Background People with high body mass index (BMI) suffer elevated rates of mortality from a variety of causes, particularly from cardiovascular disease (CVD). Some studies have also found elevated mortality at low BMI, but it is unclear whether this represents a causal association. Potential confounding by existing ill-health is of particular concern. To avoid this problem, we used the BMI of an offspring as an instrument for the parent’s BMI, as well as conducting conventional analyses of parental mortality against their own BMI.

Methods We extracted 33,011 mother-offspring and 28,142 father-offspring pairs from HUNT, a large prospective cohort study conducted in Nord-Trøndelag county, Norway. Participants’ BMI was adjusted for age, sex, secular trends and smoking status. Associations of parental mortality rates with their own BMI were estimated as hazard ratios using Cox regression. Age was the time axis, and we adjusted for smoking, alcohol, exercise, education and employment. Additionally, adjusted causal hazard ratios between parental mortality and BMI were estimated using offspring BMI as an instrument for parental BMI and cubic splines were fitted to all associations to assess their linearity.

Results There were 9,271 maternal and 9,889 paternal deaths within the follow-up period of 1984–2009. The associations of all-cause, CVD and cancer mortality with parents’ own BMI were substantially non-linear, with elevated mortality at both extremes and minima at 21–25 kg m⁻². Hazard ratios for all-cause mortality per standard deviation (4.1 kg m⁻²) of own BMI were 1.04 (95% CI 1.02, 1.06) and 1.05 (1.02, 1.08) in mothers and fathers, respectively. In contrast, associations of mortality with offspring BMI were approximately linear and positive. Causal hazard ratios per standard deviation of BMI estimated from the instrumental variable analyses were 1.20 (1.11, 1.30) and 1.11 (1.02, 1.22) for all-cause mortality in mothers and fathers, respectively. For CVD they were 1.26 (1.13, 1.41) and 1.15 (1.01, 1.30), and for cancer they were 1.23 (1.05, 1.45) and 1.07 (0.90, 1.27).

Conclusion These results confirm the elevated mortality from all causes, CVD and cancer in subjects with high or low BMI. The use of an offspring’s BMI as an instrument suggests that the elevated mortality at low BMI in this and other studies may be the result of confounding by existing ill-health, and that the causal relationship between BMI and mortality is positive and approximately linear.

Conclusion Parental SA in childhood increases the risk of self-harm with suicidal intent in offspring but is unrelated to risk of self-harm without suicide intent. Parental SA is associated with larger effect size on suicidal thoughts with a suicidal plan than without a plan. Findings provide limited evidence that maternal SA is a more potent risk factor than partner SA.