Obesity in childhood and adolescence

Last year it was reported that a 9-year-old boy had died at school from sleep apnoea and Pickwickian syndrome. He weighed between 105 and 120 kg, and despite the overseeing medical doctor insisting that he be admitted for further investigations and treatment, his parents refused. He had been falling asleep in the classroom on a regular basis for 2 months before his tragic death.

Obesity rates among children and adolescents have reached epidemic proportions in both industrialised and developing countries, with an estimated 1 out of every 5 youngsters suffering from obesity at a BMI above 30.1 Childhood obesity is a strong predictor of adult obesity, and very difficult to treat once established.1

Childhood obesity also predicts an increased risk of death, primarily due to an increased likelihood of cardiorespiratory death.2 The rise in the cardiovascular death rate is explained by the increased risk factor profile from high rates of hypertension, dyslipidaemia, type 2 diabetes and sleep apnoea.3 Longitudinal studies looking at various risk factors, including carotid intima-media thickness, have indicated that adults who became obese but had a normal BMI in childhood had a more adverse risk profile. Adults who were obese as children and then normalised their BMI had a risk profile similar to patients who had never been obese during childhood.4

Given the above, active intervention in childhood obesity is likely to have a number of benefits. However, we face the difficult scientific problem that circulating levels of biological mediators of appetite which encourage weight regain after induced weight loss do not revert to the levels recorded before weight loss.4 Long-term strategies and research to counteract this change are needed to prevent obesity relapse, and so far we have failed in this area. Various other factors have contributed to our lack of success in containing childhood obesity, including living in neighbourhoods with a high level of poverty,5 sleep deprivation during childhood,6 consumption of sugary drinks, frequent ingestion of fast foods,7 and lack of physical activity.8

Less well-recognised medical conditions associated with childhood obesity include non-alcoholic fatty liver, gastro-oesophageal reflux, slipped capital femoral epiphyses, pseudo-tumour cerebri, and a high risk of certain cancers. At the onset these medical conditions may be less destructive than the stigma of obesity and the psychological risk of certain cancers. At the onset these medical conditions may be less destructive than the stigma of obesity and the psychological trauma experienced by bullying and stigmatisation.

Morbid obesity seems virtually impossible to treat with lifestyle intervention alone. A 2-year trial of obesity treatment in primary care practice indicated that quarterly visits with brief counselling had no impact on weight loss. Enhanced weight loss counselling by lifestyle experts helped around one-third of patients to achieve a 5% weight loss.8 Enhanced weight loss counselling by lifestyle experts helped around one-third of patients to achieve a 5% weight loss.9

One approach would be to make societal changes to enhance human well being rather than to try and prevent a particular symptom such as obesity.10 Blaming, shaming and punishing the obese will not solve the problem – it is simply shooting the messenger. If parents fail to recognise that their child is obese, they are unlikely to recognise that interventions targeting obesity are relevant to the family.

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References

An eerie silence;
Birds, crickets, bees, trees are still -
Rain is imminent.

Haiku: Peter Folb