Childhood obesity: One epidemic, or two?

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A recent report from the EarlyBird Study appeared under the rather less prosaic title of "Childhood obesity: evidence for distinct early and late environmental determinants" (Mostazir et al, 2015). The findings suggested that two distinct factors were responsible for excess weight gain in different sectors of the childhood population at different ages. The successful prevention of childhood obesity requires understanding the cause, and this new research implies that fundamentally different strategies may be needed at different stages of childhood.

As recently as 10 or 15 years ago, approaches to the prevention of childhood obesity were marred by the lack of an evidence base to support them. As a result, official advice relied heavily on intuition, which targeted physical inactivity as the chief cause of childhood obesity (Sport England, 2001; National Audit Office, 2006). The advice censured youngsters for their failure to meet (arbitrary) Government goals for physical activity (Metcalf et al, 2008), while the consumption of processed food and sugar-laden drinks went largely unsullied. The epidemic of childhood obesity continued to escalate.

However, views on the causes of childhood obesity have begun to change, and the EarlyBird study has played its part. In particular, using objective accelerometry, EarlyBird was unable to demonstrate the expected relationship between opportunity for physical activity (e.g. time allocated for Physical Education at school) and overall physical activity (Frémeaux et al, 2011). It suggested that the physical activity of children is constrained by an "activitystat" (Wilkin et al, 2006), whereby more activity at one part of the day is compensated by less at another. The variation in activity among children does not appear to reflect environmental privilege, as many suggest, but a range of set-points.

One counterintuitive observation was followed by another. The association between physical inactivity and childhood obesity was always interpreted in one direction, to the detriment of the child: that inactivity was the cause of obesity. However, EarlyBird had the advantage of annual measurements over 12 years and was able to test the direction of causality by time-lapse correlation back and forth across different ages. It demonstrated that physical inactivity did not predict future weight gain, whereas weight gain repeatedly predicted future inactivity (Metcalf et al, 2011). These fundamental observations were substantiated by experimental data from the meta-analysis of two systematic reviews, which appeared to vindicate the activitystat principle (Harris et al, 2009; Metcalf et al, 2012). Physical activity interventions, often very intense, did not alter childhood BMI and, most challenging of all, physical activity interventions did not alter overall physical activity.

Nobody doubts the value of physical activity to health, but few will now advocate physical activity programmes for weight reduction – they don't work. So where does the obesity epidemic in children originate, if not with physical inactivity? With knowledge of the birth weights and annual measures of BMI from the ages of 5–16 years, EarlyBird used a statistical approach to explore the exposures responsible. Obesity rates are usually reported as a percentage or as a mean, and the popular perception of weight gain in children tends to be one of whole population shift, sometimes referred to as a "drifting iceberg". However, neither the percentage nor the mean defines the distribution, which is key to understanding environmental impact. Thus, the mean can rise without change in the median (skew, or "landslip"), and the variance can rise without change in the mean. Obesity "rates" increase with both skew and variance, as more children in both instances cross the obesity threshold, but for different reasons. The development of a skew implies that one sector only of the population is exposed (or susceptible) to an environmental risk, while increasing

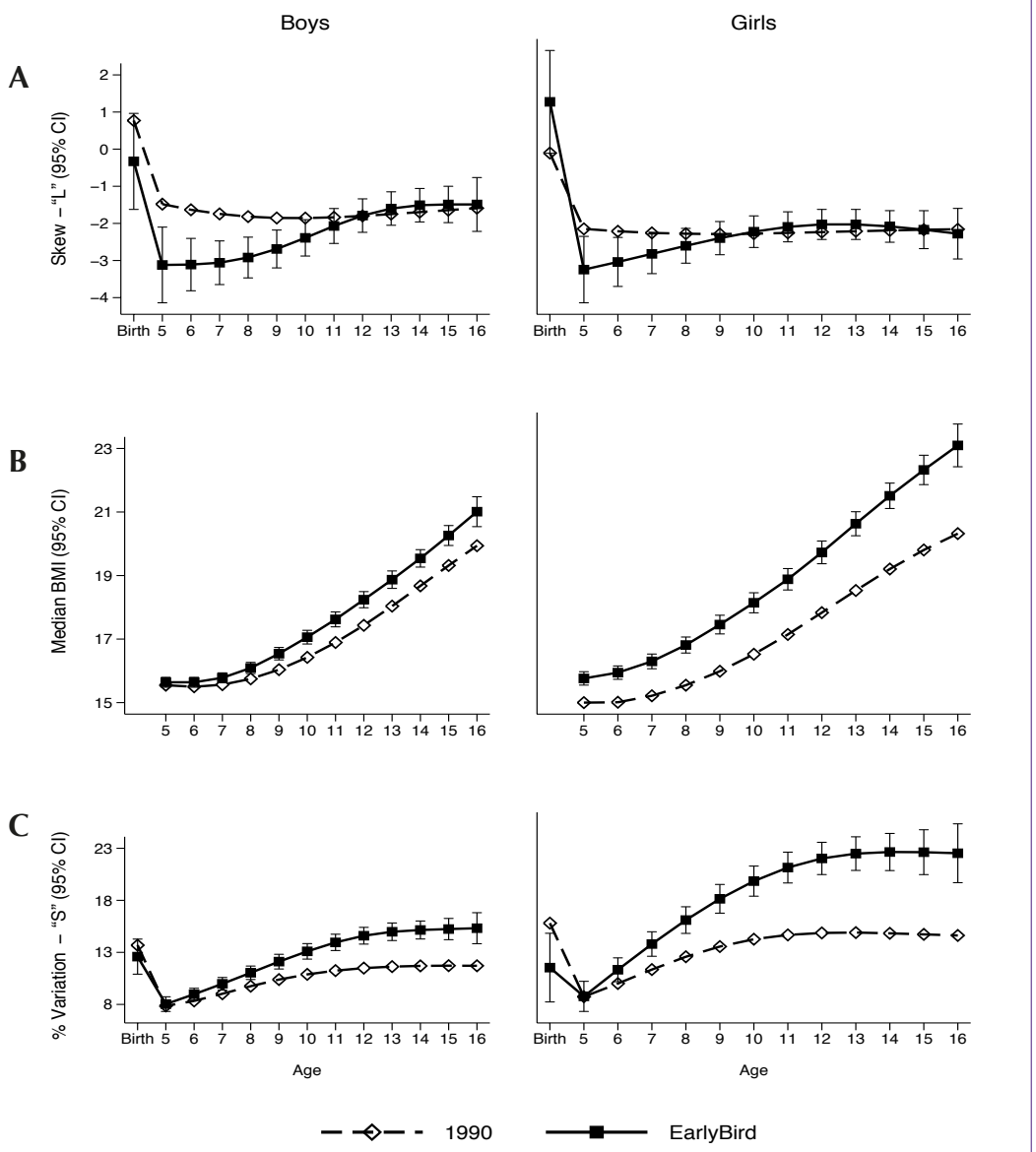

Figure 1. Comparison of the EarlyBird cohort, by gender, with the UK Growth Standards of 1990 in relation to skew in BMI (A), median (B) and variance (C). The marker between birth and age 5 years indicates non-availability of data from 1–4 years, and the estimate for "L" and "S" at birth relates to weight, not to BMI. Reproduced with permission from Mostazir et al (2015).
CI=confidence interval.

variance suggests a more generalised exposure or susceptibility.

In the latest EarlyBird report, we applied the LMS system (L for variance, M for median and S for deviation; Cole, 1990) to compare the trends in BMI of contemporary children as they grew up between 5 and 16 years of age with those of a generation ago, who had been used to establish the 1990 British Growth Standards (Cole et al, 1995). The findings were revealing. First, there was no statistically significant difference in birth weights, and the birth weight distributions were normal in both groups, suggesting random variance. By the age of 5 years, the mean BMI of the EarlyBird cohort was substantially greater, but the difference was accounted for largely by skew (landslip rather than drifting iceberg; *Figure 1A*), with little change in the median BMI (*Figure 1B*), and none in its

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variance (*Figure 1C*). The skew involved children who were predominantly the offspring of obese parents. Thus, while a rightwards (negative) skew in BMI was already established by 5 years of age in 1990, it was considerably greater 25 years later. Importantly, there was no change over this time in the wider population of toddlers.

The greater skew in the BMI of EarlyBird children at age 5 years returned to 1990 values by the onset of puberty (*Figure 1A*). At the same time, however, the variance in BMI began to rise steeply, and to diverge from the trend of 1990 (*Figure 1C*). As the variance in BMI of the EarlyBird children moved away from the trajectory of 1990, so too did the median (*Figure 1B*).

The median BMI of the EarlyBird children continued to diverge from the 1990 values as puberty progressed (*Figure 1B*). The difference in variance, on the other hand, peaked at around 13 years (*Figure 1C*). The skew in BMI established by 5 years remained strikingly stable throughout childhood in the 1990 data, whereas that of the EarlyBird children returned to the values of 1990 by early puberty, before rising again in later adolescence (*Figure 1A*).

To summarise, the mean BMI of the contemporary cohort of children was higher than that of the 1990 growth standards throughout the course of childhood, but for reasons that changed as the children grew older. Exaggerated skew accounted for the difference early on (the median changed little), while widening variance accounted for it later on (the median rose). In both instances, the mean rose progressively, and the prevalence of obesity increased. The sequence of events points to trends in the distribution of BMI over the course of contemporary childhood that are different from a generation ago, when skew and variance were relatively stable.

Observational data must be interpreted with caution. Notwithstanding, very young children are exposed to little outside the home, and the normal birth weight distribution of the EarlyBird cohort, combined with a skew by age 5 years involving predominantly the offspring of obese parents (Perez-Pastor et al, 2009), suggests that nutritional errors of the parents may be important in the early weight gain of their children. The widening variance and shift in the median that occurs with puberty, together with attenuation of the early skew, suggests that this second wave of obesity is attributable to different factors that are now affecting the childhood population more widely. Adolescence is characterised by independence, pocket money and food choices that are influenced more by peers than by parents. New numbers are added to the ranks of obese children as they enter puberty, and the challenge for health strategists is to disentangle the different factors responsible at different ages. ■

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