

ADULT

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Association of Serum Free Thyroxine and Glucose Homeostasis; Korea National Health and Nutrition Examination Survey

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INTRODUCTION

Glucose homeostasis is elaborately controlled by multiple processes in various organs. Thyroid hormones are involved in a wide range of glucose metabolism functions. Overt thyroid dysfunctions are related to altered glucose homeostasis. However, it is not conclusive as to whether subtle changes in thyroid hormones within normal ranges can induce alterations in the parameters that represent glucose homeostasis. The aim of this study was to evaluate the association between thyroid hormone and glucose homeostasis parameters in subjects without overt thyroid dysfunction based on nationwide population data.

METHODOLOGY

In the KNHANES VI 2015 (n=7,380), data were collected from subjects with insulin and thyroid function measurements who were older than 19-years-old. After the exclusion of subjects who had FT4 ranges that were beyond normal ranges, a total of 1,543 patients were included in the analysis. Subjects were categorized into quartiles of the FT4. Fasting glucose, insulin, HOMA-IR and haemoglobin A1c (HbA1c) levels were considered to be glucose homeostasis parameters.

RESULTS

No differences in serum fasting glucose levels and HOMA-IR values were observed among subjects with the different FT4 quartiles. A significant inverse correlation between FT4 and A1c levels was observed ($\beta=-0.261$, $p=0.025$). In the logistic regression analysis, the highest quartile of FT4 was demonstrated to lower the risk of HbA1c to a greater degree than the median by approximately 40%, after adjusting for confounders, compared to the lowest quartile ($p=0.028$).

CONCLUSION

Without overt thyroid dysfunctions, an association between FT4 and A1c levels was observed. A1c is therefore a relevant parameter for the measurement of the net effects of thyroid hormone on glucose metabolism in subjects without diabetes. Subjects with the lowest FT4 quartile should be cautiously managed in terms of altered glucose homeostasis.