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<th>Title</th>
<th>Risk of Coronary Heart Disease and Mortality for Adults With Subclinical Hypothyroidism</th>
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<tr>
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To the Editor:

Rodondi et al. assessed the risk of coronary heart disease (CHD) and total mortality for adults with subclinical hypothyroidism. In this study, they showed that the hazard ratio for CHD events was 1.00 (95% confidence interval [CI], 0.86-1.18) for a thyroid-stimulating hormone (TSH) level of 4.5 to 6.9 mIU/L, 1.17 (95% CI, 0.96-1.43) for a TSH level of 7.0 to 9.9 mIU/L, and 1.89 (95% CI, 1.28-2.80) for a TSH level of 10.0 to 19.9 mIU/L. They concluded that subclinical hypothyroidism is associated with an increased risk of CHD events and CHD mortality in those with higher TSH levels, particularly in those with a TSH concentration of 10 mIU/L or greater, and that minimal TSH elevations are not associated with an increased risk of CHD events and CHD mortality. On the other hand, they did not verify the CHD events and CHD mortality among those within the reference range of TSH levels.

It has been suggested that TSH levels within the reference range may be positively associated with body mass index and negatively associated with insulin sensitivity. We recently investigated the relationship between thyroid function and carotid intima-media thickness (CIMT) in a relatively large general population with euthyroid status and demonstrated that CIMT is independently associated with thyroid function within the normal reference range, which suggests increased cardiovascular risk in subjects with low-normal thyroid function. Furthermore, Åsvold et al. conducted a population-based prospective cohort study and found that TSH levels, even within the reference range, were positively and linearly associated with CHD mortality in women.

One possible explanation for this gap is the effects of confounding factors. In the study conducted by Åsvold et al., a modest attenuation of the effect of TSH levels on CHD was observed after adjustment for blood pressure and serum lipids, which suggests that the effect of TSH may be at least partially mediated by these factors. Further analysis should be carefully conducted with a similar strategy in the current study, aimed at those within the reference range of TSH.
Conflict of interest

All authors have no conflicts of interest

References


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