Relevance between *Helicobacter pylori* infection and nonalcoholic fatty liver disease in Bai minority region, Southwestern China

Ping Yan
Dali University

Bocheng Yu
Dali University

Min Li
Baoshan People's Hospital

Weidong Zhao (✉ wdzhao@dali.edu.cn)
Dali University   https://orcid.org/0000-0002-7431-2432

Research

Abstract

Objective

The association between *Helicobacter pylori* (*H. pylori*) infection and nonalcoholic fatty liver disease (NAFLD) remains a matter of debate. We conducted this study to evaluate whether *H. pylori* infection is a major risk factor for NAFLD.

Methods

A total of 1185 individuals who received health check-ups from January 2017 to June 2019 were studied. Data of each subject who underwent ^{13}C-urea breath, abdominal ultrasound, neck vascular color doppler ultrasound, and had a complete set of serum biochemical results was collected from the hospital information system. Participants were allocated to NAFLD group and non-NAFLD group based on abdominal color ultrasound for NAFLD. The baseline characteristics and serum biochemical results were compared. Logistic regression analyses were utilized to identify risk factors for NAFLD. Logistic regression models adjusted for confounding factors was performed to investigate the association between *H. pylori* infection and NAFLD.

Results

Compared to subjects without NAFLD(n = 656), those with NAFLD (n = 529) were more likely to be older, with higher weight, SBP, DBP, higher levels of TC, TG, LDL-C, FPG, AST and ALT, but with lower levels of HDL-C (all *P* values < 0.05). The levels of ALT, AST, BMI, FPG, TG, and DBP were independent risk factors for NAFLD (all *P* values < 0.05). Additionally, *H. pylori* was also an independent risk factor for NAFLD (OR = 1.35, 95%CI 1.02–1.79, *P* = 0.036). TC and LDL-C levels were significantly higher in *H. pylori*-positive group (n = 464) than that of in *H. pylori*-negative group (n = 721) (all *P* values < 0.05).

Conclusions

H. pylori infection is a key risk factor for NAFLD, and serum lipid metabolic dysfunction can be observed in the subjects with *H. pylori*, suggesting the potential role of *H. pylori* infection in the progression of NAFLD.

Background

Nonalcoholic fatty liver disease (NAFLD) has emerged as one of the greatest global health issues in the 21st century and a major cause of chronic liver diseases worldwide[1]. In China, approximately 400 million adults have NAFLD and more than 0.1 million deaths[2, 3]. It is well established that NAFLD is

closely allied to metabolic syndrome, obesity and insulin resistance[4]. It is yet unclear whether gastrointestinal bacteria have any impact on NAFLD pathogenesis[5].

H. pylori is a gram-negative, spiral-shaped bacterium present in the human stomach and infects more than 50% of world's population[6]. It is a common belief that *H. pylori* infection is strictly associated with peptic ulcer disease, chronic gastritis, gastric cancer, and mucosa-associated lymphoid tissue lymphoma[7]. In recent years, the association between *H. pylori* infection and extra-gastric diseases including coronary heart disease, stroke and obesity, has attracted increasing interest among the scientific community[8]. Also, there are several studies about the positive correlations between *H. pylori* infection and NAFLD[9–11]. However, negative correlations between *H. pylori* infection and NAFLD have also been identified in some high-quality clinical trials[12–15]. Additionally, there are no available studies regarding the association between *H. pylori* infection and NAFLD in the Bai minority area of Southwestern China. Thus, we performed this study to evaluated the link between *H. pylori* infection and NAFLD and fill this void in local researches.

Methods

Study population

It was a cross-sectional study of Chinese asymptomatic adults who underwent health check-up in the First Affiliated Hospital of Dali University form January 2017 to June 2019 and entered into current study (Figure 1). Subjects who simultaneous underwent abdominal color ultrasound, cervical color ultrasound, ^{13}C-urea breath test (UBT), complete physical examination and complete biochemical tests were included in this study. Exclusion criteria were listed as follows: (a) a long history of heavy drinking: drinking alcohol at least 210 g for male, 140 g for female per week in the past year; (b) previous treatment history of Methotrexate, amiodarone, glucocorticoid and tamoxifen; (c) subjects with viral hepatitis, autoimmune hepatitis, hepatolenticular degeneration, total parenteral nutrition, lack of beta lipoprotein, congenital lipid atrophy, and celiac disease; (d) taking proton pump inhibitors for 2 weeks, taking antibiotics and bismuth salts for 4 weeks before 13C-urea breath test; (e) a history of upper gastrointestinal; (f) a history of previous gastric surgery; (g) subjects with tumor or mental illness. This study gained approval by the ethics committees of the First Affiliated Hospital of Dali University.

Physical examination and serum biochemical examination

Height, weight, body mass index (BMI), systolic and diastolic blood pressure of each subject were measured during the physical examination.

For serum biochemical test, subjects were expected to be fasting from 7:00 p.m. of the day before, blood samples were collected (8:00-9:00 a.m.). A 5-ml fasting venous blood sample was obtained under vacuum in a serum separator tube. Then serum separator tubes were centrifuged for 10 minutes at 3500 rpm. The serum biochemical indicators, including Alanine aminotransferase (ALT), aspartate aminotransferase (AST), album (ALB), urea, creatinine (Crea), uric acid (UA), fasting plasma glucose

(FPG), total cholesterol (TC), serum triglycerides (TG), serum low-density lipoprotein cholesterol (LDL-C), and serum high-density lipoprotein cholesterol (HDL-C), were tested within two hours of blood collection.

H. pylori Infection determination

Subjects received ^{13}C-UBT using commercial kits (Headway, Zhonghe, Shenzhen, China). In brief, the exhaled breath samples were obtained at baseline and 30 minutes after the oral intake of a ^{13}C-urea capsule (75 mg). The samples were analyzed by the ^{13}C infrared spectrometry (Type HCBT-01, Headway, Zhonghe, Shenzhen, China), and the results were expressed as difference per thousand (δ‰). Test value = δ‰(30min)−δ‰(0min)[16]. A test value ≥4.0 was considered positive, and a test value <4.0 was considered negative.

Diagnosis of NAFLD

All the subjects underwent abdominal color Doppler ultrasound examination made by an ultrasound doctor using a Logiq E8 system (GE Healthcare, Chicago, IL, USA). Diagnosis of NAFLD was made based on findings from ultrasound reports. Subjects who have at least two of the following three items can be diagnosed as Fatty Liver Diffuse Fatty Liver: (a) the near-field echo of liver is diffusely enhanced and is stronger than that of the kidney. (2) The structure of intrahepatic duct is not clearly displayed. (3) The far-field echo of liver is gradually attenuated[17].

Statistical Analysis

We applied a widely used software SPSS version 23.0 for statistical analysis. Continuous data accorded with normal distribution were presented as average ± standard deviation (Mean±SD), whereas categorical data by percentage. Continuous variables conforming to a normal distribution were compared using the *t*-test, and categorical variables were compared by Chi-square test. For non-normally distributed data, comparisons of the medians between two groups were performed by *Mann-Whitney U* test. Logistic regression analysis was utilized to identify independent risk factors for NAFLD. A *P* value <0.05 was regarded as statistically significant.

Results

Basic information of the subjects

A total of 1185 subjects were enrolled in current study, with 778 men and 407 women. Of 1185 subjects, the prevalence of NAFLD was 44.6% (529/1185), with 362 men and 167 women. Compared to subjects without NAFLD, those with NAFLD were more likely to be older, with higher weight, SBP, DBP, higher levels of TC, TG, LDL-C, FPG, AST and ALT, but with lower levels of HDL-C (all *P* values <0.05). There were no significant differences in height and ALB level between NAFLD group and non-NAFLD group (*P*>0.05). Moreover, the number of *H. pylori*-positive participants in NAFLD group were higher than those in non-

NAFLD group (43.5% *v.s.* 35.7%, *P*=0.007). Collectively, these results indicate that there may be somehow related between *H. pylori* and NAFLD.

Risk factors for NAFLD

To explore which factors influence NAFLD, univariate regression analysis was performed to establish risk factors for NAFLD. As shown in table 2, the levels of ALT, AST, BMI, FPG, TG, and DBP were independent risk factors for NAFLD (all *P* values <0.05). Of note, *H. pylori* was also an independent risk factor for NAFLD (OR =1.35, 95%CI 1.02-1.79, *P*=0.036).

Association between *H. pylori* infection and NAFLD

To further clarity the effects of *H. pylori* infection on NAFLD, univariate and multivariate regression analysis were applied (Table 3). *H. pylori* infection was associated with an OR 1.38 (CI 1.09-1.75) for NAFLD incidence (Model 1, unadjusted). After adjustment for age and carotid plaque, the OR decreased to 1.33 (CI 1.05-1.68) (Model 2, adjusted). When adjusting the model for ALT, AST and UA, the OR increased to 1.42 (CI 1.10-1.83) (Model 3, adjusted). When adjustment for SBP, DBP, TC, TG, LDL-C, FPG, and BMI, *H. pylori* infection could increase the 35% NAFLD incidence (OR 1.35, CI 1.02-1.79). Accordingly, these results indicate that *H. pylori* infection is a significant risk factor for NAFLD.

Association between *H. pylori* infection and metabolic syndrome components

To investigate the possible link between *H. pylori* infection and NAFLD, some metabolic syndrome components were further analyzed. As presented in Table 4, the levels of TC and LDL-C were significantly higher in *H. pylori*-positive group than that of in *H. pylori*-negative group (all *P* values <0.05). However, there was no statistically significant difference between two groups for TG (*P*=0.409), HDL-C (*P*=0.087), SBP (*P*=0.776), DBP (*P*=0.614), FPG (*P*=0.492), and BMI (*P*=0.142). Overall, our findings demonstrate that *H. pylori* infection may be responsible for NAFLD by affecting cholesterol metabolism.

Discussion

NAFLD is a common metabolic disorder that affects approximately 13%-30% of the general population, although its underlying mechanism still remains to be defined. Several factors, such as genetic, environmental, metabolic, and behavioral factors, contribute to the pathogenesis of NAFLD[18]. In recent years, some scientific investigations have revealed possible connections between *H. pylori* infection and NAFLD[19].

In current study, we found that *H. pylori* infection was significantly associated with NAFLD. Of note, *H. pylori* infection remained an independent risk factor of NAFLD morbidity following ruling out some confounding factors, such as age, carotid plaque, blood pressure, blood lipids, blood glucose, and liver enzymes (OR = 1.35,*P* = 0.036).

This might imply that *H. pylori* infected individuals wound have a 35% higher risk of NAFLD than those without *H. pylori* infection. This is consistent with the results of meta-analysis by Zhou et al. who showed that a positive link between *H. pylori* infection and the risk of NAFLD[9].

At present, the preliminary idea that NAFLD would be the hepatic manifestation of the metabolic syndrome is changed, and it is considered that it might actually precede metabolic syndrome[20]. When analyzed in conjunction with metabolic syndrome, we found that higher weight, SBP, DBP, higher levels of TC, TG, LDL-C, FPG, and BMI were observed in NAFLD group than those of in non-NAFLD group (all *P*< 0.05). Moreover, we also found that DBP, FPG, TG, and BMI were significant risk factors for NAFLD. Accordingly, we speculate that a cross-talk may exist between the NAFLD and metabolic syndrome components. As previously reported by other studies, metabolic syndrome itself is one of the key risk factors for NAFLD, and vice versa[21].

A large body of literature has described that *H. pylori* infection is closely related to NAFLD and metabolic syndrome[22–25]. In current study, the effects of *H. pylori* infection on some key indicators of metabolic syndrome (SBP, DBP, FPG, BMI, TC, TG, HDL-C, and LDL-C) were analyzed. Interesting, we found that the levels of TC and LDL-C were higher in *H. pylori*-positive group than in *H. pylori*-negative group (*P*< 0.05). Accordingly, *H. pylori* infection may contribute to the increase of TC and LDL-C. This is in agreement with other reports. A large-scale meta-analysis shows that *H. pylori* infection significantly affects the serum lipid profile, which might lead to various severe dyslipidemia-related diseases[26]. However, in this study, we failed to identify the impacts of *H. pylori* infection on other metabolic syndrome indicators, including SBP, DBP, FPG, BMI, TG, and HDL-C. We suggest that there are some reasons why our findings may differ from other studies. Firstly, our study population with a higher level of education was drawn from government affiliated institutions in Dali region. Compared with the rural population, they are likely to be more concerned about their health conditions, and have the awareness about the disease prevention. Secondly, subjects were given advance notification of some dos and don'ts prior to blood collection, especially eating habits. To some extent, some biochemical indicators of subjects in the original living environment, such as blood lipid, blood glucose, liver and kidney function, might be underestimated after the adjustment of dietary structure. Thus, our findings may be biased by the subjects with a good living environment, good habit, better dietary structure and good health awareness.

Several potential limitations of this study merit consideration. First, the current study is a single-center, small sample size, and retrospective research. Second, no follow-up of the subjects was performed. Third, only subjects of health check-up were involved in this study, and the results may not be generalizable to other populations. Larger cohorts including different subjects with different backgrounds are needed to further validate our findings.

Conclusion

H. pylori infection is an independent risk factor for NAFLD. A possible interaction may exist between NAFLD and indicators of metabolic syndrome (FPG, BMI, TG, and DBP). *H. pylori* infection has been

implicated in NAFLD initiation possibly through regulating lipid metabolism.

Abbreviations

NAFLD: nonalcoholic fatty liver disease; *H. pylori*: *Helicobacter pylori*;

UBT: urea breath test

Declarations

Acknowledgments

Not applicable

Authors' contributions

Drafting manuscript: PY, BcY. Data collection: PY, BcY, ML. Conceiving the study and data analysis: ML, WdZ. The author (s) read and approved the final manuscript.

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Availability of data and materials

The datasets analyzed during the current study are available from the

corresponding author upon reasonable request.

Ethics approval and consent to participate

All subjects gave their oral agreement to participate in this study. This study gained approval by the ethics committees of the First Affiliated Hospital of Dali University (2019072201).

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests

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Tables

Table 1 Comparison of study subjects demographics and biochemical laboratory data between NAFLD group and non-NAFLD group

	NAFLD group	Non-NAFLD group	t	P
Sex（male/female）	（362/167）	（416/240）	3.268	0.071
H. pylori（+, %）	（230, 43.5）	（234, 35.7）	7.285	0.007
Carotid plaque（+/-）	（104/425）	（63/593）	24.461	0.000
Age（years）	43.50±9.94	40.90±11.28	-4.211	0.000
Height（cm）	164.11±8.26	163.66±7.54	-0.957	0.339
Weight（kg）	72.61±11.79	61.97±11.04	-16.484	0.000
BMI（kg/m^2）	26.83±3.48	23.02±3.12	-19.594	0.000
SBP（mmHg）	125.42±16.81	117.34±15.56	-8.573	0.000
DBP（mmHg）	81.75±11.98	76.12±10.76	-8.421	0.000
TC（mmol/L）	5.36±1.34	4.98±0.94	-6.276	0.000
TG（mmol/L）	2.24（1.57,3.26）	1.34（0.99,1.99）	-13.597	0.000
HDL-C（mmol/L）	1.22±0.34	1.44±0.42	9.866	0.000
LDL-C（mmol/L）	3.15±0.89	2.92±0.78	-4.490	0.000
FPG（mmol/L）	5.19±1.62	4.69±1.20	-6.016	0.000
ALT（U/L）	33（23,49）	21（15,33）	-11.110	0.000
AST（U/L）	25（20,32）	22（19,28）	-5.099	0.000
ALB（g/L）	47.58±3.11	47.26±3.61	-1.573	0.116

Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; FPG, fasting plasma glucose; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALB, album.

Table 2 Univariate regression analysis for risk factors associated with NAFLD

Risk factors	B	S. E.	Wald	P	OR	OR95%CI
H. Pylori	0.30	0.14	4.38	0.036	1.35	1.02~1.79
Age	0.01	0.01	3.03	0.082	1.01	0.99~1.03
Carotid plaque	0.37	0.21	2.30	0.083	1.45	0.95~2.21
ALT	0.02	0.01	17.81	0.000	1.02	1.01~1.03
AST	0.03	0.01	9.31	0.002	0.97	0.95~0.99
UA	0.00	0.00	2.46	0.117	1.00	1.00~1.00
BMI	1.48	0.15	102.19	0.000	4.38	3.29~5.84
FPG	0.83	0.28	8.53	0.003	2.29	1.31~3.40
TC	-0.14	0.19	0.52	0.472	0.87	0.60~1.27
TG	0.85	0.15	30.83	0.000	2.33	1.73~3.14
HDL-C	-0.27	0.20	1.90	0.168	0.76	0.52~1.12
LDL-C	0.32	0.20	2.56	0.109	1.38	0.93~2.04
SBP	0.06	0.23	0.06	0.800	1.06	0.68~1.66
DBP	0.55	0.20	7.56	0.006	1.73	1.17~2.56

Abbreviations: B= unstandardized coefficients; S. E., standard error; OR, odds ratio; CI, confidence interval. ALT, alanine aminotransferase; AST, aspartate aminotransferase; UA, uric acid; BMI, body mass index; FPG, fasting plasma glucose; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 3 Multivariate logistic regression analysis for *H. pylori* infection and NAFLD after adjusting effects of confounding factors

Model	B	S. E.	Wald	P	OR	OR95%CI
Model 1	0.32	0.12	7.27	0.007	1.38	1.09~1.75
Model 2	0.28	0.12	5.40	0.020	1.33	1.05~1.68
Model 3	0.35	0.13	7.14	0.008	1.42	1.10~1.83
Model 4	0.30	0.14	4.38	0.036	1.35	1.02~1.79

Abbreviations: B= unstandardized coefficients; S. E., standard error; OR, odds ratio; CI, confidence interval.

Model 1, *H. pylori* infection only;

Model 2, adjusted for age and carotid plaque;

Model 3, adjusted for ALT, AST and UA; Model 4, adjusted for SBP, DBP, TC, TG, LDL-C, FPG, and BMI.

Table 4 Effect of *H. pylori* infection on metabolic syndrome indicators

	H. pylori（+）	*H. pylori*（-）	*t / U*	*P*
TC（mmol/L）	5.24±1.10	5.09±1.02	-2.47	0.014
TG（mmol/L）	1.71（1.16,2.71）	1.67（1.15,2.46）	-0.826	0.409
HDL-C（mmol/L）	1.31±0.37	1.35±0.42	1.71	0.087
LDL-C（mmol/L）	3.10±0.86	2.98±0.82	-2.51	0.012
SBP（mmHg）	121.14±16.29	120.86±16.79	-0.29	0.776
DBP（mmHg）	78.85±11.74	78.50±11.61	-0.50	0.614
FPG（mmol/L）	4.95±1.34	4.89±1.48	-0.69	0.492
BMI（kg/m^2）	24.92±3.64	24.59±3.89	-1.47	0.142

Abbreviations: TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; BMI, body mass index.
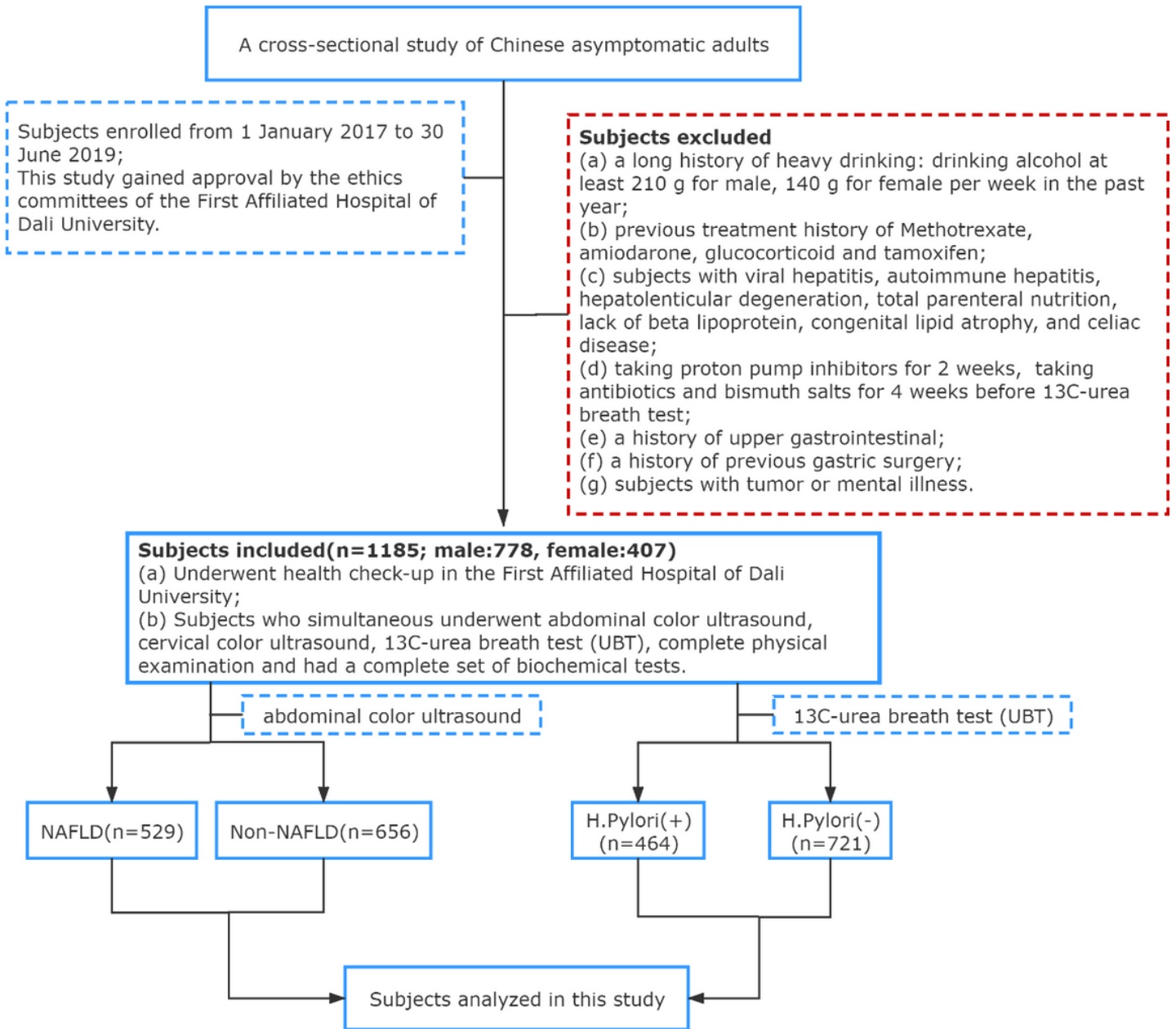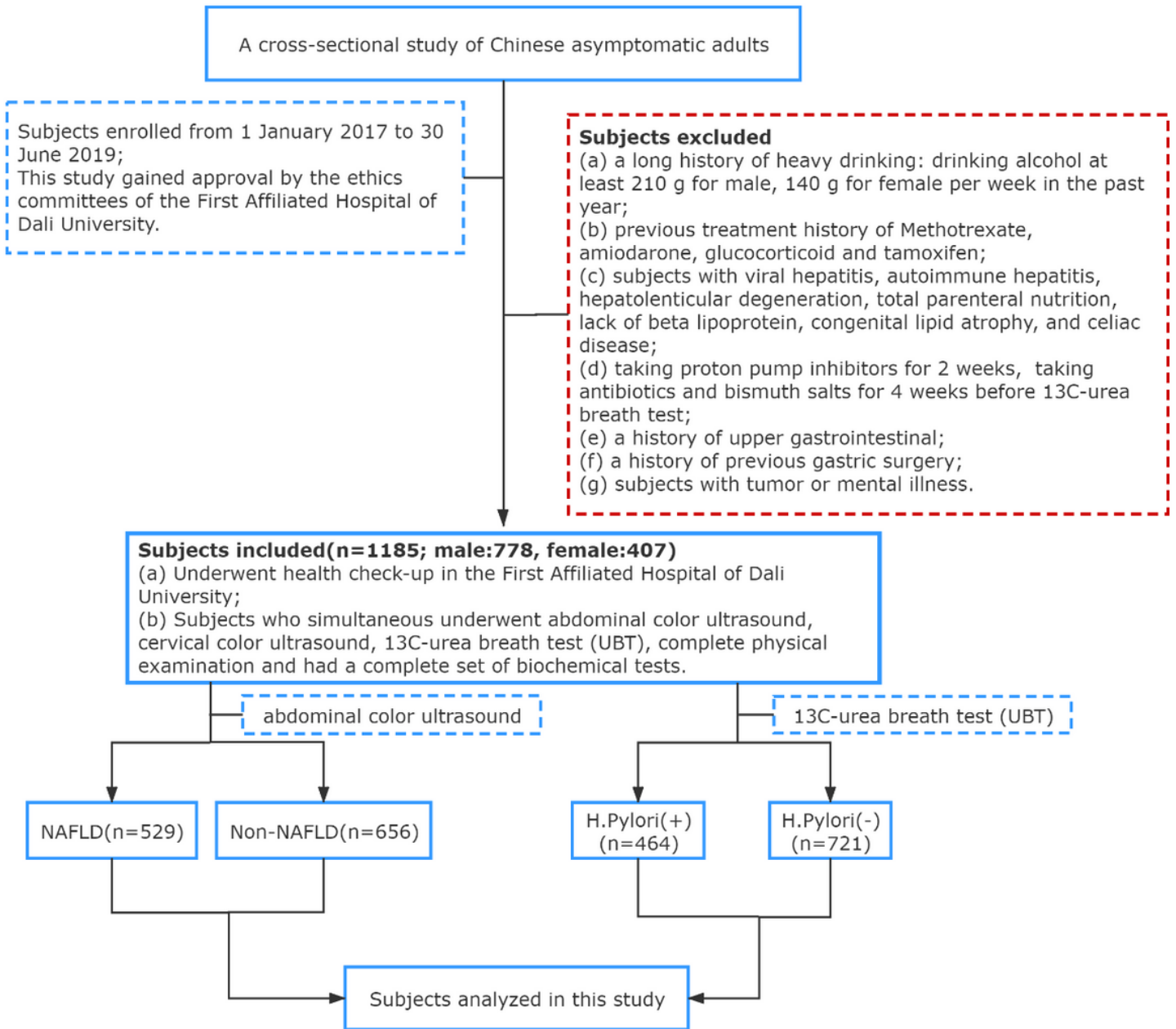
Figures

Figure 1

Flowchart of study population selection flowchart. H. pylori, Helicobacter pylori; NAFLD, nonalcoholic fatty liver disease.

Figure 1

Flowchart of study population selection flowchart. H. pylori, Helicobacter pylori; NAFLD, nonalcoholic fatty liver disease.