Progressive weight gain to a BMI of 89 kg/m^2 following a road traffic accident:

a collusion of nature and nurture?

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We report the case of a 23-year Emirati man who presented to our endocrinology outpatient department with morbid obesity. His weight at the time of his first visit was 230.8 kg (BMI 89 kg/m^2). Notable in his history was hyperphagia which was more prominent following a road traffic accident (RTA) he had sustained at the age of 13; he had major injuries to his left side of his body when he was dragged by the school bus for several meters. He reported no direct head injury. He underwent several operations followed by a prolonged recovery phase in hospital. He dated much of his weight gain (55 kg to over 100 kg) to this period of four months or so, although that continued progressively since. Other notable parts of the medical history included type 2 diabetes and hypertension. On examination there were extensive scars on the left side of his body from previous RTA. His testes were noted to be small (8 mls); facial and body hair were scant. He also had a borderline elevated blood pressure (142/72 mmHg). There was a strong family history of obesity; six (of seven) of his siblings from the same mother were obese and had had bariatric surgery. One sister died at an early age. His other siblings (total 16) did not have a weight problem.

Initial investigations showed a glycosylated haemoglobin of 7.2 % (8.89 mmol/l); evidence of subclinical hypothyroidism (TSH 5.8 uIU/ml; free T4 14.91 pmol/L), vitamin D deficiency and low serum testosterone (2.23 nmol/L). He was commenced on exenatide, metformin, cholecalciferol and valsartan. His glycemic control improved (HbA1c 6.4 %) in the ensuing months and there was a modest weight loss of 2 kg.

He failed to attend for his follow-up appointment and re-presented seven years later, having had sleeve gastrectomy and gastric bypass plus cholecystectomy the previous year. He had had a good response with a weight reduction down to 181 Kg (BMI 69.83 kg/m^2). His diabetes had resolved with glycosylated haemoglobin of 5.4 % on no antidiabetic medication. His resting metabolic rate was checked and noted to be substantially lower than the expected RMR (2123 kcal/day vs 3156 kcal/day) for his weight. Further investigations indicated hypergonadotrophic hypogonadism (FSH 19.66 IU/mL and LH 20.05 IU/mL). Prolactin and cortisol levels were normal. Follow-up pituitary MRI is awaited.

Given the strong family history, monogenic obesity is a major possibility, which may have put him at a much higher risk of weight gain following his RTA and his recovery phase from it. Whether this case represents an unusual presentation of hypothalamic obesity remains unclear. Further investigations including gut hormone profile and genetic testing are planned. This case may have important insights into hormonal pathways in obesity and warrant further studies.