PEER REVIEW HISTORY

BMJ Open publishes all reviews undertaken for accepted manuscripts. Reviewers are asked to complete a checklist review form (see an example) and are provided with free text boxes to elaborate on their assessment. These free text comments are reproduced below. Some articles will have been accepted based in part or entirely on reviews undertaken for other BMJ Group journals. These will be reproduced where possible.

ARTICLE DETAILS

TITLE (PROVISIONAL)	The effect of a glucagon-like peptide-1 receptor agonist on glucose tolerance in women with previous gestational diabetes mellitus: Protocol for an investigator-initiated, randomised, placebo-controlled, double-blinded, parallel intervention trial
AUTHORS	Foghsgaard, Signe; Vedtofte, Louise; Mathiesen, Elisabeth; Svare, Jens; Gluud, Lise; Holst, Jens; Damm, Peter; Knop, Filip; Vilsbøll, Tina

VERSION 1 - REVIEW

REVIEWER	Katrien Benhalima Department of Endocrinology, UZ Leuven and KU Leuven, Belgium No competing Interests
REVIEW RETURNED	19-Sep-2013

- The reviewer completed the checklist but made no further comments.

REVIEWER	Adam Deane Uinversity of Adelaide None Declared
REVIEW RETURNED	21-Sep-2013

GENERAL COMMENTS	Visboll and colleagues have submitted their trial design for a phase II study of a glucagon-like peptide-1 agonist, liraglutide, on the development of type 2 diabetes in a defined at-risk population (women that had gestational diabetes). The rationale for the study is logical and persuasive, the study is ethically appropriate, randomisation process is valid, allocation concealment is excellent, and the proposed outcomes and analyses are well-founded. I have only two minor comments. 1. The authors may wish to comment on what they will do with 'dropouts'. It is conceivable that with a cohort of 100 women of this age, many still within their child-bearing years, that the occasional subject will get pregnant (and need to withdraw) even though they are not planning another pregnancy during the study period. 2. With the sentences (p.5) 'Patients with T2DM exhibit impaired incretin effect. The cause of this pathophysiological trait is unclear (21-23)'. There has been recent suggestions from Meier and Nauck [1] that the impaired incretin effect observed in patients with type 2 diabetes is just epi-phenomenon related to reduced β-cell function, supported by data from Horowitz and colleagues suggesting that secretion of incretin hormones is unaffected in diabetic patients when the variable of gastric emptying is removed [2]. The authors

	may wish to identify this when discussing the pathophysiology of the impaired incretin effect in this group. 1. Meier JJ, Nauck MA: Is the diminished incretin effect in type 2 diabetes just an epi-phenomenon of impaired beta-cell function? Diabetes 2010, 59(5):1117-1125. 2. Marathe CS, Rayner CK, Jones KL, Horowitz M: Relationships between gastric emptying, postprandial glycemia, and incretin hormones. Diabetes Care 2013, 36(5):1396-1405.