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One year of liraglutide treatment offers sustained and more effective glycaemic control and weight reduction compared with sitagliptin, both in combination with metformin, in patients with type 2 diabetes: a randomised, parallel-group, open-label trial

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SUMMARY

Aim: The aim of this study was to compare the efficacy and safety of once-daily human glucagon-like peptide-1 analogue liraglutide with dipeptidyl peptidase-4 inhibitor sitagliptin, each added to metformin, over 52 weeks in individuals with type 2 diabetes. **Methods:** In an open-label, parallel-group trial, metformin-treated participants were randomised to liraglutide 1.2 mg/day ($n = 225$), liraglutide 1.8 mg/day ($n = 221$) or sitagliptin 100 mg/day ($n = 219$) for 26 weeks (main phase). Participants continued the same treatment in a 26-week extension. **Results:** Liraglutide (1.2 or 1.8 mg) was superior to sitagliptin for reducing HbA_{1c} from baseline (8.4–8.5%) to 52 weeks: −1.29% and −1.51% vs. −0.88% respectively. Estimated mean treatment differences between liraglutide and sitagliptin were as follows: −0.40% (95% confidence interval −0.59 to −0.22) for 1.2 mg and −0.63% (−0.81 to −0.44) for 1.8 mg (both $p < 0.0001$). Weight loss was greater with liraglutide 1.2 mg (−2.78 kg) and 1.8 mg (−3.68 kg) than sitagliptin (−1.16 kg) (both $p < 0.0001$). Diabetes Treatment Satisfaction Questionnaire scores increased significantly more with liraglutide 1.8 mg than with sitagliptin ($p = 0.03$). Proportions of participants reporting adverse events were generally comparable; minor hypoglycaemia was 8.1%, 8.3% and 6.4% for liraglutide 1.2 mg, 1.8 mg and sitagliptin respectively. Gastrointestinal side effects, mainly nausea, initially occurred more frequently with liraglutide, but declined after several weeks. **Conclusion:** Liraglutide provides greater sustained glycaemic control and body weight reduction over 52 weeks. Treatment satisfaction was significantly greater with 1.8 mg liraglutide, similar to 26-week results. The safety profiles of liraglutide and sitagliptin are consistent with previous reports.

What's known

Results of independent trials and several 26-week head-to-head trials suggest that GLP-1 receptor agonists produce greater glycaemic and weight reductions compared with DPP-4 inhibitors. Our 26-week trial showed that the human once-daily GLP-1 analogue liraglutide effected greater glycaemic control and weight loss than the DPP-4 inhibitor sitagliptin.

What's new

Longer-term sustainability of the 26-week efficacy and safety results with liraglutide and sitagliptin, as well as the maintenance of the greater comparative efficacy with liraglutide, was not known. This report shows that 26-week improvements were sustained after 52 weeks of treatment, with liraglutide producing significantly greater glycaemic and weight reductions than sitagliptin.

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RP has attended advisory panels for GlaxoSmithKline; has received research

Introduction

Metformin is now standard first-line treatment (in addition to lifestyle modifications) for type 2 diabetes (T2D) (1). The progressive nature of T2D, including declining beta-cell function, usually necessitates addition of other antihyperglycaemic agents to metformin, as blood glucose levels rise. However, current guidelines vary with respect to second-line therapy (1,2). A meta-analysis of currently available non-insulin antihyperglycaemic agents added to metformin revealed that, while reductions in glycosylated haemoglobin (HbA_{1c}) were similar across several drug classes (including sulphonylureas, thiazolinediones and alpha-glucosidase inhibitors; reduction range: 0.64–0.97%), treatment side effects (such as weight gain and/or hypoglycaemia) varied considerably (3). Therefore, head-to-head studies of glucose-lowering agents are needed to compare overall clinical efficacy and safety when added to metformin.

Treatment intensification with incretin-based therapies is appealing given that they provide good glycaemic control with a low risk of hypoglycaemia, because of the glucose-dependent stimulation of

grants/participated in clinical trials for Merck & Co., Novartis Pharmaceuticals, Novo Nordisk Inc., Roche, Takeda Global Research Development Center Inc., Eli Lilly & Co., Merck & Co., Novartis Pharmaceuticals Corporation, Novo Nordisk A/S, Pfizer Inc., sanofi-aventis; has acted as consultant for GlaxoSmithKline; has received honoraria from Novartis Pharmaceuticals Corp, Novo Nordisk Inc., Roche and Takeda Global Research Development Center and has attended speakers' bureaux for Merck & Co. MN has received research grants from Bayer Vital Pharma, Eli Lilly & Co., Menarini/Berlin-Chemie, Merck Sharp & Dohme, Novartis Pharma and Novo Nordisk A/S; has accepted honoraria for membership in advisory boards and consulting, and has received honoraria for speaking on incretin-based antidiabetic medications from Amylin Pharmaceuticals, AstraZeneca, Bayer Vital Pharma, Berlin-Chemie/Menarini, Biovitrum, Boehringer Ingelheim, Eli Lilly & Co., GlaxoSmithKline, Hoffman La Roche, Novartis Pharma, Novo Nordisk A/S, sanofi-aventis Pharma and Takeda. TB has attended advisory panels for Amylin Pharmaceuticals; has received research support from Animas Corporation, Becton Dickinson, CPEX Pharmaceuticals, Dexcom, Eli Lilly & Co., GlaxoSmithKline, Medtronic MiniMed, Merck, Novo Nordisk Inc., Resmed and sanofi-aventis; and has attended speakers' bureaux for Amylin Pharmaceuticals Inc., Dexcom, Eli Lilly & Co., Medtronic MiniMed, Novo Nordisk Inc., Roche Diagnostics and sanofi-aventis. EM has attended advisory panels for Merck Sharp & Dohme, Novartis, Novo Nordisk and sanofi-aventis. RC has attended advisory boards for Novo Nordisk, Bayer, Roche, CeQur, Eli Lilly and Abbott; has received support for educational activities from Lifescan, Eli Lilly, Merck, Novartis and sanofi-aventis; has or is principal or co-investigator for sponsored clinical trials research for Amylin, Abbott, Bayer, Daiichi Sankyo, Dexcom, Edwards Lifesciences, Eli Lilly, Hygeia, Intarcia, Johnson & Johnson/Lifescan, Mannkind, Medtronic, Merck, Novo Nordisk, Quotient Diagnostics, ResMed, Roche, sanofi-aventis,

insulin secretion and inhibition of glucagon release, and do not produce weight gain (3–6). Glucagon-like peptide-1 (GLP-1) receptor agonists and dipeptidyl peptidase-4 (DPP-4) inhibitors are two distinct classes of incretin-based therapies. While 26-week, head-to-head studies suggest that GLP-1 receptor agonists have greater glycaemic and weight reduction efficacy than DPP-4 inhibitors (7–9), longer-term results have not been reported.

In a 26-week, head-to-head trial of the once-daily human GLP-1 analogue liraglutide and the DPP-4 inhibitor sitagliptin, both in combination with metformin, liraglutide (1.2 or 1.8 mg/day) was significantly more effective than sitagliptin (100 mg/day) for reducing HbA_{1c} (−1.24% and −1.50% vs. −0.90% respectively), fasting plasma glucose (FPG) (−1.87 mmol/l [−33.66 mg/dl] and −2.14 mmol/l [−38.52 mg/dl] vs. −0.83 mmol/l [−14.94 mg/dl], respectively) and body weight (−2.86 and −3.38 kg vs. −0.96 kg respectively) (8). Incidence of minor hypoglycaemia was low (around 5%) and comparable across treatment groups. Nausea incidence was greater with liraglutide than with sitagliptin during therapy initiation, but generally declined after several weeks of treatment.

Trial participants could continue treatment in a 26-week extension phase designed to evaluate the sustainability of efficacy and safety effects of liraglutide and sitagliptin. This report shows that 26-week improvements were sustained after 52 weeks of treatment, with liraglutide producing greater glycaemic and weight reductions than sitagliptin.

Methods

Study design

Details of study design and participant inclusion/exclusion criteria have been reported previously (8). Briefly, in a multinational, randomised, parallel-group, open-label, active-comparator trial, participants with T2D previously treated with metformin monotherapy (≥ 1500 mg/day) for a minimum of 3 months, but with suboptimal glycaemic control (HbA_{1c} 7.5–10%), were randomised (1 : 1 : 1) to treatment with either liraglutide 1.2 or 1.8 mg/day (subcutaneous injection) or sitagliptin 100 mg/day (orally) while continuing on existing metformin therapy.

After completing the 26-week main phase, participants choosing to enrol in the extension provided written informed consent and continued for another 26 weeks in their originally assigned treatment groups. The protocol, including the extension, was institutional review board-approved, followed Good Clinical Practice guidelines and conformed to the Declaration of Helsinki. The 52-week trial was initiated on 16 June 2008 and completed on 10 December 2009.

Additional withdrawal criteria during the extension were: elevated FPG > 11.1 mmol/l (200 mg/dl) with no treatable intercurrent cause or acute pancreatitis (defined as a minimum two out of three of the following: characteristic abdominal pain, amylase and/or lipase > 3 × upper normal range or characteristic findings on computed tomography/magnetic resonance imaging).

Outcomes

Efficacy outcomes assessed at 52 weeks included change in HbA_{1c}, FPG, body weight, proportion of participants achieving HbA_{1c} < 7% or ≤ 6.5%, proportion of participants reaching the composite end-point of HbA_{1c} < 7.0% with no weight gain and no confirmed major (participant unable to treat him/herself) or minor (plasma glucose < 3.1 mmol/l [56 mg/dl]) hypoglycaemia. Other measures included fasting C-peptide, fasting pro-insulin : insulin ratio, and homeostasis model assessment analyses of beta-cell function (HOMA-B) and insulin resistance (HOMA-IR). Change in Diabetes Treatment Satisfaction Questionnaire (DTSQ) scores from baseline was not assessed in participants from Slovakia, Serbia or Slovenia (118/665 [17.7%]) because of the lack of validated questionnaires in their native languages.

Safety and tolerability assessments at 52 weeks included incidence of adverse events (AEs) and hypoglycaemia, as well as various clinical and laboratory variables. AEs of special interest included nausea, thyroid AEs and pancreatitis.

Statistical analysis

Methods for statistical analyses were similar to those reported for the first 26 weeks (8). Glycaemic efficacy, as measured by change in HbA_{1c} from baseline to week 52 of liraglutide vs. sitagliptin treatment, was assessed by a non-inferiority comparison with a margin of 0.4%, followed by a superiority comparison. Both tests used two-sided hypotheses, with a p-value of < 0.05 considered significant. Analysis of covariance, with treatment and country as fixed effects and baseline measure as a covariate, was used for continuous efficacy end-points. Logistic regression was used to analyse categorical variables, including the participant proportions achieving HbA_{1c} targets and composite end-point (HbA_{1c} < 7.0% with no weight gain and no confirmed major or minor hypoglycaemia), with treatment and country as fixed effects, and baseline HbA_{1c} (and body weight for composite) as covariates. Efficacy assessments were performed on the full analysis set: all randomised participants exposed to at least one dose of the drug.

Missing data were imputed using the last observation carried forward (LOCF) method.

The safety analysis set included all participants exposed to at least one dose of the drug they were randomised to. Serum calcitonin values were analysed using a repeated measures model, with time, gender, treatment and treatment-by-time interaction as fixed effects and participant as a random effect. Hypoglycaemia was analysed using a general linear model with treatment as a fixed effect. For each week of the extension, the proportions of participants experiencing nausea were analysed using Fisher's exact test. Only summary statistics are reported for other safety parameters. Data are reported as least square means with 95% confidence interval (CI), unless otherwise noted. The significance level is $p < 0.05$.

Results

Participant disposition and baseline characteristics

After screening, 665 participants were randomised into three treatment arms: liraglutide 1.2 mg, 1.8 mg and sitagliptin (Figure 1). As previously reported, the groups were well matched for baseline characteristics (8). Of participants completing 26 weeks, 497/554 (90%) entered into the extension, with 436/497 (88%) completing 52 weeks. A lower proportion of randomised participants withdrew from the extension compared with the main phase, and withdrawal because of AEs was also lower in the extension. Patient withdrawal because of AEs in the main phase was higher for both liraglutide groups than for sitagliptin, whereas only the liraglutide 1.8 mg group had a slightly higher AE withdrawal rate in the extension.

Efficacy outcomes

Mean HbA_{1c} decreased more substantially with either dose of liraglutide compared with sitagliptin during the first 12 weeks, and these reductions were generally maintained up to week 52 (Figure 2A). Mean reductions in HbA_{1c} from baseline to week 52 with liraglutide 1.2 mg (−1.29% [95% CI: −1.43 to −1.15]) and 1.8 mg (−1.51% [−1.65 to −1.37]) were significantly greater compared with sitagliptin (−0.88% [−1.02 to −0.74]). Estimated mean treatment differences were −0.40% (95% CI −0.59 to −0.22) for liraglutide 1.2 mg vs. sitagliptin and −0.63% (−0.81 to −0.44) for liraglutide 1.8 mg vs. sitagliptin ($p < 0.0001$ for both doses.)

As with HbA_{1c}, liraglutide was more effective for reducing FPG compared with sitagliptin (Figure 2B). FPG declined rapidly from baseline during weeks 0–4 in all treatment groups and the reductions were generally sustained up to 52 weeks. FPG reductions from baseline at week 52 were −1.71 mmol/l (95% CI −2.04 to −1.38) (−30.78 mg/dl [−36.78 to −24.78]) for 1.2 mg liraglutide and −2.04 mmol/l (−2.37 to −1.71) (−36.72 mg/dl [−42.72 to −30.72]) for 1.8 mg liraglutide vs. −0.59 mmol/l (−0.92 to −0.26) (−10.62 mg/dl [−16.62 to −4.62]) for sitagliptin. Estimated mean treatment differences between liraglutide and sitagliptin were −1.13 mmol/l (95% CI −1.57 to −0.68) (−20.34 mg/dl [−28.26 to −12.24]) for 1.2 mg and −1.45 mmol/l (−1.89 to −1.01) (−26.1 mg/dl [−34.02 to −18.18]) for 1.8 mg ($p < 0.0001$ vs. sitagliptin for both doses).

Weight loss was considerably greater with liraglutide compared with sitagliptin (Figure 2C). Most weight loss occurred during the first 26 weeks and was sustained in the extension in all treatment groups (Figure 2C). At week 52, weight loss with liraglutide 1.2 mg was −2.78 kg (95% CI −3.39 to −2.17) compared with −3.68 kg (−4.29 to −3.07) for 1.8 mg and −1.16 kg (−1.77 to −0.55) for sitagliptin. Estimated mean treatment differences were −1.62 kg (95% CI −2.43 to −0.82) for liraglutide 1.2 mg and −2.53 kg (−3.33 to −1.72) for liraglutide 1.8 mg vs. sitagliptin ($p < 0.0001$ for both doses). Weight loss with liraglutide 1.8 mg was significantly greater than that with liraglutide 1.2 mg ($p = 0.03$). The 26-week-reductions in waist circumference were generally maintained at week 52 in all groups and were significantly larger with liraglutide (both doses) than sitagliptin (Table 1).

As with the main study results (8), postprandial plasma glucose data were highly variable and difficult to interpret, and are excluded from this report. As this was a multinational study, data variability may have resulted from the varying meal content, time of meals and timing of postprandial glucose measurements across different countries.

Overall, the magnitude of HbA_{1c} reduction from baseline increased with the higher baseline HbA_{1c} categories in all groups (Figure 3A). After 52 weeks, mean reductions in HbA_{1c} were significantly greater with liraglutide 1.8 mg than with sitagliptin across all baseline HbA_{1c} categories. The reductions were significantly larger with liraglutide 1.2 mg than with sitagliptin for two baseline HbA_{1c} categories: > 8% to ≤ 8.5% and > 9%.

Proportions of participants achieving target HbA_{1c} < 7% (American Diabetes Association [ADA] target) or ≤ 6.5% (American Association of Clinical Endocrinologists [AACE] target) increased during the extension in all treatment groups (Figure 3B). Overall, liraglutide (both doses) was significantly more effective than sitagliptin in allowing patients to reach target HbA_{1c} after 52 weeks.

Takeda and Valeritas; and is an employee of the International Diabetes Center at Park Nicollet. All honoraria, speaking fees, consulting fees and research and educational support are paid directly to the non-profit International Diabetes Center of which RP is a salaried employee; he receives no personal payments for any of these activities. SF has acted as consultant and attended speakers' bureaux for Novo Nordisk A/S. AG is an advisor and speaker for Novo Nordisk, Merck, GlaxoSmithKline and Sankyo. ABT is an employee of Novo Nordisk and was directly involved in study conduct. HH is an employee of Novo Nordisk and was directly involved in study conduct. MD has attended advisory panels for Eli Lilly & Co., Merck Sharp & Dohme Ltd., Novartis Pharmaceuticals, Novo Nordisk Pharma Ltd., Schering-Plough, Roche and Bristol-Myers Squibb; has received research support from Eli Lilly & Co., Merck Sharp & Dohme Ltd., Novartis Pharmaceuticals, Novo Nordisk Pharma UK and GlaxoSmithKline; and has received honoraria for lectures from Eli Lilly & Co., Merck Sharp & Dohme Ltd., Novartis Pharmaceuticals and Novo Nordisk Pharma Ltd.

Clinical trial registration number: clinicaltrials.gov, NCT00700817

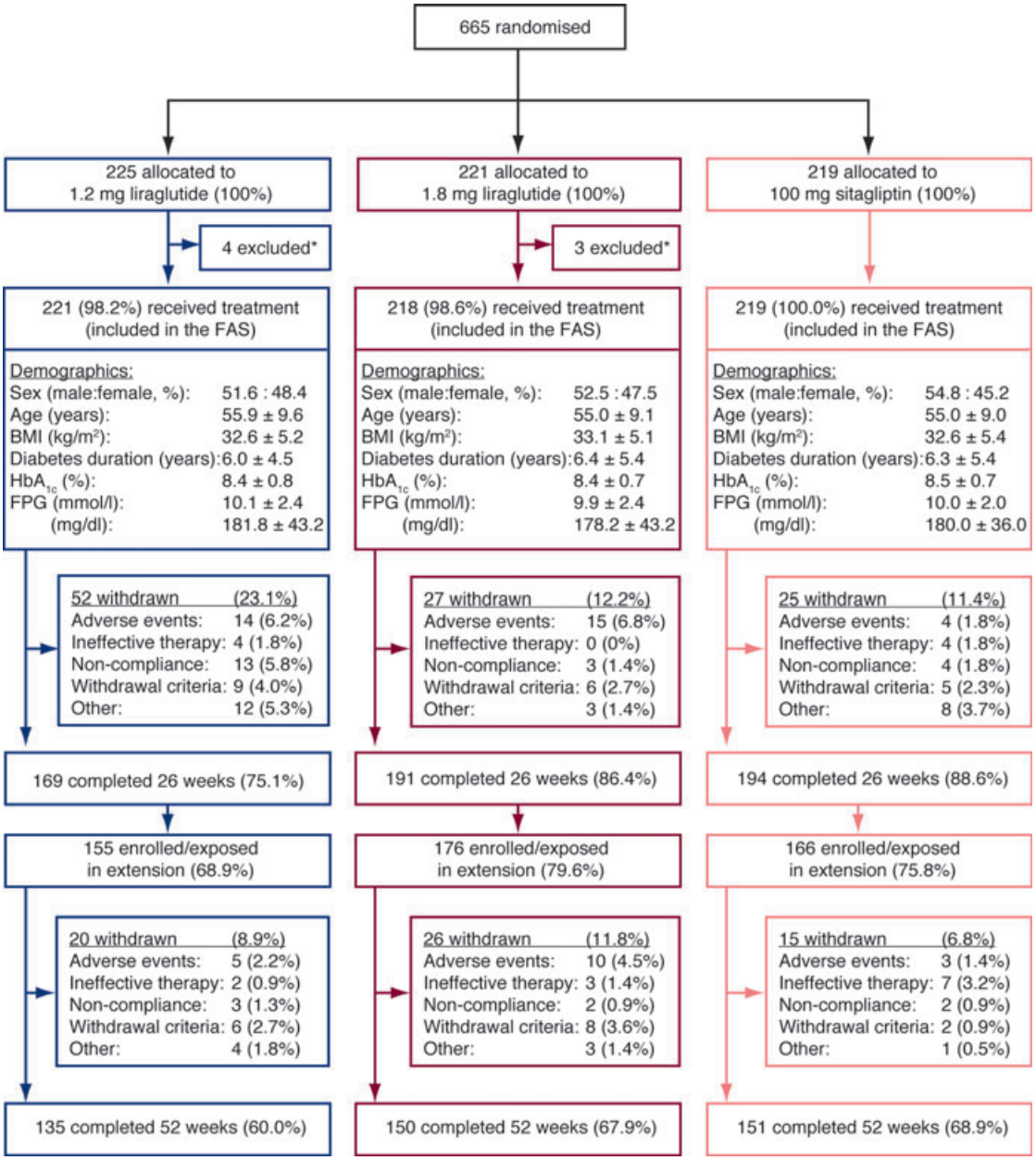

Figure 1 Trial flow chart with participant demographics at baseline. Demographic data are mean ± SD, unless otherwise noted. *Participants were withdrawn if they fulfilled withdrawal criteria, decided that they no longer wanted to participate, or did not attend any visit after randomisation. BMI, body mass index; FAS, full analysis set; FPG, fasting plasma glucose

The estimated proportion of participants reaching the composite end-point of HbA_{1c} < 7.0%, with no weight gain and no confirmed major or minor hypoglycaemia, increased during the extension in all treatment groups (Figure 3C). After 52 weeks, a significantly greater percentage of participants achieved the composite end-point with liraglutide (both groups) than with sitagliptin, with an odds ratio (OR) vs. sitagliptin of 2.80 (95% CI 1.74 to 4.48) and 4.37 (2.74 to 6.98) for 1.2 and 1.8 mg liraglutide respectively (both doses $p < 0.0001$). Liraglutide 1.8 mg was more effective than liraglutide 1.2 mg (OR: 1.56 [1.04 to 2.35], p = 0.03).

Overall, the improved status of several indicators of beta-cell function (fasting C-peptide, fasting proinsulin:insulin ratio and HOMA-B) at week 26 was maintained at week 52, with liraglutide effecting significantly greater improvements than sitagliptin (Table 1). The reduction in HOMA-IR became significantly greater with liraglutide 1.8 mg than sitagliptin during the extension. As observed at week 26, mean heart rate continued to be slightly but signifi-

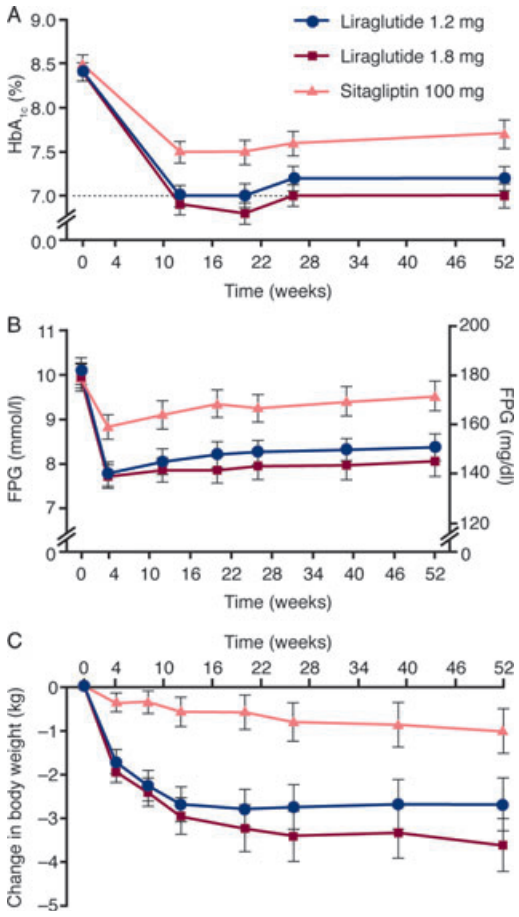

Figure 2 Effect of 1.2 mg liraglutide, 1.8 mg liraglutide or 100 mg sitagliptin on glycaemic control and body weight from baseline to 52 weeks. (A) Mean HbA_{1c} values. (B) Mean fasting plasma glucose (FPG) values. (C) Mean change in body weight. Error bars are 1.96 × SE, corresponding to the 95% CI

cantly elevated with liraglutide compared with sitagliptin at week 52 (Table 1).

The increase in DTSQ scores at week 26 was generally sustained at week 52 in all treatment groups. The improvement in overall treatment satisfaction, measured as the increase in DTSQ scores between weeks 0 and 52, was significantly higher with liraglutide 1.8 mg (baseline: 28.0) than with sitagliptin (baseline: 27.1): 4.3 (95% CI 3.3 to 5.3) vs. 3.0 (2.0 to 4.0) ($p = 0.03$). By contrast, the increase from baseline (27.8) with liraglutide 1.2 mg (3.3 [2.3 to 4.3]) was not statistically different from sitagliptin.

Safety outcomes

The majority (≥ 97%) of treatment-emergent AEs in all groups over 52 weeks were mild or moderate. The proportion of participants reporting serious AEs was low and comparable between treatment groups (4.5%, 6.0% and 5.5% for liraglutide 1.2 mg, 1.8 mg and sitagliptin respectively) with no consistent pattern with respect to system organ class (Table 2).

Three deaths occurred during the 52-week period. Two deaths during the first 26 weeks, one in a participant with pancreatic carcinoma (liraglutide 1.8 mg) and one because of cardiac arrest (sitagliptin), were reported previously and considered unlikely to be related to the trial drugs (8). One sudden cardiac death during the extension occurred in a 66-year-old man randomised to sitagliptin and was judged as unlikely to be related to the trial drug by the investigator.

Gastrointestinal disorders, as well as infections and infestations, were the most commonly reported mild-to-moderate AEs with liraglutide. The incidence of nausea, the most prevalent gastrointestinal AE with liraglutide, declined after the first 3 weeks of treatment and remained low during the extension (Figure 4). For each week of the extension, the proportions of participants experiencing nausea did not differ significantly between liraglutide (1.2 or 1.8 mg) and sitagliptin treatment groups.

One episode of major hypoglycaemia (blood glucose 3.6 mmol/l [64.8 mg/dl]) occurred during the first 26 weeks in a participant on liraglutide 1.2 mg (8). Third-party assistance was required, but no seizures or coma occurred. The participant recovered and the episode was categorised as possibly related to the trial product by the investigator. No major hypoglycaemic episodes occurred during the extension. Minor hypoglycaemia rates were low and comparable between treatment groups over 52 weeks, after excluding an outlier in the 1.8 mg liraglutide group with 21 minor events during the first 26 weeks and two events in the extension (leading to participant withdrawal from the trial). Adjusted minor hypoglycaemia rates were 0.143, 0.154 and 0.137 hypoglycaemic episodes per patient per year for liraglutide 1.2 mg, 1.8 mg and sitagliptin respectively.

One case of 'non-acute' pancreatitis was reported during the extension in a 54-year-old man, with a medical history of hepatitis and hyperlipidaemia, treated with liraglutide 1.8 mg for 227 days. Initially, the participant experienced abdominal pain, nausea, vomiting for 1 day and black stools for 3 days. The participant was instructed to stop aspirin and initiate omeprazole treatment. Upon later hospitalisation for a different condition, laboratory tests showed slightly increased levels of amylase (2.6 μkat/l, normal range: 0–1.67 μkat/l) and lipase (1.44 μkat/l, normal range: 0–1 μkat/l). The investigator decided to withdraw the participant, although the specific withdrawal criteria for acute pancreatitis were not met. The event was rated as mild and possibly related to the trial drug by the investigator.

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