TABLE 1 Participant characteristics of the two study groups

	NGR (n = 12)	IGR (n = 15)	p value
Age (years)	57 ± 12	62 ± 9	0.21
Weight (kg)	107.1 ± 18.5	106.6 ± 14.6	0.94
BMI (kg/m^2)	34.0 ± 4.5	34.3 ± 3.1	0.84
Waist circumference (cm)	111.4 ± 9.3	113.1 ± 10.4	0.67
Body fat (%)	34.1 ± 3.5	35.1 ± 3.7	0.46
HTGC (%)	15.9 ± 8.8	15.0 ± 4.8	0.74
HbA1c (mmol/mol)	37 ± 4	50 ± 6	<0.001
HbA1c (%)	5.6 ± 0.3	6.7 ± 0.5	<0.001
Peak oxygen uptake (ml/kg/min)	28.9 ± 4.1	26.3 ± 3.5	0.11

Note. Data are means ± SD.
Abbreviations: BMI, body mass index; HbA1c, glycated haemoglobin; HTGC, hepatic triglyceride content; IGR, impaired glucose regulation; NGR, normal glucose regulation.

calculated. Participants underwent a maximal incremental exercise test for assessment of peak oxygen uptake, and a venous blood sample was obtained for measurement of plasma HbA1c.

RESULTS: As intended, plasma HbA1c was higher in the IGR group compared to the NGR group (p < 0.001; ES = 2.64), while all other characteristics were similar between groups (Table 1). Hepatic SI tended to be higher (p = 0.055; ES = 0.79; Figure 1A), whereas hepatic UI and PUI tended to be lower in the IGR group compared to the NGR group (both $p \leq 0.055$; ES ≥ 0.79; Figure 1B,C). No differences in hepatic SCI were observed between the two groups (Figure 1D). Across the whole study population, peak oxygen uptake was positively related to hepatic UI and PUI (both $r \geq 0.44$; $p \leq 0.033$) and inversely related to hepatic SI (r = –0.44; p = 0.033).

CONCLUSION: This study suggests that the hepatic lipids of patients with NAFLD and IGR may be composed of more saturated and less polyunsaturated lipids than patients with NGR. Furthermore, this lipid profile may be associated with lower cardiorespiratory fitness. Additional analyses are needed to determine the relationship between hepatic lipid composition and metabolic function in these patient groups.

CONFLICT OF INTEREST: None.

FUNDING: This research was funded by Diabetes UK and the NIHR Leicester and Nottingham Biomedical Research Centres.

AD02-06 | Effects of maternal obesity and direct exposure to high-fat diet on bowel functions in offsprings

F. Garelli[1]; A. Nericcio[1]; L. Antonioli[2]; L. Benvenuti[2]; V. D'antongiovanni[2]; C. Pellegrini[2]; M. Fornai[2]; C. Blandizzi[2]; R. Colucci[1]

[1]Department of Pharmacological and Pharmaceutical Science, University of Padova, Padova, Italy; [2]Division of Pharmacology and Pharmacovigilance, Department of Clinical and Experimental Medicine, University of Pisa, Pisa, Italy

BACKGROUND and AIM: The prevalence of overweight and obesity in children and among women in reproductive age has increased substantially representing a critical issue. Because maternal obesity is known to exert detrimental consequences on offspring, this study investigated the impact of maternal obesity and high-fat diet exposure on offspring gastrointestinal (GI) system.

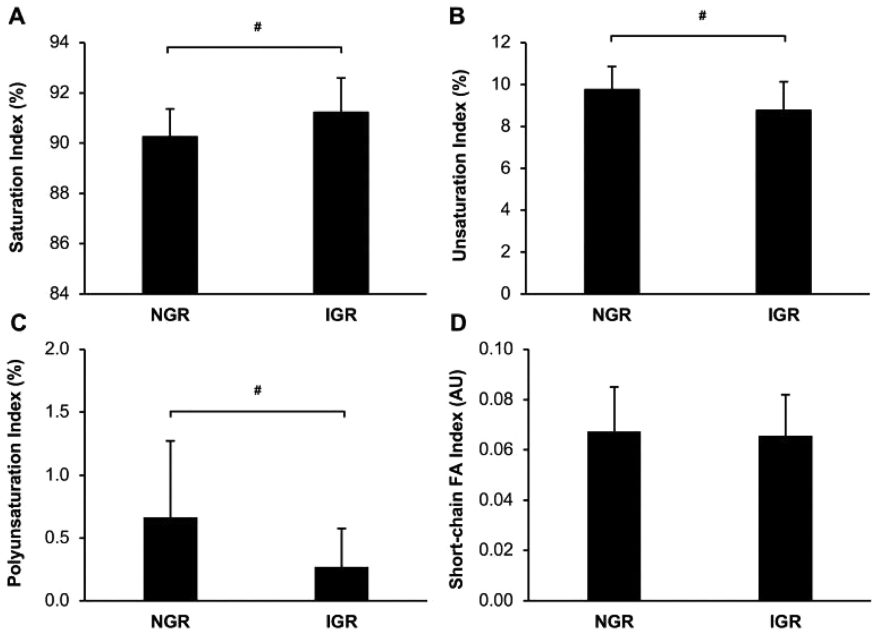

FIGURE 1

METHODS: C57BL/6J female mice were fed with standard (SD) or high-fat (HFD) diet for 8 weeks, mated with SD males and exposed to SD or HFD throughout pregnancy and lactation. At weaning, pups were randomly assigned to SD or HFD for 8 weeks. Body weight and food intake were monitored once a week. Blood glucose, triglycerides, cholesterol, insulin and leptin levels and faecal pellet expulsion frequency were evaluated the day before sacrifice. Interleukins levels were assayed by ELISA. GI transit was assessed after intragastric administration of non-absorbable fluorescein isothiocyanate labelled dextran (FITC-dextran 70 kDa). Intestinal permeability was determined by plasma level assessment of low molecular weight FITC-dextran and zonulin-1 by ELISA. Immunofluorescence staining for the neuronal (HuC/D), neuronal nitric oxide synthase (nNOS) and glial (S100β) markers was evaluated in longitudinal muscle myenteric plexus preparations of ileum. The microbiota composition of colonic faecal pellets was analysed by 16rRNA amplicon sequencing.

RESULTS: HFD pups developed increased body weight and metabolic indexes, decreased food intake and changes in microbiota (increased abundance of Firmicutes and Proteobacteria and reduction of Bacteroidetes) irrespectively of the maternal diet. They also displayed an increment in serum IL-1β, IL-23, IL-6 and a decrease in IL-10 levels, more pronounced in female and male pups born to obese dams and fed with SD. HFD female pups showed a delay in GI transit, more severe in those born to HFD mothers, confirmed by a reduction of stool expulsion frequency. Male pups born from SD dams and fed with HFD displayed both a delay in GI transit and a decrease in faecal pellet expulsion, more severe in those born from HFD dams and fed with SD. The increase in intestinal permeability, observed in HFD offspring, was further enhanced in pups born from obese mothers. Staining for HuC/D and nNOS was decreased in both sexes fed with HFD and among the offspring born from HFD mothers; these changes were more pronounced in SD pups. The immunoreactivity to S100β was increased in HFD pups but was reduced in pups born from obese dams.

CONCLUSION: Body weight gain, alterations of metabolic parameters and changes in microbiota occur mainly in offspring with direct exposure to HFD. Maternal HFD consumption confers to the offspring a higher susceptibility to develop bowel dysfunctions and among the offspring from obese dams; the most detrimental impact was observed in pups fed with SD.

CONFLICT OF INTEREST: None disclosed.

AD02-07 | Association between rs174537 *FADS1* genotype and immune cell profiles in human abdominal and femoral subcutaneous adipose tissue

C. Wang[1]; J. Murphy[2,5,6]; K.Z. Delaney[2,5,6]; J.A. Morais[3]; P. Garneau[4]; H. Atlas[4]; R. Pescarus[4]; R. Denis[4]; D.E. Lowry[1]; D.M. Mutch[1]; S. Santosa[2,5,6]

[1]Department of Human Health and Nutritional Sciences, University of Guelph, Guelph, Canada; [2]Department of Health, Kinesiology and Applied Physiology, Concordia University, Montreal, Canada; [3]Division of Geriatric Medicine, McGill University Health Centre, Montreal, Canada; [4]Departement du Chirurgie, Hôpital du Sacré-Coeur de Montréal, Montréal, Canada; [5]Metabolism, Obesity, and Nutrition Lab, PERFORM Centre, Concordia University, Montreal, Canada; [6]Research Centre, Centre intégré universitarie de santé et de services sociaux du Nord-de-l'Île-de-Montréal, Hôpital du Sacré-Cœur de Monréal (CIUSS-NIM, HSCM), Montreal, Canada

INTRODUCTION: Chronic low-grade inflammation in adipose tissue is characteristic of obesity and its related complications. Polyunsaturated fatty acids (PUFAs) regulate adipose tissue inflammatory status. Evidence suggests that single nucleotide polymorphisms (SNPs) in the *FADS1/2* gene cluster alter PUFA content in adipose tissue and may therefore influence adipose tissue inflammation. However, data on the relationship between *FADS1/2* SNPs and adipose tissue inflammation to date are limited and conflicting. Immune cell infiltration is a key determinant of inflammatory status in adipose tissue; however, no data have examined the relationship between *FADS1/2* genotypes and immune cell profiles. To advance this area, we investigated whether the common rs174537 SNP in the *FADS1* gene was associated with immune cell profiles in abdominal and femoral subcutaneous adipose tissue (SAT) in individuals with obesity.

METHODS: Flow cytometry was used to identify and quantity macrophages and T cells, as well as their subsets, in abdominal and femoral SAT from 35 healthy men and women with obesity stratified by their rs174537 genotype. Immune cell differences between genotypes were assessed by the Mann–Whitney *U* test and linear regression accounting for participant age, sex and body mass index (BMI).

RESULTS: No differences were observed between the major (GG) and minor (GT + TT) allele carriers regarding the percentage of identified immune cells, the ratios of M1/M2-like macrophages and the ratios of active/naïve T cells in either SAT depot.

CONCLUSIONS: Our study provides new evidence that genetic variation in *FADS1* gene does not appear to associate with inflammatory status in various obese SAT depots. It also provides a foundation for future research examining the interaction between PUFA, genetic variants and adipose tissue inflammation.

AD02-08 | Supplementation of a high-fat diet with acesulfame-k results in sex-specific effects on insulin concentrations and adipose tissue morphology in C57BL6 mice

P.E. Bridge Comer[1]; M.H. Vickers[1]; J.F. Plows[2]; C.M. Reynolds[3]

[1]Liggins Institute, University of Auckland, Auckland, New Zealand; [2]Children's Hospital Los Angeles, Saban Research Institute, Los Angeles,