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Clinical Studies

Association between abnormal gastric function risk and *Helicobacter pylori* infection assessed by ELISA and ^{14}C-urea breath test

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ABSTRACT

Objective: Epidemiological studies found a significant correlation between *Helicobacter pylori* infection and elevated serum pepsinogen, especially pepsinogen II (PGII), and reduced pepsinogen I (PGI)/PGII ratio. The study aimed to evaluate the association between abnormal gastric function risk and *H. pylori* infection assessed by *H. pylori* IgG assay and ^{14}C-urea breath test (UBT).
Methods: A total of 1555 subjects who underwent a health check were enrolled. Serum PGI, serum PGII, PGI/PGII ratio, gastrin 17 (G17), *H. pylori* IgG antibody titer, and UBT results were collected.
Results: Median PGII and G17 levels were higher, but PGI/PGII ratio was lower in *H. pylori*–seropositive compared with seronegative participants ($P < 0.001$, respectively). Similar effects were demonstrated by UBT. The consistency between *H. pylori* IgG assay, and UBT results were 86.9%, 82.29%, and 84.64% in individuals with normal gastric function, but only 73.4%, 67.98%, and 74.6% in those with abnormal gastric function. The correlation coefficients for *H. pylori* infection and abnormal gastric function diagnosed by PGI/PGII <7 were 0.336 ($P < 0.001$) by *H. pylori* IgG assay and 0.231 ($P < 0.001$) by UBT, diagnosed by PGII ≥8.25 μg/L were 0.594($P < 0.001$) by *H. pylori* IgG assay and 0.493 ($P < 0.001$) by UBT, diagnosed by G17 >3 pmol/L was 0.469 ($P < 0.001$) by *H. pylori* IgG assay and 0.394 ($P < 0.001$) by UBT. The odds ratios (ORs) (95% confidence intervals) of abnormal gastric function were 7.477 (5.278–10.594), 19.204 (14.526–25.387), and 7.921 (6.286–9.982) comparing positive versus negative by *H. pylori* IgG assay and 4.084 (2.98–5.598), 9.552 (7.494–12.174), and 5.402 (4.335–6.731) comparing positive versus negative by UBT.
Conclusions: H. pylori infection assessments by antibody-based or bacterial component–based detection are both related with abnormal gastric function. Moreover, serum *H. pylori* IgG assay was stronger associated with abnormal gastric function than UBT assay.

1. Introduction

Helicobacter pylori is one of the most common bacterial infections of humans. It is well known that *H. pylori* infection has been associated with the development of gastric diseases. The crucial role of *H. pylori* makes its detection necessary in various situations. So far, multiple methods have been introduced including invasive and noninvasive assays. Among the noninvasive assays, the methods most widely used are antibody-based detection and bacterial component–based detection (Koletzko et al., 2003; Leal et al., 2008; Shimizu et al., 2003). Common design of antibody-based detection test is the serum IgG assay, which has the advantages that many serum samples can be tested in parallel, the process can be completely automated and the test is less expensive. The sensitivity and specificity of serological *H. pylori* IgG assay are reported at 85% and 79%, respectively (Loy et al., 1996). Serological IgG is useful in diagnosis of *H. pylori* in patients taking proton pump inhibitors (PPIs) or other antireflux medications; those recently treated with antibiotics; and patients with upper gastrointestinal bleeding, gastric atrophy, or gastric malignancy. It is the only test that is not affected by localized changes in the stomach that affect *H. pylori* load. However, serologic testing is not suitable for the diagnosis of active *H. pylori* infection or as a follow-up test after *H. pylori* eradication therapy. Common design of bacterial component detection test is urease activity by urea breath test (UBT) (Kato et al., 2002). UBT exploits the ability of *H. pylori* to convert urea into carbon dioxide (Chey, 2000), which is simple, innocuous, easy to repeat, and easy to collect and can even be sent by mail to a central laboratory for analysis. The sensitivity and specificity of UBT commonly exceed 95% (Leodolter et al., 1999). Additionally, The UBT has been used to screen patients before endoscopy and to assess the success of therapies aimed at eradicating *H. pylori* (Gisbert and Pajares, 2004). However, UBT in patients on long-term PPI therapy may result in false-negative results because PPI use leads to a rise in gastric PH and a subsequent reduction in *H. pylori* load. Some previous study also compared serum IgG assay and UBT to each other for diagnosis with histology stain and/or a positive culture as gold standard. They found

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that the sensitivity, specificity, and accuracy rates for *H. pylori* diagnosis were higher by UBT than by *H. pylori* IgG assay (Cohen et al., 1999).

As we know, *H. pylori* can colonize in the gastric mucosa, leading to the changes in the morphology and function of gastric mucosa (Suerbaum and Michetti, 2002; Sun et al., 2007). Morphological changes usually can be observed by endoscopic biopsy, and functional changes often are judged by enzymes and hormones secreted by the gastric mucosa. Serum pepsinogen (sPG) and gastrin levels seem to be related to the functional changes in the stomach, and their use as "serological biopsy" has been reported for over 20 years (Samloff, 1982; Samloff and Taggart, 1987; Samloff et al., 1982). Recently in our group, we further defined the cut-off values to differentiate Chinese patients with healthy and diseased stomach mucosa by 8.25 μg/L for serum pepsinogen II (sPGII) or 3 pmol/L for serum gastrin 17 (sG17) (He et al., 2011; Liping Sun et al., 2014; Sun et al., 2007) or atrophic and nonatrophic stomach by 6.9 for the pepsinogen I (PGI)/pepsinogen II (PGII) ratio (Sun et al., 2007). Establishment of these cut-off values made it possible to compare healthy and diseased stomach with changes of gastric function.

Although epidemiological studies have shown significant correlations between *H. pylori* infection and elevated sPG, especially sPGII, gastrin 17 (G17), and reduced PGI/PGII ratio (Ohkusa et al., 2004). However, what kind of *H. pylori* detecting assay represents gastric function change is unclear, which has important value to guide antibiotic therapy. This study, therefore, compared antibodies detected by serum *H. pylori* IgG assay and bacterial component detected by UBT to assess the associations between *H. pylori* infection and risk of abnormal gastric function in a health check-up population.

2. Methods

2.1. Subjects

This retrospective study included 1555 asymptomatic subjects (930 men, 625 women, and median age 48 years [16–86]) who were selected into our present study as follows. First, a total of 6207 participants (3476 males and 2731 females) who underwent PGI, PGII, PGI/II ratio, G17 examination, and *H. pylori* IgG assay during a health check-up program from September 2011 to May 2012 were included. Among the 6207 that had undergone serologic testing, 1555 had also previously undergone ^{14}C-UBT. We had no role in the determination of which patients had these tests performed. We then retrieved serologic and ^{14}C-UBT results from the laboratory information system for these 1555 patients. The participants in our study had no history of antibiotic treatment within the last 1 month.

2.2. Determination of sPGI, sPGII, sG17, and H. pylori IgG levels

Approximately 5 mL of fasting venous blood was collected from each participant. The samples were centrifuged immediately at 3500*g for 10 min. The serum aliquot was stored at −20 °C or used at once. Serum concentrations of PGI, PGII, G17, and *H. pylori* IgG levels were carried out by enzyme linked immunosorbent assay (ELISA) (Biohit Plc, Helsinki, Finland). For example of *H. pylori* IgG detection, briefly, a 1/200 dilution of serum in buffer was introduced into *H. pylori*–coated microtiter wells. After 30 min incubation at 37 °C, the wells were washed, and a peroxidase-conjugated anti-human IgG was added and incubated at 37 °C for a further 30 min. After washing, tetramethylbenzidine substrate was added for 30 min, and the optical density (OD) was measured at 450 nM. Duplicate negative and positive controls were included in each 96-well plate. Samples with a reading >42 enzyme immune units were regarded as *H. pylori* seropositive. The procedures of sPGI, sPGII, and sG17 detection see the Supplementary methods.

2.3. The cut-off values defined by sPGII, sPGI/II, and sG17 levels

The cut-off value defined by sPGII (8.25 μg/L), sG17 (3 pmol/L), and PGI/II ratio (6.9, approximately to 7) in this research based on preliminary data published by our group with histological diagnosis of endoscopic biopsy as gold standard (He et al., 2011; Liping Sun et al., 2014; Sun et al., 2007).The "healthy stomach" refers to normal mucosae without evidence of *H. pylori* infection and gastrointestinal symptoms. "Diseased stomach" is defined as mucosae with all kind of stomach diseases except "healthy stomach".

2.4. Determination of H. pylori status by ^{14}C-UBT

H. pylori infection was also determined using the ^{14}C-UBT. Granular powder (27.8 kBq [0.75 μCi]/grain) was administered orally with 100-mL tap water. Exhaled breath samples were taken at 20 min after consumption of the urea. The ^{14}C enrichment in the breath was measured using diagnostic equipment for *H. pylori* (HUBT-20; Zhonghe Headway Bio-Sci & Tech, Shenzhen, China). Samples were considered *H. pylori*–positive if the disintegration per minute/mmol CO_2 value ≥100.

2.5. Ethical statement

Ethical approval to collect data from the laboratory information system was obtained from the Human Ethics Review Committee of the First Affiliated Hospital of China Medical University (Shenyang, China). The Research Ethics Committee considered that written or verbal consent was not necessary because of the secondary use of detected data from the health-check program and because the analyzed data were de-identified.

2.6. Statistical analysis

All statistical analyses were performed using SPSS version 13.0 software (SPSS, Chicago, IL, USA). The distribution of variables was assessed by the Kolmogorov–Smirnov test. Descriptive analysis was performed to evaluate continuous variables using medians and interquartile ranges. Qualitative variables were expressed as percentages. χ^2 Tests were used to compare positivity rates for qualitative data. The Mann–Whitney U-test was used to compare medians of variables. The relationships between *H. pylori* status and gastric function were analyzed using bivariate correlation analysis. Conditional logistic regression was used to estimate the OR of *H. pylori* infection for abnormal gastric function. A 2-sided P value <0.05 was considered to be statistically significant.

3. Results

3.1. General characteristics of the study population

Among the 1555 participants, 618 (39.74%) were *H. pylori* seropositive and 937 (60.26%) were *H. pylori* seronegative, while 650 (41.8%) were *H. pylori* positive and 905 (58.2%) were *H. pylori* negative by UBT. sPGI, sPGII, and sG17 levels were significantly skewed from the normal distribution in the whole study. The median values (ranges) of sPGI, sPGII, PGI/PGII, and sG17 were 94.8 (74–121.4) μg/L, 8.2 (5.5–13) μg/L, 11.43 (8.22–15.02), and 2.43 (0.85–6.11) pmol/L, respectively (Table 1). Comparing *H. pylori* IgG assay–positive versus *H. pylori* IgG assay–negative status, there was no significant difference in abnormal gastric function according to PGII or PGI/II levels between the 1555 who underwent serologic testing and UBT compared to the 4652 who only underwent serologic testing, although there was a small but statistically significant OR difference for sG17 comparing the 1555 and 4652 groups (7.9: 6.29–9.98 versus 5.3: 4.64–6.19, respectively, $P < 0.05$).

3.2. Associations between gastric function and H. pylori status assessed by H. pylori IgG assay and UBT

Age, sex, sPGII, PGI/II, and sG17 were compared between individuals assessed as *H. pylori* positive and negative according to *H. pylori* IgG assay or UBT. The median age was significantly higher in the *H. pylori*-positive group, according to both *H. pylori* IgG assay and UBT results ($P < 0.05$). Median PGII and G17 levels were higher in *H. pylori*–seropositive compared with *H. pylori*–seronegative participants (13.0 versus 6.1 μg/L,

Table 1
Baseline characteristics.

Parameter	
Total n	1555
H. pylori IgG	
Seropositive, n (%)	618 (39.74)
Seronegative, n (%)	937 (60.26)
^{14}C-UBT	
Positive, n (%)	650 (41.8)
Negative, n (%)	905 (58.2)
sPGI (μg/L), median (range)	94.8 (74.0–121.4)
sPGII (μg/L), median (range)	8.2 (5.5–13)
PGI/II, median (range)	11.43 (8.22–15.02)
sG17 (pmol/L), median (range)	2.43 (0.85–6.11)

Table 3
Consistency between *H. pylori* IgG assay and UBT in terms of sex, age and gastric function.

Parameters		Concordance, n (%)	Discrepancy, n (%)	χ^2	*P* value	Consistency rate (%)
Sex	Male	737 (59.1)	193 (62.7)	1.303	0.25	79.2 (737/930)
	Female	510 (40.9)	115 (37.3)			81.6 (510/625)
Age	<48	607 (48.8)	144 (46.8)	0.396	0.53	80.8 (607/751)
	≥48	638 (51.2)	164 (53.2)			79.6 (638/802)
sPGII	<8.25	682 (54.7)	103 (33.4)	44.62	0.00	86.9 (682/785)
	≥8.25	565 (45.3)	205 (66.6)			73.4 (565/770)
PGI/II	≥7	1092 (87.6)	235 (76.3)	25.079	0.00	82.29 (1092/1327)
	<7	155 (12.4)	73 (23.7)			67.98 (155/228)
sG17	≤3	733 (58.8)	133 (43.2)	24.357	0.00	84.64 (733/866)
	>3	514 (41.2)	175 (56.8)			74.6 (514/689)

5.5 versus 1.15 pmol/L, $P < 0.001$, respectively), while the median PGI/II was lower in *H. pylori*–seropositive than *H. pylori*–seronegative participants (8.35 versus 13.64, $P < 0.001$, respectively). Similar results were observed when *H. pylori* status was defined by UBT (Table 2).

3.3. Consistency between H. pylori IgG assay and UBT in terms of sex, age and gastric function

Participants were divided into 2 groups: "concordance" and "discrepancy", based on *H. pylori* infection determined by *H. pylori* IgG assay and UBT. The "concordance" group included participants who were either *H. pylori* positive or negative according to both *H. pylori* IgG assay and UBT. All other participants were included in the "discrepancy" group and were either positive by *H. pylori* IgG assay but negative by UBT or negative by *H. pylori* IgG assay but positive by UBT.

The consistency of the results of both the tests was 79.2% for male and 81.6% for female. No significant difference was found between the consistencies of the 2 tests in different sex groups. The consistency of the results of both the tests was 80.8% for persons aged <48 years and 79.6% for persons aged ≥48 years. No significant difference was found between the consistencies of the 2 tests in different age groups (Table 3).

Stratification analysis of sPGI, sPGII, PGI/II, and sG17 between the "concordance" and "discrepancy" groups was carried out. The difference remained significant in individuals with sPGII levels ≥8.25 or <8.25 μg/L, PGI/II ≥7 or <7, and sG17 ≤3 or >3 pmol/L ($P < 0.05$). The consistency rates were as high as 86.9% for sPGII <8.25 μg/L, 82.29% for PGI/II ≥7, and 84.64% for sG17 ≤3 pmol/L but were only 73.4% for sPGII ≥8.25 μg/L, 67.98% for PGI/II <7, and 74.6% for sG17 >3 pmol/L (Table 3). These results indicated that normal gastric function was associated with greater consistency between the results of *H. pylori* IgG assay and UBT.

3.4. The correlation of H. pylori detection by H. pylori IgG assay or UBT and gastric function

The relationships between *H. pylori* status and gastric function were calculated by correlation analysis. The correlation coefficients for *H. pylori* infection and abnormal gastric function diagnosed by PGI/II <7 was 0.336 ($P < 0.001$) by *H. pylori* IgG assay and 0.231 ($P < 0.001$) by UBT, diagnosed by PGII ≥8.25 was 0.594($P < 0.001$) by *H. pylori* IgG assay and 0.493 ($P < 0.001$) by UBT, and diagnosed by G17 >3 was 0.469 ($P < 0.001$) by *H. pylori* IgG assay and 0.394 ($P < 0.001$) by UBT. These results indicated that gastric function correlated better with *H. pylori* infection assessed by *H. pylori* IgG assay than by UBT (Table 4).

Tables 5 show the ORs of abnormal gastric function indicated by PGI/II <7, PGII ≥8.25, or G17 >3 pmol/L associated with *H. pylori* seropositivity by IgG assay or positivity by UBT. The ORs (95% confidence intervals) were 7.477 (5.278–10.594), 19.204 (14.526–25.387), and 7.921 (6.286–9.982) comparing positive versus negative by *H. pylori* IgG assay and 4.084 (2.98–5.598), 9.552 (7.494–12.174), and 5.402 (4.335–6.731) comparing positive versus negative by UBT. The ORs by *H. pylori* IgG assay were more than 1–2-fold higher than those assessed by UBT. These results supports the evidence that measuring only by UBT to classify abnormal gastric function associated with *H. pylori* infection may lead to a substantial underestimation of the effect of *H. pylori* infection in abnormal gastric function risk.

4. Discussion

Among all the diagnostic methods of *H. pylori*, the detection target basically comprises 2 kinds: one is to detect the bacterial components itself, e.g., urease, to directly prove that whether the human body is infected with the bacterium; another is to detect human antibodies produced against bacterial infection, e.g., *H. pylori* IgG assay, to indicate whether there is a response against *H. pylori* infection in the human body. Unlike other invasive detection methods, such as biopsy-PCR method and others, UBT and *H. pylori* IgG assay as the noninvasive methods have been widely recognized and applied in clinical situation. In the present study, we compared *H. pylori* infection assessments by *H. pylori* IgG assay and UBT and evaluated the associations between *H. pylori* infection diagnosed by these 2 methods and abnormal gastric function risk in a health check-up population. Our results indicated that *H. pylori*–positive individuals had more abnormal gastric function than *H. pylori*–negative ones. Furthermore, normal gastric function was associated with greater consistency between *H. pylori* IgG assay and UBT results, and *H. pylori* status assessed by *H. pylori* IgG assay was more closely correlated with abnormal gastric function than status assessed by UBT.

Changes in serum levels of the biomarkers that reflect changes in the function of the gastric mucosa, i.e., abnormal levels are signs of a "sick" stomach mucosa and indicate failures in the feedback mechanism controlling secretion in the stomach. The current results indicated that sPGII and sG17 levels were higher, while PGI/II was lower, in *H. pylori*–seropositive individuals compared with *H. pylori*–seronegative participants. Similar results were observed in participants defined according to UBT. *H. pylori* has been reported to stimulate gastrin cells in the antrum, thus increasing the level of gastrin secretion (Lorente et al., 2002). Gastrin stimulates the main cells directly and is able to stimulate the synthesis and secretion of pepsinogens, especially PGII, by increasing calciumion flux, cyclic adenosine monophosphate, and phosphoinositide inside the main cells. This explains the associations between increased

Table 2
Gastric function parameters according to *H. pylori* status assessed by *H. pylori* IgG assay and ^{14}C-UBT.

Parameter	IgG assay		P value	^{14}C-UBT		*P* value
	+	−		+	−	
Age (years)	49 (43–56)	47 (41–55)	0.003	48 (42–56)	47 (41–55)	0.048
Sex						
Male	365 (59.1)	565 (60.3)	0.635	396 (60.9)	534 (59)	0.463
Female	253 (40.9)	372 (39.7)		254 (39.1)	371 (41)	
sPGII	13.0 (9.78–19.3)	6.1 (4.7–8.3)	<0.001	12.1 (8.8-17.6)	6.2 (4.7–8.7)	<0.001
PGI/II	8.35 (6.67–10.84)	13.64 (11–16.68)	<0.001	8.87 (7.04–11.39)	13.43 (10.64–16.48)	<0.001
sG17	5.5 (2.85–10.56)	1.15 (0.5–3.08)	<0.001	4.9 (2.25–8.88)	1.25 (0.5–3.45)	<0.001

Table 4
Spearman correlation coefficient for the relationship between *H. pylori* infection and gastric function.

H. pylori infection of noninfection diagnosed by	Gastric function normal or abnormal evaluated by	Correlation coefficient	*P* value
IgG antibody	sPGII cut off 8.25	0.594	0.000
	PGI/II cut off 7	0.336	0.000
	sG17 cut off 3	0.469	0.000
^{14}C-UBT	sPGII cut off 8.25	0.493	0.000
	PGI/II cut off 7	0.231	0.000
	sG17 cut off 3	0.394	0.000

sPGII and sG17 levels and decreased PGI/II in the presence of *H. pylori* infection and abnormal gastric function.

Most previous studies compared the consistency of these 2 methods mentioned above in terms of *H. pylori* infection itself. Traditional opinion is that *H. pylori* IgG assay is not synchronized with the *H. pylori* infection process and is less accurate owing to its delaying generation after an acute infection and persistent for several months after the end of eradication (Kosunen et al., 1992), while UBT is considered to synchronize with the *H. pylori* infection process and is the most accurate method to determine *H pylori* infection. Therefore, the discrepancy between the results of the 2 tests seemed to be caused by the false seropositive results. Unfortunately, few investigators considered the consistency of 2 methods in terms of the change of gastric function. In the present study, we found that abnormal gastric function was associated with lower consistency between the results of *H. pylori* IgG assay and UBT. In 1 case, the "discrepancy" group included individuals identified as seropositive by *H. pylori* IgG assay, but *H. pylori* negative by UBT. This apparent discrepancy may be related to antibody levels not yet returned to normal after the disappearance of infection. In the other case, the "discrepancy" included a negative result by *H. pylori* IgG assay but positive result by UBT, which may be associated with the acute phase of infection. In addition, the third possible underlying mechanism is related to the fact that abnormal gastric function is usually associated with gastric diseases, which conditions are inhospitable for *H. pylori*. Changes in the stomach environment caused by gastric disease will thus affect the sensitivity and specificity of *H. pylori* diagnosis and may have been responsible for the lower consistency in individuals with abnormal gastric function in our study.

Furthermore, the present results suggested that serologic antibody demonstrated more obvious correlation with the change of gastric function. UBT assay is the detection based on bacterial component itself but unable to determine whether there is a response against infection in the body and represent the existing infection. However, that *H. pylori* IgG–positive subjects enclose both existing and past infection. *H. pylori* infection induces specific IgG antibody responses against the bacteria in the peripheral blood as well as in the gastric mucosa (Futagami et al., 1998), which indicated a response against *H. pylori* infection in the body. The amount of these antibodies against self- or autoantibodies has been shown to be proportional to the degree of gastritis in infected individuals (Kreuning et al., 1994; Tu et al., 2014). Therefore, despite the production of such antibodies, the microorganism usually persists and gastritis progresses chronically. With the occurrence of superficial gastritis and the development of mucosal atrophy and intestinal metaplasia, the abnormal levels of gastric function will emerge. Worsening gastric function will render gastric conditions inhospitable for *H. pylori*, and this may result in false-negative results for UBT and other tests that detect bacteria or bacterial products (Kokkola et al., 2000; Salomaa-Rasanen et al., 2004; Zhang et al., 2005). However, the reduced number of *H. pylori* in the gastric mucosa has no effect on the diagnostic accuracy of serological assays (Herbrink and van Doorn, 2000). So, serologic tests are recommended for assessing *H. pylori* in patients with a low bacterial density (Kokkola et al., 2000; Malfertheiner et al., 2007). Measuring *H. pylori* infection only by UBT may lead to a substantial underestimation of the effect of *H. pylori* infection on abnormal gastric function risk.

As have discussed above, although UBT assay is a more accurate determinant for *H. pylori* infection than *H. pylori* IgG assay, the less correlation with abnormal gastric function inevitably leads to overlook of some patients with gastric diseases. Although *H. pylori*, *H. pylori* IgG assay is more correlative with abnormal gastric function, but the less accurate determination of *H. pylori* infection makes it inevitable to neglect some patients with *H. pylori* infection. We suggest that serum gastric function tests should be obtained, in addition to *H. pylori* IgG or UBT assays, as a normal course of clinical care and diagnostic workup.

This study had several limitations. First, participants included in this study were limited from the health check-up population. It is probable that the current conclusions may not generalize general or diseased population due to the differences of *H. pylori* infection rate among them. However, in our study, *H. pylori* infection rate is about 40%, which is close to infection rate of general population(Suerbaum and Michetti, 2002), and infection rate of clinical patients in the department of gastroenterology of our hospital in the same period(41%, data unpublished). Therefore, we consider that the current study has certain representativeness. Second, the lack of information on medication use, such as proton pump inhibitors and H_2 receptor antagonists, inevitably precludes us from excluding the impact of drugs on the diagnosis of *H. pylori* infection. However, given that this analysis was based on a health check-up population, the number of individuals receiving regular medication would be expected to be small and would, therefore, have little impact on *H. pylori* diagnosis. Third, the scarcity of information regarding pathological diagnoses represents another limitation. However, our designed aim is to compare the correlation of gastric function with 2 methods in parallel. Therefore, scarcity of information regarding pathological diagnoses would have little impact on our results.

In conclusion, the results of this study suggested that *H. pylori* infection assessments by antibody-based or bacterial component–based detection are both related with abnormal gastric function. Moreover, serum *H. pylori* IgG assay was stronger associated with abnormal gastric function than UBT assay.

Table 5
Association between gastric function according to sPGII, PGI/II,sG17 levels and *H. pylori* infection detected by *H. pylori* IgG assay or ^{14}C-UBT.

Parameter	sPGII		OR (95% CI)[a]	PGI/II		OR (95% CI)[a]	sG17		OR (95% CI)[a]
	<8.25	≥8.25		≥7	<7		≤3	>3	
	(n =785)	(n =770)		(n = 1327)	(n = 228)		(n = 866)	(n = 689)	
IgG negative (ELISA)	699	238	1.0(reference)	890	47	1.0(reference)	699	238	1.0(reference)
IgG positive (ELISA)	86	532	19.204 (14.526–25.387)	437	181	7.477 (5.278–10.594)	167	451	7.921 (6.286–9.982)
^{14}C-UBT negative	646	259	1.0(reference)	835	70	1.0(reference)	654	251	1.0(reference)
^{14}C-UBT positive	139	511	9.552 (7.494–12.174)	492	158	4.084 (2.98–5.598)	212	438	5.402 (4.335–6.731)

CI = confidence interval.
[a] Adjusted by age and sex.

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Statement

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