samples. Although $CD8^+/CD45^+/perf^-$ and $CD56^+/CD45^+/perf^-$ cells were identified. Next, we analysed the same cells for cytotoxic activation by 107a. The percentage of 107a positivity was low in CD 8^+ (7% and 4 % respectively in cases and control) and CD 56^+ cells (10% and 9 % respectively in cases and control),Although clinically type 2 DM subjects were obese and had inflammation (i.e higher hsCRP), there was no difference in VAT activation of immune cells studied. Also, we could not delineate perforin in any of the samples. **Conclusion:** Taken together this work suggests VAT T cell immune milieu in human Type 2 DM is different from mouse model. It is neither characterised by perforin deficiency nor activation of T cell/NK cell. This study points towards the probability that, the role of T cell/NK cells in human VAT infiltration could be fundamentally different from mice models. Further studies should be focussed on functional characteristics of these cells and interaction with VAT macrophages. **References** 1. Xavier S. Revelo et al Diabetes 2015;64:90–103 2. Wetzels S et al J Vis Exp. 2018 Mar 6;(133):57319.

Adipose Tissue, Appetite, and Obesity
INTEGRATED PHYSIOLOGY OF OBESITY AND METABOLIC DISEASE

Impact of Morbid Obesity on Patients With Hypertriglyceridemia Induced Acute Pancreatitis

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Introduction: Obesity is reportedly associated with worse outcome in patients with acute pancreatitis (AP). However, AP has varying etiologies. Hypertriglyceridemia induced acute pancreatitis (HTGP) has sociodemographic variations compared to AP from biliary stones or alcohol. This study aimed to determine the impact of obesity on outcomes of patients with HTGP. **Methods:** This was a retrospective cohort study of the combined Nationwide Inpatient Sample database for 2016 and 2017. Hospital discharges of patients 18 years and over with HTGP were included. This cohort was divided based on presence of comorbid obesity into three groups- patients without obesity, mild-moderate obesity (MMO) (BMI: 30.0 - 39.9) and morbid obesity (MO) (BMI >=40.0). Primary outcome was inpatient mortality. Secondary outcomes included length of hospital stay (LOS), total hospital charges (THC), discharge diagnoses of hypocalcemia, sepsis, septic shock, acute renal failure (AKI) and acute respiratory failure (ARF). Multivariate regression analysis was used to adjust for patients' sociodemographic factors, Charlson comorbidity index as well as hospital characteristics as confounders. **Results:** A total of 104,465 hospitalizations were principally for HTGP, accounting for 18.2% of patients with acute pancreatitis during the study period. Of the patients with HTGP, 13.7% and 10.9% of these patients classified as having MMO and MO respectively. Patients with obesity were significantly younger than patients without obesity.
In patients with MO, there was higher odds of mortality (aOR=1.83, 95% CI: 1.090 – 3.083, p=0.022), while there was no difference in mortality in patients with MMO (aOR 1.09 95% CI: 0.609 – 1.940, p=0.777), both compared with patients without obesity. Patients with MO had increased mean LOS of 0.5 days (95% CI: 0.3 – 0.7, p<0.001) as well as increased THC of $3977 (95% CI: 1467 – 6487, p=0.002) compared to those without obesity. There was no difference in mortality, THC and LOS in patients with MMO. Morbidly obese patients also had increased odds of septic shock (aOR=2.27, 95% CI: 1.297 – 3.972, p=0.007), AKI (aOR=1.28, 95% CI: 1.120 – 1.459, p<0.001), and ARF (aOR=1.94, 95% CI: 1.491 – 2.524, p<0.001). Conclusion: Morbid obesity is associated with higher mortality and poor outcomes in patient with hypertriglyceridemia induced pancreatitis.

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Impact of Semaglutide on Body Composition in Adults With Overweight or Obesity: Exploratory Analysis of the STEP 1 Study

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Background: Central obesity is associated with increased risk of cardiometabolic disease. Weight loss reduces lean muscle mass, potentially impacting resting energy expenditure and/or physical functioning. This analysis of the STEP 1 trial evaluated the impact of subcutaneous (s.c.) semaglutide, a glucagon-like peptide-1 analogue, on body composition in adults with overweight/obesity using dual energy X-ray absorptiometry (DEXA).

Methods: In STEP 1, 1961 adults aged ≥18 years with body mass index (BMI) ≥27 kg/m^2 with ≥1 weight-related comorbidity or BMI ≥30 kg/m^2, without diabetes, were randomized to s.c. semaglutide 2.4 mg once-weekly or matched placebo (2:1) for 68 weeks, plus lifestyle intervention. Participants with BMI ≤40 kg/m^2 from 9 sites were eligible for the substudy. Total fat mass, total lean body mass and regional visceral fat mass were measured using DEXA at screening and week 68; visceral fat mass was calculated in the L4 region (both males/females), android region (males), or gynoid region (females), depending on site scanner methodology. Proportions of total fat and lean body mass are shown relative to total body mass; proportion of visceral fat mass is expressed relative to region assessed.
Results: This analysis included 140 participants (semaglutide n=95; placebo n=45) (mean weight 98.4 kg, BMI 34.8 kg/m^2; 76% female). Baseline body composition was similar in those receiving semaglutide and placebo (total fat mass proportion: 43.4% vs 44.6%; regional visceral fat mass proportion: 33.8% vs 36.3%; total lean body mass proportion: 53.9% vs 52.7%; respectively). Percentage change in body weight from baseline to week 68 was -15.0% with semaglutide vs -3.6% with placebo. This resulted in reductions from baseline with semaglutide in total fat mass (-19.3%) and regional visceral fat mass (-27.4%), leading to 3.5%-point and 2.0%-point reductions in the proportions of total fat mass and visceral fat mass, respectively. Total lean body mass decreased from baseline (-9.7%); however, the proportion relative to total body mass increased by 3.0%-points. An increasing improvement in lean body mass:fat mass ratio was seen with semaglutide with increasing weight loss from baseline to week 68 (continuous data). Overall, the ratio increased from baseline (1.34 [95% CI: 1.22, 1.47]) to week 68 by 0.23 [0.14, 0.32], with greater improvement in those with ≥15% weight loss (n=44; 0.41 [0.28, 0.53]) vs <15% weight loss (n=39; 0.03 [-0.05, 0.12]) (observed, dichotomized data; no imputation for missing data). There were no major changes in body composition with placebo from baseline to week 68.
Conclusion: In adults with overweight/obesity, semaglutide 2.4 mg was associated with reduced total fat mass and regional visceral fat mass, and an increased proportion of lean body mass. Greater weight loss was associated with greater improvement in body composition (lean body mass:fat mass ratio).

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Incidence of Insulin Resistance in Obese Adolescent of Type-2 Diabetes Mellitus Patients

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Background: Insulin resistance is a reduced response of tissue to insulin-mediated action on cells. It may be due to many reasons, including the surplus of adipose tissue, which cause a resistance of insulin. Aims and **Objectives:** To find the incidence of insulin resistance in obese adolescent of type-2 diabetes mellitus patients.
Material and **Methods:** The study involved 50 adolescents aged 14–20 years old. Adolescents with BMI > 26.0 Kg/m2 were included in the study. Levels of fasting blood sugar, Hb A1c and serum insulin were estimated. The index of Homeostatic model assessment for insulin resistance or HOMA-IR was calculated. The cut-off value of HOMA-IR was > 3.16 for both genders.
Results: It was observed that the values of BMI and level of fasting blood sugar of first degree relatives of diabetics was significantly higher as compared to their controls. Levels of both blood HbA1c and serum insulin were increased but significant difference was observed only in case of serum insulin when compared with their controls.
Conclusion: Obesity in adolescents of first degree relatives of diabetics shows a major reason of insulin resistance. The incidence of insulin resistance in obese adolescents signals a perturbing trend for the burden of type 2 diabetes in our country.

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Insulin Resistance Moderates the Association Between BMI and Metabolic Syndrome Severity in Women 4–10 Years After Pregnancy, Independent of Gestational Diabetes Status

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Objective: Obesity and gestational diabetes mellitus (GDM) increase the risk for metabolic syndrome (MetS). Insulin resistance (IR) is associated with obesity, contributes to risk for GDM, and persists after pregnancy even when glucose tolerance returns. Further, IR may enhance the risk of MetS associated with obesity and GDM. The purpose of this study was to test the hypothesis that IR moderates the relationship between BMI and MetS severity 4–10 years after pregnancy, independent of prior GDM, such that the positive association between BMI and MetS severity is stronger among women with greater IR. **Methods:** This hypothesis was tested in a secondary analysis of data collected from women enrolled in a study of the intergenerational transmission of obesity, 4–10 years after the index pregnancy. Recruitment in the parent study was stratified to include women with normal weight without GDM (NW), overweight or obesity without GDM (OwOB), and women with GDM during the index pregnancy. Standard clinical procedures were used to measure height, weight, waist circumference and blood pressure, and a fasting blood draw was obtained with which to measure glucose, insulin, triglycerides, and HDL-cholesterol. MetS was evaluated as