RATES of pediatric obesity have increased substantially in recent years (1) and now pose a major risk for increased morbidity and premature mortality in adulthood for those affected, with cardiovascular disease (CVD) being a major contributor. In the current issue, Libman et al. (2) examine the relationship between glucose and insulin in overweight children and adolescents and a range of biomarkers for cardiovascular disease. They report that increased fasting and postprandial insulin are related to CVD risk factors, including systolic blood pressure and triglycerides, whereas glucose is not.

These results are perhaps not surprising given what we already know about glucose homeostasis in obese children and adolescents (3) and recent evidence supporting a relatively weak relationship between elevations in fasting glucose within the normal range and incident CVD events in adults (4). Hyperinsulinemia is the hallmark of metabolic dysfunction in children and adolescents, with hyperglycemia not manifesting until a very late stage of β-cell failure (3), making the process far lengthier than in adults and reflecting the ability of the young pancreas to better compensate.

Given the epidemic of childhood and adolescent obesity, there is an urgent need for a strong evidence base to help guide clinicians and other health professions in their assessment and treatment of children with these conditions. In this regard, Libman et al. conclude that “there is a need to determine the best marker for early identification of CVD morbidity in this population.” But is their suggested approach reasonable? Oral glucose tolerance tests including insulin are time-consuming and costly, and they involve a clinic visit and venepuncture which is stressful for children. Are there clear clinical advantages to this complicated, stressful test over simple measures such as body mass? The authors do not report the associations between body mass or percentage fat mass and cardiovascular risk factors, though power may be limited in their small, heterogeneous sample consisting of several ethnicities and a wide age range that spans puberty. Fortunately, such data already exist from several sources, with a number of studies (5–8) showing a strong relationship between children’s body mass and cardiovascular risk markers, carotid intima-media thickness, and adult heart mass. Perhaps more importantly, new prospective data from the Avon Longitudinal Study of Parents and Children (ALSPAC) reports strong relationships between several measures of adiposity at age 9 years and subsequent CVD risk markers at age 15 years, with BMI being equally as good an indicator as fat mass and waist circumference (9). These latter data, in turn, concur with epidemiological evidence linking adiposity in childhood with CVD risk in later life (10).

An early marker of insulin resistance might be useful if there was a specific targeted treatment option that improved outcomes in these individuals above others. Currently, no such treatment exists, though there may be temptation to use medications such as metformin or even statins in this population. Caution should be heeded: until these treatments are fully investigated in prospective, randomized trials there can be little justification for their use. Would such treatments be life-long? Although some evidence exists on the short-term relative safety of statin therapy in children (11) the unknowns of using metformin and statins are not only the effect on future cardiovascular outcomes, but also the effect in such a young cohort on future pregnancies, long-term growth, or increased diabetes risk associated with using statins (12), not forgetting the potential psychological and societal consequences of burdening children with a medical diagnosis for what is only a potential future risk. Quality of life has to be considered at all times.

Of course, what is actually required is weight loss or weight maintenance in growing children. Childhood obesity is not only likely to increase the future risk of CVD and diabetes, but also that of cancers, liver disease, osteoarthritis, depression, and anxiety—with huge implications for not only the individual but also for society through lost days of work and increased health costs. The point here is that all overweight and obese children, not just those at the highest risk for CVD or diabetes, need healthy weight interventions because weight loss will treat all potential risks, not only CVD or diabetes. Nevertheless, it was reassuring to note that children in ALSPAC who favorably altered their overweight status between childhood and adolescence improved their cardiovascular risk profile (9).

Weight loss interventions in any age-group are neither simple nor inexpensive. Best evidence suggests they need to be multicomponent, fully involving the whole family, and supported by schools and the community (13). There is some evidence for the use of bariatric surgery in severely obese adolescents (14) but no data as to the long term consequences of such action. There is an urgent need for the efforts against childhood obesity to increase several-fold; prevention of future obesity is paramount, and government intervention in the food industry is the linchpin of this. For those already affected, evidence-based weight management programs that result in global risk reduction are required. There may be a case for more targeted cardiovascular risk reduction in the future, but we must not forget that because energy imbalance is causal, it should be the primary focus of our efforts.
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