# Insulin treatment and longer diabetes duration both predict poorer glycaemic response to liraglutide treatment in type 2 diabetes: the Association of British Clinical Diabetologists Nationwide Liraglutide Audit

KEN Y THONG,[1] BARBARA M MCGOWAN,[2] THEIN HTAY,[3] ANDREW PERNET,[4] CHRIS KELLY,[5] CHINNADORAI RAJESWARAN,[6] JILL HOWELL,[7] CATRIONA DUNCAN,[8] BERIT INKSTER,[9] LINDA BUCHANAN,[10] SAIFUL KASSIM,[11] RAHUL NAYER,[12] NICHOLAS D BARWELL,[10] CHRISTOPHER WALTON,[13] ROBERT EJ RYDER,[14] ABCD NATIONWIDE LIRAGLUTIDE AUDIT CONTRIBUTORS[15]

**Abstract**

Background: **Liraglutide may be less effective in patients with more advanced type 2 diabetes. This study from the Association of British Clinical Diabetologists Nationwide Liraglutide Audit analysed changes in $HbA_{1c}$ of patients after 26 weeks of treatment with liraglutide 1.2 mg, stratified according to the intensity of their background diabetes therapy, or according to their duration of diabetes.**

Methods: **Patients using liraglutide as add-on therapy were stratified for receipt to one, two or three oral antidiabetic agents (OADs) or insulin (± OAD), or for diabetes duration of 0–5 years, 6–10 years, or >10 years. Changes in $HbA_{1c}$ were compared across groups after adjusting for baseline $HbA_{1c}$.**

Results: **After exclusions to standardise comparisons, 937 patients with background diabetes treatment and 802 patients with recorded diabetes duration were analysed. Least-squares adjusted mean changes in $HbA_{1c}$ (± SEM) were –1.8% ± 0.1 for 135 patients on one OAD, –1.7% ± 0.1 for 284 patients on two OADs,–1.9% ± 0.1 for 94 patients on three OADs (n=94) and –1.0% ± 0.1 for 424 patients receiving insulin. $HbA_{1c}$ changes did not differ significantly between OAD groups, but all OAD groups had greater $HbA_{1c}$ reductions compared with the insulin group (all p<0.00001). Adjusted mean $HbA_{1c}$ changes were –2.0% ± 0.1 for patients with diabetes duration 0–5 years (n=147, p<0.05 vs. longer diabetes durations), –1.6% ± 0.1 for 6–10 years (n=256), and –1.2% ± 0.1 for >10 years (n=399).**

Conclusion: **The need for insulin and long diabetes duration, but not the number of OADs taken, predicted a smaller treatment response to liraglutide.**

*Br J Diabetes Vasc Dis* 2015;**15**:169-172

**Key words:** type 2 diabetes, liraglutide, insulin, diabetes duration, oral antidiabetic drug

[1] School of Medicine and Pharmacology, University of Western Australia, Perth, Australia
[2] Guy's and St Thomas' NHS Trust, London, UK
[3] Queen Elizabeth II, Welwyn Garden City, UK
[4] King's College Hospital, London, UK
[5] Stirling Community Hospital, UK
[6] Mid Yorkshire NHS Trust, UK
[7] Pontefract General Infirmary, Yorkshire, UK
[8] Kirkcaldy Acute Hospitals NHS Trust, UK
[9] Royal Infirmary of Edinburgh, UK
[10] Forth Valley Royal Hospital, Larbert, UK
[11] Causeway Hospital, Northern Ireland, UK
[12] City Hospitals Sunderland, UK
[13] Hull Royal Infirmary, Hull, UK
[14] Sandwell & West Birmingham NHS Trust, Birmingham, UK
[15] Appendix 1 (see online version of article at www.bjdvd.com)

**Address for correspondence:** Dr Ken Yan Thong
Department of Diabetes and Endocrinology, Rockingham General Hospital, Elanora Drive, Rockingham, WA 6168 Australia
Tel: +618 95994697
E-mail: kythong@gmail.com

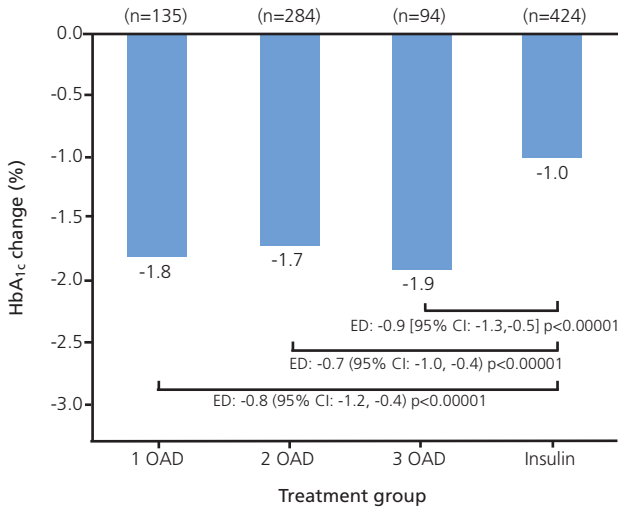
http://dx.doi.org/10.15277/bjdvd.2015.046

## Introduction

Guidelines for the management of type 2 diabetes place a strong emphasis on the need for personalised antidiabetic treatment.[1] Accordingly, it is important to identify factors which predispose to an optimum treatment response to a given antidiabetic therapy. Liraglutide is a once-daily GLP-1 receptor agonist approved for use alongside diet and exercise in combination with one or more oral antidiabetic agents (OADs) or with basal insulin for the management of type 2 diabetes.[2,3]

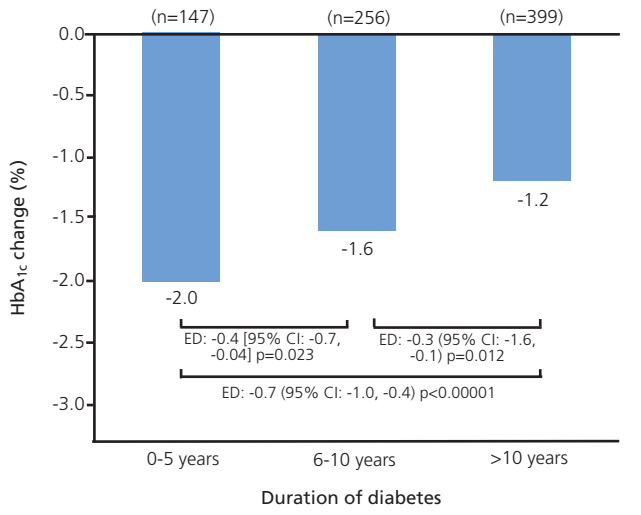
Studies currently reported in abstract form point towards a

**Figure 1.** Mean $HbA_{1c}$ changes after 26 weeks of liraglutide treatment, stratified according to intensity of background diabetes treatment




Columns show adjusted mean changes analysed by ANCOVA with baseline $HbA_{1c}$ as a covariate. CI: confidence interval; ED: estimated difference; OAD: oral antidiabetic drug(s).

**Figure 2.** Mean $HbA_{1c}$ changes after 26 weeks of liraglutide treatment, stratified according to duration of diabetes




Columns show adjusted mean changes analysed by ANCOVA with baseline $HbA_{1c}$ as a covariate. ED: estimated difference; CI: confidence interval

diminished glycaemic response to a GLP-1 receptor agonist treatment among patients with more advanced diabetes, based on assessments of the intensity of antidiabetic treatment or the duration of diabetes.[4-6] We studied the influence of these indices on the therapeutic response to liraglutide in a large cohort of patients who received liraglutide 1.2 mg in the UK.

## Patients and methods

This study was part of a nationwide audit of the use of liraglutide in the UK carried out on behalf of the Association of British Clinical Diabetologists (ABCD).[7] Participating physicians provided anonymised information on demographic data (age, gender, ethnicity, height, weight), duration of diabetes, cardiometabolic parameters (glycaemia, blood pressure, lipids, alanine aminotransferase and creatinine) and treatments prescribed, before and after treatment with liraglutide. Only effects on $HbA_{1c}$ are discussed here. Data capture was via an online audit tool or paper forms conveying identical information. By 2013, 117 diabetes centres in the UK had participated and submitted baseline data on 6,238 patients treated with liraglutide.

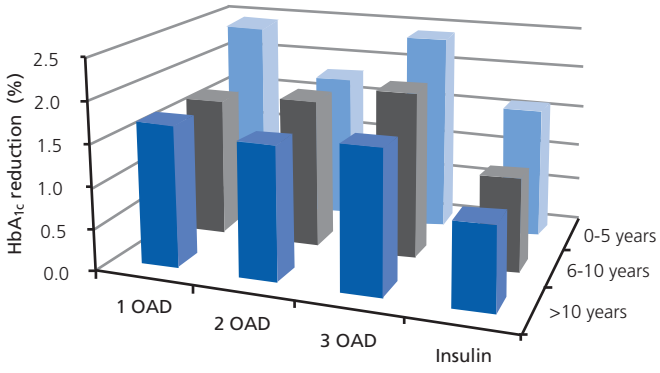
Patients were stratified for receipt of one, two or three OADs or insulin (with or without concomitant treatment with an OAD), or for diabetes duration 0–5 years, 6–10 years, or >10 years. Patients without at least one follow-up data submission (n=1,296) were excluded from the analysis. To standardise comparisons and to represent the effects of liraglutide add-on therapy, we excluded patients who switched from exenatide (n=870), received liraglutide 1.8mg (n=343), stopped an OAD at liraglutide initiation (n=947), or reduced their total daily insulin by >20% at liraglutide initiation (n=451). We also excluded patients with $HbA_{1c}$ <7.0% at baseline (n=102) and patients without relevant $HbA_{1c}$ data (n=1,173). The latest $HbA_{1c}$ data at 26 weeks of liraglutide treatment prior to any rescue treatment and with a minimum of 13 weeks after liraglutide treatment were used in the analysis. Changes in $HbA_{1c}$ according to diabetes treatment or duration were analysed using ANCOVA, after checking that the statistical requirements for valid ANCOVA had been met. Due to incomplete concurrent data on diabetes treatment and diabetes duration, separate analyses were first performed for these parameters as independent variables, without adjustment for one another, and with baseline $HbA_{1c}$ as a covariate (Figures 1 and 2). Combined ANCOVA analyses were subsequently performed among patients with complete data for both diabetes treatment and duration (Figure 3).

This was a purely observational study, and no investigations or treatments other than those required for the routine management of the patients were performed. Accordingly, formal ethical approval of the study was not required.

## Results

After exclusions, 1,056 patients were analysed (937 patients with background diabetes treatment with 1–3 OADs or insulin, and 802 patients with information on diabetes duration). All diabetes treatment or duration groups achieved mean $HbA_{1c}$ reductions at 26 weeks that were statistically significant compared with baseline (all p<0.01). Mean changes in $HbA_{1c}$ were significantly larger for patients receiving treatment based on one, two or three OADs, compared with patients receiving insulin

**Figure 3.** Mean $HbA_{1c}$ reductions according to diabetes treatment and diabetes duration in 785 patients with data on both parameters




Columns show adjusted mean changes analysed by ANCOVA with baseline $HbA_{1c}$ as a covariate. CI: confidence interval; ED: estimated difference; OAD: oral antidiabetic drug(s).

(Figure 1); no differences were seen comparing patients on one versus two versus three OADs.

Liraglutide was significantly more effective in patients with duration of diabetes of 0–5 years compared with patients with duration of diabetes of 6–10 years or >10 years (Figure 2). Insulin treatment and diabetes duration remained independent predictors of the magnitude of changes in mean $HbA_{1c}$ for patients with data on both antidiabetic treatment and diabetes duration (Figure 3).

## Discussion

Observational studies provide a different perspective to randomised trials on the therapeutic profiles of antidiabetic treatments and can add important information on their use in routine, "real world" clinical practice.[8] We found in clinical practice that the effect of liraglutide on glycaemia was reduced in type 2 diabetes with more advanced disease, as indicated by the need for insulin treatment or a longer duration of diabetes.

Our results showing a diminished treatment response to a GLP-1 agonist among insulin-treated patients are consistent with previous findings of smaller $HbA_{1c}$ reductions in patients with versus without insulin treatment after starting exenatide twice daily. This was in a similar audit to the current study, in a "real world" setting.[9] However, we did not find any difference in glycaemic efficacy when comparing the addition of liraglutide to patients on one, two or three OADs. This contrasts with earlier findings from the Liraglutide Effect and Action in Diabetes (LEAD)-2 study.[10] There, liraglutide was more effective in reducing $HbA_{1c}$ when added to OAD monotherapy as opposed to combinations of OAD,[10] a finding supported by subsequent meta-analyses.[4,5] The reason for the difference in findings is not known but may be due to methodological differences between clinical trials and an audit. The use of thiazolidinediones in the audit (18.0% of patients on two OADs and 87.2% of patients on three OADs) may have also contributed to the difference since this class of treatment has been associated with greater glycaemic durability over some other OADs.[11]

Longer diabetes duration predicted a poorer glycaemic response to liraglutide therapy in our study, and is consistent with findings of a meta-analysis of the LEAD studies[4] and a report from a small observational study.[12] In contrast, in a trial consisting only of patients on insulin, glycaemic reduction with the addition of exenatide twice daily was greater among patients with longer rather than shorter duration of disease.[13]

Our findings might suggest that the therapeutic response to liraglutide may be influenced by background β-cell function, for which background diabetes treatment and duration of diabetes may act as surrogate markers. Consistent with this view, studies have shown that higher C-peptide secretion is predictive of a larger treatment response to liraglutide,[12,14] or a greater likelihood of success when switching patients to liraglutide from insulin.[15,16]

The mean reduction in $HbA_{1c}$ with liraglutide was smaller for patients treated with insulin, relative to patients receiving other therapies, but nevertheless remained clinically significant. Recent clinical studies have shown that combinations of GLP-1 agonists and insulin are a rational treatment choice, as the inclusion of the GLP-1 agonist improves antihyperglycaemic efficacy while limiting the weight gain and hypoglycaemia associated with insulin.[17,18] Hence, the efficacy of liraglutide should both be judged across patients with different stages of diabetes, as well as judged at the level of the individual patient against other available therapeutic options at their stage of disease.

**Key messages**

- The glycaemic response to liraglutide was smaller in magnitude in patients with type 2 diabetes receiving insulin compared with those on oral therapies only
- Liraglutide was also less effective in patients with longer duration of diabetes
- Patients on insulin or with long duration of diabetes still achieved a clinically significant therapeutic response with liraglutide treatment

## Conclusion

Our study confirms and extends previous findings that patients with more advanced type 2 diabetes respond less well to liraglutide compared with patients with earlier-stage disease.

**Acknowledgements** Dr Mike Gwilt (then Submissions Editor, BJDVD) provided editorial assistance in producing the first draft of this article and played no further role in the journal's peer review procedures. We would like to thank all contributors to the nationwide liraglutide audit.

**Conflict of interest** The ABCD Nationwide Liraglutide Audit program has previously received unrestricted grants from Eli Lilly and Novo Nordisk.

The authors take full responsibility for the content of this article.

**Funding** This audit was independently initiated and performed by ABCD, and the authors remained independent in the analysis and writing of this report.

## References

1. Inzucchi SE, Bergenstal RM, Buse JB, *et al*. Management of hyperglycaemia in type 2 diabetes: a patient-centered approach. Position statement of the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). *Diabetologia* 2012;**55**:1577-96. http://dx.doi.org/10.1007/s00125-012-2534-0
2. Novo Nordisk. Liraglutide Summary of Product Characteristics (Europe). Available at http://www.medicines.org.uk/emc/medicine/21986 (accessed May 2015).
3. Victoza® (liraglutide) US Prescribing Information. Available at www.victoza.com (accessed October 2015).
4. Ratner R, Brett J, Khutoryansky N, Aroda VR. Identifying predictors of response to liraglutide in type 2 diabetes using recursive partitioning analysis. *Diabetologia* 2012 (55 Suppl 1):S332. http://dx.doi.org/10.1007/s00125-012-2688-9
5. Garber A, Matthews DR, Zinman B, *et al*. The impact of disease stage, indicated by number of previous oral antidiabetic agents, on the clinical benefits of liraglutide in the treatment of type 2 diabetes. *Diabetologia* 2011;**54** (Suppl1):S320. http://dx.doi.org/10.1007/s00125-011-2276-4
6. Penfornis A, Gourdy P, Martinez L, *et al*. Diabetes duration and background diabetes therapies in predicting liraglutide treatment response: data from the post-marketing EVIDENCE study. *Diabetologia* 2013;**56**:(Suppl1):S356. http://dx.doi.org/10.1007/s00125-013-3012-z
7. Association of British Clinical Diabetologists. ABCD Nationwide Liraglutide Audit. Available at http://www.diabetologists-abcd.org.uk/GLP1_Audits/Liraglutide_Audit.htm (accessed October 2015).
8. Ligthelm RJ, Borzì V, Gumprecht J, *et al*. Importance of observational studies in clinical practice. *Clin Ther* 2007;**29** (Themed issue):1284-92. http://dx.doi.org/10.1016/j.clinthera.2007.07.004
9. Thong KY, Jose B, Sukumar N, *et al*. Safety, efficacy and tolerability of exenatide in combination with insulin in the Association of British Clinical Diabetologists nationwide exenatide audit. *Diabetes Obes Metab* 2011;**13**:703-10. http://dx.doi.org/10.1111/j.1463-1326.2011.01393.x
10. Nauck M, Frid A, Hermansen K, *et al*. Efficacy and safety comparison of liraglutide, glimepiride, and placebo, all in combination with metformin, in type 2 diabetes: the LEAD (liraglutide effect and action in diabetes)-2 study. *Diabetes Care* 2009;**32**:84-90. http://dx.doi.org/10.2337/dc08-1355
11. Kahn SE, Haffner SM, Heise MA, *et al*. Glycemic durability of rosiglitazone, metformin, or glyburide monotherapy. *N Engl J Med* 2006;**355**:2427-2443. http://dx.doi.org/10.1056/NEJMoa066224
12. Kozawa J, Inoue K, Iwamoto R, *et al*. Liraglutide is effective in type 2 diabetic patients with sustained endogenous insulin-secreting capacity. *J Diabetes Investig* 2012;**3**:294-7. http://dx.doi.org/10.1111/j.2040-1124.2011.00168.x
13. Rosenstock TR, Shenouda SK, Bergenstal RM, *et al*. Baseline factors associated with glycaemic control and weight loss when exenatide twice daily is added to optimized insulin glargine in patients with type 2 diabetes. *Diabetes Care* 2012;**35**:955-958. http://dx.doi.org/10.2337/dc11-1434
14. Thong KY, McDonald TJ, Hattersley AT, *et al*. The association between postprandial urinary C-peptide creatinine ratio and the treatment response to liraglutide: a multi-centre observational study. *Diabet Med* 2014;**31**:403-11. http://dx.doi.org/10.1111/dme.12367
15. Usui R, Yabe D, Kuwata H, *et al*. Retrospective analysis of safety and efficacy of insulin-to-liraglutide switch in Japanese type 2 diabetes: A caution against inappropriate use in patients with reduced β-cell function. *J Diabetes Investig* 2013;**4**:585-94. http://dx.doi.org/10.1111/jdi.12111
16. Araki H, Tanaka Y, Yoshida S, *et al*. Oral glucose-stimulated serum C-peptide predicts successful switching from insulin therapy to liraglutide monotherapy in Japanese patients with type 2 diabetes and renal impairment. *J Diabetes Investig* 2014;**5**:435-41. http://dx.doi.org/10.1111/jdi.12169
17. Meier JJ, Rosenstock J, Hincelin-Méry A, *et al*. Contrasting effects of lixisenatide and liraglutide on postprandial glycemic control, gastric emptying, and safety parameters in patients with type 2 diabetes on optimized insulin glargine with or without metformin: a randomized, open-label trial. *Diabetes Care* 2015;**38**:1263-73. http://dx.doi.org/ 10.2337/dc14-1984
18. Morales J, Merker L. Minimizing hypoglycemia and weight gain with intensive glucose control: potential benefits of a new combination therapy (IDegLira). *Adv Ther* 2015;**32**:391-403. http://dx.doi.org/10.1007/s12325-015-0208-2

**Appendix 1.** ABCD nationwide prospective liraglutide audit contributors
The following are those whom we know about.

**ABCD nationwide liraglutide audit – initial setup, maintenance and nationwide analysis**: Ryder REJ, Walton C, Thong KY, Sen Gupta P, Cull ML, Mills AP. Statistician: Blann A.

**Addenbrookes Hospital**: Adler A, Bejinariu E, Park A, Parker V, Sarker A, Simmons D. **Altnagelvin Area Hospital**: Black R N, Caskey H, Cooke B, Early R, Giff K, Hamilton L, Helmy A F, King L, Lindsay J R, McCarroll F, McDaid A-M, McIlvor E, Moles K W, Morahan S, O'Kane M, Williams L. **Antrim Area Hospital**: Kennedy A. **BaNES NHS primary care trust**: Catchpole S, Wylie S. **Barnsley Hospital NHS Foundation Trust**: Uchegbu E. **Barnet General, London**: Cohen M, Katz J, Kola B, Tanday R, Seenandan J, Steuer L. **Basildon University Hospital**: Mulcahy M. **Bassetlaw Hospital**: Kela R, Woods H. **Bearwood Medical Practice**: Alderman J, Bhanderi S, Matthews J, Newhouse R, Purcell J Sen Gupta P. **Belfast City Hospital**: Henry RW, McMullan P, Nugent A. **Bensham General Hospital**: Narayanan K R. **Birmingham Community Healthcare NHS Trust**: Bhanderi S, Cunningham B, Haughton K, Matthews J, Muralidhara K, Sen Gupta P, Shahid S, Thomas A. **Bradford Royal Infirmary**: González S. **Brighton General Hospital**: Duff B. **Brighton Sussex University Hospital NHS Trust, Royal Sussex County Hospital**: Burberry A. **Bristol General Hospital**: Croxson S. **Bristol Royal Infirmary**: John H, Jones L, Pople J A, Richards G. **Bronglais hospital**: Evans C, Jones A M, Kotonya C, Phillips L, Powell P, Saunders H. **Calderdale Royal Hospital**: Mon Zin Tun E. **Cape Hill Medical Centre**: Bhanderi S, Child D, Chitnis J, Gardner G, Maan P, Matthews J, Merali A, Sen Gupta P. **Causeway Hospital, Coleraine**: Davidson E, Diong K L, Glass M, Hutchinson K, Kassim S B, McKee M, Ryan M F, Spiers K, Woodend J. **Chase Farm Hospital**: Baynes, C, Lomas J, Russell S. **Cheltenham General Hospital**: Evans A, Gray H, Lock-Pullan P, Phillips S. **City Hospital Birmingham (SWBH)**: Basu A, Bedi T, Bhanderi S, Blann A, Burbridge W, Cull M L, Cutler J, De P, Guthrie S, Irwin S, Lee B, Lloyd F, Matthews J, Mehrali T, Mills A P, Ryder R E J, Sen Gupta P, Stevenson-Mort J, Thong K, Zzizinger A. **City Hospitals, Sunderland**: Carey P, Coates J A, Lee A, Nayar R, Ogilvie P, Purvis A, Todd J, Walton K. **Conquest Hospital**: Batson D, Castro E, Combes A, Dashora E, Edwards V, Govindan R, Kumar S, Morris R. **Cumberland Infirmary Centre**: Graham S, Higgins N, Mason J, Redgate J, Routledge A, Simpson E, Vithian K. **Darlington Memorial Hospital**: Bishop D. **Derriford Hospital**: English P, Fox T, Tambal A, Wotton F. **Dewsbury District Hospital**: Bissell J. **Downe Hospital Northern Ireland**: Whitehead H. **East Lancs Hospitals NHS Trust**: Ali A, Demssie Y, Glew M, Jones G, Jostel A, Littley M, Mishra M, Ramtoola S, Wilkinson R. **East Surrey Hospital**: Chinnasamy E, Prajapati C, Sennik D. **Eastbourne District General**: O'Donnell H. **ELPCT**: McKane C, Procter W, Sarsfield J, Wilkinson R. **Forth Valley Royal Hospital**: Barwell N, Bramley A, Buchanan L, Currie J, Davidson E, Devlin K, Doig J, Kelly C, MacDonald P, Mackenzie A, Mackintosh L, Peden N, Ryan L, Simpson C, Whitty H. **Friarage Hospital**: Kamaruddin M S, Leek C, Owen K. **Frimley Park Hospital**: Beebeejaun M, Tringham J. **Furness General Hospital**: Banerjee M, Obale B, Pearce D, Tong M. **George Eliot Hospital**: Patel V. **Gloucestershire Royal Hospital**: Gan K S, Mahajan T, Saunders S, Ulahannan T. **Guy's and St Thomas' Hospital London (Guy's & St. Thomas' NHS Trust)**: McGowan B, Abbas N, Sen Gupta P, Da Costa R, Georgieva E. **Harrogate Hospital**: Brown D, El-Laboudi A, Hammond P. **Hinchingbrooke Health Care NHS Trust**: Bejinariu E, Krishnan S, Mathews A, Walland K. **Huddersfield Royal Infirmary**: Moisey R. **Hull Royal Infirmary**: Marinceu D, Sathyapalan T, Sugunendran S, Walton C, Waqas. **Hunslet Health Centre**: Muneer K. **King's College Hospital**: Amiel, SA, Hunt K F, Lee M, Nathan Y, Pernet A, Raeburn J, Sen Gupta P, Stothard B, Vitello S. **Lagan Valley Hospital**: Au S, Brennan U, Carr S, Harding J, Harper R, MacDonald P, McLaughlin D, Moore L, Mulligan C, Whitehead H. **Lancashire Teaching Hospital, Chorley Hospital**: Rajbhandari S M, Whittaker J. **Lancashire Teaching Hospital, Royal Preston Hospital**: Rajbhandari S M, Whittaker J. **Leicester General Hospital**: Gregory R, Jackson S, Kong M-F, Tarigopula G. **Leicester Royal Infirmary**: Htike ZZ. **Leigh Infirmary**: Fatima J, Pearce S. **Lister Hospital**: Barker L, O' Donnell L. **Llandridod Wells**: Powell P. **London Medical (Private Medical Centre)**: Abraham C, Abraham R, Bowden J, Genovezos S, King L, Spahiu E, Thomas S. **Mid Yorkshire Hospitals NHS Trust (Pinderfield Hospital, Wakefield, West Yorkshire)**: D'Costa R, Kadis T, Maycock J, Nagi D, Seddon L. **Minerva Centre**: Caunce K. **Monklands Hospital**: Sandeep T C, White A. **Musgrove Park Hospital (Taunton & Somerset NHS Foundation Trust)**: Adams S, Andrews R, Close C, Douek I, Dunlop A, Lambert P, Thomas J, Watson J. **New Cross Hospital Wolverhampton**: Katreddy V, Khalid Y, Krishnasamy S, Nayak A U, Singh B M. **Newham University Hospital**: Balakumar Y, Gelding S, Menon R, Rayanagoudar G. **NHS Tayside (Ninewells Hospital/Perth Royal Infirmary)**: George P, Leese G. **Northumbria Diabetes Service**: Strey C. **Orpington Hospital**: Casiglia D. **Pendyffryn Medical Group**: Morrison C L. **Pennine Acute Hospitals Trust**: Adams L, Aherne D, Ahmad M, Allen G, Anderson K, Asam M, Atherton L, Balmuri M, Benton M, Berry M J, Bhatnagar D, Bood A, Broude H, Byrom J, Cheer K, Dang C, Emsley C, Farook S, Fletcher M, Flight W, Garg R, Hafeez K, Hall D, Higham C, Holland K, Hunsdale D, Jagadhish, Jani M, Jennings R, Jostel A, Joyce P, Kalavalapalli S, Khan S, Khurana R, Kouta S, Kumar S, Lea S, Lewthwaite P, MacDonald L, Malik I, Mawdsley J, McAllister G, Meredith K, Meth-Cohn D, Mishra B, Moore J, Mustafa A, Narasimhan S, Naray S T K, Nazir K, Norris A, Nune A, Picton M, Prakash P K, Prouten J, Rathur H, Roberts K, Rothwell N, Rowles S, S Rashid S, Savage M, Shah S, Shingler W, Smith G, Smith K V, Smithurst H, S-Samavi M, Stott R, Sudagani J, Suliman M, Tarpey S, Taylor A, Taylor E, Weaver A, West A, Wild J, Wiles P. **Pilgrim Hospital**: Htwe N, Jacob K. **Pontefract General Infirmary**: Bissell J. **QE2 Hospital, Welwyn Garden City**: Ali S, Chirayath H, Darzy K, Ford M, George S, Kaplan F, Lecamwasam V, Perera S, Qureshi S A, Scott R, Thay T, Winocour P, Wyman D, Zalin B. **Queens Romford**: Khan K, Nkonge F. **Roebuck House (Surgery 1)**: Dicker C, Rowan J, White T. **Rotherham General Hospital**: Franke B, Muzulu S, Salam S. **Royal Blackburn Hospital**: Demssie Y, Glew M, Jones G, Jostel, A, Littley M, Mishra M, Prouten J, Ramtoola S, Wilkinson R. **Royal Devon and Exeter Hospital**: Aziz A, Babiker T, Brooks A, Lockett H. **Royal Gwent Hospital**: DaCruz T, Kamath C, Obuobie K. **Royal Infirmary of Edinburgh**: Inkster B, McLaren J, Zammitt N. **Royal United Hospital Bath**: Allen K, Higgs E, Naik S, Robinson A, Ward A. **Royal Victoria Hospital Belfast**: Cooke B, Hunter S, McErlean U. **Sandwell General Hospital (SWBH)**: Bhanderi S, Davies P,

Matthews J, Rock K, Sen Gupta P, Thong K Y. **Sedlescombe House Surgery St. Leonards-on-Sea**: Cooper S, Joyce L, Kaliniecki J. **Singleton Hospital, Swansea**: Udiawar M. **Smethwick Medical Centre (GP) (SWBH)**: Bhanderi S, Harrington J, Matthews J, Sen Gupta P. **Southern General Hospital**: Gallagher S, Hutchieson A, Kennon B, Kernohan A, Semple C, Struthers S. **Southmead Hospital**: Gaffar I. **St Bartholomew's and The London NHS Trust**: Coppack S, Gouveia C, Khan R, Waugh J. **St George's Hospital NHS Trust**: Ahmed FW, Bano G, Firth P, Flanagan A, O'Brien J, Patel N, Wilson Z. **St John's Hospital Livingston**: Adamson K, Teoh WL. **St Mary's Hospital, IOW**: Al-Mrayat M, Verlekar P. **St Mary's Hospital, London**: Qureshi S A. **St Stephens Gate Medical Practice (Norfolk PCT) (SSGMP)**: Haylock C. **Stepping Hill Hospital**: Kong N, Mumby C. **Stirling Community Hospital (Stirling Royal Infirmary)**:, Barwell N, Bramley A, Buchanan L, Currie J, Davidson E, Devlin K, Dewar L, Doig J, Kelly C, MacDonald P, Mackenzie A, Mackintosh L, Peden N, Ryan L, Simpson C, Whitty H. **Stobhill Hospital, Glasgow**: Acquah R, Drummond R, Gordon D, Leggett G, MacEwen A, McKenzie J, McLaren J, Smith C. **Stoke Mandeville**: Stokes V. **The Ipswich Hospital**: Astle J, Fowler D, Morris D, Parkinson C, Rayman G, Thomas M. **Torbay Hospital**: Dimitropoulos I, Dyer R, Lissett K, Paisey R, Smith J, Weekes C. **Trafford General Hospital**: George A, Hopewell L, Snell A, Stephens W P. **Tyrone County Hospital**: Bradley P, Evans H, Hameed A, Helmy A, McGirr B, Monaghan S, Patterson H. **Ulster Hospital**: Au S, Brennan U, Carr S, Donnelly R, Harding J, Harper R, MacDonald P, McIlwaine W, McLaughlin D, Moore L, Mulligan C, Trinick T, Whitehead H. **University College Hospital, London**: Lunken C, Patel D. **University Hospital of Durham**: Kashif M. **University Hospital of Hartlepool**: Anthony S, Ijaz S, Jones S, Sinclair J, Worrall E. **University Hospital of North Tees**: Dobson M, MacLeod J, Manohar S P, Mehaffy J, Presgrave M, Pye S, Robinson M, Roper N, Worrall E. **Victoria Hospital Kirkcaldy (Kirkcaldy Acute Hospitals NHS Trust)**: Burns D, Chalmers J, Duncan C. **Warrior Square Surgery**: Adams S, Dunlop A, Ottaway L. **West Suffolk Hospital**: Clarke J, Moss A. **Western General Hospital**: Inkster B, Kochhar R S, Mathur S, Mclaren, Zammitt N. **Western Isles Hospital**: Achar K N. **Westmoreland General Hospital**: Banerjee M, Obale B, Pearce D, Tong M. **Wharfedale Hospital**: Amery C. **Wiltshire NHS Primary Care Trust**: Hall B, Hillier N. **Wrexham Maelor**: Dixon A. **Wythenshawe Hospital (UHSM)**: Younis N. **Yeovil District Hospital NHS Foundation Trust**: Bickerton A, Crocker M, Pramodh S. **Ysbyty Ystrad Mynach**: Premawardhana L D.

Acknowledgment
The ABCD nationwide liraglutide audit is an independent audit supported by an unrestricted grant from Novo Nordisk Ltd