To gain further insight into the regulatory role of insulin and leptin on plasma ghrelin, 56 normal weight, 128 normoinsulinemic obese, 121 hyperinsulinemic obese, and 30 type 2 diabetic normoinsulinemic and 75 type 2 diabetic hyperinsulinemic obese patients were examined. In the obese subjects, basal hyperinsulinemia was associated with significantly lower ghrelin independent of BMI, age, and leptin. In normoinsulinemic (normal weight and normoinsulinemic obese) subjects, ghrelin was inversely related to stepwise increasing leptin. Multiple regression analysis and matching for insulin revealed a significant negative interaction of ghrelin with leptin but not insulin. In type 2 diabetic normoinsulinemic subjects, ghrelin was significantly lower compared with that in normoinsulinemic obese subjects. In type 2 diabetic hyperinsulinemic subjects, ghrelin was significantly lower than in normoinsulinemic subjects, whereas no further reduction was observed compared with hyperinsulinemic obese subjects. The postprandial decrease was significantly attenuated in normoinsulinemic obese and hyperinsulinemic obese subjects (-214.8 +/- 247 pg/ml [normal weight], -137.6 +/- 107 pg/ml [normoinsulinemic obese], -85.5 +/- 69 pg/ml [hyperinsulinemic obese], P<0.001; mean +/- SD), whereas type 2 diabetes had no independent postprandial effect. In conclusion, the present data support the concept that leptin could be of importance for
suppression of basal ghrelin during moderate weight gain in normoinsulinemic subjects, whereas hyperinsulinemia but not leptin is responsible in more severe obesity. Postprandial suppression of ghrelin is attenuated by as yet unknown mechanisms that are related to body weight but not to insulin or type 2 diabetes.

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