

Weight and weight change and risk of acute myocardial infarction and heart failure: The HUNT study

Imre Janszky

Norwegian University of Science and Technology, Norway

Objectives: To delineate the association of weight with cardiovascular health throughout adulthood.

Methods: We conducted a population-based prospective cohort study of 26 097 community-dwelling individuals who were followed for 11.4 years with measurements of cardiovascular risk factors and common chronic disorders. Body weight and height were directly measured at baseline in 1995–1997 as they had been 10 and 30 years prior to baseline. From these measurements, we estimated average body mass index (BMI) over time and calculated weight change.

Results: The association of average BMI with acute myocardial infarction (AMI) became weaker with adjustment for the most recent BMI measurement, whilst this adjustment had a more limited effect on associations with heart failure (HF) risk. For example, the multi-adjusted hazard ratios for AMI in a comparison of individuals with average BMI until baseline ≥ 35 kg m⁻² and between 18.5 and 22.4 kg m⁻² decreased from 1.75 [95% confidence interval (CI) 1.04–2.95] to 1.32 (0.73–2.40). The corresponding numbers for HF were 3.12 (1.85–5.27) and 2.95 (1.53–5.71), respectively. The associations between weight change and risk of AMI and HF were U-shaped, with stable weight showing the lowest risk.

Conclusion: Sustained overweight or obesity over time is associated with increased risk of HF, even after adjustment for the most recent BMI. For AMI risk, the most recent BMI appears to be the most important. Weight change also increases risks for both outcomes beyond the effects of BMI. Our results suggest that a global epidemic of obesity is likely to increase the incidence of HF, even if BMI in middle age can be controlled.

Introduction: Overweight is often regarded as one of the most important modifiable cardiovascular risk factors, but its association with cardiovascular health over a lifespan remains incompletely understood. Not all studies have demonstrated adverse effects of overweight, and it is unclear whether overweight has

cumulative effects and whether overweight or obesity at different life stages has different effects on cardiovascular health 2, 3. The effect of weight change is also controversial 4–6.

Previous longitudinal studies on body weight and cardiovascular disorders have had several potential limitations. In many of these studies, body weight was self-reported, and even if current weight was measured, weight earlier in life was estimated by self-report rather than by direct measurement. Self-reported current BMI is generally highly accurate, but recalling about body weight from decades earlier might be less reliable; the accuracy of recall depends on several characteristics, including the actual body weight values 7, 8. Studies with repeated direct anthropometric measurements over time have been small, with short-term follow-up and/or restriction to younger populations with few clinical events 3, 5, 9–14.

Morbidity from chronic disorders that may lead to weight loss is a potentially important confounder for studies of body weight. These disorders have not been systematically assessed in most studies. Smoking causes leanness, but it is simultaneously a risk factor for coronary heart disease and its role in the association between long-term body weight and cardiovascular health is also not clear. Furthermore, in comparison with studies examining mortality, fewer studies have investigated well-validated cases of incident acute myocardial infarction (AMI) or congestive heart failure (HF). Finally, the cumulative effects of BMI and the effects of weight change on incident HF are largely unknown.

In this large, population-based study, we investigated the cumulative effect of overweight and the effect of change in BMI on the risks of AMI and HF. Body weight and height had been directly measured 10 and 30 years prior to baseline and were measured again at baseline. Our analysis also included data on established cardiovascular risk factors and common chronic disorders.

Methods: Adults 20 years of age and older in Nord-Trøndelag County in Norway were invited to participate in the second wave of the HUNT Study (HUNT-2) from August 1995 to June 1997. Briefly, 93 898 individuals were eligible to participate, and 64 726 (69%) returned the questionnaire that was included with the invitation letter and underwent a clinical examination conducted by trained nurses 18-20. In the current analysis, we excluded 480 participants with missing information on BMI and 2947 individuals with a history of AMI, HF or stroke at baseline.

Before the baseline measurement in the HUNT-2 Study, height and weight had been measured in a comparable and standardized manner twice before. Between 1984 and 1986, in the first wave of the HUNT Study, the adult population of the county was invited, and 77 212 (89% of those invited) participated. Between 1966 and 1969, height and weight were measured in conjunction with a mandatory tuberculosis screening that was conducted in the county. Amongst the 61 299 eligible participants in HUNT-2, information on BMI from HUNT-1 and from the tuberculosis screening was available for 27 196 individuals who were old enough to have participated in the earlier surveys. In our main analyses, we excluded all individuals who had a BMI measure below 18.5 kg m⁻² at any time-point, resulting in 26 097 individuals. Our results were essentially the same when we included all individuals with a valid BMI.

Body weight and height measurements for 182 individuals who were adolescents at the time of the tuberculosis survey were converted to adult BMI categories using internationally recognized cut-off points. The present study was approved by the regional committee for ethics in medical research.

In this work, we refer to BMI measurement at baseline, that is, at HUNT-2 conducted in 1995–1997 as BMI-96, to BMI measurement at HUNT-1 conducted in 1984–1986 as BMI-85 and to BMI measurement in 1966–1969 as BMI-67.