To the Editor,

A recent work proposed a new index (SlisOGTT index), derived from glucose and insulin values obtained during an oral glucose tolerance test (OGTT) in a population of non-diabetic overweight and obese postmenopausal women [1]. This easily calculated index provided more accurate information than did a simple fasting-based formula, was well correlated with the clamp \((r = 0.68, \ P < 0.0001)\) and was not affected by the way in which clamp results are expressed [1]. As medical problems related to insulin resistance increase, the prompt diagnosis of such a state has become the main objective of much research work. For this reason, we tested the SlisOGTT index in a different population of 39 (age = 30–57 years) non-obese (BMI = 17–25 kg/m\(^2\)), non-diabetic and non-impaired glucose-tolerant (fasting glycaemia <5.6 mmol/L, 2-h post-glucose-load glycaemia <7.8 mmol/L), French male \((n = 12)\) and female \((n = 27)\) subjects from the EGIR-RISC study cohort [2] at the Human Nutrition Research Centre of Rhône-Alpes, France.

The patients all submitted to an OGTT with glucose and insulin measurements, and to a hyperinsulinaemic-euglycaemic (HIEG) clamp with an insulin infusion rate of 40 mU/m\(^2\) min for 120 min, which allowed estimation of insulin resistance. Correlations were calculated using Spearman’s rank test. The difference between correlations was assessed by the using the following formula: 
\[ t = (r_{xy} - r_{xz})\sqrt{(n-3)(1 + r_{yz})/2[(1 - r_{xy})^2 - (r_{xz})^2 - (r_{yz})^2 + 2(r_{xy} \times r_{xz} \times r_{yz})]} \] as reported in Dawson and Trapp [3].

We found a significant correlation \((r = 0.60, \ P < 0.001)\) between the SlisOGTT and HIEG clamp results, expressed as \(M\) (mg/min/kg fat-free mass) divided by \(I\) (steady-state insulin concentration) (Fig. 1A). Although, this correlation was not statistically different to those of Matsuda \((r = 0.58, \ P < 0.001)\), Belfiore \((r = 0.49, \ P < 0.01)\), Stumvoll \((r = 0.37, \ P < 0.05)\) and HOMA index \((r = 0.39, \ P = 0.01)\), it was higher \((P < 0.05)\) than that observed with Cederholm index \((r = 0.30, \ P = 0.06)\). In a Bland–Altman plot, we observed good agreement between the
HIEG clamp and SlisOGTT, with less than 6% disagreement (Fig. 1B).

As has been shown before in an obese and overweight, postmenopausal, non-diabetic population [1], the SlisOGTT-derived index is well correlated with the current gold standard measure of insulin sensitivity in a healthy population, which may also render it suitable for general population screening. However, studies in populations that are glucose-intolerant or diabetic are needed to further validate its utility as an insulin-resistance diagnostic tool.

Conflict of interest

None of the authors has a conflict of interest to declare.

Acknowledgements

The RISC Study is partly supported by EU grant QLG1-CT-2001-01252. Additional support was provided by AstraZeneca (Sweden). B. Antuna-Puente is receiving a scholarship from CONACYT, and R. Rabasa-Lhoret and M.-E. Lavoie are supported by a fellowship from the Fonds de recherche en santé du Québec (FRSQ). R. Rabasa-Lhoret has also received funding as the holder of the J.-A. DeSève Chair for clinical research.

References


To the editor,

We recently performed the first genome-wide fluorescence-based microsatellite screen in diabetic nephropathy (DN). Persons of Irish descent with type 1 diabetes and DN (cases; n = 200) were compared to individuals with type 1 diabetes but without DN (controls; n = 200) [1]. A DNA pooling strategy, based on the genetic analysis of multiple sclerosis in Europeans (GAMES) methodology [2], was employed. The top ranked markers (n = 50) were assessed by individual genotyping using the same DNA samples comprising the pools. Two markers on chromosome 10 (D10S558 and D10S1435) were significantly associated with DN even after correction for multiple testing; in addition, a number of markers with suggestive evidence of association warranted further investigation. In the present report, we have performed replication studies for the D10S558 and D10S1435 markers, together with the next 10 markers most significantly associated with DN from our initial study [1], in a total of 570 cases and 611 controls derived from the British Isles. The clinical definition of cases and controls was as previously reported [3]. The majority of these cases and controls were derived from the UK Genetics of Kidneys in Diabetics (GoKinD) collection [3]. Ethical approval was obtained from the appropriate Research Ethics Committees and written informed consent obtained from participants prior to conducting the study.

Fluorescence-based microsatellite genotyping was performed for all markers in cases and controls [1] using Qiagen Multiplex PCR kits (Qiagen, Crawley, UK). Amplification products were resolved by capillary electrophoresis and alleles scored, using an ABI 3730 Genetic Analyser (Applied Biosystems, Warrington, UK). Clinical characteristics of cases and