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ABO BLOOD TYPES OF BABCOCK UNIVERSITY STUDENTS, NIGERIA AND THEIR LINK WITH *HELICOBACTER PYLORI* INFECTION

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ABSTRACT

Five millilitres (5ml) of venous whole blood was collected from one hundred and eighty three students made up of 93 (50.8%) male and 90 (49.2%) female students of Babcock University, Ilishan Remo randomly selected across various Departments. Whole blood samples were dispensed into sequestrinized (EDTA anticoagulated) blood containers, properly mixed and labeled. Plasma samples were tested for *Helicobacter pylori* specific antibodies using the *H. pylori* Rapid Test Devices Kit. ABO blood phenotyping was carried out with monoclonal Antisera A, B and D by tile agglutination. A total of 169 (92.3%) and 14 (7.7%) students were rhesus positive and negative respectively of which 92 (54.4%) and 77 (45.6%) samples were rhesus positive male and female students respectively and of which 1 (7.1%) and 13 (92.9%) students were rhesus negative male and female students respectively. One hundred and thirty five (73.8%), 36 (19.7%) and 12 (6.5%) of the sampled student population belonged to 17-20, 21-24 and 25-30 yr age brackets respectively. One hundred and ten (60.1%), 38 (20.8%), 29 (15.9%) and 6 (3.3%) students were of O, A, B and AB blood phenotypes respectively. A total of 9 (4.9%) male students were seropositive for *H. pylori* infection of which 3 (10.3%) and 6 (5.5%) belonged to groups B and O respectively with no female students infected. Chi square analysis showed that sex was significantly associated with *H. pylori* infection (X^2 = 137.571, Critical (P) value of $X^2_{0.05(1)}$ = 3.841, $X^2_{0.01(1)}$ = 6.635, P<0.05, P<0.01). Chi square analysis also indicated that ABO phenotypes were significantly associated with *H. pylori* infection with respect to types B and O (X^2 = 178.211, Critical (P) value of $X^2_{0.05(1)}$ = 3.841, $X^2_{0.01(1)}$ = 6.635, P<0.05, P<0.01).

KEYWORDS: ABO types, University students, link, *H. pylori*, infection.

1. INTRODUCTION

Helicobacter pylori (H. pylori) is a Gram negative bacillus that regularly colonizes the human stomach.[1,2] It is present in 20 to 50% of the population in developed countries and 80% of the population in developing countries.[3] *H. pylori* are gram negative microaerophilic, spiral, rod-shaped bacteria which are a major health problem worldwide.[4] Gastritis, peptic ulcer disease, gastric carcinoma and mucosa associated lymphoid tissue (MALT) lymphoma are recognized complications of H. pylori infection.[4] According to Suerbaum *et al.*[5] and Oluwasola *et al.*,[6] *Helicobacter pylori* is a microaerophilic, gram negative, motile, spiral, flagellated bacterium with a capability for abundant urease production which has been implicated in several upper gastro-intestinal diseases that present a dyspepsia. *H. pylori* is a cork-screw shaped microaerophilic Gram negative cocccobacillus (0.5um by 3um) equipped with 2-6 flagella that are lophotrichously positioned.[7,8,9]

When a new slow-growing Campylobacter-like organism (CLO) was cultured by Marshall in 1982 from mucosal and stomach specimens of patients with gastritis, it was classified as *Campylobacter pylori* and shortly after, corrected to *Campylobacter pylori.*[10] New intestinal CLOs were discovered at the same time and C. pylori was sometimes referred to as gastric CLO (GCLO) and GCLO-1 when another CLO was isolated from the human stomach.[11,12] It soon became clear that even though *C. pylori* resemble *Campylobacter* in many aspects, it differed in important features such as flagellum morphology, fatty acid content and 16S rRNA sequence.[13,14] *C. pylori* was transferred to a new genus – Helicobacter and later named *Helicobacter pylori* in 1989 together with *Campylobacter fennelliae* and *Campylobacter cinaedae.*[7] Helicobacter was eventually placed in the phylum *Proteobacteria*, class *Epsilon Proteobacteria*, order *Campylobacterales* and family *Helicobacteraceae.* Currently, this genus Helicobacter consists of over 20 recognized species. *H. pylori* an *H. felis* are the only species known to infect the human host.[15,16]

H. pylori enter the digestive tract through the mouth and attaches to the gastric mucosa causing a persistent infection that is known to be closely associated with the development of disorders such as atrophic gastritis, gastric ulcers, stomach cancer.[17,18] These bacteria can

recognize and bind to blood group antigens expressed on the surface of the gastric mucosa which may play a critical role in the persistence of infection.[19,20,21] In recent years, lactic acid probiotics have gained attention as a method of preventing *H. pylori* infection and studies show that probiotics can inhibit attachment of *H. pylori* in the stomach.[22,23,24]

Among a number of adhesins, this organism uses bacterial adhesion protein called sialic acid binding adhesion (SabA) to recognize a molecule associated with inflammation and a molecule known as Lewis B antigen binding adhesion (BabA) to adhere to the inflamed cells of the glandular lining.[25,26,27] The ability of *H. pylori* to adjust its adherence properties to the level of inflammation it causes at the stomach surface could help explain how this bacterium maintains its persistence in the stomach of millions worldwide.

Several studies have highlighted a high prevalence of this organism in the developing World including Africa.[28,29,30,31,32] It has been estimated that over 80% of Africans are infected with *H. pylori* but their rate of developing gastric cancer is low.[33,34] Dating back as early as the 90s, a high prevalence (80%) of the organism and gastritis in an asymptomatic population was documented.[35] Careful surveys have also revealed that most persons in the developing world are infected with the bacterium at the early stages of their lives. *H. pylori* infections have been documented in several studies carried out in developing countries such as Egypt where a high prevalence (72.4%) was noted among children.[36] In yet another study, *H. pylori* IgG antibodies were also detected in South African children and their mothers.[37]

In a study involving hospitalized patients conducted in Venda region, Polokwane, PCR revealed a 50.6% prevalence rate of *H. pylori* DNA in faecal samples.[38] In another study carried out in Tanzania, seropositivity rose steeply with age from 76% in children aged 0-4yrs to 99% in adults.[39] A similar trend was recorded in Libya where prevalence rose with age up to 94% in age above 70yrs.[28] In Cameroon, a high incidence of *H. pylori* was recorded in both asymptomatic and symptomatic individuals using the HpSA technique.[8] High colonization rates have also been recorded asymptomatic individuals based in Tunisia.[40] In Nigerian children, the seropositivity rate rose from 57-82% in children between 5-9yrs of age.[35]

Immigration is responsible for isolated areas of high prevalence in certain Western countries. However, prevalence of the pathogen correlates more with socioeconomic status rather than with ethnicity.

About thirty major blood groups have been recognized by The International Society of Blood Transfusion (ISBT) and among this thirty are the ABO and Rh blood groups.[41] The ABO system is the most investigated erythrocyte antigen system for all populations and due to the ease of identifying its phenotypes, it has been used as a genetic marker in studies of associations with infectious and non-infectious diseases.[42,43] The ABO is the most clinically important antigen classification system to date. Its recognition is central to the practice of transfusion medicine, because of the immediate recognition and rejection of major incompatible non-self-cells.

Blood group antigens are either sugars or proteins and they are attached to various components in the red blood cell membrane. The antigens of the ABO blood group are sugars. They are produced by a series of reactions in which enzymes catalyze the transfer of sugar units. A person's DNA determines the type of enzymes they have and therefore, the type of sugar antigens that end up on their red blood cells. In contrast, the antigens of the Rh blood group are proteins. A person's DNA holds the information for producing the protein antigens. The RhD gene encodes the D antigen, which is a large protein on the red blood cell membrane. Some people have a version of the gene that does not produce D antigen, and therefore the RhD protein is absent from their red blood cells. If this protein is present on a particular blood type, that blood type is called positive and if absent, it is called negative.[44]

Many studies have shown adherence of the *H. pylori* to blood group O and Le^b antigen secretor in gastric mucosa and babA on the outer membrane of *H. pylori* mediates adherence of *H. pylori* to Le^b antigen expressed on the mucosa.[45,46,47] In colonizing the human host, *H. pylori* bind to gastric mucins rather than directly to mucosal epithelium in order to protect itself from luminal acidity and shedding.[48] The most efficient binding occurs on mucin Lewis b antigens with some secondary binding to H type 1 antigen.[49] Both antigens contain the terminal Fuca 1, 2 residue on which binding occurs. Blood groups A and B antigen determinants (GalNAcα 1, 3 and Galα 1, 3 respectively) are attached at the third position of the penultimate Galβ 1, 3 moiety immediately adjacent to the Lewis (b) Fucα 1, 2 residue.[21] For example, some strains of *H. pylori* that bind to the Lewis (b) antigen do not bind to A-Lewis (b) antigen.[21]

H. pylori have several lipopolysaccharides such as O antigen on its outer membrane. In addition, expression of Le b antigen of the gastric mucosa may play a role as a receptor to the bacterial adhesion. Binding of the *H. pylori* to H (on blood group O) and Le antigens in gastric mucosa probably describe higher incidence of chronic gastritis and adenocarcinoma in O blood group phenotype. Mohammad,[50] studied the relationship between ABO blood groups and *Helicobacter pylori* infection in symptomatic patients. The role of the lewis and ABO blood group antigens in *Helicobacter pylori* infection was investigated by Mohammad *et al*.[51] and Kamran et al.[52] examined the association of *Helicobacter pylori* infection with the lewis and ABO blood groups in dyspeptic patients. Takashi et al.[53] worked on association between *Helicobacter pylori*

infection and ABO blood groups in a cross-sectional study on subjects in a metropolitan town of Japan; Debebe and Deresse[54] reported did a systematic review and meta-analysis on association between O blood group individuals and *Helicobacter pylori* infection and some authors have also investigated and reported their findings on association between *Helicobacter pylori* infection, ABO blood groups and rhesus factor in peptic ulcer disease patients in a town in Central Sudan.[55] No known study has been done in which *Helicobacter pylori* infection was linked with ABO blood variants of students of Babcock University. This study therefore is aimed at linking *H. pylori* infection with ABO blood variants of Babcock University students with the below objectives:

(1) To determine the phenotypic ABO blood types and rhesus frequency distribution among the students recruited for study with respect to sex.
(2) To determine the age distribution of ABO blood types of Babcock University students recruited for the study.
(3) To determine the sex distribution of ABO blood types of Babcock University students recruited for the study.
(4) To determine any possible association of *H. pylori* infection with ABO phenotypic blood types of students recruited.

2. MATERIALS AND METHODS

2.1. Ethical Clearance

Ethical clearance was obtained from Babcock University Health Research Ethics Committee for the approval of the research proposal and other related materials after the necessary reviews and corrections. The students who were recruited for the study signed informed consent forms to show their approval before blood samples were collected from them.

2.2. Criteria for Selection of Subjects

In selecting the students to be used for this study, the aim of the study and benefits were explained to them. In addition, subjects were verbally asked questions such as: (1). do you have any history of peptic/duodenal ulcer? (2). If yes, are you currently on any anti-ulcer medications? Hence, subjects with history of peptic/duodenal ulcer and who were on anti-ulcer medications were excluded from the study. Consequently, subjects who did not have any history of peptic ulcer and who were not on any form of anti-ulcer treatment and who had voluntarily accepted to be recruited into the study were included and given consent forms to fill. All information obtained from participating subjects was treated with strict confidentiality.

2.3. Sampling

One hundred and eighty three students made up of 93 (50.8%) male and 90 (49.2%) female students of Babcock University Ilishan Remo, Ogun state were randomly selected across various departments and used for the study. Blood collection was done by venous puncture. With the aid of a tourniquet, a prominent vein was located on the fore arm and the vein area was sterilized with 70% ethanol soaked cotton wool swabs. Using sterile 5ml syringe and needle, five milliliters (5ml) of venous blood was withdrawn from subjects into appropriately labeled ethylene diamine tetra-acetic acid (ETDA) blood containers. All containers were properly mixed by standard method in order to sufficiently mix the anticoagulant with the blood to stop coagulation from taking place. All collected blood samples were allowed to stand for about 10mins to allow separation of blood into plasma and red cells by gravity. The plasma supernatant was carefully transferred into plain containers and appropriately labeled. Blood samples were collected by a qualified and licensed Medical Laboratory Scientist who is a Babcock University staff.

At the end of the research, the results (*H. pylori* status and ABO blood types) were written and given to participants as compensation for participation.

2.4. Specimen Handling/Disposal

Sterile Hand gloves and knee length laboratory coats were worn all through blood collection and disposal period. Specimens were properly labeled, packaged and kept in a functional refrigerator before and after use. All sera (after separation), were properly packaged and frozen before and after use. All red cell sediments were well packaged and appropriately disposed of. All used syringes and needles were also well packaged and disposed of by standard methods.

2.5. Duration/Venue of Study

This study was carried out between when ethical clearance was obtained and end of February, 2016. The venue of the research was the Microbiology laboratory of Bioscience Department of Babcock University, Ogun State. The plasma samples were tested for *Helicobacter pylori* specific antibodies using the *H. pylori* Rapid Test Devices kit.

2.6. Helicobacter pylori Rapid Test Procedure

The test device contained disposable specimen droppers, buffer and test cassettes inside foil pouch. The test device or set were placed on a clean and level surface. The dropper was held vertically and 4 drops of plasma (approximately 100µl) were transferred to the specimen well of the cassettes or test device and then a timer was started. In the case of hanging drop, two hanging drops of finger stick of whole blood specimen (approximately 50µl) were allowed to fall into the center of the specimen well for the cassette and the time started. The results were read after 10mins for red lines to appear. Red lines that appeared after 20mins were considered unreliable and not used.

2.6.1. Quality Control

A red line appearing in the control region C was an internal positive procedural control.

2.6.2. Limitations

The *H. pylori* Rapid Test Device is for invitro diagnostic

use only. The test was used for the detection of *H. pylori* antibodies in the whole blood, serum or plasma specimens only. The test device only indicated the presence of *H. pylori* antibodies in the specimen and was not used as the sole criteria for the diagnosis of *H. pylori* infecton.

2.6.3. Sensitivity

The *H. pylori* Rapid Test Device has been evaluated in specimen obtained from a population of symptomatic and asymptomatic individuals who presented for endoscopic examination. The result showed that the sensitivity of H. pylori Rapid Test Device is 93%.

2.6.4. Specificity

The *H. pylori* Rapid Test Device uses an antigen that is highly specific for *H. pylori* antibodies. The result showed that the specificity of *H. pylori* Rapid Test Device is 89.2%.

2.7. ABO Blood Group Typing

Monoclonal antiserum A, antiserum B and antiserum D reagent bottles which are commercially available were used for blood grouping as well as rhesus factor typing of all specimens collected.

2.8. Statistical Analysis of Data

Data obtained were analyzed by chi square at both 95% and 99% confidence intervals

3. RESULTS

The data on ABO blood groups and Rhesus factor frequency distribution among Babcock University students are shown in Table 1. A total of 183 whole blood samples obtained from 93 (50.8%) and 90 (49.2%) male and female students respectively were processed for ABO blood types. Out of this sample size, whereas 92 (54.4%) and 77 (45.6%) students were rhesus positive males and females respectively, 1 (7.1%) and 13 (92.9%) students were rhesus negative males and females respectively. On the whole, a total of 169 (92.3%) and 14 (7.7%) students were rhesus positive and negative respectively.

A total of 54 (58.1%), 24 (25.8%), 12 (12.9%) and 3 (3.2%) male students belonged to groups O, A, B and AB blood types respectively while a total of 56 (62.2%), 17 (18.9%), 14 (15.6%) and 3 (3.3%) female students were grouped into O, B, A and AB blood types respectively in that descending order. This showed that the most predominant blood group among Babcock University students is group O while the least is AB type. In the male students' population, the next highest occurring blood group was group A while it was group B in the female students' population.

Table 1: Phenotypic ABO blood types and rhesus frequency distribution among students recruited for study with respect to sex.

ABO blood types	Sex	Rhesus positive (%)	Rhesus Negative (%)	Total
A	M	24 (26.1)	0 (0.0)	24 (25.8)
	F	11 (14.3)	3 (23.1)	14 (15.6)
B	M	12 (13.0)	0 (0.0)	12 (12.9)
	F	12 (15.6)	5 (38.5)	17 (18.9)
AB	M	3 (3.3)	0 (0.0)	3 (3.2)
	F	3 (3.9)	0 (0.0)	3 (3.3)
O	M	53 (57.6)	1 (100)	54 (58.1)
	F	51 (66.2)	5 (38.5)	56 (62.2)
Total	M	92 (54.4)	1 (7.1)	93 (50.8)
	F	77 (45.6)	13 (92.9)	90 (49.2)
Overall total		169 (92.3)	14 (7.7)	183 (100)

The results of the age distribution of ABO blood types of Babcock University students recruited for the study are shown in Table 2. The subjects were grouped into 17-20, 21-24 and 25-30 age brackets of which 135 (73.8%), 36 (19.7%) and 12 (6.5%) students belonged to each group respectively. This suggested that the highest of subjects involved in the study belonged to the 17-20yr age bracket with an average age of 19yrs. This was distantly followed by 21-24 age bracket with an average age of 23yrs.

Table 2 clearly shows in decreasing order, that 110 (60.1%), 38 (20.8%), 29 (15.9%) and 6 (3.3%) students belonged to blood types O, A, B and AB respectively indicating that the highest and next highest occurring blood types in the studied population were groups O and A while the least occurring was clearly AB group.

In decreasing order, 77 (57.0%), 21 (58.3%) and 12 (100.0%) group O students were of 17-20, 21-24 and 25-30yr age brackets respectively. Twenty nine (21.5%), 9 (25.0%) and 0.0% group A students belonged to the same age groups respectively. Similarly, 23 (17.0%), 6 (16.7%) and 0.0% blood group B students were of the same age brackets respectively. Vertically, the distribution of ABO blood types in the 17-20 age group included 77 (57.0%), 29 (21.5%), 23 (17.0%) and 6 (4.4%) belonging to types O, A, B and AB respectively. In the 21-24 age group, 21 (58.3%), 9 (25.0%), 6 (16.7%) and 0.0% students were of O, A, B and AB groups respectively and lastly, in the 25-30 bracket, only 12 (100.0%) students were of group O blood type (Table 2).

Table 2: Age distribution of ABO blood types of Babcock University students recruited for the study.

ABO Blood Types	17-20yr (%)	21-24yr (%)	25-30yr (%)	Total (%)
A	29 (21.5)	9 (25.0)	0 (0.0)	38 (20.8)
B	23 (17.0)	6 (16.7)	0 (0.0)	29 (15.9)
AB	6 (4.4)	0 (0.0)	0 (0.0)	6 (3.3)
O	77 (57.0)	21 (58.3)	12 (100.0)	110 (60.1)
Total	135 (73.8)	36 (19.7)	12 (6.5)	183 (100.0)

In Table 3, data showing sex distribution of *H. pylori* infection among Babcock University students recruited for the study are presented. A total of 9 (4.9%) students were seropositive for *H. pylori* infection of which there was no female student infected. This implied that all the infected nine students were males. The possible association between *H. pylori* infection and ABO blood types was statistically analysed by Chi square and the critical (P) values of $X^2_{0.05(1)}$ and $X^2_{0.01(1)}$ were 3.841 and 6.635 at both 95% and 99% confidence intervals respectively. Calculated X^2 was 137.571 and hence, $P<0.05$ and $P<0.01$. Results showed that sex was significantly associated with *H. pylori* infection at both 95% and 99% confidence intervals.

Table 3: Sex distribution of H. pylori infection among Babcock University students recruited for the study.

Sex	Positive *H. pylori* infection (%)	Negative *H. pylori* infection (%)	Total (%)
Males	9 (100)	84 (48.3)	93 (50.8)
Females	0 (0.0)	90 (51.7)	90 (49.2)
Total	9 (4.9)	174 (95.1)	183 (100.0)

Critical (P) value of $X^2_{0.05(1)} = 3.841$
Critical (P) value of $X^2_{0.01(1)} = 6.635$
Calculated $X^2 = 137.571$
Hence, $P<0.05$ (at 95% confidence interval)
$P<0.01$ (at 99% confidence interval)

Table 4 represents summarizes the results on *H. pylori* infection as it related to the ABO blood types. There were a total of 9 (4.9%) infected students of which 3 (10.3%) and 6 (5.5%) belonged to groups B and O respectively. There were no infected groups A and AB students. As a consequence, 26 (89.7%) and 104 (94.5%) groups B and O students were un-infected respectively. The association between *H. pylori* infection and ABO blood types was statistically analyzed by Chi square and the critical (P) values of $X^2_{0.05(1)}$ and $X^2_{0.01(1)}$ were 3.841 and 6.635 at both 95% and 99% confidence intervals respectively. Calculated X^2 was 178.211 and hence, $P<0.05$ and $P<0.01$. Results showed that ABO blood phenotypes were significantly associated with *H. pylori* infection at both 95% and 99% confidence intervals with regard to groups B and O in which *H. pylori* infection was recorded.

Table 4: Association of H. pylori infection with ABO phenotypic blood types of students Recruited.

H. pylori status	ABO BLOOD TYPES				
	A	B	AB	O	Total
Infected (%)	0 (0.0)	3 (10.3)	0 (0.0)	6 (5.5)	9 (4.9)
Uninfected (%)	38 (100)	26 (89.7)	6 (100)	104 (94.5)	174 (95.1)
Total	38 (20.8)	29 (15.8)	6 (3.3)	110 (60.1)	183 (100)

Critical (P) value of $X^2_{0.05(1)} = 3.841$
Critical (P) value of $X^2_{0.01(1)} = 6.635$
Calculated $X^2 = 178.211$
Hence, $P<0.05$ (at 95% confidence interval)
$P<0.01$ (at 99% confidence interval)

4. DISCUSSION

In this study, 169 (92.3%) and 14 (7.7%) students were rhesus positive and rhesus negative respectively of which 92 (54.4%) and 1 (7.1%) male students were rhesus positive and rhesus negative respectively. Similarly, 77 (45.6%) and 13 (92.9%) female students were rhesus positive and rhesus negative respectively. Rhesus grouping is based on the presence or absence of the D antigen on red blood cells.[56,57] Whereas there were more rhesus positive male students than female students, there were more rhesus negative females than the males. This finding is not in agreement with the report of a previous study.[58] The 7.7% rhesus negative prevalence among the female students in this study is worrisome as it suggests a steady increase in rhesus negative factor frequency. This prevalence rate is high compared to 5.8% recorded by a previous author[59] and its occurrence to that level in females has serious

medical implications in terms of child birth and still birth which may arise from haemolytic disease of newborn (HDN). This increasing rhesus negative prevalence suggests the need for relevant health care providers as well as Ministry of health to track down people with this factor through compulsory blood typing test.

Many previous studies have however reported that rhesus positive population are much more frequent compared to that of rhesus negative although in varying proportions based on varied locations.[60,61,62,63] The findings in this study also show that the frequency occurrence of the ABO blood types were 60.1%, 20.8%, 15.9% and 3.3% for groups O, A, B and AB respectively (Table 2). This further validates the reports of previous authors which stated that blood groups O and AB are the most and least prevalent in any population.[60,61,62,63,64,65,66,67,68,69,70,71,72,73,74,75] These findings are not however consistent with an earlier report which stated that groups O and A are the highest and least occurring groups.[76] These differences may be due to ethnic, racial and geographical disparities inherent in various populations.

ABO blood types as well as rhesus blood types have attracted enormous attention regarding their association with genetic and infectious diseases.[57] Previous studies on patients of cancer and tumor,[77] heart disease,[78] parasitic and viral infections[79] indicated associations of ABO and rhesus blood groups. In particular, the ABO antigens regulate cellular activities suggesting their impact on determining susceptibility and severity of certain diseases.[80]

Helicobacter pylori has been linked or associated with the ABO blood groups particularly blood group O.[81] This organism is a major cause of upper gastrointestinal diseases such as gastritis, peptic ulcer and gastric cancer.[82,83] It has been suggested that up to 95% of duodenal and 70% of gastric ulcers are attributable to this infection and most cases occur in the middle aged subjects.[45] *H. pylori* enter the digestive tract through the mouth and attaches to the gastric mucosa causing a persistent infection that is known to be closely associated with the development of disorders such as atrophic gastritis, gastric ulcers, stomach cancer.[17,18] These bacteria can recognize and bind to blood group antigens expressed on the surface of the gastric mucosa which may play a critical role in the persistence of infection.[84,20,21] Some authors have reported that 80% of Africans are infected with *H. pylori* but their rate of developing cancer is low.[33,34]

In this study, the infection rate or prevalence rate of *H. pylori* among the studied population was 100% with respect to 9 (4.9%) students. This result is similar to 57-82% *H. pylori* infection rates recorded in children between 5-9yrs in Nigeria by some authors.[35] This particular finding in this work is also consistent with prevalence rates of 76% recorded for 0-4yr old and 99% recorded for adults in Tanzania by some authors,[39] 94% prevalence rate in Libya[28] and 72.4% in Egyptian children.[36] The 100% prevalence rate finding in this study is however, too high when compared with 50.6% infection rate recorded in Venda by some previous workers.[38]

A chi square statistical analysis of association of sex and *H. pylori* infection among students recruited showed that there was significant association at both 95% and 99% confidence intervals (critical or p value = 3.841 and 6.635 and calculated value = 137.571 suggesting $P<0.05$ and $P<0.01$). Hence, in this study, sex is significantly associated with *H. pylori* infection. This study also showed a significant association between *H. pylori* infection and ABO blood types with regard to blood groups O and B as these were the only groups in which seropositivity was recorded (critical or p value = 3.841 and 6.635 and calculated value = 178.211 suggesting $P<0.05$ and $P<0.01$). According to some previous authors, *H. pylori* bind to the H and Le^b blood group antigens in gastric mucosa. This binding, most likely explains the increased incidence of gastritis and gastric cancer in individuals with type O blood and in secretors who express the Le^b antigen.[81,85]

5. CONCLUSION

The 7.7% prevalence rate of rhesus negative subjects recorded in this study is high and its occurrence to that level in females has serious medical implications in terms of child birth and still birth which may arise from haemolytic disease of newborn (HDN) or erystoblastosis fetalis. Also, the 9 (4.9%) *Helicobacter pylori* infection rate implicated seems to be high. This increasing rhesus negative prevalence and apparently high *H. pylori* seropositivity suggest the need for relevant health care providers as well as Ministry of health to track down people with this factor through compulsory blood typing and screening tests.

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