which would also contribute to no significant relationship between SUA and CVD in men.Reportedly, exacerbation of glucose metabolism resulted in the decrease in SUA levels [39].Conversely, the pathological roles of hypouricemia would be caused by the attenu ation of beneficial effects of UA. Since UA is one of the major endoge nous antioxidants in humans [4], hypouricemia would result in exacerbation of oxidative stress and subsequent vascular dysfunction, thereby leading to the increased risk for CVD [37].In this study, a U-shaped relationship was observed between the SUA levels and incident CVD events in both sexes, suggesting the patholog ical roles of hypouricemia and hyperuricemia in CVD events develop ment in obese patients.Thus, reduc tion of UA-related beneficial activities and/or aggravation of glucose metabolism in hypouricemia would be implicated in a high CVD risk in obese patients.The SUA values corresponding to the lowest risk of incident CVD events were lower in women (5.2 mg/dL) than in men (6.6 mg/dL) with obesity in this study, suggesting that the optimal SUA values for male obese patients rather increased the CVD risk for female obese patients, as previously reported in a general pop ulation [19].

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