Association between dyslipidaemia and the risk of hyperuricaemia: A six-year longitudinal cohort study of elderly individuals in China

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Research Article

Keywords: Dyslipidaemia, Hyperuricaemia, Longitudinal cohort study, Stratification analyses

Posted Date: April 22nd, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1573800/v1

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Abstract

Background

Despite abundant evidence indicating that dyslipidaemia are associated with increased risks of hyperuricaemia, the relationship between each component of dyslipidaemia and hyperuricaemia remains controversial. Thus, we aimed to explore the correlation of dyslipidaemia and its components with hyperuricaemia in Chinese elderly individuals aged 60 years or older.

Methods

In the cohort study, 4081 participants aged 60 years or older without hyperuricaemia were investigated from 2014 to 2020. Cox proportional hazards models were used to investigate the associations between dyslipidaemia and its components (hypercholesterolaemia, hypertriglyceridaemia, high low-density lipoprotein cholesterol, and low high-density lipoprotein cholesterol) and hyperuricaemia.

Results

A total of 1155 participants suffered from hyperuricaemia (28.75%) at the time of the 6-year follow-up survey. In multivariable adjusted analyses, compared to participants with normal lipid levels, those with dyslipidaemia had 1.28 times the risk (95% confidence interval 1.12 to 1.47) of experiencing hyperuricaemia. The hazard ratios comparing hypercholesterolaemia, hypertriglyceridaemia, high low-density lipoprotein cholesterol, and low high-density lipoprotein cholesterol of dyslipidaemia with the normal group were 0.99 (0.72 to 1.37), 1.30 (1.07 to 1.57), 1.02 (0.70 to 1.50), and 1.20 (1.00 to 1.44), respectively.

Conclusions

In this prospective cohort, dyslipidaemia and its two different types, hypertriglyceridaemia and low HDL-C, increased the risk of hyperuricaemia.

Background

Hyperuricaemia (HUA), a major public health problem in the world, is of increasing concern due to its high prevalence and increased risk associated with dyslipidaemia, hypertension, and cardiovascular disease\textsuperscript{1–3}. Its prevalence is approximately 20% worldwide\textsuperscript{4}. Nevertheless, a clearly defined pathological mechanism for hyperuricaemia has not been fully elucidated until now and understanding the risk or protective factors of hyperuricaemia, especially modifiable ones, is important for asymptomatic
individuals with a high risk of developing hyperuricaemia to effectively prevent the condition and the subsequent condition of gouty arthritis.

Dyslipidaemia is a state of disorder caused by abnormal lipid metabolism in the body and the excessive or insufficient production of lipoproteins in the plasma, including hypertriglyceridaemia, hypercholesterolaemia, high low-density lipoprotein cholesterol (LDL-C), and low high-density lipoprotein cholesterol (HDL-C) [5]. A plethora of evidence has indicated that dyslipidaemia is significantly associated with elevated serum uric acid levels or hyperuricaemia [6–8]. However, the relationship between each component of dyslipidaemia and hyperuricaemia remains controversial. Some studies have found that triglyceride levels, but not HDL-C levels, were significantly closely correlated with hyperuricaemia [9,10], whereas other studies demonstrated that HDL-C was inversely related to serum uric acid levels [11]. Several cross-sectional studies have reported that dyslipidaemia may be associated with the development of hyperuricaemia, hampering causal inferences [12–14]. Consequently, scientifically sound studies are needed to investigate the relationship between dyslipidaemia and its different types with hyperuricaemia.

The aim of this research was to explore the causal association of dyslipidaemia and its components with hyperuricaemia. Therefore, we performed a prospective cohort study based on elderly individuals aged more than 60 years with a six-year follow-up from 2014 to 2020.

**Methods**

**Setting and participants**

Data were derived from the Weitang Geriatric Diseases Study, which is a community-based survey conducted in Weitang Town located in Suzhou, a metropolis in East China. Based on the official records, 5493 individuals aged 60 years and older were enrolled at baseline in 2014. From this cohort, we excluded 27 subjects with missing baseline serum uric acid values, 645 subjects who had missing health behaviour variables, 60 subjects with outliers, and 734 subjects with hyperuricaemia at baseline, leaving 4018 participants who were free of hyperuricaemia to take part in the study between 2014 and 2020. The Weitang Geriatric Diseases Study was conducted following the tenets of the Helsinki Declaration and approved by the Institutional Review Board of Soochow University. Written informed consent was obtained from all participants.

According to the initial blood lipid level, participants with abnormal blood lipids were included in the exposure group, and those with normal blood lipids were classified in the control group. Of the 844 people who had dyslipidaemia, 71 had hypertriglyceridaemia, 155 had hypercholesterolaemia, 22 had high LDL-C levels, 292 had low HDL-C levels, and 304 suffered from more than one dyslipidaemia component. The incidence of hyperuricaemia in each year was collected for the two groups. Individuals contributed person-time from study entry until the first hyperuricaemia event, loss to follow-up, or the end of the study, which ever occurred first (Additional file 1: Fig.S1).
Data Collection

We invited all eligible individuals to participate in the survey and to undergo a physical examination with laboratory testing at local community centers. Trained research staff administered a standard questionnaire to obtain data on sociodemographic variables and health behaviours. Factors collected in the survey were sociodemographic variables including age and sex; health behaviour variables including smoking status (yes/no), alcohol intake (yes/no) and physical activity (yes/no); anthropometric indicators including body mass index (BMI), systolic blood pressure (SBP), and diastolic blood pressure (DBP); and laboratory examination indices including levels of fasting plasma glucose (FPG), total cholesterol (TC), triglycerides (TG), high HDL-C, low LDL-C, creatinine (CRE), blood urea nitrogen (BUN), uric acid (UA), alanine aminotransferase (ALT), and aspartate transaminase (AST).

Definitions

Hyperuricaemia was defined as a serum uric acid level $\geq 420$ mmol/L ($\geq 7.0$ mg/dL) for men and $\geq 360$ mmol/L (6.0 mg/dL) for women$^{[15]}$. Dyslipidaemia was categorized into hypercholesterolaemia, hypertriglyceridaemia, low HDL-C levels, or high LDL-C levels$^{[5]}$. The cut-off values for hypercholesterolaemia, hypertriglyceridaemia, high LDL-C levels, and low HDL-C levels were TC $\geq 6.22$ mmol/L ($\geq 240$ mg/dL), TG $\geq 2.26$ mmol/L ($\geq 200$ mg/dL), LDL-C $\geq 4.14$ mmol/L ($\geq 160$, mg/dL), and HDL-C $\leq 1.04$ mmol/L (40 mg/dL), respectively. Diabetes mellitus was defined as a fasting blood glucose level $\geq 7.0$ mmol/L, taking antidiabetic medicines, or a diagnosis of diabetes mellitus$^{[16]}$. Hypertension was defined as a measured SBP $\geq 140$ mmHg, a DBP $\geq 90$ mmHg, and/or taking antihypertensive medications$^{[17]}$.

Statistical analysis

Baseline characteristics are reported as the means with standard deviations (SDs) for continuous variables and percentages for categorical variables. The characteristics of the participants with and without dyslipidaemia were compared using Student’s t test for continuous variables and the chi-square test for categorical variables.

Cox proportional hazards regression models were further fitted to calculate the hazard ratios (HRs) and 95% confidence intervals (CIs) of incident hyperuricaemia events to analyse the relationship between dyslipidaemia and hyperuricaemia after controlling for covariates by stepwise adjustments in three models: Model 1 was adjusted for age and sex; Model 2 was similar to Model 1, with the addition of smoking status, alcohol intake, physical activity, hypertension, diabetes, and levels of CRE, BUN, ALT, and AST; Model 3 was similar to Model 2, but was further adjusted for BMI, TC, TG, HDL-C and LDL-C levels. We repeated the analysis with TC, TG, LDL-C, and HDL-C levels as continuous variables to quantify the dose–response relationships of specific risk factors with hyperuricaemia.

Further stratified analyses, which depended on age (60–69 years or $\geq 70$ years), sex (female or male), BMI ($< 24$ or $\geq 24$), and hypertension (yes or no), were performed to evaluate potential confounding
factors that could have an impact on the correlation between dyslipidaemia and hyperuricaemia.

To minimize the influence of potential bias, we also performed sensitivity analyses that excluded participants who were confirmed as having hyperuricaemia within two years of follow-up or those with baseline cardiovascular disease, hypertension, or diabetes.

All statistical analyses were performed using STATA 15.0 and SAS software (version 9.4), and a P < 0.05 (two-sided) was indicated to be statistically significant.

**Results**

**Baseline characteristics**

In total, 4081 people who effectively completed the physical examination were included in this research, with an average age of 67.70 ± 6.43 years. A total of 1895 (47.16%) men with an average age of 67.63 ± 6.23 years, and 2123 (52.84%) women with an average age of 67.76 ± 6.61 years, were included in the study. By the end of the research, there were 1155 new cases of hyperuricaemia, with a 6-year cumulative incidence of 28.75%. We divided all participants into two groups based on their lipid levels: the dyslipidaemia group and the normal lipid group. BMI, SBP, DBP, FPG, and ALT levels, alcohol intake, history of hypertension, and history of diabetes were significantly higher among those who had dyslipidaemia (Table 1). There was no significant difference in other variables, such as age, sex, smoking status, physical activity, AST level, and CRE level, between the two groups (p > 0.05).
Table 1
Baseline characteristics of subjects participants by the group of blood lipid

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Dyslipidaemia (n = 844)</th>
<th>Normal lipid level (n = 3174)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years, mean ± SD</td>
<td>67.30 ± 6.10</td>
<td>67.80 ± 6.52</td>
<td>0.1197</td>
</tr>
<tr>
<td>Gender, n (%)</td>
<td></td>
<td></td>
<td>0.2130</td>
</tr>
<tr>
<td>man</td>
<td>382 (45.26)</td>
<td>1513 (47.67)</td>
<td></td>
</tr>
<tr>
<td>woman</td>
<td>462 (54.74)</td>
<td>1661 (52.33)</td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>24.31 ± 3.27</td>
<td>22.77 ± 3.06</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Smoking status, n (%)</td>
<td></td>
<td></td>
<td>0.8055</td>
</tr>
<tr>
<td>yes</td>
<td>291 (34.48)</td>
<td>1080 (34.03)</td>
<td></td>
</tr>
<tr>
<td>no</td>
<td>553 (65.52)</td>
<td>2094 (65.97)</td>
<td></td>
</tr>
<tr>
<td>Alcohol intake, n (%)</td>
<td></td>
<td></td>
<td>0.0006</td>
</tr>
<tr>
<td>yes</td>
<td>145 (17.18)</td>
<td>718 (22.62)</td>
<td></td>
</tr>
<tr>
<td>no</td>
<td>699 (82.82)</td>
<td>2456 (77.38)</td>
<td></td>
</tr>
<tr>
<td>Physical activity, n (%)</td>
<td></td>
<td></td>
<td>0.3484</td>
</tr>
<tr>
<td>yes</td>
<td>368 (43.60)</td>
<td>1327 (41.81)</td>
<td></td>
</tr>
<tr>
<td>no</td>
<td>476 (56.40)</td>
<td>1847 (58.19)</td>
<td></td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>146.22 ± 19.80</td>
<td>142.72 ± 19.00</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>86.57 ± 11.19</td>
<td>85.13 ± 11.20</td>
<td>0.0006</td>
</tr>
<tr>
<td>FPG, mmol/L</td>
<td>5.74 ± 1.31</td>
<td>5.52 ± 1.16</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>ALT, U/L</td>
<td>21.30 ± 13.56</td>
<td>18.33 ± 10.82</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>AST, U/L</td>
<td>24.14 ± 10.05</td>
<td>24.06 ± 8.07</td>
<td>0.0463</td>
</tr>
<tr>
<td>Creatinine, μmol/L</td>
<td>68.24 ± 14.79</td>
<td>67.73 ± 15.12</td>
<td>0.2303</td>
</tr>
<tr>
<td>BUN, mmol/L</td>
<td>4.94 ± 1.27</td>
<td>5.29 ± 1.42</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td></td>
<td></td>
<td>0.0006</td>
</tr>
<tr>
<td>yes</td>
<td>569 (67.42)</td>
<td>1936 (61.00)</td>
<td></td>
</tr>
<tr>
<td>no</td>
<td>275 (32.58)</td>
<td>1238 (39.00)</td>
<td></td>
</tr>
</tbody>
</table>

SD, standard deviation; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; ALT, alanine aminotransferase; AST, aspartate transaminase; BUN: blood urea nitrogen.
<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Dyslipidaemia (n = 844)</th>
<th>Normal lipid level (n = 3174)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes, n (%)</td>
<td></td>
<td></td>
<td>&lt; .0001</td>
</tr>
<tr>
<td>yes</td>
<td>93 (11.02)</td>
<td>206 (6.49)</td>
<td></td>
</tr>
<tr>
<td>no</td>
<td>751 (88.98)</td>
<td>2968 (93.51)</td>
<td></td>
</tr>
</tbody>
</table>

SD, standard deviation; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; ALT, alanine aminotransferase; AST, aspartate transaminase; BUN: blood urea nitrogen.

**Associations between the components of dyslipidaemia and hyperuricaemia.**

Table 2 demonstrates the association between the components of dyslipidaemia and hyperuricaemia in all groups by multivariate Cox proportional hazard regression analyses. Hazard ratios (HRs) and 95% confidence intervals (CIs) of hyperuricaemia to dyslipidaemia and each of its components were determined. After the full adjustment for potential confounders in Model 3, dyslipidaemia and its components of hypertriglyceridaemia and low HDL-C levels remained significantly correlated with hyperuricaemia, with hazard ratios of 1.28 (1.12 to 1.47), 1.30 (1.07 to 1.57) and 1.20 (1.00 to 1.44), respectively.

We observed no significant associations between hypercholesterolaemia, high LDL-C and hyperuricaemia in any of the models.

We found evidence of nonlinear associations (P < 0.001) for TG levels, with a strong association with hyperuricaemia at higher concentrations but a weaker association at low to moderate concentrations, and HDL-C levels, with a strong inverse association with hyperuricaemia at low to moderate concentrations but a weaker association at higher concentrations (Fig. 1).

Table 3 presents the association between the increasing number of dyslipidaemia components and hyperuricaemia. The HRs for hyperuricaemia were statistically significant for one, two or more components of each cumulative number compared with reference values for nondyslipidaemia components: the hazard ratios were 1.27 (1.08 to 1.49) and 1.30 (1.07 to 1.58), respectively.
Table 2
Hazard ratio for the incidence of hyperuricaemia by dyslipidaemia and its components

<table>
<thead>
<tr>
<th>Dyslipidaemia</th>
<th>Normal group</th>
<th>Exposure group</th>
<th>For each SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incident rate 10000 person-years</td>
<td>1840.74/10000</td>
<td>1981.61/10000</td>
<td>-</td>
</tr>
<tr>
<td>model1^a</td>
<td>1.0 (ref)</td>
<td>1.52 (1.33–1.73)</td>
<td>-</td>
</tr>
<tr>
<td>model2^b</td>
<td>1.0 (ref)</td>
<td>1.40 (1.23–1.60)</td>
<td>-</td>
</tr>
<tr>
<td>model3^c</td>
<td>1.0 (ref)</td>
<td>1.28 (1.12–1.47)</td>
<td>-</td>
</tr>
</tbody>
</table>

**High TC**

| Incident rate 10000 person-years | 2001.89/10000 | 2019.54/10000 | -           |
| model1^a      | 1.0 (ref)    | 1.18 (0.94–1.47) | 1.06 (1.00-1.14) |
| model2^b      | 1.0 (ref)    | 1.06 (0.84–1.32) | 1.06 (0.99–1.16) |
| model3^c1     | 1.0 (ref)    | 0.99 (0.72–1.37) | 0.88 (0.64–1.19) |

**High TG**

| Incident rate 10000 person-years | 2004.03/10000 | 1992.06/10000 | -           |
| model1^a      | 1.0 (ref)    | 1.66 (1.39–1.98) | 1.24 (1.17–1.31) |
| model2^b      | 1.0 (ref)    | 1.48 (1.24–1.78) | 1.18 (1.12–1.26) |
| model3^c2     | 1.0 (ref)    | 1.30 (1.07–1.57) | 1.15 (1.00-1.32) |

**High LDL-C**

| Incident rate 10000 person-years | 2003.00/10000 | 2002.92/10000 | -           |
| model1^a      | 1.0 (ref)    | 1.17 (0.90–1.53) | 1.14 (1.05–1.23) |
| model2^b      | 1.0 (ref)    | 1.07 (0.82–1.40) | 1.05 (0.96–1.15) |
| model3^c3     | 1.0 (ref)    | 1.02 (0.70–1.50) | 1.15 (0.76–1.73) |

**Low HDL-C**

| Incident rate 10000 person-years | 1851.33/10000 | 1945.95/10000 | -           |
| model1^a      | 1.0 (ref)    | 1.52 (1.29–1.79) | 0.56 (0.48–0.66) |
| model2^b      | 1.0 (ref)    | 1.44 (1.22–1.71) | 0.59 (0.50–0.70) |
### Table 3
Multivariable Cox regression analysis of between number of dyslipidaemia components and hyperuricaemia

<table>
<thead>
<tr>
<th>Number of dyslipidaemia components</th>
<th>Case(n)</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>3174</td>
<td>1(ref)</td>
<td>1(ref)</td>
<td>1(ref)</td>
</tr>
<tr>
<td>1</td>
<td>540</td>
<td>1.49 (1.28–1.75)</td>
<td>1.40(1.21–1.65)</td>
<td>1.27 (1.08–1.49)</td>
</tr>
<tr>
<td>2 and more</td>
<td>304</td>
<td>1.54 (1.27–1.87)</td>
<td>1.39 (1.14–1.69)</td>
<td>1.30 (1.07–1.58)</td>
</tr>
</tbody>
</table>

Model 1: adjusted for age and gender.

Model 2: adjusted for age and gender, smoking status, alcohol intake, physical activity, hypertension, diabetes, creatinine, blood urea nitrogen, alanine aminotransferase, and aspartate transaminase.

Model 3: adjusted for age and gender, smoking status, alcohol intake, physical activity, hypertension, diabetes, creatinine, blood urea nitrogen, alanine aminotransferase, aspartate transaminase and body mass index.
Subgroup analyses

Stratified analyses were performed to investigate whether the relationship between dyslipidaemia and hyperuricaemia was influenced by age, sex, BMI level, and hypertension. As shown in Fig. 2, after controlling for possible confounding factors, the stratification analyses showed that the association between dyslipidaemia and the presence of hyperuricaemia was more pronounced among individuals aged 70 years and older (hazard ratio 1.44, 95% confidence interval 1.13 to 1.82), men (1.38, 1.12 to 1.72), individuals with a BMI $\geq$ 24 (1.33, 1.11 to 1.60) and people with hypertension (1.32, 1.13 to 1.55). However, dyslipidaemia did not have significant interactions with any of the variables mentioned above.

Figure 2 Stratified analyses for the association between dyslipidaemia and hyperuricaemia

BMI: body max index; HR: hazard ratio; CI: confidence interval.

When analyzing a subgroup variable, age, gender, BMI, smoking status, alcohol intake, physical activity, creatinine, blood urea nitrogen, alanine aminotransferase, aspartate transaminase, diabetes, and hypertension were all adjusted except the variable itself.

Sensitivity analyses

In our sensitivity analyses, after mutual adjustment, the association between dyslipidaemia and hyperuricaemia was attenuated after excluding the first 2 years of incident hyperuricaemia or baseline diabetes or cardiovascular cases (Table 4). When we excluded participants who developed hypertension events, dyslipidaemia was no longer significantly associated with hyperuricaemia.

<table>
<thead>
<tr>
<th>Model</th>
<th>Number</th>
<th>Hazard ratios (95% Confidence Intervals)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Normal lipid level</td>
</tr>
<tr>
<td>Model 3c, excluding first 2 years' incident hyperuricaemia</td>
<td>3694</td>
<td>1.0 (ref)</td>
</tr>
<tr>
<td>Model 3c, excluding baseline cardiovascular cases</td>
<td>3728</td>
<td>1.0 (ref)</td>
</tr>
<tr>
<td>Model 3c, excluding baseline hypertension cases</td>
<td>1513</td>
<td>1.0 (ref)</td>
</tr>
<tr>
<td>Model 3c, excluding baseline diabetes cases</td>
<td>3719</td>
<td>1.0 (ref)</td>
</tr>
</tbody>
</table>

Discussion
In this 6-year prospective study of elderly individuals aged more than 60 years living in China, we clarified the relationship between dyslipidaemia and hyperuricaemia, suggesting that dyslipidaemia is significantly and independently associated with the risk of developing hyperuricaemia even after adjusting for possible confounding factors. We found evidence of associations with TG and HDL-C levels, suggesting that an elevated TG level and decreased HDL-C level may increase the risk of hyperuricaemia. In addition, the association between the increasing numbers of dyslipidaemia components and hyperuricaemia seemed to be somewhat stronger in mixed dyslipidaemia participants.

Many studies have confirmed that dyslipidaemia may cause an increase in hyperuricaemia. However, whether all dyslipidaemia components are involved in hyperuricaemia is still open to debate. The positive association between TG levels and hyperuricaemia risk is supported by previous studies[9,18,19]. Nakanishi et al[20] found that basal triglycerides remained an independent predictor of new-onset hyperuricaemia even after excluding patients with diabetes mellitus and those on long-term medications for certain chronic conditions, which was consistent with our study. The specific mechanism of elevated TG levels and hyperuricaemia has not been elaborated. One potential explanation that has been suggested is that increased free fatty acid production and utilization will be induced in the body with the increase in TG levels, and the catabolism of adenosine triphosphate will be accelerated, causing an increase in UA production[21].

Scholars have concluded that there are differences in the correlation between HDL-C levels and hyperuricaemia. Abbas Dehghan et al[22] concluded that there was no correlation between HDL-C levels and blood uric acid. However, we found a clear dose-response trend of increasing incidence of hyperuricaemia with decreasing HDL-C levels in our study, suggesting that a low HDL-C level is a risk factor for hyperuricaemia. HDL-C has anti-inflammatory, antioxidant and anti-apoptotic effects[23]. Some basic studies have shown that HDL-C inhibits urate crystallization-induced inflammation, which suggests that HDL may be related to the inflammatory response involving uric acid[24].

In a population aged 70 years and older, dyslipidaemia was associated with higher hazard ratios for hyperuricaemia than in people younger than 70 years. Because the activity of various chemical enzymes involved in the metabolic process of the body becomes abnormal with increasing age, the activity of some of the enzymes involved in the process of purine metabolism increases accordingly, giving rise to the cumulative concentration of blood uric acid in the elderly population.

With respect to sex differences, it is well known that males have higher uric acid levels than females[25,26]. Several previous studies have suggested that the association between dyslipidaemia and hyperuricaemia is not significant in females[27]. In contrast, we found this association in both male and female participants, and the correlation was even more pronounced among men. This phenomenon is mostly related to biological differences[28]. Androgens can promote the reabsorption of blood uric acid in the body and have a critical influence in hindering the excretion of blood uric acid[29]. In addition, men face significantly higher work and life pressure than women, which increases their chances of smoking, drinking alcohol and eating purine-rich and protein-rich foods, resulting in more exogenous purines.
entering the body. The result is a large accumulation of lactic acid in the body during metabolism, which prevents the normal excretion of uric acid in the blood, thus triggering an increase in blood uric acid concentration. This pattern of behaviour gradually raises the blood uric acid level in the male population.

A considerable amount of literature has been published showing that obesity and hypertension are closely related to dyslipidaemia and hyperuricaemia\cite{2,30,31}. Previous studies have shown that insulin resistance or hyperinsulinaemia has a pivotal role in the correlation between dyslipidaemia and the risk of hyperuricaemia. One possible explanation is that insulin resistance partially overlaps with the pathophysiological features of hypertension, obesity, dyslipidaemia and hyperuricaemia, as higher insulin levels reduce renal uric acid excretion\cite{32–34}. A longitudinal study of nondiabetic Japanese men showed that insulin resistance itself or hyperinsulinaemia (increased triglyceride concentrations and decreased HDL-C concentrations) may lead to the incidence of hyperuricaemia\cite{35}, which is similar to our study.

The present work has several limitations. First, since this study was based on one community cohort, the results need to be further validated in a multicentre, long-term prospective cohort study. Second, the sample size of participants with physical examination data was not large enough to satisfy the completeness of the survey. In addition, this study only measured the relevant indices at 2 time points of baseline follow-up and did not analyse the effect of dynamic changes in the indices in the follow-up results.

**Conclusions**

In conclusion, this cohort investigation demonstrated that the dyslipidaemia components of hypertriglyceridaemia and low HDL-C levels are positively correlated with the incidence of hyperuricaemia in the elderly population. Findings from this and previous studies provide important information for the early prevention of dyslipidaemia, which could be beneficial for reducing the incidence of hyperuricaemia and gout arthritis.

**Abbreviations**

HUA
Hyperuricaemia
TC
total cholesterol
TG
triglycerides
LDL-C
low-density lipoprotein cholesterolaemia
HDL-C
high-density lipoprotein cholesterolaemia
BMI
Declarations

Ethics approval and consent to participate

The study was conducted following the tenets of the Helsinki Declaration and approved by the Institutional Review Board of Soochow University. Written informed consent was obtained from all participants.

Authors' contributions

HS had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. HD, BZ and JZ participated in the acquisition of data, performed analysis and prepared all tables. YX participated in the study design and wrote the main manuscript text. QM helped develop the idea for the study and critically revised the manuscript. All authors read and approved the final manuscript.

Acknowledgments
The authors thank all subjects who took part in this study.

Consent for publication

Not applicable.

Availability of data and material

All the data and materials used in our article are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

This study was funded by funds from the Priority Academic Program Development of Jiangsu Higher Education Institutions at Soochow University, the National Key Laboratory of Radiation Medicine and Radiation Protection (GZK1201919), Suzhou science and technology development project (SS201811), Suzhou Xiangcheng district people's livelihood science and technology project (XJ201655, XJ201706), Jiangsu Key Laboratory of Preventive and Translational Medicine for Geriatric Diseases (KJS1513)

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References


Figures
Figure 1

Risk of incident hyperuricaemia associated with different dyslipidaemia types during the 6 years

The black solid line and the dashed area represent estimates of hazard ratios and the 95% confidence intervals, respectively, for each dyslipidaemia type. Covariates included age, gender, body mass index,
smoking status, alcohol intake, physical activity, hypertension, diabetes, creatinine, blood urea nitrogen, alanine aminotransferase, and aspartate transaminase.

**Figure 2**

Stratified analyses for the association between dyslipidaemia and hyperuricaemia
BMI: body mass index; HR: hazard ratio; CI: confidence interval.

When analyzing a subgroup variable, age, gender, BMI, smoking status, alcohol intake, physical activity, creatinine, blood urea nitrogen, alanine aminotransferase, aspartate transaminase, diabetes, and hypertension were all adjusted except the variable itself.

**Supplementary Files**

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