Prolactin release is enhanced in proportion to excess visceral fat in obese women

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Prolactin (PRL) promotes (visceral) fat accrual in a variety of animal models. The release of PRL by the pituitary is tonically inhibited by dopamine through activation of the dopamine D2 receptor (D2R) of lactotroph cells and obese humans appear to have reduced D2R binding sites in their brain. Therefore, we hypothesized that spontaneous PRL release is enhanced in obese humans. To evaluate this hypothesis, we measured 24h plasma PRL concentrations at 10 min intervals in eleven obese premenopausal women (BMI 33.3 ± 0.7 kg/m^2) and ten lean premenopausal women of similar age (BMI 21.2 ± 0.6 kg/m^2). Total body fat was determined using DEXA and subcutaneous and visceral fat area was measured by MRI in ten obese subjects. PRL secretion rate was estimated by deconvolution analysis. All subjects were studied in the early follicular stage of their menstrual cycle. PRL secretion was significantly enhanced in obese women (total daily release 137 ± 8 *vs.* lean controls 92 ± 8 µg/L/24 h, P = 0.001) in proportion to their BMI (R^2 = 0.55, P < 0.001). Interestingly, PRL release was particularly associated with the size of the visceral fat mass (total PRL secretion *vs.* visceral fat area R^2 = 0.64, P = 0.006). These data show that spontaneous PRL release is considerably enhanced in obese women in proportion to the size of their visceral fat mass. Since PRL is inhibited by D2R activation we speculate that elevated PRL secretion may be due to reduced D2R availability in the brain.

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