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 pre-pubertal 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 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 ± 5.26 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

be considered in obese children with low or undetectable leptin levels.

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IPV - Doubts Persist

Despite sustained efforts by the entire medical community, elimination of wild polio virus from India remains an enigma. The introduction of injectable polio vaccine[IPV] in the fray leads to a number of doubts and altered approaches on our part.

The Policy Update [1] on IPV does not come a day too soon and would be of immense benefit as a guide to the practicing pediatrician. However certain lacunae still remain.

The committee mentions "the risk of Vaccine associated paralytic polio (VAPP)" associated with OPV is lower in India. The committee fails to quantify what "lower" implies. It needs to be carefully noted that the risk is lower and not non-existent. Furthermore even if the risk is lower per dose of OPV given, is the risk really lower per patient vaccinated. We should not just calculate the risk per dose of OPV but the risk per child, each of whom may receive a large number of OPV doses in his first few years of life. In that case the risk rises exponentially. Are we ethically justified in exposing any child to OPV before adequate coverage with IPV is achieved (minimum of two doses of IPV)?

It would be ideal to avoid any dose of OPV till the child has completed his primary IPV schedule and achieved adequate antibody titers and only then allow OPV administration. Would we as pediatricians be able to defend ourselves medicolegally if a child develops VAPP due to OPV when IPV is commercially available? Also, why does the booster at 18 months and 5 years continue to remain OPV. It should be replaced by IPV which is a superior vaccine.

Another query; as exposure to the wild virus reduces, is there a risk to the population over five years and and is there any role for an additional IPV dose at ten years of age?

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