

Media Responsibility and Child Health

One of the most daunting and often frustrating tasks for a pediatrician is convincing a parent to avoid using a bottle for feeding a child. Doctors are taught from their undergraduate days about the propensity of bottle feeding to lead to infection, malnutrition, lactation failure through nipple confusion, dental caries, delayed development of chewing and swallowing, economic deprivation, and problems in young infant and toddlers(1).

To enhance breastfeeding practices, the World Health Organization discourages pacifiers and bottle-feeding. Indeed, the UNICEF/WHO Baby Friendly Hospital Initiative specifically proscribes two such exposures, pacifier use and bottle-feeding, citing their avoidance as important to the successful establishment of breastfeeding(2). Cup feeding has been recommended by the Baby Friendly Hospital Initiative, and the International Infant Food Action Network as a method for supplementing breastfed infants who require supplemental feedings(3).

Unfortunately, this message has not been percolated adequately and effectively to the public. It is said that a picture speaks a thousand words. The attractive imagery of advertising campaigns by manufacturers of artificial foods and bottles often wins over the pediatrician's pleas. On occasions a

seemingly innocent looking picture may influence a large sub set of population in the negative sense, the President's picture of display of affection by bottlefeeding a child (published in the Indian Express, June 17, 2006) being a classical example of the same. The media has, on several occasions done a yeoman task by focusing public attention on burning issues plaguing the country. With their cooperation, it is possible to take important health messages to the community. It is also pertinent to note that the media would be well advised to screen out images that legitimize practices harmful to child health.

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Leptin Resistance in Obese Indian Girls?

We read with interest the article on serum leptin concentrations by the Menon group(1). The authors have clearly reported the association of elevated serum leptin with obesity in a well designed study in Indian children.

In pre-pubertal normal weight children, serum leptin concentrations, as measured by radio-immunoassay, have been noted to be equal in girls and boys(2). In contrast, the authors(1) have observed higher leptin levels in girls than boys (23.5

± 1.78 v 18.0 ± 7.6 ng/mL) with obesity. Assuming that the cohort of obese Indian children is almost entirely pre-pubertal (90% in Tanner stage 1), the hypothesis that gender dimorphism is likely to be due to a testosterone effect may not be correct. However, this assumption may be fallacious if there were greater proportions of children with Tanner stage 2 and 3 on the female side. One wonders whether higher leptin levels were due to greater adiposity in obese girls (as a cause, not an effect); if so, leptin levels adjusted for body mass index (BMI) and BMI standard deviation scores (BMI SDS) may have provided further information.

In this study(1), in spite of a relatively small sample size (10 girls vs 26 boys), a nominal significance of difference ($P = 0.04$), a wide standard deviation in boys (± 7.6) and probable inclusion of 3 outliers with abnormal biochemical phenotypes, there remains the possibility that prepubertal Indian girls with obesity have greater leptin concentrations (adjusted for BMI SDS) than their male counterparts. This may indicate greater leptin resistance in Indian obese girls before the onset of puberty.

To improve our understanding of the origins and pathogenesis of leptin resistance and obesity in the Indian context, we suggest further study of leptin concentrations in boys and girls, with longitudinal follow up through puberty, supplemented by serial measurements of BMI, pubertal staging and measures of insulin resistance in comparison with healthy normal weight controls.

As for the 3 children with high BMI ($>30 \text{ kg/m}^2$) and unusually low serum leptin concentrations(1), it would be advisable to repeat the leptin measurement in these individuals, perhaps using a different assay(2). If serum leptin levels are truly low or undetectable, one must consider the possibility of leptin mutations in these children(3) and hence proceed to leptin gene sequencing.

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Reply

We thank Dr. Indraneel Banerjee and Dr. Dilip K Mukherjee for their observations on possible leptin resistance in Indian girls based on our study published earlier(1).

1. *Higher leptin levels in girls compared to boys:* Higher leptin levels were also observed in control girls compared to boys (7.5 ± 3.7 versus

$4.3 \pm 5.26 \text{ ng/mL}$). Higher leptin levels have been reported in prepubertal normal-weight girls compared to boys in a previous study(2). This gender difference in leptin levels in children might be related to the greater pubertal maturation in girls. This should be looked into in future studies.

2. *The hypothesis that gender dimorphism is likely to be due to a testosterone effect may not be correct.* This can be confirmed only by long-term studies on obese girls and boys. In boys, studies had shown that the leptin levels rose in parallel to weight till the age of 10 years, when a striking decrease was observed as testosterone levels rose(2). Given the proposed role of leptin in induction of puberty, higher levels are expected in girls who achieve puberty earlier than boys.
3. *One wonders whether higher leptin levels were due to greater adiposity in obese girls (as a cause, not an effect):* It is unclear whether elevated leptin levels are a cause or effect of obesity from the present study. Mere demonstration of elevated leptin levels in obese individuals as in our study does not prove a causative role of leptin resistance in the pathogenesis of obesity.
4. *Leptin levels adjusted for body mass index (BMI) and BMI standard deviation scores (BMI SDS) may have provided further information.* This may indeed be the case as leptin levels correlated well with BMI(1). Importantly the rise in leptin levels was independent of Tanner stage when controlling for adiposity. Similar observations have been reported by Hassink, *et al* previously(3). Long-term studies with comparison of leptin levels in obese girls and boys after adjustment of BMI as suggested would be helpful in deciding whether true leptin resistance is present in obese Indian girls. However such a study would be resource-intensive and demanding.
5. *If serum leptin levels are truly low or undetectable, one must consider the possibility of leptin mutations in these children and hence proceed to leptin gene sequencing.* We agree with the suggestion that leptin deficiency should