49 to 0.87) in quartile 3 and 0.65 (95% CI 0.49 to 0.86) in quartile 4.

The adjusted hazard in obese men (BMI >30 kg/m^2) was 0.72 (95% CI 0.48 to 1.08) compared with those who were underweight (BMI <20 kg/m^2). To formally test for non-linearity a quadratic term for BMI was fitted to the fully

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**Table 1** Deaths attributed to coronary disease all causes following myocardial infarction by height and body mass index in men enrolled in the Diet and Reinfarction Trial (DART) 1983–2000

<table>
<thead>
<tr>
<th>Hazard for quartiles of height (cm)</th>
<th>First Mean (range)</th>
<th>Second Mean (range)</th>
<th>Third Mean (range)</th>
<th>Fourth Mean (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cause mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.00</td>
<td>1.03 (0.86 to 1.22)</td>
<td>0.99 (0.85 to 1.16)</td>
<td>0.97 (0.82 to 1.14)</td>
</tr>
<tr>
<td>Multiply adjusted*</td>
<td>1.00</td>
<td>1.07 (0.90 to 1.28)</td>
<td>1.00 (0.85 to 1.17)</td>
<td>0.99 (0.84 to 1.17)</td>
</tr>
<tr>
<td>Coronary heart disease mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.00</td>
<td>1.06 (0.85 to 1.30)</td>
<td>1.05 (0.87 to 1.26)</td>
<td>0.97 (0.79 to 1.19)</td>
</tr>
<tr>
<td>Multiply adjusted*</td>
<td>1.00</td>
<td>1.09 (0.88 to 1.35)</td>
<td>1.06 (0.87 to 1.28)</td>
<td>0.98 (0.80 to 1.21)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Body mass index (BMI)</th>
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<tbody>
<tr>
<td>All cause mortality</td>
<td></td>
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</tr>
<tr>
<td>Age adjusted</td>
<td>1.00</td>
<td>0.75 (0.64 to 0.89)</td>
<td>0.73 (0.61 to 0.86)</td>
<td>0.85 (0.72 to 1.00)</td>
</tr>
<tr>
<td>Multiply adjusted*</td>
<td>1.00</td>
<td>0.76 (0.64 to 0.90)</td>
<td>0.69 (0.58 to 0.82)</td>
<td>0.79 (0.66 to 0.93)</td>
</tr>
<tr>
<td>Coronary heart disease mortality</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Age adjusted</td>
<td>1.00</td>
<td>0.72 (0.58 to 0.88)</td>
<td>0.69 (0.56 to 0.85)</td>
<td>0.86 (0.70 to 1.04)</td>
</tr>
<tr>
<td>Multiply adjusted*</td>
<td>1.00</td>
<td>0.71 (0.58 to 0.87)</td>
<td>0.64 (0.52 to 0.79)</td>
<td>0.76 (0.62 to 0.93)</td>
</tr>
</tbody>
</table>

*Adjusted for the following baseline variables: history of MI, angina, hypertension at baseline; radiographic evidence of cardiomegaly, pulmonary congestion or pulmonary oedema at baseline; treatment (β blockers, other antihypertensives, digoxin/antiarrhythmics, or anticoagulants), smoking, diet randomisation, height or BMI.
Height, body mass index, and survival in men with coronary disease

To examine the effect of pre-existing disease we repeated the analyses in men with a past history of myocardial infarction (MI) (n=424) and in men with no past history of MI (n=1609). Compared with those with the lowest BMI the adjusted hazard for all cause mortality in men with a history of MI was 0.73 (95% CI 0.52 to 1.01) in quartile 2, 0.64 (95% CI 0.46 to 0.90) in quartile 3 and 0.56 (95% CI 0.40 to 0.77) in quartile 4. While, compared with those with the lowest BMI the adjusted hazard for all cause mortality in men with no past history of MI was 0.73 (95% CI 0.60 to 0.88) in quartile 2, 0.69 (95% CI 0.56 to 0.84) in quartile 3 and 0.86 (95% CI 0.71 to 1.04) in quartile 4. To test formally for interaction a term was fitted to the fully adjusted model. This indicated a significant interaction (p=0.009) between BMI and past history of MI. There was a similar interaction for deaths attributed to coronary disease (p=0.01).

COMMENT

We found no association between height and mortality and a reverse J shaped association between BMI and mortality. Though cross sectional angiographic studies have reported that taller men have less coronary atheroma, we found no evidence of a survival advantage in taller men with symptomatic coronary disease.

The apparent lack of an excess risk in men with a higher BMI may be attributed to increased inhospital and early post-discharge death rates among overweight men before recruitment to DART. Surviving overweight men may therefore be those with better cardiovascular risk profiles. Alternatively, infarct survivors with a high BMI may be those who have a higher lean body mass, rather than high fat mass. A well recognised limitation of BMI as a measure of body fatness is that it is based on body weight, which includes both fat mass and lean body mass. Lean body mass is a measure of muscularity and physical fitness, which are cardioprotective. These explanations would be consistent with the observation that the protective effect of being overweight was strongest in those with a past history of myocardial infarction.

The observed excess mortality in those with a low BMI was more pronounced than in other studies. The excess mortality observed in men with low BMI may represent reverse causality with pre-existing illness (including cardiac cachexia) being associated with a lower weight at recruitment. Alternatively the adverse effect of low BMI may either reflect the fact that lean men cannot reduce their mortality by weight reduction or that low BMI in older people is a marker of biological aging.

ACKNOWLEDGEMENTS

Dr Burr, Ms Hughes and Professor Elwood were part of the team that set up the original study. Dr Ness performed the analyses. Dr Ness wrote the first draft of the paper and all other authors commented on further drafts. Dr Ness is guarantor for this study.

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Conflicts of interest: none.

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