measures, including fasting plasma glucose, 2-h plasma glucose post 75 g oral glucose load, glycosylated haemoglobin (HbA1c), and homeostatic model assessment of insulin resistance (HOMA-IR), classifying 61 of 62 subjects correctly.

Conclusions: We provide a simple novel tool based on circulating lipids and metabolites to guide physicians to the most effective insulin-sensitising treatment in individuals with obesity. Future studies are necessary to validate these findings and to compare the efficacy of the biomarker-guided therapy with the traditional treatment.

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Vitamin D supplementation improves adipokine concentrations in overweight or obese adults

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Background: Adipokine dysregulation is a feature of obesity and related cardiometabolic disorders including type 2 diabetes. Vitamin D regulates adipokine production *in vitro*; however, clinical trials have been inconclusive.

Objective: To examine the effects of vitamin D supplementation on serum adipokine concentrations in overweight or obese and vitamin D-deficient adults, using data from a randomized controlled trial (RCT).

Design: Sixty-five individuals with a body mass index (BMI) $\geq$25 kg/m^2 and 25-hydroxyvitamin D (25(OH)D) $\leq$50 nmol/L were randomized to an oral bolus dose of 100,000 IU followed by 4,000 IU daily of cholecalciferol or matching placebo for 16 weeks. Before and after the intervention, we measured BMI, waist-to-hip ratio (WHR), % body fat (dual energy X-ray absorptiometry), serum concentrations of 25(OH)D (chemiluminescent immunoassay) and total adiponectin, leptin, resistin, and adipsin (multiplex assay; flow cytometry). Sun exposure habits, physical activity, and diet were assessed using questionnaires.

Results: Fifty-four participants completed the study (35 M/19F; age = 31.9 $\pm$ 8.5 years; BMI = 30.9 $\pm$ 4.4 kg/m^2 [mean $\pm$ SD]). After 16 weeks, vitamin D supplementation increased serum 25(OH)D compared with placebo (57.0 $\pm$ 21.3 versus 1.9 $\pm$ 15.1 nmol/L, $p < 0.001$). There were no differences between vitamin D and placebo groups for changes in adiponectin, leptin, resistin, or adipsin in unadjusted analyses (all $p > 0.05$). After adjustment for baseline values, season, sun exposure, and dietary vitamin D intake, there was a greater increase in adiponectin (β [95%CI] = 13.7 [2.0, 25.5], $p = 0.02$) and leptin (β [95%CI] = 22.3 [3.8, 40.9], $p = 0.02$) concentrations in the vitamin D group compared with placebo. Results remained significant after additional adjustment for age, sex, and % body fat (both $p < 0.02$).

Conclusions: Vitamin D may increase adiponectin and leptin concentrations in vitamin D-deficient and overweight or obese adults. Further studies are needed to clarify the molecular interactions between vitamin D and adipokines, and to establish the clinical implications of these interactions in the context of obesity.

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Modification of cognitive biases in overweight and obesity

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Introduction: Obesity is partly driven by unhealthy food choices underpinned by cognitive biases, including approach bias (an automatic tendency to move toward rather than away from appetitive food cues) and delay discounting (a preference for smaller, immediate over larger, delayed rewards). Cognitive training strategies aimed at modifying these biases, namely, approach–avoidance training (AAT) and episodic future thinking (EFT) have been shown to improve food choice. However, previous studies tested these training strategies in single laboratory-based sessions among healthy participants. We conducted a pilot randomised trial to examine the effect of these two trainings, delivered daily for one week via smartphone apps, on approach bias for healthy and unhealthy food, delay discounting, and food choice.

Method: Sixty participants with overweight or obesity (39F; age = 26.93 $\pm$ 6.73 years; BMI = 30.34 $\pm$ 3.75 kg/m^2) were randomly allocated to AAT, EFT, or a waitlist control. Outcomes were measured at pre-training, post-training, and at 6-week follow-up. Additional measurements included weight (kgs) and training engagement.

Results: Training session completion rates were high for AAT (85.71%) and EFT (86.43%), t (38) = $-$0.11, $p = 0.92$. Approach bias for unhealthy food was lower in AAT than EFT at post-training ($M_{Diff} = -64.56$, $p = 0.02$, 95% CI [$-$118.83, $-$10.28]). Healthy food choice (%) was higher for AAT than controls at post-training ($M_{Diff} = 23.45$, $p = 0.01$, 95% CI [7.26, 39.64], $d = 1.26$), and 6-week follow-up ($M_{Diff} = 23.92$, $p = 0.01$, 95% CI [5.37, 42.48], $d = 1.24$), and weight reduced from pre-training to 6-week follow-up in AAT ($M_{Diff} = -0.74$, $p = 0.03$, 95% CI [$-$1.40, $-$0.090], $d = 0.47$). However, EFT did not affect delay discounting, food choice, or weight (all p's > 0.1).

Conclusion: AAT is a useful training strategy for improving food choice in obesity and smartphones are a feasible, engaging way to deliver training.

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Intermittent compared to continuous energy restriction on weight loss and weight maintenance: effects after 12 months

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Background and aim: There are few long-term trials comparing intermittent energy restriction (IER) to continuous energy restriction (CER) for weight loss. We compared the effects of CER to two forms of IER; a week-on-week-off energy restriction and a 5:2 program on weight loss, body composition, blood lipids and glucose.