In adults with Prader-Willi syndrome, elevated ghrelin levels are more consistent with hyperphagia than high PYY and GLP-1 levels

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OBJECTIVE
Prader-Willi syndrome (PWS) is a leading genetic cause of obesity, characterized by hyperphagia, endocrine and developmental disorders. It is suggested that the intense hyperphagia could stem, in part, from impaired gut hormone signaling. Previous studies produced conflicting results, being confounded by differences in body composition between PWS and control subjects.

DESIGN
Fasting and postprandial gut hormone responses were investigated in a cross-sectional cohort study including 10 adult PWS, 12 obese subjects matched for percentage body fat and central abdominal fat, and 10 healthy normal weight subjects.

METHODS
PYY[total], PYY[3-36], GLP-1[active] and ghrelin[total] were measured by ELISA or radioimmunoassay. Body composition was assessed by dual energy X-ray absorptiometry. Visual analog scales were used to assess hunger and satiety.

RESULTS
In contrast to lean subjects (p<0.05), PWS and obese subjects were similarly insulin resistant and had similar insulin levels. Ghrelin[total] levels were significantly higher in PWS compared to obese subjects before and during the meal (p<0.05). PYY[3-36] meal responses were higher in PWS than in lean subjects (p=0.01), but not significantly different to obese (p=0.08), with an additional non-significant trend in PYY[total] levels. There were no significant differences in self-reported satiety between groups, however PWS subjects reported more hunger throughout (p=0.003), and exhibited a markedly reduced meal-induced suppression of hunger (p=0.01) compared to lean or obese subjects.

CONCLUSIONS
Compared to adiposity-matched control subjects, hyperphagia in PWS is not related to a lower postprandial GLP-1 or PYY response. Elevated ghrelin levels in PWS are consistent with increased hunger and are unrelated to insulin levels.

type: journal paper/review (English)
date of publishing: 01-07-2011
journal title: Neuropeptides (45/4)
ISSN electronic: 1532-2785
pages: 301-7