ISPOR 20th Annual International Meeting, Philadelphia, US, 16-20 May 2015| PRM 74

# Assessing the relative contribution to changes in quality adjusted life expectancy associated with HbA1c, weight and hypoglycaemia across multiple risk equations with the CORE Diabetes Model.

**Authors:** Phil McEwan,[1] Marc Evans[2], Mark Lamotte[3] and Volker Foos[4]
1: Centre for Health Economics, Swansea University. 2: Llandough Hospital, Cardiff; 3: IMS Health, Brussels, Belgium; 4: IMS Health, Basel, Switzerland;

## Introduction

The health economic benefit associated with diabetes management is often driven by a combination of lower HbA1c, weight and hypoglycaemia event rates.
The choice of cardiovascular risk equation (RE) used by a diabetes model is an important consideration when performing an economic evaluation of any new health technologies for the treatment of type 2 diabetes (T2DM). The IMS CORE Diabetes Model (CDM) is a validated and widely used simulation model (1-3) that utilizes REs derived from the UKPDS Outcomes model (UK68-RE) (4).

More recently, a number of cardiovascular (CV) risk prediction equations suitable for use in those with T2DM have been developed, in particular the UKPDS 82 REs (UK82-RE) (5) which are derived from the extended UKPDS dataset including the 10 year observational study extension, equations based on the Swedish National Diabetes Registry (SNDR-RE) (6) and the ADVANCE Risk Engine (ADV-RE) (7). The CDM has been recently updated to include these new equations.

## Objective

The objective of this study was to attribute the predicted gain in quality adjusted life years (QALYs) to HbA1c, weight and hypoglycaemia for each RE (UK68-RE; UK82-RE; SNDR-RE and ADV-RE) embedded within the CDM to assess the role of equation choice in predicting health benefit within a diabetes model.

## Methods

This study used the CDM version 9.0 and published real-world audit data for patients with type 2 diabetes switching to insulin degludec (IDeg) from either insulin glargine or detemir (Baseline). Mean (± SD) baseline profiles were age 62.8 years (± 14.7); diabetes duration 16.2 years (± 5.0); HbA1c 9.4% (±1.1); weight 102.8 kg (±23.0) and 1.0 hypoglycaemia events per week (±1.4).

Mean change in clinical variables was HbA1c -0.7% (±0.3); weight -1.3kg (±1.1) and hypoglycaemia events/week -1.0 (±1.3) (Table 1). Body mass index modeled within the CDM was 34.0 kg/m2 (IDeg) compared to 33.6 kg/m2 (Baseline). Non-severe hypoglycaemia (NSHE) per 100 patient years was 416 (IDeg) compared to 5200 (Baseline).

A baseline cohort was projected over a lifetime using the CDM with and without treatment switch effects applied in order to calculate absolute and incremental quality adjusted life expectancy (QALE) associated with either the UK-68-RE; UK-82-RE and S-NDR RE. Results were discounted at 3%.

### Table 1: Baseline characteristics and change in clinical variables

| Baseline | Value |
|---|---|
| Age (years) | 62.8 ±14.7 |
| Proportion male | 0.56 |
| Diabetes duration (years) | 16.2 ±5.0 |
| HbA1c (%) | 9.4 ±1.1 |
| Weight (kg) | 102.8 ±23.0 |
| Episodes of hypoglycaemia per week | 1.00 ±1.4 |
| **Clinical variables post switch** | |
| HbA1c (%) | 8.7 ±1.0 |
| Weight (kg) | 104.1 ±23.0 |
| Episodes of hypoglycaemia per week | 0.08 ±0.1 |

## Results

Baseline discounted predicted QALE were 4.441, 4.449, 3.327 and 4.048 for the UK-68-RE; UK-82-RE, SNDR-RE and ADV-RE respectively.

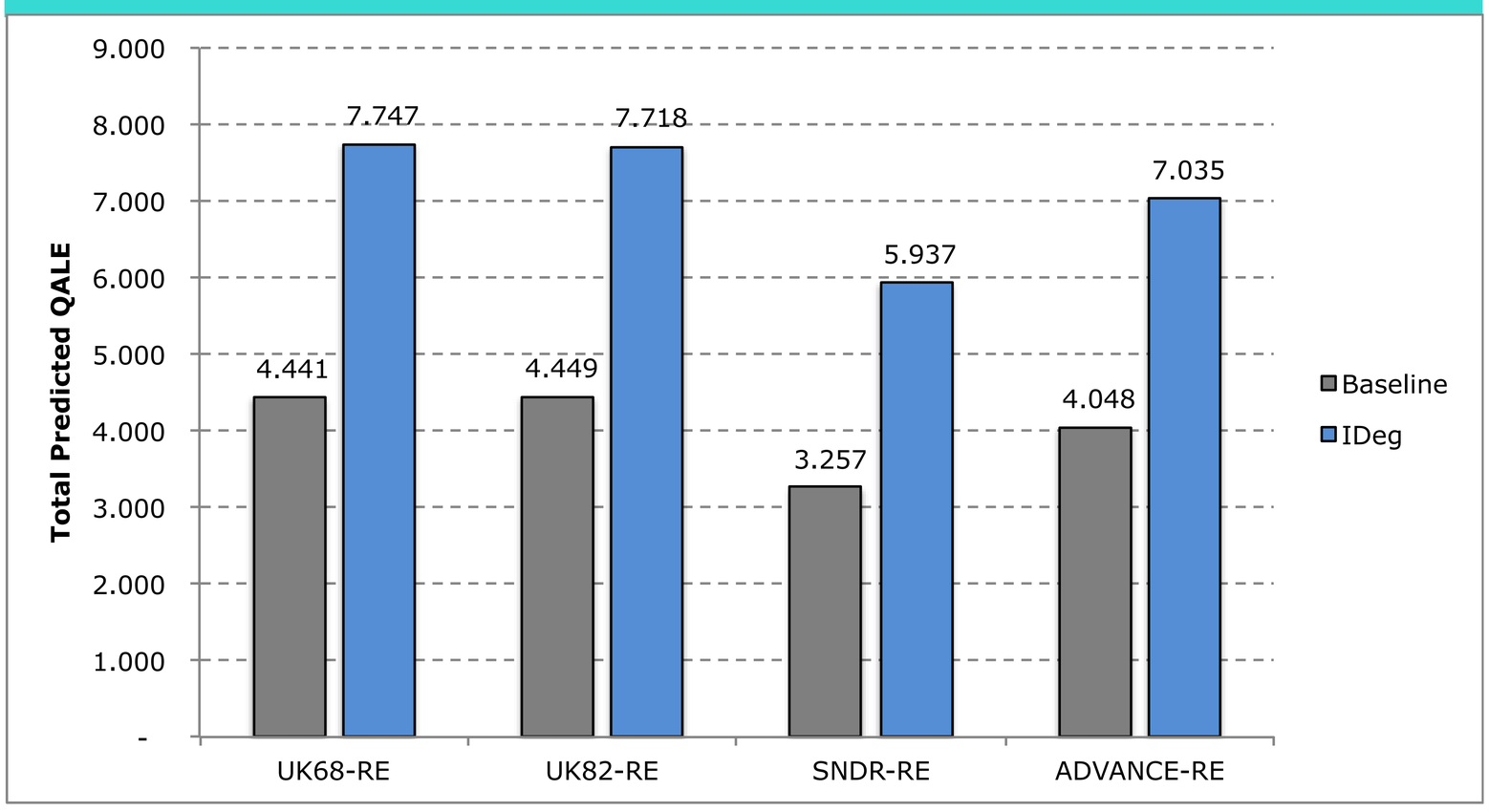
When using the UK-68-RE total predicted QALYs for IDeg were 7.747 with a QALY gain of 3.306 vs. basal insulin. 2.84% and 97.73% were attributable to improvements in HbA1c and hypoglycaemia while weight gain introduced a -0.57% reduction in the QALE benefit.

When using the UK-82-RE total predicted QALYs were 7.718 and QALY gains 3.269; 2.9% and 97.43% of this QALY gain was attributable to HbA1c and hypoglycaemia, respectively and -0.58% to weight increase.

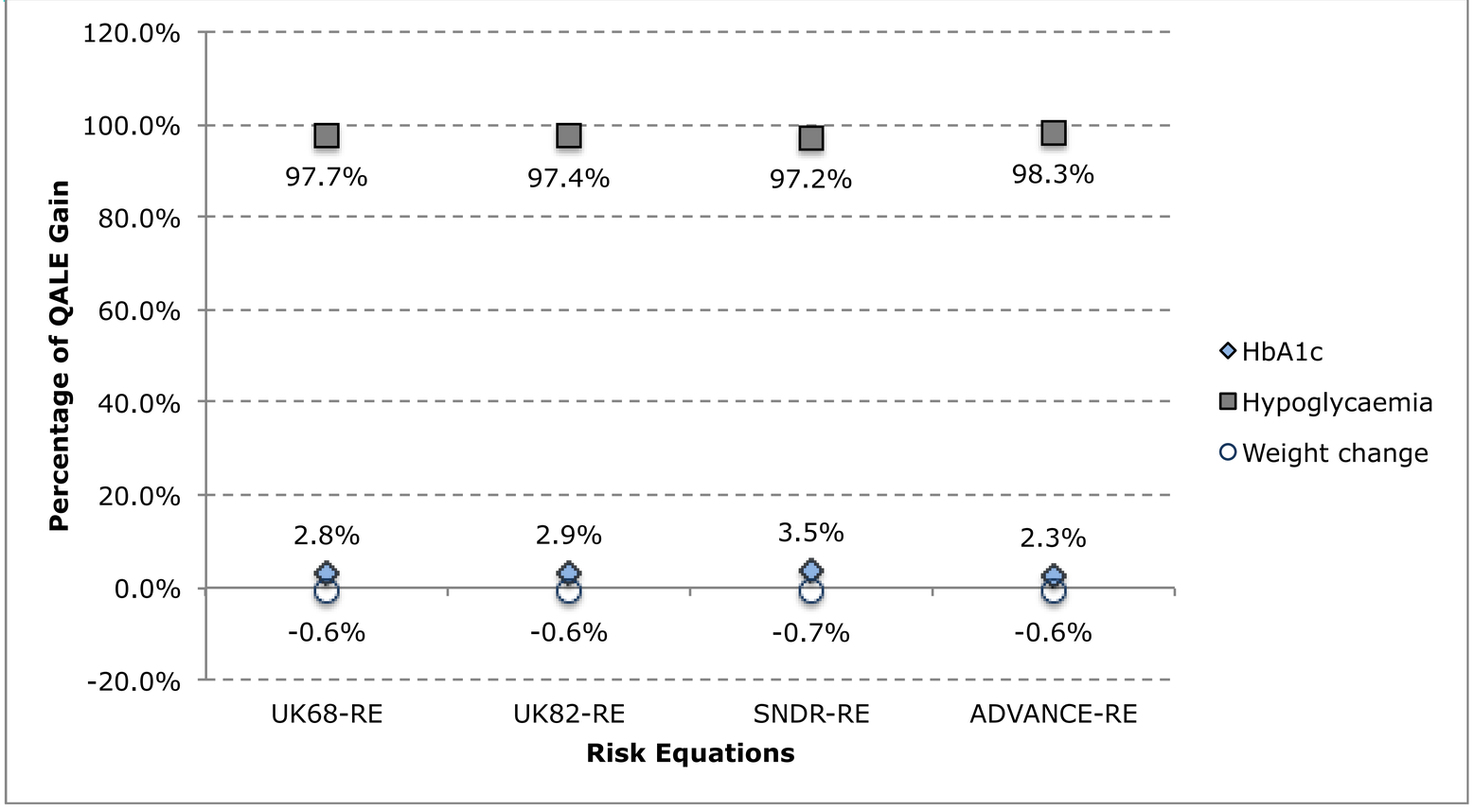
When using the S-NDR total predicted QALYs were 5.937 and QALY gains of 2.68. 3.47% and 97.24% of this gain was attributable to HbA1c and hypoglycaemia and -0.7% to weight increase.

When using the ADVANCE-RE total predicted QALYs were 7.035 and QALY gains 2.987; 2.31% and 98.32% of this gain was attributable to HbA1c and hypoglycaemia and -0.63% to weight increase.

### Figure 1: Probability of endpoint associated with baseline cohort characteristics




### Figure 2: Relative risk of complications associated with fixed risk factor changes across




## Conclusions

The treatment difference in HbA1c (-0.7%) modeled in this analysis is of a similar order of magnitude to HbA1c changes typically modeled with health technology assessments. The predicted absolute change in QALE attributable to this 0.7% reduction in HbA1c ranged from 0.069 (ADV-RE) to 0.095 (UK82-RE) with SNDR-RE and UK68-RE providing very similar incremental gains (0.093 and 0.094 respectively).

The reduction in the rate of hypoglycaemia in the audit data used in this study was substantial and the key driver of cost effectiveness. Consequently, the choice of risk equation was influential with respect to the prediction of absolute QALE but far less relevant when assessing incremental QALE.

Published mixed-treatment comparisons in second and third line treatment settings in T2DMtend to show relatively modest differences in HbA1c reduction across various therapeutic options with most of the treatment differences relating to change in weight and hyoglycaemia risk [9,10]. Consequently, the choice of risk equation may be of more relevance for predicting absolute event rates rather than incremental changes.

## References

[1] Palmer et al. Curr Med Res Opin 2004;20:S5–S26.
[2] Palmer et al. Curr Med Res Opin 2004;20:S27–40.
[3] McEwan et al. Value in Health 2014;17(6):714-24
[4] Clarke PM et al. Diabetologia 2004; 47(10):1747-59
[5] Hayes AJ et al. Diabetologia 2013; 56(9):1925-33
[6] Kiadaliri A et al. PLoS One. 2013; 8(5):e62650
[7] Kenge AP et al. Cardiovasc J Afr. 2013; 24(9): 376-381
[8] Evans et al. JME. 2014:1-10
[9] McIntosh et al. Open Medicine 2011;5(1):e35
[10] McIntosh et al. Open Medicine 2012;6(2)e62

**FOR FURTHER INFORMATION:** Please contact – Volker Foos (vfoos@ch.imshealth.com) or Phil McEwan (Phil.McEwan@heor.co.uk)
IMS HEALTH | 210 PENTONVILLE ROAD, LONDON N1 9JY, UNITED KINGDOM