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Normal Glucose Tolerance with a High 1-Hour Postload Plasma Glucose Level Exhibits Decreased β-Cell Function Similar to Impaired Glucose Tolerance (*Diabetes Metab J* 2015;39:147-53)

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We appreciate Dr. Hee Kyung Kim's comments on our study entitled "Normal glucose tolerance with a high 1-hour postload plasma glucose level exhibits decreased β-cell function similar to impaired glucose tolerance," which was published in *Diabetes and Metabolism Journal* [1]. Our responses to Dr. Kim's comments are below.

First, in our study, insulin resistance or insulin sensitivity presented by the homeostasis model assessment for insulin resistance (HOMA-IR) and Matsuda index was not different between the subjects who had 1-hour glucose levels of ≥155 mg/dL with normal glucose tolerance (NGT 1 hour-high) and the subjects with NGT 1 hour-low (<155 mg/dL). In addition, simple indices representing β-cell function such as HOMA-β cell function (HOMA-β) and insulinogenic index were also comparable between the two groups. However, β-cell function should be viewed in the context of insulin sensitivity to reflect the β-cell capacity of compensation for insulin resistance [2]. As such, indices of β-cell function adjusted by insulin sensitivity (e.g., oral disposition index and insulin secretion-sensitivity index-2) were different between the NGT 1 hour-high and NGT 1 hour-low subjects. Therefore, the data presented in our study indicated that insulin resistance (or sensitivity) and simple β-cell function indices were not different, but indices for β-cell function adjusted by insulin sensitivity were different, which consistently substantiated our conclusion.

Second, we did not compare NGT 1 hour-high with impaired fasting glucose (IFG). Because there are different metabolic abnormalities between IFG and IGT [3], it would be worthwhile to examine the differences in insulin sensitivity and β-cell function between NGT 1 hour-high and IFG.

Lastly, Dr. Kim suggested an important implication of our study: NGT 1 hour-high subjects may be associated with nonalcoholic fatty liver disease and dyslipidemia and could be potential candidates for pharmacological or non-pharmacological intervention even though they are classified as normal based on fasting plasma glucose and 2-hour postload plasma glucose levels. Indeed, we showed that fasting plasma glucose and triglyceride levels were significantly higher in NGT 1 hour-high subjects than in NGT 1 hour-low subjects. These parameters are components of metabolic syndrome, which is considered as a risk factor for type 2 diabetes [4]. Further studies are necessary regarding the cost-effectiveness of testing 1-hour postload glucose levels during the standard 75 g oral glucose tolerance test. We all appreciate Dr. Kim's valuable comments and suggestions.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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REFERENCES

1. Oh TJ, Min SH, Ahn CH, Kim EK, Kwak SH, Jung HS, Park KS, Cho YM. Normal glucose tolerance with a high 1-hour postload plasma glucose level exhibits decreased β-cell function similar to impaired glucose tolerance. Diabetes Metab J 2015; 39:147-53.
2. Bergman RN. Orchestration of glucose homeostasis: from a small acorn to the California oak. Diabetes 2007;56:1489-501.
3. Abdul-Ghani MA, Tripathy D, DeFronzo RA. Contributions of beta-cell dysfunction and insulin resistance to the pathogenesis of impaired glucose tolerance and impaired fasting glucose. Diabetes Care 2006;29:1130-9.
4. Lorenzo C, Okoloise M, Williams K, Stern MP, Haffner SM; San Antonio Heart Study. The metabolic syndrome as predictor of type 2 diabetes: the San Antonio heart study. Diabetes Care 2003;26:3153-9.