	Subject Age			
	0–6 Years	6–10 Years	10–12 Years	12–18 Years
Clinical assessments	Patient medical history Physical examination Blood pressure Information about ULSCO	Patient medical history Physical examination Blood pressure Information about ULSCO	Patient medical history Physical examination Blood pressure Information about ULSCO	Patient medical history Physical examination Blood pressure Information about ULSCO
Clinical examinations	Weight Height Waist/hip circumference Skinfold thickness (caliper) Bioimpedance Indirect calorimetry Accelerometer	Weight Height Waist/hip circumference Skinfold thickness (caliper) Bioimpedance Indirect calorimetry Accelerometer Bodpod 6-minute-walk test	Weight Height Waist/hip circumference Skinfold thickness (caliper) Bioimpedance Indirect calorimetry Accelerometer Bodpod 6-minute-walk test	Weight Height Waist/hip circumference Skinfold thickness (caliper) Bioimpedance Indirect calorimetry Accelerometer Bodpod 6-minute-walk-test
Questionnaires	Goals Nutrition Sleep Well-being/welfare Stress Physical activity Relationships/network	Goals Nutrition Sleep Well-being/welfare Stress Physical activity Relationships/network	Goals Nutrition Sleep Well-being/welfare Stress Physical activity Relationships/network	Goals Nutrition Sleep Well-being/welfare Stress Physical activity Relationships/network
Fasting blood samples	2–3 EDTA/P800 tubes,[a] plasma and whole blood	3–4 EDTA/P800 tubes, plasma and whole blood	4–5 EDTA/P800 tubes, plasma and whole blood	4–5 EDTA/P800 tubes, plasma and whole blood
Oral glucose tolerance test	Samples obtained at 5, 10, 15, 30, 60, 90, and 120 min using EDTA tubes	Samples obtained at 5, 10, 15, 30, 60, 90, and 120 min using EDTA/P800 tubes	Samples obtained at 5, 10, 15, 30, 60, 90, and 120 min using EDTA/P800 tubes	Samples obtained at 5, 10, 15, 30, 60, 90, and 120 min using EDTA/P800 tubes
Frequent blood sampling[b]	—	Sampling every 1–2 min using EDTA tubes	Sampling every 1–2 min using EDTA tubes	Sampling every 1–2 min using EDTA tubes
MRI[b]	—	—	DSAT/SSAT and VAT, BAT, liver fat, pancreatic fat	DSAT/SSAT and VAT, BAT, liver fat, pancreatic fat
Clamps[b]	—	—	—	Using hyperinsulinemic and hyperglycemic clamp techniques
Tissue biopsies[b]	—	—	—	SSAT

Table 1: Procedures performed in the Beta-JUDO project in obese and lean children of different ages. From (1).

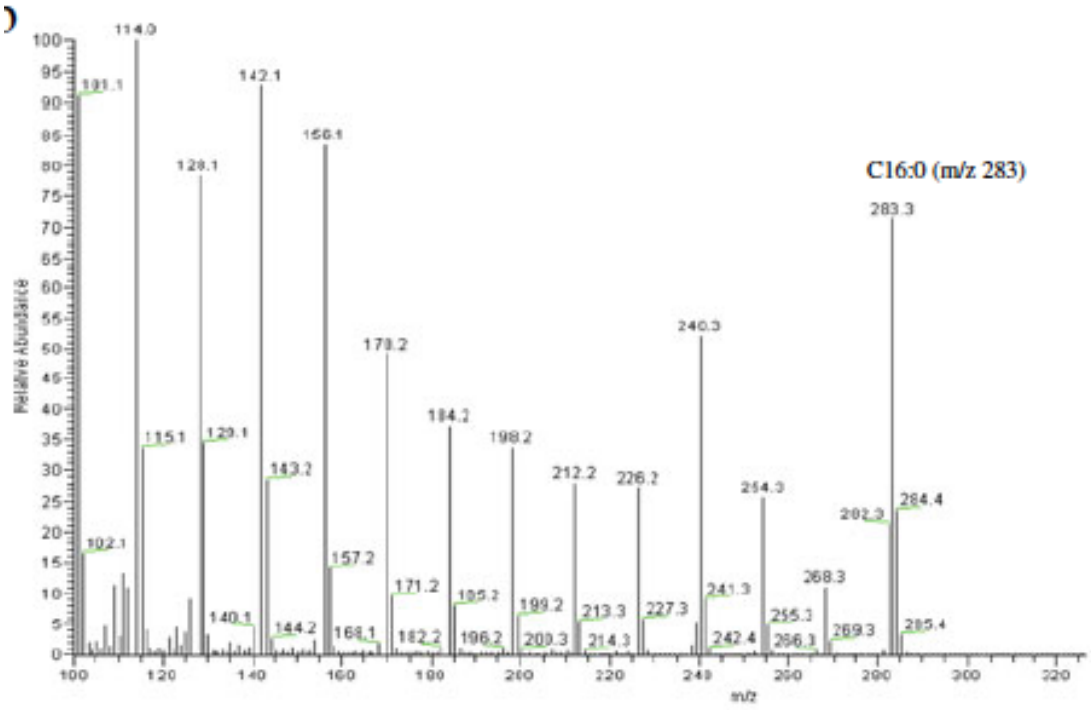

Figure 1: MS spectrum scan of palmitic acid. From (2).

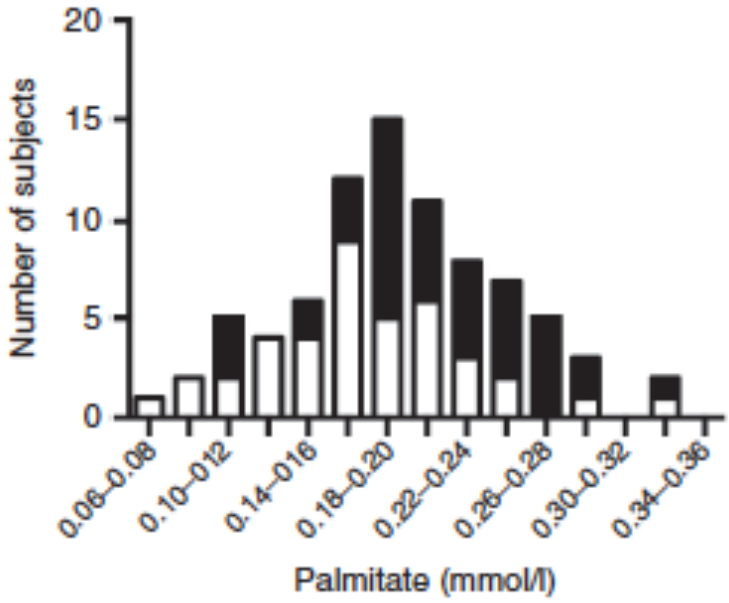

Figure 2: Fasting palmitate levels in obese (black bars) and lean (white bars) children and adolescents. From (3).

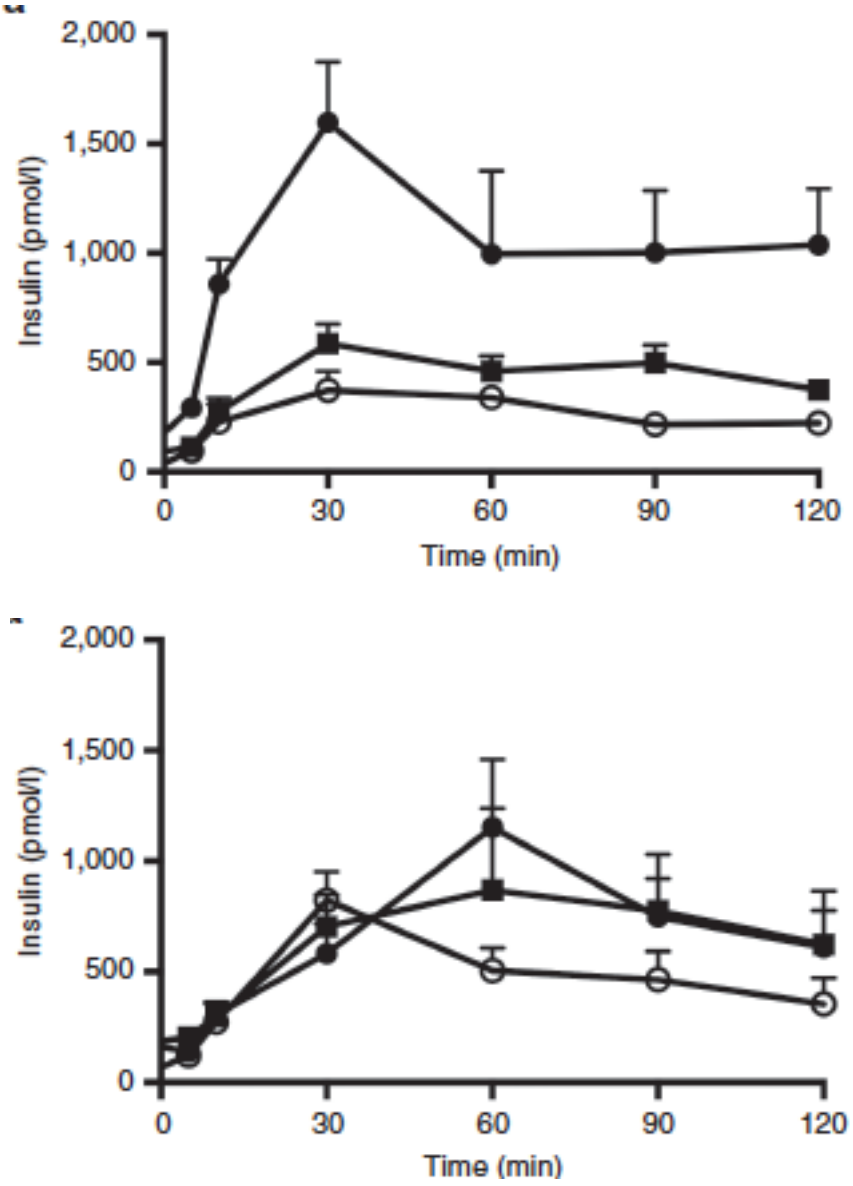

Figure 3: Insulin secretion during OGTT in obese and lean children (top panel) and adolescents (bottom panel) with high (black circles) or low (black squares) palmitate levels and lean children (open circles). From (3).

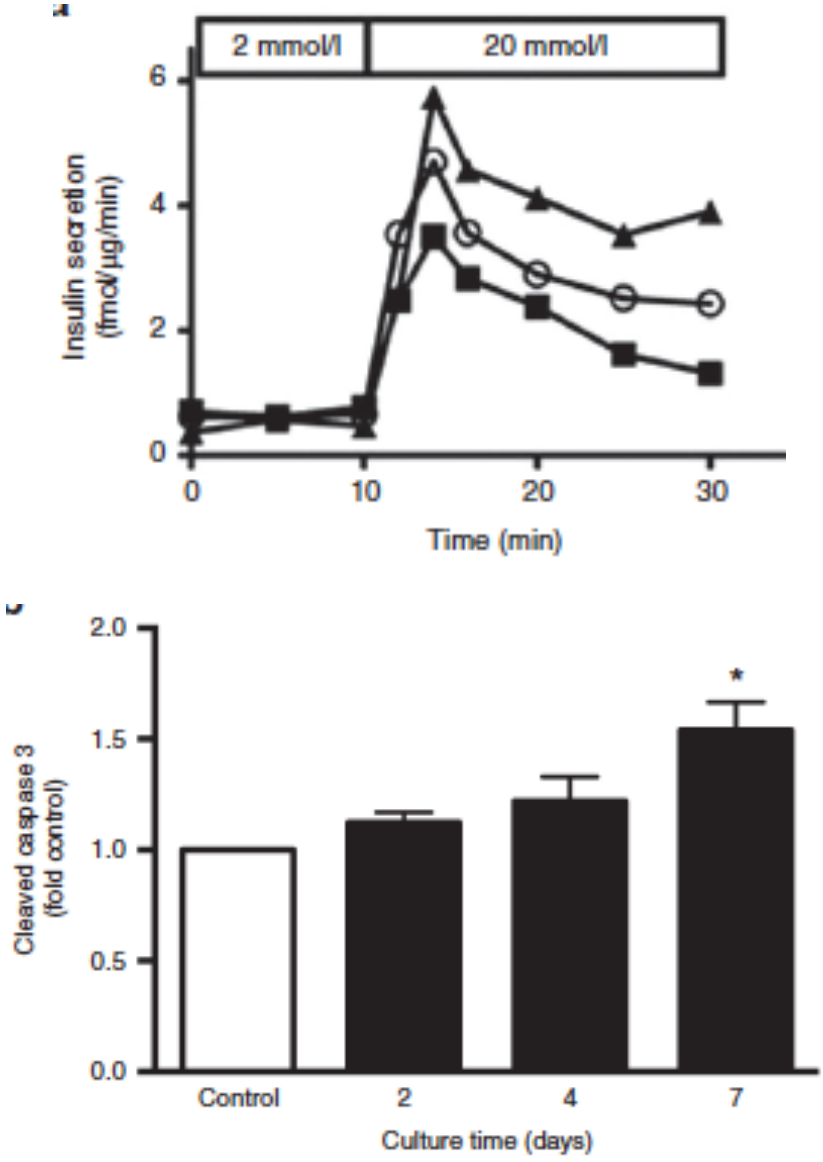

Figure 4: Glucose-stimulated insulin secretion (top panel) from islets cultured in the presence of palmitate for 0 (open circles), 2 (black triangles) or 7 (black squares) days. Apoptosis (bottom panel) was measured in islets cultured in the presence of palmitate for the indicated number of days. Control cells were not exposed to the fatty acids. From (3).

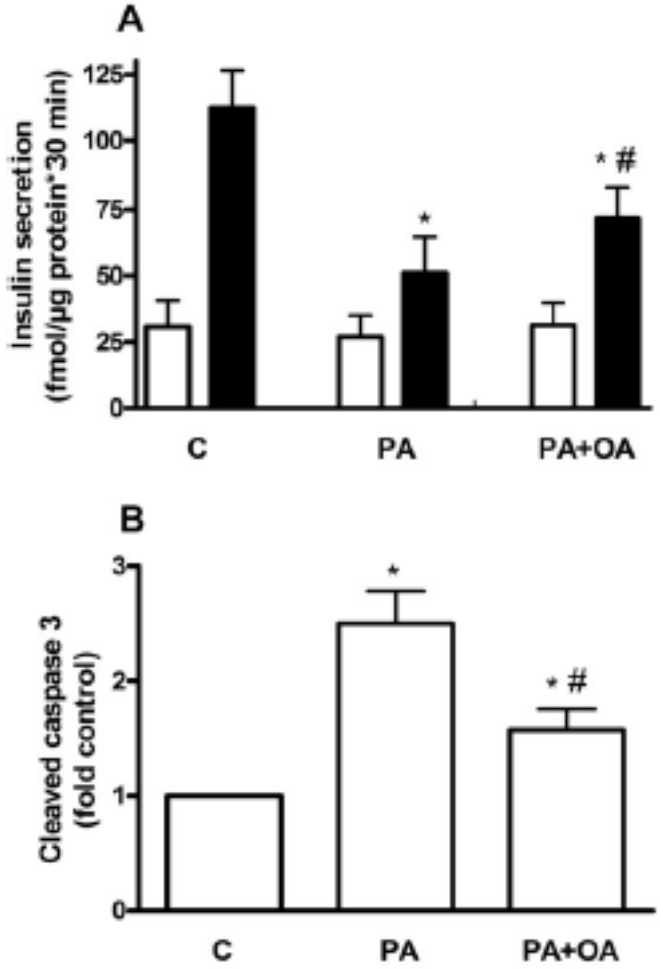

Figure 5: Insulin secretion (panel A) at 2 (white bars) or 20 (black bars) mM glucose from insulin-producing MIN6 cells exposed to palmitate (PA) and oleate (OA) for 2 days. Apoptosis (panel B) was measured in the cells after 2 days. Control (C) cells were not exposed to the fatty acids. From (4).

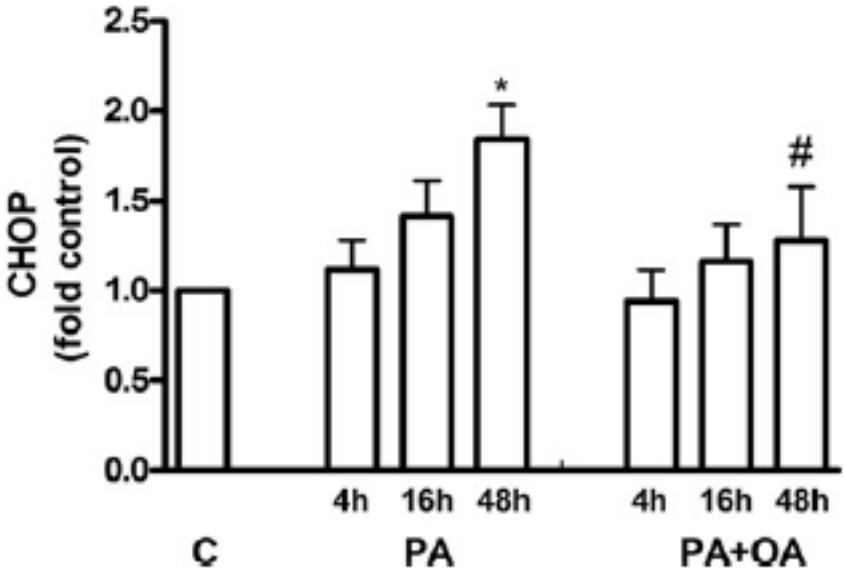

Figure 6: Levels of the pro-apoptotic ER stress factor CHOP in insulin-producing MIN6 cells exposed to palmitate (PA) and oleate (OA) for the indicated time periods. Control (C) cells were not exposed to the fatty acids. From (4).

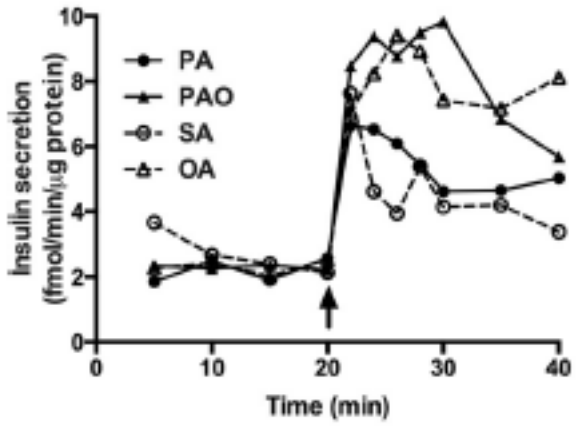

Figure 7: Effects of fatty acids on insulin secretion from human islets after acutely exposing to palmitate (PA), palmitoleete (PAO), sterate (SA) or oleate (OA). From (5).

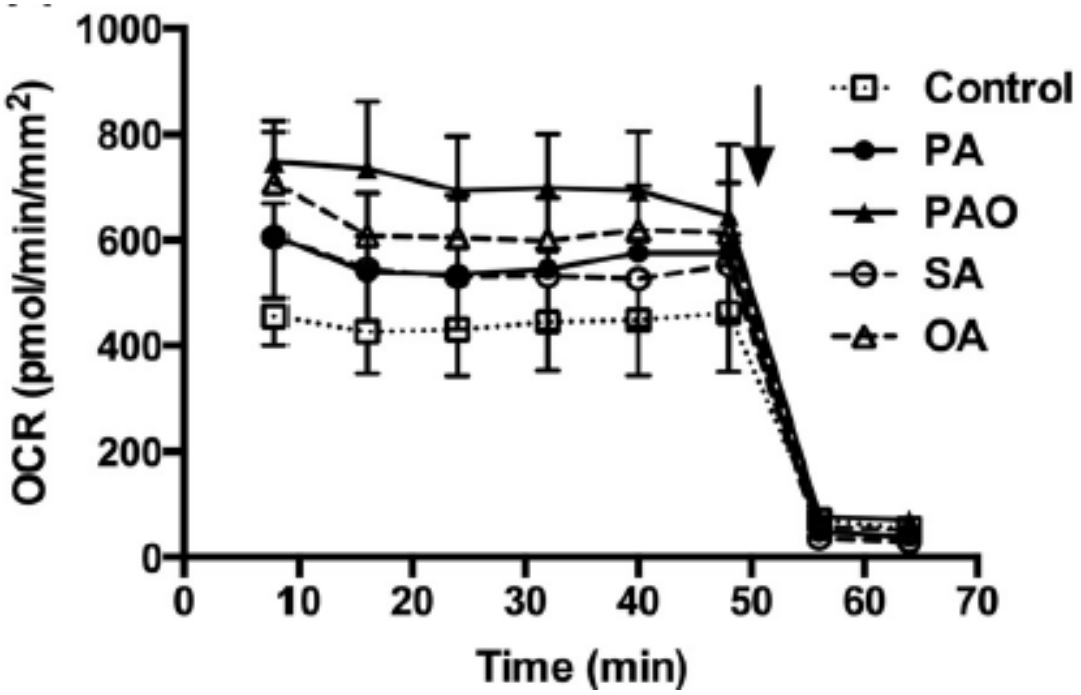

Figure 8: Effects of fatty acids on metabolism in human islets after acutely exposing to palmitate (PA), palmitoleete (PAO), sterate (SA) or oleate (OA). From (5).

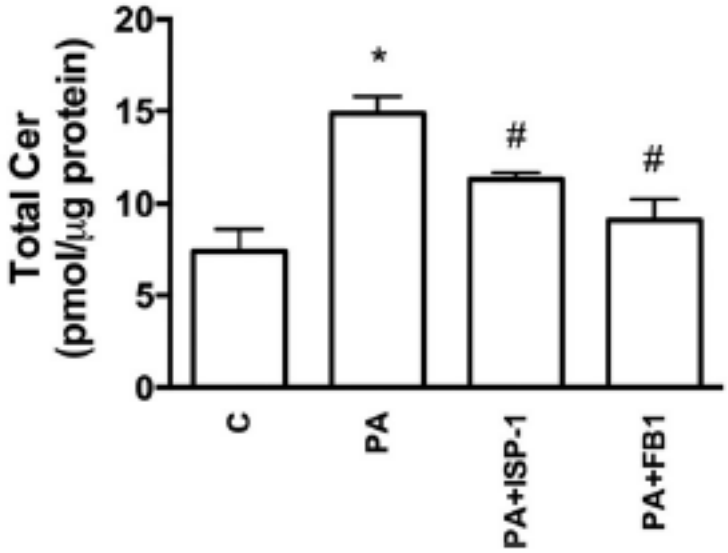

Figure 9: Ceramide amounts in insulin-secreting cells exposed to palmitate (PA) in the presence of not (C) in presence of SPT inhibitor ISP-1 or ceramide synthases FB1. From (6).

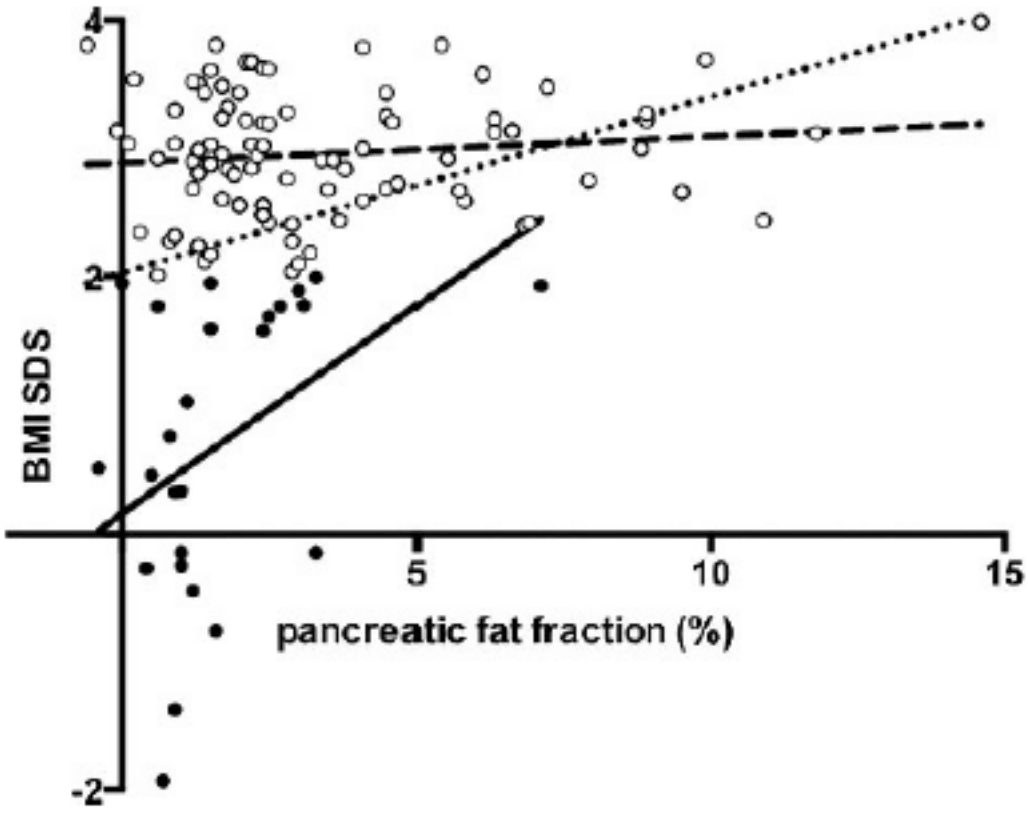

Figure 10: Scatter plot and regression lines of BMI-SDS and pancreas fat in non-obese (black circles) and obese (white circles) adolescents. From (7).

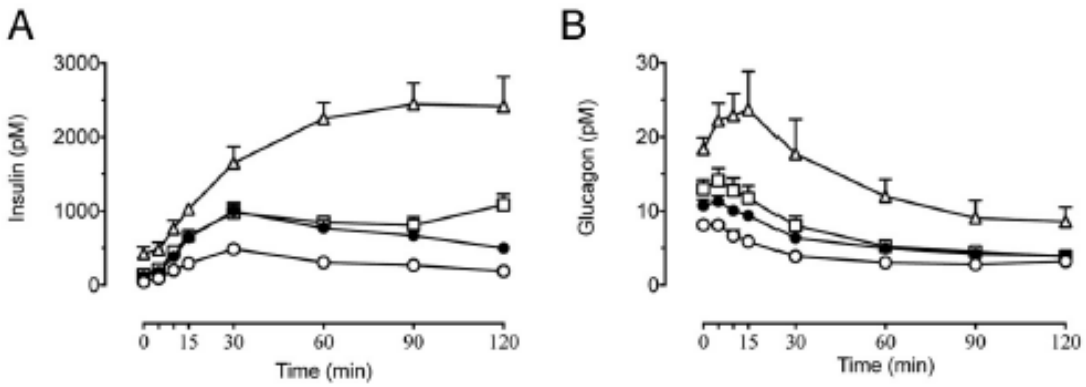

Figure 11: Circulating insulin (panel A) and glucagon (panel B) in lean control children (open circles), obese children with NGT (black circles), obese with IFG or OGT (white squares) and obese children with T2DM (white triangles).

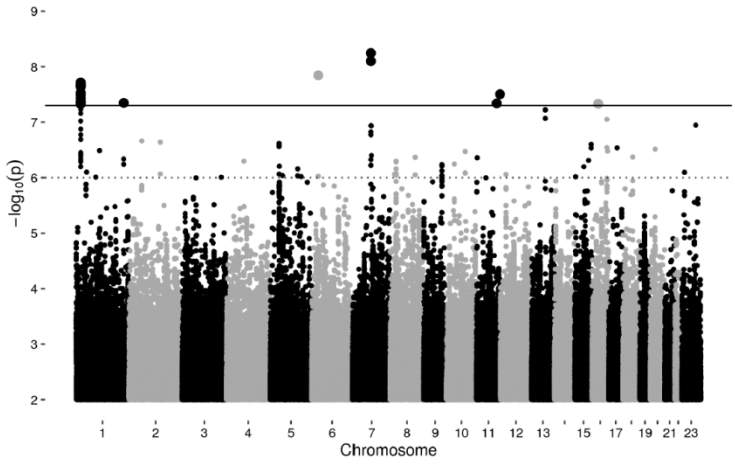

Figure 12: GWAS in childhood obesity cohorts has identified 45 independent variants with $p_{log}>6$.

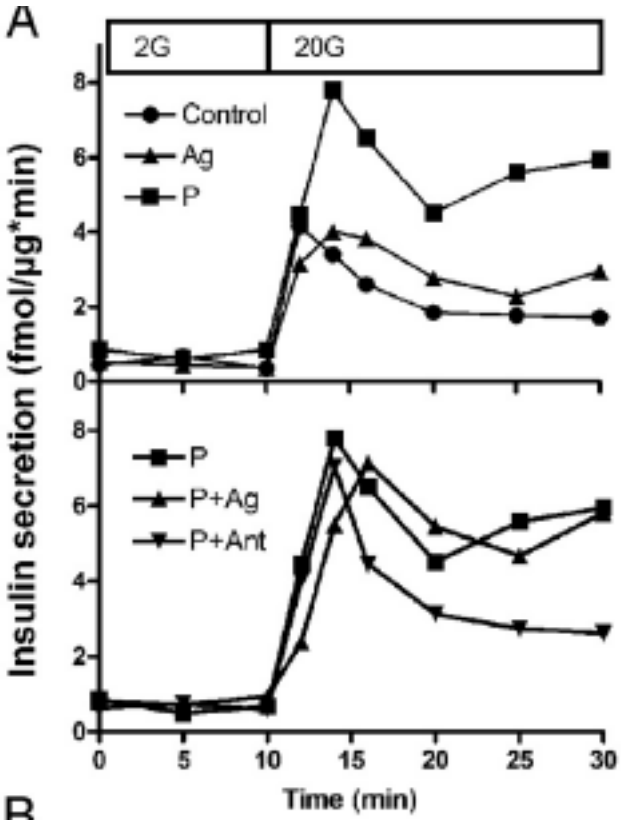

Figure 13: Glucose-stimulated insulin secretion from human islets acutely exposed to palmitate (P) and FFAR1 antagonist (Ant) or FFAR1 agonist (Ag). From (8).

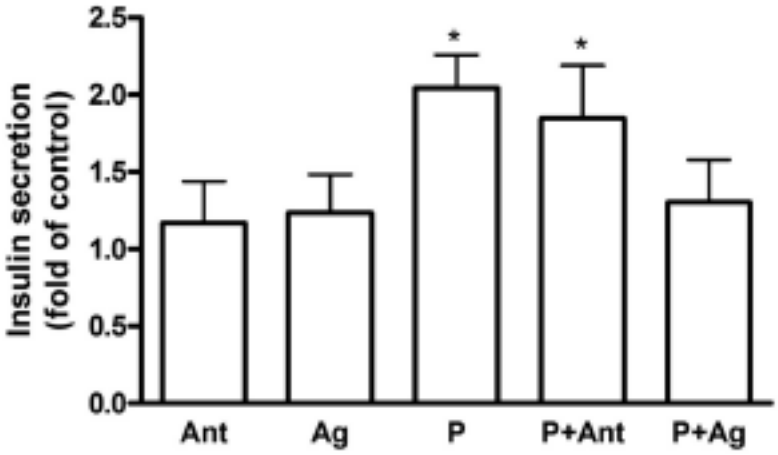

Figure 14: Glucose-stimulated insulin secretion from human islets exposed to palmitate (P) and FFAR1 antagonist (Ant) or FFAR1 agonist (Ag) for 2 days. From (8).

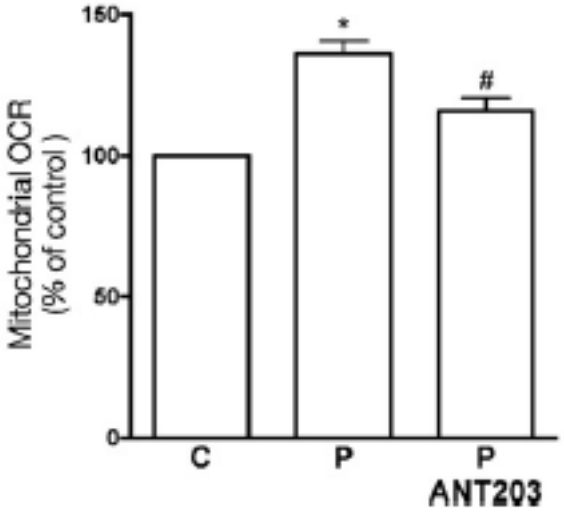

Figure 15: Effects of palmitate (P) and FFAR1 antagonist (ANT203) on metabolism in human islets exposing to the fatty acid for 2 days. From (9).

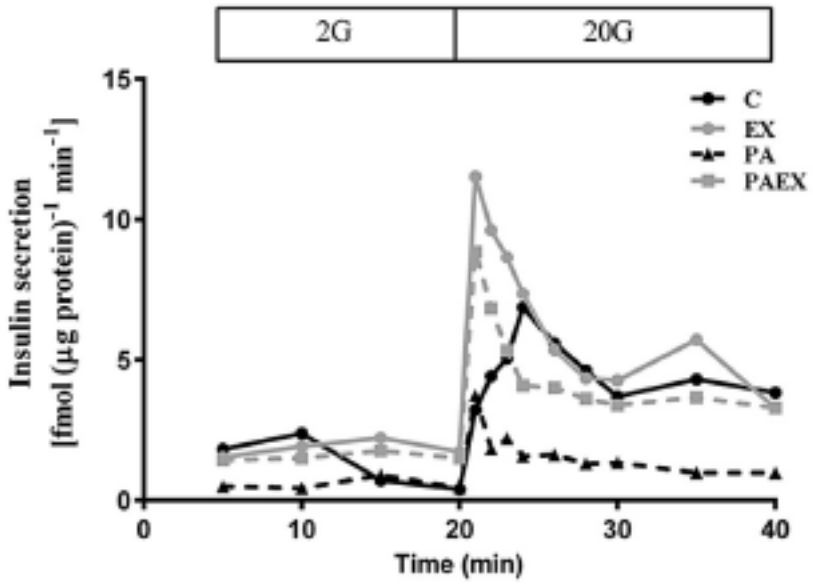

Figure 16: Glucose-stimulated insulin secretion from human islets exposed to palmitate (PA) and GLP-1 analogue exendin-4 (EX). From (10).

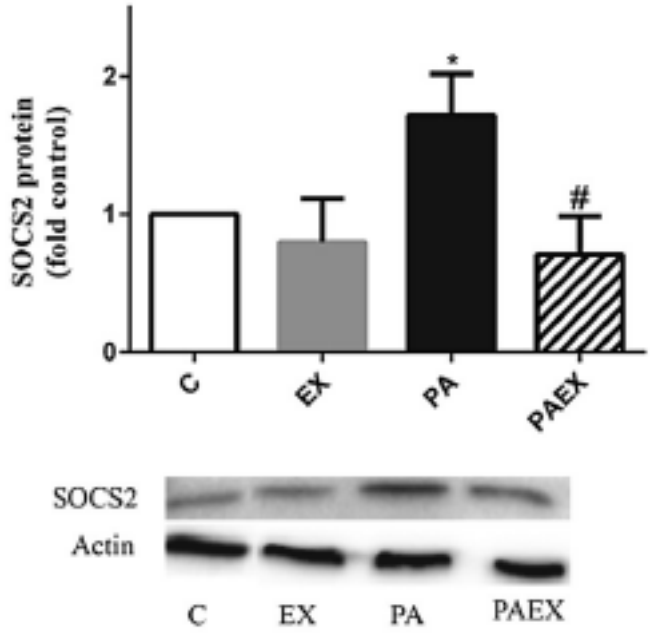

Figure 17: SOCS2 protein levels in human islets exposed to palmitate (PA) and GLP-1 analogue exendin-4 (EX). From (10).