

## **Children with Prader-Willi syndrome exhibit more evident meal-induced responses in plasma ghrelin and peptide YY levels than obese and lean children.**

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Background and aims: Ghrelin is an orexigenic 28-amino acid peptide produced by the stomach. Circulating ghrelin levels rise shortly before and fall shortly after every meal. Peptide YY (PYY), an anorexigenic 36-amino acid peptide, is secreted primarily from the intestinal mucosa of the ileum and large intestine. Plasma PYY levels begin to rise within 15 min after starting to eat and plateau within ~ 90 min, remaining elevated for up to 6 h. Recently, some studies have tried to evaluate the potential role of ghrelin and PYY in the hyperphagia of patients with Prader-Willi syndrome (PWS). While hyperghrelinemia is well characterized in PWS, conflicting results have been reported for PYY. The aim of the study was to investigate ghrelin and PYY responses to a standard liquid high-fat meal in children with PWS.

Patients and methods: Circulating levels of total ghrelin and PYY levels were assayed by RIA after overnight fasting and 45, 60, 90, and 180 min following a standard meal (Ensure 6 ml/kg) in 16 patients with PWS (11 boys and five girls, aged 4.6-10.7 years, including ten receiving 0.02 mg/kg per day rhGH for 2-18 months; body mass index (BMI) z-score:  $0.6 \pm 0.2$  and  $1.6 \pm 0.5$  for children treated or not treated with rhGH respectively), ten obese (eight boys and two girls, aged 9.2-15.6 years; BMI z-score:  $2.4 \pm 0.2$ , i.e. BMI > 97th centile for chronological age and sex) subjects, and 16 normalweight controls (five boys and 11 girls, aged 5.8-17.3 years; BMI z-score:  $0.6 \pm 0.2$ ).

Results: PWS children showed higher fasting levels of ghrelin than obese and lean controls. Postprandial ghrelin drop was more pronounced in PWS than in the other study groups. No significant difference on fasting levels of PYY was found among groups. PWS showed a higher postprandial PYY rise than obese and lean controls. PWS patients treated and not treated with GH showed similar fasting and postprandial levels of ghrelin and PYY. Fasting PYY levels correlated negatively ( $P < 0.05$ ;  $r = -0.68$ ) with those of ghrelin only in PWS.

Conclusions: The results of this study confirm fasting hyperghrelinemia in PWS. Since in PWS adults an impaired postprandial suppression of plasma ghrelin was previously reported to be associated with a blunted postprandial PYY response, the finding of a meal-induced decrease and increase in ghrelin and PYY levels respectively in PWS children would imply that the regulation of appetite/satiety of these peptides is operative during childhood, and it progressively deteriorates and vanishes in adulthood when hyperphagia and obesity worsen.

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