

EuroQoL-5D (EQ-5D) index and the SF-36 Physical (PCS) and Mental (MCS) Component Summary scores were analysed.

Results: At Week 160, individuals on liraglutide 3.0mg had greater weight loss from baseline ($-7.1 \pm 8.4\%$) compared with placebo ($-2.7 \pm 7.2\%$); estimated treatment difference (ETD) -4.3% [95%CI -4.9 ; -3.7], $p < 0.0001$. SF-6D score at baseline [mean (SD)] was 0.76 (0.11) and 0.75 (0.11), and at Week 160 change from baseline was 0.02 (0.12) and 0.01 (0.12) for liraglutide 3.0mg and placebo, respectively. At week 160 ETD was 0.014 [95%CI 0.002; 0.025], $p = 0.0182$. The EQ-5D score supported these findings, with a higher score for liraglutide 3.0mg versus placebo; ED 0.007 [95%CI: 0.002; 0.013], $p = 0.0116$. Change in SF-36 PCS score was significantly higher (better) at Week 160 compared with placebo: ETD 0.87 [95%CI 0.17; 1.58], $p = 0.0156$.

Conclusion: Liraglutide 3.0mg is associated with improved health utility compared with placebo for weight management in people with prediabetes over 3 years.

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Fathers' interest in participating in a healthy eating program – Preference for online and family-focused programs



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Background: In child obesity research and nutrition interventions that aim to reduce child obesity risk the specific inputs of fathers are under-represented. Yet studies that have included fathers suggest that they play a unique role in the feeding environment and child health outcomes. This study aimed to assess fathers' interest in participating in healthy eating programs and specifically to identify their preferred intervention focus and mode of delivery.

Methods: Recruitment of fathers was via a university email list and two community-based family

research cohorts. Fathers ($N = 436$) aged 37 ± 6 years (34% university educated; 89% living with child) of 2–5 year old children (mean age 3.5 ± 0.9 years, 53% boys) completed questions identifying their confidence and knowledge of healthy eating, willingness to participate in healthy eating programs, with focus on type and mode of delivery.

Results: Most fathers ($\geq 80\%$) knew what and how much their children should eat and were confident about providing healthy food. Interest was greater in learning about healthy eating for the child (67%) than themselves (51%). Fathers preferred a focus on the family (58%), compared to individual (32%), group (24%) or fathers-only (23%) programs. Perceived usefulness varied between online (45%), written information (28%), information DVD (28%), interactive social network (11%) and mobile phone (5%) programs. University educated fathers rated the online program and written information as more useful compared to fathers with no university degree ($p < 0.05$).

Conclusion: Successful access and engagement of fathers in child feeding interventions might increase via an online and family-focused program that focuses on learning about healthy eating for the child. This is in line with other research indicating that fathers prefer to be targeted in interventions to support their family, rather than undergoing a 'self-help' program.

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Metabolic improvement from switching to saccharin or water following chronic consumption by rats of 10% sucrose solution



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High consumption of sugar-sweetened beverages (SSBs) is acknowledged as a risk factor for weight gain and metabolic disease. A common strategy for reducing this risk is to switch from consumption of SSBs to 'diet' beverages containing non-nutritive sweeteners (NNS). However, research on the effects of NNS is mixed: While a majority of studies indicate no harm, some animal and cross-sectional data suggest NNS confer *increased* risk of metabolic

disease. The present experiment modelled whether the switch from SSB to NNS beverages described above conferred positive outcomes on behavioural and metabolic measures. Thus, in Stage 1, thirty adult female rats received *ad-libitum* access to 10% sucrose solution in addition to chow and water for 4 weeks. In Stage 2, rats were switched either to 0.1% Saccharin (*Suc-Sacch*) or to Water (*Suc-Water*) or remained on 10% Sucrose (*Suc-Suc*) for a further 4 weeks. Weight gain in Stage 2 was reduced for *Suc-Sacch* and *Suc-Water* groups relative to the *Suc-Suc* group. At cull, the *Suc-Suc* group showed poorer insulin sensitivity and greater g/kg fat than *Suc-Water* and *Suc-Sacch* groups. There were no group differences in either fasting glucose or liver and plasma triglyceride content. Short-term recognition memory, assessed using the place/object task, found no group differences but poorer performance overall on the hippocampal-dependent place task, consistent with all rats' access to sucrose in Stage 1. These results show beneficial effects on weight gain, insulin resistance and adiposity after switching from Sucrose to either Saccharin or Water. Our and others' past research suggests that Stage 2 may have been too short to observe group differences on other measures.

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IL-22 therapy reverses high fat diet induced colonic epithelial cell stress and inflammation



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In Type 2 diabetes β -cell dysfunction is accompanied by adverse cellular responses to high concentration of lipids and glucose, oxidative stress, endoplasmic reticulum (ER) stress and local

inflammation. Furthermore, prolonged high fat diets (HFD) induce low-grade chronic intestinal inflammation in mice, and diets high in saturated fat are a risk factor for the development of human inflammatory bowel diseases. Previously we showed that IL-22 is a natural regulator of β -cell insulin biosynthesis and secretion, protecting the β -cell from stress, preventing hypersecretion of poor quality insulin, and suppressing innate islet inflammation [1]. To further investigate extra-pancreatic effects of IL-22 we hypothesized that HFD-induced endoplasmic reticulum (ER)/oxidative stress occur in intestinal secretory goblet cells, triggering inflammatory signalling and reducing synthesis/secretion of proteins that form the protective mucus barrier.

A prolonged HFD was accompanied by an increase in circulating triglyceride levels *in-vitro* in cultured intestinal goblet cells. Non-esterified long-chain saturated fatty acids directly increased oxidative/ER stress leading to protein misfolding. We noted an increase in the intestinal inflammatory cytokine signature, alongside compromised mucosal barrier integrity, loss in the tight junction protein, claudin-1 and increased serum endotoxin levels when mice were kept on HFD for 22 weeks. Obese mice treated with IL-22, not only had an improvement in hyperglycaemia but this was accompanied by reduced ER/oxidative stress in the intestine which overall led to an improvement in the integrity of the mucosal barrier. IL-22 treatment also reversed microbial changes associated with obesity and diabetes with an increase in *Akkermansia muciniphila* and decrease in *E. coli* and *Prevotella* spp. Consistent with epidemiological studies, our experiments suggest that HFDs are likely to impair intestinal barrier function, particularly in early life, which partially involves direct effects of free fatty acids on intestinal secretory cells, and this can be reversed by IL-22 therapy.

Reference

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