Incidence of metabolic risk factors among healthy obese adults: 20-year follow-up

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Incidence of Metabolic Risk Factors Among Healthy Obese Adults

20-Year Follow-Up

There is growing evidence that obese adults without metabolic risk factor clustering (the so-called “healthy obese”) progress to unhealthy obesity over time (1). However, the pathophysiological changes underlying the long-term transition into an unhealthy obese state have not been well characterized. To inform clinical management of healthy obesity, we aimed to identify the metabolic risk factors responsible for this transition, as well as the timing of their onset.

Repeat clinical data were drawn from the Whitehall II cohort study of British adults. We grouped participants as normal-weight (body mass index [BMI] 18.5 to 24.9 kg/m²), overweight (BMI 25 to 29 kg/m²), or obese (BMI ≥ 30 kg/m²), and as healthy (2) if they were free of any the following characteristics: high-density lipoprotein cholesterol < 1.03 mmol/l (men) and < 1.29 mmol/l (women); blood pressure ≥ 130/85 mm Hg or antihypertension medication use; fasting plasma glucose ≥ 5.6 mmol/l or diabetic medication use; triglycerides ≥ 1.7 mmol/l; and homeostatic model-assessed insulin resistance > 2.83 (baseline 90th percentile value). Participants provided written

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**FIGURE 1** Incidence of Metabolic Risk Factors Among Initially Healthy Obese Compared With Initially Healthy Normal-Weight Adults Over 20 Years (n = 1,120)

Results are incidence ratios and 95% confidence intervals (CI) for having each metabolic risk factor at follow-up, on the basis of Poisson regression models with robust error variances. Models are adjusted for age, sex, and ethnicity. Little difference in high-density lipoprotein cholesterol or triglycerides was observed between groups. Baseline healthy status is defined as having none of 5 metabolic risk factors (hypertension, low-density lipoprotein cholesterol, high triglycerides, insulin resistance, and high blood glucose).
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normal-weight counterparts. That insulin resistance is an established indicator of future impaired glucose metabolism (5) may explain their much higher incidence of type 2 diabetes (relative risk near 4.0) (2) and slightly higher incidence of cardiovascular disease (relative risk near 1.2) (4), given that earlier onset of risk factors leads to a greater cumulated exposure and higher disease risk. Overall, our findings suggest that healthy obesity is strongly linked with future insulin resistance that subsequently causes cardiometabolic pathology.

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REFERENCES