Original Article

Alcohol Drinking, Dyslipidemia, and Diabetes: A Population-based Prospective Cohort Study among Inner Mongolians in China

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Abstract

Objective  No previous studies have evaluated the association between dyslipidemia, alcohol drinking, and diabetes in an Inner Mongolian population. We aimed to evaluate the co-effects of drinking and dyslipidemia on diabetes incidence in this population.

Methods  The present study was based on 1880 participants from a population-based prospective cohort study among Inner Mongolians living in China. Participants were classified into four subgroups according to their drinking status and dyslipidemia. Multivariate logistic regression analysis and receiver operating characteristic (ROC) curves were used to evaluate the association between alcohol drinking, dyslipidemia, and diabetes.

Results  During the follow-up period, 203 participants were found to have developed diabetes. The multivariable-adjusted odds ratios (95% confidence interval) for the incidence of non-dyslipidemia/drinkers, dyslipidemia/non-drinkers, and dyslipidemia/drinkers in diabetic patients were 1.40 (0.82-2.37), 1.73 (1.17-2.55), and 2.31 (1.38-3.87), respectively, when compared with non-dyslipidemia/non-drinkers. The area under the ROC curve for a model containing dyslipidemia and drinking status along with conventional factors (AUC=0.746) was significantly (P=0.003) larger than the one containing only conventional factors (AUC=0.711).

Conclusion  The present study showed that dyslipidemia was an independent risk factor for diabetes, and that drinkers with dyslipidemia had the highest risk of diabetes in the Mongolian population. These findings suggest that dyslipidemia and drinking status may be valuable in predicting diabetes incidence.

Key words: Diabetes; Dyslipidemia; Drinking; Prospective; Cohort study
INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major risk factor for cardiovascular disease and can have devastating consequences on quality of life. In China, the prevalence of diabetes is high and increasing\[^1\]-\[^2\], a development that has followed rapid economic growth and changes in lifestyle. Diabetes has reached epidemic proportions in the general Chinese adult population and about 92.4 million adults 20 years of age or older (9.7% of the adult population) have diabetes\[^3\]. Dyslipidemia is a common co-morbidity in T2DM patients\[^4\], and over 70% of adults with T2DM have one or more lipid abnormalities\[^5\]. Low levels of high-density lipoprotein (HDL) cholesterol, often associated with elevated triglyceride levels, is the most prevalent form of dyslipidemia in T2DM patients. It has been shown that poor glycemic control increases triglyceride levels and decreases HDL levels in T2DM\[^6\]. In addition, emerging evidence suggests that dyslipidemia is a significant risk factor for the future development of T2DM\[^7\]-\[^8\].

Alcohol is also a potential risk factor for diabetes, and with its influence depending on the type of alcohol consumed and the level of consumption\[^9\]-\[^10\]. Alcohol consumption may also play an important role in changing lipid levels\[^10\]-\[^11\]. A Japanese study found that alcohol drinking influences components of metabolic syndrome, such as dyslipidemia. Furthermore, in heavy drinkers, the prevalence of high triglyceride levels was higher than that in non-drinkers\[^12\]. Our previous study showed that Mongolian population had a higher prevalence of alcohol drinking and a significant association existed between alcohol drinking and dyslipidemia\[^13\]. However, there have been no reports specifically analyzing the co-effects of dyslipidemia and alcohol drinking on diabetes risk among the Mongolian population (an ethnic minority in China). Considering the potential interplay between dyslipidemia and drinking, we analyzed the association between drinking, dyslipidemia, and diabetes incidence on the basis of a 10-year-follow-up study in an Inner Mongolian population in China.

METHODS

Study Participants

This study was established to evaluate potential risk factors for chronic diseases from 2002 to 2003 in an Inner Mongolia, an autonomous region in northern China. The methods used to recruit study participants and collect baseline data have been described elsewhere\[^14\]. Study participants were recruited from 32 villages in two adjacent townships, Kezouhou Banner rural and Naiman Banner. The majority of the residents were Inner Mongolians who had lived there for a long time and a number of generations and maintained a traditional diet and lifestyle. In total, there were 3475 Mongolian people aged over 20 years living in the selected villages. Among them, 886 people were excluded because of the presence of cardiovascular diseases, endocrine diseases including hyper/hypothyroidism, and antihypertensive drug use, or refusal to participate. Moreover, 94 diabetic patients and 61 non-diabetics without complete key variables were excluded. Finally, 2434 individuals were included in this study at baseline. Written informed consent was obtained for all study participants. This study was approved by the ethics committee at Soochow University in China.

Data Collection

The trained staff interviewed participants in Chinese using a standard questionnaire to obtain information on demographic characteristics, medical history, and lifestyle risk factors. Cigarette smoking was defined as having smoked at least one cigarette per day for 1 year or longer. Almost all residents we investigated drank the native distilled liquor that has an alcohol concentration of approximately 50%. Information regarding the amount and type of alcohol consumed over the past several years was collected, and alcohol drinking was defined as consuming at least 50 g distilled spirits (about 50% alcohol concentration, i.e., 25 g alcohol) per day for at least 1 year. Three blood pressure (BP) measurements were conducted for each participant using a mercury sphygmomanometer while participants were seated according to a standard protocol\[^15\]. The first and fifth Korotkoff sounds were recorded as the systolic (SBP) and diastolic blood pressures (DBP), respectively. The mean of these three blood pressure measurements was used in the data analysis. Height and body weight were measured by trained staff using a balance beam scale after subjects removed their shoes and wore light clothing. The body mass index (BMI) was calculated as the weight in kilograms divided by the square of the height in meters (kg/m\(^2\)).

Blood samples were obtained in the morning after at least 8 h of fasting. All plasma and serum
samples were frozen at -80 °C until laboratory testing. The concentrations of total cholesterol, HDL cholesterol, and triglycerides were assessed enzymatically on a Beckman Synchromy CX5 Delta Clinical System (Beckman Coulter, Fullerton, CA, USA) using commercial reagents. The low-density lipoprotein (LDL) cholesterol concentration was calculated using the Friedewald equation for participants who had a triglyceride level below 400 mg/dL. Dyslipidemia was defined as an LDL cholesterol level of 3.64 mmol/L (100 mg/dL) or greater, HDL cholesterol level of 0.91 mmol/L or less, triglyceride level of 1.70 mmol/L or greater, or total cholesterol of 5.72 mmol/L or greater. A modified hexokinase enzymatic method was applied to test plasma glucose levels. The concentration of C-reactive protein (CRP) was determined using an immunoturbidimetric assay on a Beckman Synchrony CX5 Delta Clinical System using commercial reagents. Serum insulin was measured using a radioimmunoassay method, and the homeostasis model assessment method was used to calculate the insulin resistance index (HOMA-IR): HOMA-IR = insulin (mU/L) × glucose(mmol/L) / 22.5. 

Follow-up and Outcome Assessment

Cohort individuals were re-investigated from 2013 to 2014. If it was reported that a participant had developed T2DM during the period between the baseline survey and follow-up, the research staff contacted the subject’s general practitioner and reviewed the subject’s medical records or death certificate to confirm the diagnosis. All other living participants were instructed to maintain their usual physical activity and diet for at least 3 days before an oral glucose tolerance test (OGTT) performed according to a standard protocol. After at least 10 hours of overnight fasting, a fasting venous blood glucose sample was obtained and a standard 75 g glucose solution was administered. Blood samples were drawn at 120 minutes after the glucose load to measure glucose concentrations. The plasma glucose level was measured with the use of a modified hexokinase enzymatic method. Incident diabetes was defined according to the 1999 World Health Organization (WHO) criteria (≥7.0 mmol/L fasting glucose level or ≥11.1 mmol/L 2 h glucose level) or a validated physician diagnosis or the use of antidiabetic medication or a diagnosis of diabetes recorded in the medical records or death certificate.

Statistical Analysis

All participants were classified into four subgroups: non-dyslipidemia/non-drinkers, non-dyslipidemia/drinkers, dyslipidemia/non-drinkers, and dyslipidemia/drinkers. Conventional cardiovascular risk factors among the four subgroups were compared using analysis of variance for continuous variables and chi-squared tests for categorical variables. Multivariate logistic regression analysis was used to compute the odds ratios (ORs) of diabetes among the four subgroups by adjusting for important confounding factors including age, sex, smoking, systolic blood pressure, diastolic blood pressure, BMI, CRP, and HOMA-IR. We set a multiplicative interaction term of dyslipidemia and drinking in the multivariate logistic regression model and tested its effect on diabetes incidence, independent of dyslipidemia, drinking, and other confounding factors. We also assessed the discriminatory value of dyslipidemia/drinking status by computing the area under the receiver operating characteristic curve (AUC) and compared a model including only conventional risk factors with a model including dyslipidemia and drinking status subgroup in addition to conventional risk factors. All P values were two-tailed and a significance level of 0.05 was used. All statistical analyses were conducted using SAS statistical software (version 9.2: SAS Institute, Cary, North Carolina, USA) and R statistical software (version 2.15).

RESULTS

Among the original 2434 participants, 274 died, and 280 were lost to follow-up. Therefore, 1880 people were included in the final analysis, and a total of 203 patients with diabetes were observed. The cumulative incidence rate was 10.79%.

Table 1 describes the baseline characteristics of the participants in each of the four study subgroups. Conventional diabetes risk factors such as age, sex, smoking status, blood pressure, BMI, CRP, and HOMA-IR, were significantly different between the four subgroups with dyslipidemic participants in either the drinking or non-drinking group tending to have higher BMI, CRP levels, and HOMA-IR. Drinkers with dyslipidemia or non-dyslipidemia tended to be elderly and male, have higher rates of smoking, and have higher SBP and DBP.

Over the 10 years between the baseline measurements and follow-up survey, the cumulative
incidences of T2DM for non-dyslipidemia/non-drinkers, non-dyslipidemia/drinkers, dyslipidemia/non-drinkers and non-dyslipidemia/drinkers were 7.04%, 9.09%, 15.42%, and 19.41%, respectively ($P<0.0001$).

Table 2 presents the age-sex-adjusted and multivariate-adjusted odds ratios and 95% confidence intervals (95% CI) for diabetes according to dyslipidemia and drinking status. Compared with the non-dyslipidemia/non-drinkers subgroup, the age-sex-adjusted odds ratios (95% CI) of diabetes in the non-dyslipidemia/drinkers, dyslipidemia/non-drinkers, and dyslipidemia/drinkers subgroups were 1.19 (0.72-1.97), 2.38 (1.64-3.45), and 3.02 (1.89-4.84), respectively. After adjusting for other confounding factors, the ORs (95% CI) of non-dyslipidemia/drinkers, dyslipidemia/non-drinkers and dyslipidemia/drinkers were 1.40 (0.82-2.37), 1.73 (1.17-2.55), and 2.31 (1.38-3.87), respectively, compared with the reference subgroup. Drinkers with dyslipidemia were at the highest risk of diabetes.

The independent effects of drinking and dyslipidemia on the risk of diabetes incidence were also analyzed and the multivariable-adjusted ORs (95% CI) of diabetes for drinking and dyslipidemia were 1.37 (0.91-2.05) and 1.70 (1.23-2.34), respectively (Table 3). Low HDL cholesterol and high triglyceride levels were associated with diabetes [ORs (95% CI): 1.50 (1.06-2.12) and 1.38 (1.01-2.05), respectively]. We further calculated ORs for diabetes according to the quartile of alcohol consumption [ORs (95% CI): 1.47 (0.89-2.41) for the second quartile, 1.43 (0.83-2.48) for the third quartile, and 1.30 (0.72-2.35) for the top quartile]. No significant interaction was detected between drinking and dyslipidemia on diabetes risk [ORs (95% CI): 1.02 (0.54-1.93); $P=0.958$].

### Table 1. Baseline Characteristics According to Dyslipidemia/Drinking Status in Inner Mongolia, China

<table>
<thead>
<tr>
<th>variables</th>
<th>Nondyslipidemia/Nondrinkers</th>
<th>Nondyslipidemia/Drinkers</th>
<th>Dyslipidemia/Nondrinkers</th>
<th>Dyslipidemia/Drinkers</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$n$</td>
<td>895</td>
<td>333</td>
<td>415</td>
<td>237</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>42.6±10.9</td>
<td>47.3±10.7</td>
<td>43.4±11.2</td>
<td>46.5±10.1</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>Male, %</td>
<td>19.2</td>
<td>77.5</td>
<td>22.7</td>
<td>81.0</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>Smoking, %</td>
<td>29.4</td>
<td>71.2</td>
<td>31.1</td>
<td>70.5</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>123±21.0</td>
<td>128±21.3</td>
<td>128±22.9</td>
<td>133±22.4</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>81.0±11.5</td>
<td>85.0±12.1</td>
<td>83.4±12.0</td>
<td>89.6±12.5</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)</td>
<td>22.1±3.30</td>
<td>21.3±2.73</td>
<td>23.3±3.40</td>
<td>23.0±3.49</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>2.75±1.65</td>
<td>2.77±1.68</td>
<td>3.19±2.17</td>
<td>2.99±1.67</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>CRP</td>
<td>6.80±6.18</td>
<td>7.23±5.96</td>
<td>10.24±9.13</td>
<td>13.83±11.47</td>
<td>$&lt;0.0001^*$</td>
</tr>
</tbody>
</table>

**Note.** SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; HOMA-IR, insulin resistance index; CRP, C-reactive protein. $^*$ These $P$ values were mutually adjusted for sex and age. $^*$ These $P$ values were adjusted for age and sex.

### Table 2. Odds Ratios for Diabetes Incidence According to the Four Subgroups

<table>
<thead>
<tr>
<th>Item</th>
<th>Case</th>
<th>Age-gender Adjusted OR 95% CI</th>
<th>Multivariable Adjusted$^+$ OR 95% CI</th>
<th>Multivariable Adjusted$^*$ OR 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nondyslipidemia/nondrinkers</td>
<td>63</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Nondyslipidemia/drinkers</td>
<td>30</td>
<td>1.19</td>
<td>0.72-1.97</td>
<td>1.32</td>
</tr>
<tr>
<td>Dyslipidemia/nondrinkers</td>
<td>64</td>
<td>2.38</td>
<td>1.64-3.45</td>
<td>2.38</td>
</tr>
<tr>
<td>Dyslipidemia/drinkers</td>
<td>46</td>
<td>3.02</td>
<td>1.89-4.84</td>
<td>3.34</td>
</tr>
</tbody>
</table>

**Note.** $^+$ Multivariable model adjusted for age, sex, and smoking. $^*$ Multivariable model adjusted for age, sex, body mass index, smoking status, systolic blood pressure, diastolic blood pressure, CRP, and HOMA-IR. CI, confidential interval.
Table 3. Odds Ratios for Diabetes According to Drinking and Blood Lipids

<table>
<thead>
<tr>
<th>Item</th>
<th>Age-gender Adjusted OR</th>
<th>95% CI</th>
<th>Multivariable Adjusted OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low HDL-C</td>
<td>1.70</td>
<td>1.22-2.37</td>
<td>1.50</td>
<td>1.06-2.12</td>
</tr>
<tr>
<td>High LDL-C</td>
<td>1.49</td>
<td>0.98-2.28</td>
<td>1.33</td>
<td>0.86-2.05</td>
</tr>
<tr>
<td>High TG</td>
<td>2.32</td>
<td>1.64-3.30</td>
<td>1.38</td>
<td>1.01-2.05</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>2.43</td>
<td>1.81-3.28</td>
<td>1.70</td>
<td>1.23-2.34</td>
</tr>
<tr>
<td>Drinking</td>
<td>1.23</td>
<td>0.84-1.79</td>
<td>1.37</td>
<td>0.91-2.05</td>
</tr>
</tbody>
</table>

Note. *Multivariable model adjusted for age, sex, body mass index, smoking status, drinking status, systolic blood pressure, diastolic blood pressure, CRP, and HOMA-IR. ^Multivariable model adjusted for age, sex, body mass index, smoking status, systolic blood pressure, diastolic blood pressure, CRP, and HOMA-IR. CI, confidential, interval.

A receiver operating characteristic curve was used to evaluate the predictive power of the logistic regression model. The conventional risk factors included age, sex, body mass index, smoking status, systolic blood pressure, diastolic blood pressure, CRP, and HOMA-IR. The AUCs for the model including only the conventional risk factors and the model including dyslipidemia and drinking status subgroup in addition to the conventional risk factors achieved some extent of discrimination with the AUCs equal to 0.711 and 0.746, respectively. After adding dyslipidemia and the drinking status subgroup, the discriminatory value significantly improved by 0.035 (P=0.003; Figure 1).

**DISCUSSION**

In this population-based prospective cohort study in an Inner Mongolian population, dyslipidemia/non-drinkers and dyslipidemia/drinkers subgroups were at a significantly higher risk for diabetes than the non-dyslipidemia/non-drinkers subgroup. Drinkers with dyslipidemia were at the highest risk in this population. Dyslipidemia was an independent risk factor for diabetes in this study, while drinking was not independently associated with diabetes. This study is the first to examine the co-effects of dyslipidemia and drinking on the incidence of diabetes in an Inner Mongolian population. The novelty of this study was that these two factors were combined to form a new variable and the co-effects of both factors were tested, compared with previous studies that analyzed dyslipidemia and alcohol drinking separately.

Several prospective studies have shown that dyslipidemia is associated with T2DM [20-23], which was present in more than one-third of our participants. A cohort study of 2447 patients with T2DM and 3052 control participants of European ancestry showed that low HDL cholesterol or high triglycerides was related to elevated T2DM risk with ORs of 1.39 (1.17-1.65) and 1.19 (1.01-1.41), respectively [24]. Several other prospective studies have also shown that low HDL cholesterol and high triglyceride levels are independent risk factors for T2DM, and the values of HDL cholesterol or triglycerides have been used in risk-scoring systems to predict incident diabetes [25-27]. These findings are consistent with our study outcomes. In China, higher...
HDL cholesterol levels were found to strongly predict diabetes incidence in a 10-year cohort study\textsuperscript{[28]}. Moreover, multiple clinical trials have demonstrated the benefits of the pharmacological treatment of dyslipidemia in preventing diabetes, both in primary and secondary prevention\textsuperscript{[29]}. 

Drinking, especially heavy drinking, may partly account for the development of diabetes. A meta-analysis found that moderate alcohol consumption lowered the risk of T2DM, whereas heavy alcohol drinking was a risk factor for diabetes\textsuperscript{[30-32]}. A recent study showed that increasingly higher quantities of alcohol usually consumed per episode increased the risk of diabetes, and binge drinking (≥3 drinks per episode) significantly increased the risk of future diabetes compared with <1 drink per episode\textsuperscript{[33]}. There is evidence to suggest that drinking can adversely affect the lipid profile. For example, Wakabayashi found that heavy drinking, even if occasional, showed detrimental effects on the triglyceride/HDL cholesterol ratio and the lipid accumulation product (LAP)\textsuperscript{[34-35]}. Moreover, Criqui and Golomb reported that large amounts of acute or long-term alcohol ingestion increased triglyceride levels and insulin resistance\textsuperscript{[36]}. Although alcohol drinking alone was not an independent risk factor of diabetes in our study, the cumulative incidence of diabetes for dyslipidemia drinkers was 19.41%, and appeared higher than the other categories. Logistic models indicated that drinkers with dyslipidemia had the highest risk of diabetes among the four subgroups, with a 2.31-fold increased risk compared with non-dyslipidemic non-drinkers. Meanwhile, the risk of the drinker group without dyslipidemia was non-significant. It seems that drinking alone is not an independent risk factor for diabetes, and only when combined with dyslipidemia does alcohol consumption potentially increase the risk of diabetes. Alcohol drinking probably amplifies the effect of dyslipidemia on diabetes. A plausible mechanism is that there is a decrease in the breakdown of chylomicrons and very low-density lipoprotein (VLDL) remnants due to acute inhibitory effect of alcohol on lipoprotein lipase activity. Furthermore, alcohol improves the synthesis of large VLDL particles in the liver, which is the main source of triglycerides\textsuperscript{[37]}, and further increases the risk of T2DM. The coexistence of drinking and dyslipidemia is a notable issue in diabetes prevention and it might be more important for people with dyslipidemia to avoid alcohol drinking to reduce the risk of future diabetes. Our study population comprised ethnic Inner Mongolians who live in northern China. Their lifestyle and living environment are comparable with those of the other residents of northern China. Considering the incidence rate of diabetes is relatively higher in northern China than in other regions, our findings are at least valuable for populations in northern China.

Our findings have an important preventive meaning for diabetes. At present, diabetes is considered to be an incurable disease that gradually causes increasing dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels, and results in an enormous economic burden to society. Therefore, it is obvious that early prevention and intervention is the most effective strategy. Healthy lifestyles, such as drinking moderately, a healthy balanced diet, exercise, and controlling body weight may be effective in lowering the risk of diabetes and diabetes-related complications.

Our study has several strengths that deserve mention. To our knowledge, it is the first study to examine the association between drinking, dyslipidemia, and diabetes in a minority population in China. The participants were homogeneous with respect to their environmental exposures and genetic background, and the study data were collected with rigid quality control. In addition, our follow-up time is relatively long, allowing us to obtain a less biased association between exposure variables and outcome events. However, there are also some limitations that should be mentioned. About 25% of the population from these villages did not participate, which may have introduced some selection bias. However, this bias is minimal because it is unlikely that participants decided not to participate because of their drinking or dyslipidemia status. Information on lipids and drinking status was recorded only once at baseline. However, because of the underdeveloped economy and low level of health awareness in Inner Mongolia, the rates of awareness and lifestyle changes for diabetes risk factors such as dyslipidemia are low. Thus, dyslipidemia and drinking status are not expected to have varied greatly during the follow-up. Furthermore, several important confounding variables, such as lifestyle and socioeconomic factors, were not measured in our study.

In conclusion, we found that dyslipidemia was an independent risk factor for diabetes, and drinkers with dyslipidemia had the highest risk of diabetes.
among an Inner Mongolian population. These findings suggest that dyslipidemia and drinking status may be valuable in predicting diabetes incidence.

CONFLICT OF INTEREST

None of the authors have any potential conflict of interest associated with this research.

AUTHORS CONTRIBUTIONS

LIANG Zhu and QIU Qiao Yan collected and analyzed the data, and wrote the manuscript. ZHOU Jing Wen and WU JiaHui collected and researched the data. XU Tian and ZHANG Ming Zhi researched and collected data. ZHANG Shao Yan designed the study, collected and researched data, and revised the manuscript. ZHANG Shao Yan is the guarantor of this work and, as such, had full access to the data and takes responsibility for the accuracy of data analysis.

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