Prevalence and Risk Factors Associated with *Helicobacter pylori* Infection among Patients with Dyspepsia Attending Kalkaal Hospital in Mogadishu, Somalia

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DECLARATION

This is my own novel work never presented for any award of a degree in any University

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LIST OF ABBREVIATIONS AND ACRONYMS

ANC	Antenatal Care
DHS	Demographic and Health survey
H. pylori	*Helicobacter Pylori*
IQR	Interquartile Range
IRB	Institutional Review Board
MOH	Ministry of Health
ODK	Open Data Kit
OR	Odds Ratio
PCR	Polymerase Chain Reaction
RAs	Research Assistants
RDT	Rapid Diagnostic Kit
SD	Standard Deviation
SSA	Sub-Saharan Africa
VIF	Variance Inflation Factor
WHO	World Health Organization

ABSTRACT

Background: Globally, about fifty percent of the adult population is colonized by *Helicobacter pylori*, with an estimated eighty percent prevalence among adults in low-income households. The high infection rate in low-income countries can be attributed to factors such as inadequate clean water supply, poor sanitation, overcrowding, low literacy levels, and poverty within the population. In Somalia, there is limited information available regarding the prevalence of *Helicobacter pylori* and its associated risk factors, especially among patients with dyspepsia.

Aim: To determine the prevalence of *H. pylori* infection and associated risk factors among patients with dyspepsia seeking medical services at Kalkaal Hospital in Mogadishu, Somalia

Methods: This was a cross-sectional hospital-based study conducted at Kalkaal Hospital in Mogadishu, Somalia. A total of 417 patients presenting with dyspepsia were randomly and systematically sampled. Socio-demographic and clinical characteristics were collected from the patients Data was collected using a pre-tested interviewer-administered questionnaire designed electronically in an open data kit (ODK). Laboratory testing involved the use of *H. pylori* one-step Rapid Diagnostic Kit antigen to test for *H. pylori* in fresh stool specimens. Data analysis was conducted using R language version 4.2.2. Summarization of results was done in a descriptive manner. Other analytical techniques entailed chi square and binary logistic models.

Results: The prevalence of *H. pylori* infection among patients with dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia was recorded at 56.2%. Statistically significant risk factors associated with *H. pylori* infection include drinking bottled water (adjOR = 1.93, 95% CI: 1.07–3.49), a history of peptic ulcers (adjOR = 3.15, 95% CI: 1.63-6.07), nausea with vomiting (adjOR = 4.89, 95% CI: 2.60–9.19), and heartburn (adjOR = 11.24, 95% CI: 6.19-20.42). The study noted that patients who exhibit dyspepsia when seeking medical services in Mogadishu, Somalia have a 97% likelihood of testing positive for *H. pylori* in their stool.

Conclusions: There is a need for more health awareness campaigns among residents residing in Mogadishu regarding the following risk factors associated with *H. pylori* infection: drinking bottled water, a family history of peptic ulcers, nausea with vomiting, and heartburn. These campaigns should focus on encouraging early testing and treatment of *H. pylori*, which is moderately prevalent in Mogadishu.

moderately prevalent in Mogadishu.

CHAPTER ONE

1.1 Introduction

Helicobacter pylori is a ubiquitous organism that poses a public health concern globally (Peleteiro *et al.*, 2014). There is an estimated 4.4 billion persons suffering from *H. pylori* infection in the world (Hooi *et al.*, 2017). This suggests that by 2017, over 50% of the total global population was *H. pylori* positive. However, varied prevalence have been reported across the world with Southern Asia particularly Pakistan reporting the highest burden (81%), followed by Africa (70.1%) and India (63.5%)(Hooi *et al.*, 2017). In developing countries, *H. pylori* prevalence is much higher (Hooi *et al.*, 2017) than in the developed countries (Kodaman *et al.*, 2014). Amongst the African countries, *H. pylori* prevalence of upto 73% in all age groups in Kenya (Shmuely *et al.*, 2003), 75.4 % amongst Ghanaian dyspeptic patients (Archampong *et al.*, 2017), 64% in Nigeria (Jemilohun *et al.*, 2010) and 75% in Rwanda (Walker *et al.*, 2014) have been documented (Fuccio *et al.*, 2010). Of concern is, even with reported prevalence of *H. pylori* in many other regions and in African countries, there's no recent prevalence data on *H. pylori* in Somalia.

The prevalence values in LMICs ranges from 85% to 95% (Correa & Piazuelo, 2011). *H. pylori* statistics may remain significantly unchanged in developing countries characterized by lower socio-economic status and maintaining proper sanitation and hygiene may be hard due to over population and poor standards of living (Kumar *et al.*, 2020). Previously, *H. pylori* infections have also been strongly associated with poverty (Khalifa *et al.*, 2010; Kumar *et al.*, 2020; Malcolm *et al.*, 2004). In developing countries like Somalia, where women have high levels of fecundity and thus a resultant high number of a child population, *H. pylori* infection prevalence may be high with respect to previous evidence that suggested failure to maintain proper hygiene and sanitation when an area is over populated (Kumar *et al.*, 2020). With regard to socio-economic status, nearly 50% of the Somali population are economically inactive with most women being full time housewives (Somali National Bureau of Statistics, 2021).

H. pylori is a gram-negative fastidious bacteria that was first isolated in 1982 in Perth, Australia (Marshall & Warren, 1984). This pathogen is believed to colonize the gastric mucosa of approximately 50% of the world's adult population (Peek & Blaser, 2002). Schwarz et al in 2008 reported that *H. pylori* is transmitted through the feco-oral, oro-oralor intra-familial route (Schwarz *et al.*, 2008), thus associating the infection with sanitation, food and personal hygiene. This has been echoed by other researchers confirming fecal-oral transmission as the most common especially in early childhood in developing countries (Das & Paul, 2007). In developed countries on the other hand, person-to-person mode of transmission through physical contact is considered to be most frequent (van Duynhoven & Jonge, 2001). Without proper or no administration of antibiotics, *H. pylori* infection may persist till adulthood in individuals who acquire it during childhood (Hooi *et al.*, 2017). Furthermore, infected patients maybe asymptomaticfor quite a long period of time (Hooi *et al.*, 2017). As a result, when *H. pylori* colonizes its host for a long time, the gastric mucosa may get inflamed and this may lead to peptic ulcer (Muhammad *et al.*, 2013). Longer colonization of the bacterium can also lead gastric cancer (Cover & Blaser, 2009; Muhammad *et al.*, 2013).

Predisposing factors such age, income and living standards, size of family, ethnic group, being a migrant from *H. pylori* endemic areas, having infected household members, and sanitation have been identified (The EUROGAST Study Group., 1993; Venneman *et al.*, 2018). Previously, *H. pylori* infection has also been linked to nutritional deficiency conditions for instance anemia (Muhammad *et al.*, 2017). Based on the available knowledge, the conducted study held novelty as it represented the first of its kind to investigate the prevalence of *H. pylori* infection and associated risk factors in dyspepsia patients attending Kalkaal Hospital in Mogadishu, Somalia. It was crucial, therefore, to conduct this study in order to expand insights in this area.

1.2 Problem Statement

Helicobacter pylori infection is of public health significance globally. Early detection and proper treatment are paramount due to possible progression to cancer or even death. If not detected early enough and without proper or no administration of antibiotics for the treatment, *H. pylori* infection may persist until adulthood even with infected patients being asymptomatic for a long period of time. *Helicobacter pylori* infection can cause inflammation of the gastric mucosa hence resulting into various diseases like peptic ulcer. Longer colonization of the bacterium can also lead to development of gastric cancer. Such morbidity can lead to poor life satisfaction and reduced quality of life which may further result in severe suffering and death. Somalia being one of the countries in sub-Saharan Africa is characterized by poverty, poor sanitation and high population density. Preventive strategies for *H. pylori* transmission would be challenging. Somalia has also been reported to have a high fecundity rate which results into high numbers of children households. Because of this, it is anticipated that *H. pylori* infection prevalence may be high.

1.3 Justification

There is a lack of recent local studies conducted in Somalia to provide updated insights into the prevalence of *H. pylori* and its associated factors. The study conducted by Omar and Abdirahman took place in 2016, making it crucial to obtain the latest findings regarding the prevalence of *H. pylori*. Before the fall of Siad-Barre regime in the 1990s, hospitals were running, although there are no proper records that one can rely on regarding *H. pylori* epidemiology and infection burden.

Helicobacter pylori is a ubiquitous organism that is believed to have infected about 50% of the global population, posing a public health concern around the world (Peleteiro *et al.*, 2014). Results from the present study, which seeks to determine the prevalence *H. pylori* in Mogadishu and its associated factors may provide thebasis for designing, revising, or implementing public health policies to ensure proper prevention and control, detection, treatment, andmanagement of *H. pylori* infections.

1.4 Research Questions

i. What is the prevalence of *H. pylori* infection among patients with dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia?

ii. What are the risk factors associated with *H. pylori* infection among patients with dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia?

1.5 Study Objectives

1.5.1 General Objective

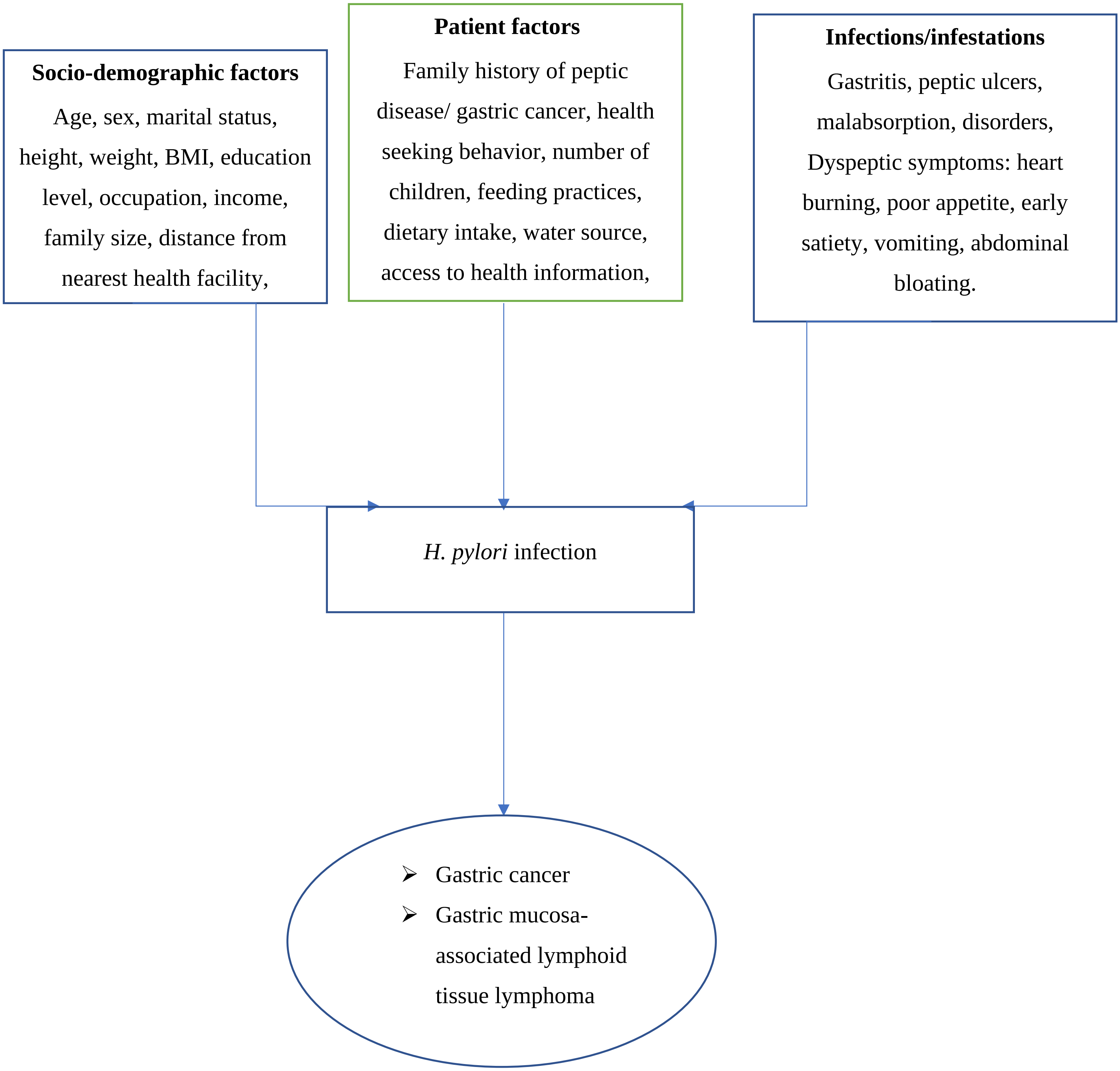
To determine the prevalence and risk factors associated with *H. pylori* infection among patients with dyspepsia attending Kalkaal Hospital, Somalia.

1.5.2 Specific Objectives

i. To determine the prevalence of *H. pylori* infection among patients with dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia

ii. To establish the risk factors associated with *H. pylori* infection among patients with dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia

1.6 Conceptual Framework



1.7 Explanation ofthe conceptual framework and study scope

High prevalence of *H. pylori* has been reported in many studies in developing countries, including the sub-Saharan African region. *Helicobacter pylori* infection is reported to occur usually in early childhood in Africa and may persist for years if not treated. This may consequently lead to severe gastrointestinal disorders, cancer, or even death. The dependent variable in this study will be *H. pylori* infection while the independent variable will be the predisposing characteristics ranging from socio-demographic characteristics, to patient characteristics and other infections or infestations. *Helicobacter pylori* is considered the causative agent of peptic ulcers, gastritis, and malabsorption. The majority of patients develop dyspepsia.

CHAPTER TWO: LITERATURE REVIEW

2.1 Burden of *H. pylori*

Helicobacter pylori was first identified in Western Australia by Robin J warren in 1979 while examining biopsy from patients with gastritis (Rubin, 2008). It is a colonizer in up to 50% of the population worldwide (Dube *et al.*, 2009). According to WHO 1994, *H. pylori* was categorized as a first class carcinogen (Mitchell & pylori, 1999). Over the years, epidemiological studies have linked *H. pylori* infections with gastric and peptic cancer (Leja *et al.*, 2019; Li *et al.*, 2010). Different variations in *H. pylori* infection prevalence worldwide have been registered (Peleteiro *et al.*, 2014). These differences are thought to be associated with the uniqueness of the environments, host genetics, circulating bacterial strains and socioeconomic status of the population (Segal *et al.*, 2001). This is also reflected in high distribution of associated disease outcomes such asgastric cancer especially *H. pylori* endemic regions (Peleteiro et al., 2014).

High rate of *H. pylori* infections are reportedly found in East and South Asia (Yamaoka & Infection, 2009) with high sero-prevalence (63.4%) in rural areas among the aborigines in Taiwan who have been reported to have high rate of gastric cancer (Yim *et al.*, 2007). A recent study in Asia reported high prevalence of infection ranging from 54% to 76% (Leja *et al.*, 2019) though previously, a lower prevalence had been reported in India (58% to 62%) (Adlekha *et al.*, 2013) and Kazakhstan (76.5%)(Benberin *et al.*, 2013) indicating a rising trend in prevalence over years.

Infection by *H. pylori* among children is generally rare before the age of 10 in the European populations (Salih, 2009). For instance, in the US, the number of new *H. pylori* cases among children aged less than five yearsis below 5% while just about 10 in every 100 children are infected by the time they become adolescents (Muhammad *et al.*, 2017). Albeit, *H. pylori* infection among children in developing countries is reported to be about 50% (Muhammad et al., 2017)

Despite the alarming rate of *H. pylori* infection in developing nations, only a small percentage of the population suffer from severe form of the disease (Correa *et al.*, 2011). The contributing factors such ashost genetic predisposition, gene regulation, environmental factors have not been studied (Hooi *et al.*, 2017). In China a sero-prevalence of 63.4% was reported amongst healthy individuals aged between 30 and 69 years (Zhu *et al.*, 2014). Whereas in Saudi Arabia, much lower prevalence of about 28% was revealed in a study carried out amongst healthy individuals (Hanafi & Mohamed, 2013).

In South and Eastern Europe, *H. pylori* prevalence is estimated to be well above 50% compared to the countries found in the northern part where lower prevalence were reported (Leja *et al.*, 2019). But in Portugal, high *H. pylori* prevalence was reported with an infection rate of 84.2% amongst the general population (Bastos *et al.*, 2013). The high prevalence of *H. pylori* among the Portuguese suggest that unknown factors could be implicated in the spread of the pathogen hence the need for more study.

Prevalence of *H. pylori* infection is reported to be on the decrease in the western countries (25 - 50%) in the recent past (Leja *et al.*, 2019). For example in urban Ontario, about 23.1% prevalence was reported amongst adults (Naja *et al.*, 2007b) and much lower prevalence in Canada (Sethi *et al.*, 2013). This could have been due to the extensive preventive measures put in place to eliminate *H. pylori* infection and improved living standards.

In sub-Saharan Africa, infection rates of 61-100% in the population have been reported (Holcombe, 1992). For instance, sub-Saharan African countries such as Morocco and Ethiopia have 75.5% and 65.7% respective *H. pylori* infection rates (Benajah *et al.*, 2013; Mathewos *et al.*, 2013). In Nigeria, 86% prevalence was reported in patients with dyspepsia (Olokoba *et al.*, 2013). Similarly a study carried out by Cover TL and colleagues in West Africa registered high incidence rate (75.0%) of *H. pylori* infection amongst Ghanaian dyspeptic patients (Archampong *et al.*, 2017). This may suggest that *H. pylori* infection is endemic in some or many regions of Africa.

Likewise, among the East African countries, *H. pylori* prevalence has remained high. In the Rwandese population, Walker and colleagues reported a 75.3% prevalence (Walker *et al.*, 2014). Meanwhile in the neighboring Kenya, high incidence was also reported amongst both dyspeptic (71%)and asymptomatic (51%) patients from the same population (Kimang'a *et al.*, 2010). On the other hand, studies carried out in Northern Tanzania by Ayana SM and colleague (2014) showed *H. pylori* prevalence at 57% amongst patients with upper gastro-intestinal disorder.

The occurrence of *H. pylori* varies across different geographical regions; within a nation, divergent prevalence rates may be observed based on factors such as age, educational level, dietary habits, and location (Smith *et al.*, 2022).

In northern Nigeria, the prevalence of *H. pylori* has been documented at 87.8%, in southeast Nigeria at 34.2%, and in southern Nigeria at 51.4% (Adedoyin & David, 2020). These rates have been linked to risk factors such as low economic status, unclear water sources, overcrowding, and cigarette smoking (Bello *et al.*, 2018; Chukwuma *et al.*, 2020; Daniyan Olapeju *et al.*, 2020). Burundi reported a prevalence rate of 70.8% (Ntagirabiri *et al.*, 2014), Rwanda at 75% (Walker *et al.*, 2014), Togo at 70.4% (Lawson–Ananissoh*et al.*, 2015), Congo Brazzaville at 93.1% (Bossali *et al.*, 2017), Morocco at 63.8% (Sokpon *et al.*, 2016), Ghana at 88% (Awuku *et al.*, 2017), and Egypt at 66.1% (Galal *et al.*, 2019). The Republic of Benin reported a prevalence rate of 71.5%, which was not associated with age, sex, marital status, religion, occupation, or education (Kpossou *et al.*, 2020). In Cameroon, a prevalence of 73.2% was significantly associated with factors such as age, socioeconomic status, alcohol consumption, family history, and nonsteroidal anti-inflammatory drugs. Additionally, common conditions like anaemia, duodenal ulcer, and chronic gastritis have been reported in patients with *H. pylori* infection (Bertrant *et al.*, 2020). Algeria reported a prevalence of 71.43% (Kasmi *et al.*, 2020), while in Ethiopia, the prevalence was 88.9% among males and 82.8% among females (Asfaw, 2018).

The reported prevalence of *H. pylori* in Europe (Germany) in 2018 ranged between 20-40% (Fischbach & Malfertheiner, 2018), North America at 23.1% (Naja *et al.*, 2007a), Australia at 24.6% (Hooi *et al.*, 2017), and Asia at 48.8% (Nguyen, 2017; Venneman *et al.*, 2018). It is noteworthy that the rates reported in Africa were higher than those reported in other continents.

With all the published data that have been reported from different parts of the world, there are few representative studies on the prevalence of *H. pylori* carried out in Somalia. One study indicated a prevalence of 42.7%in a medical outpatient unit at Aden Abdulle Hospital in Mogadishu (Omar Hussein, 2016). In the neighboring Ethiopia, the prevalence of *H. pylori* infection range from 7.7% (Ayele & Molla, 2017) to 91% (Asrat *et al.*, 2004). A systematic review including a total of 37 studies reported a prevalence of *H. pylori* infection as being 52.2% with Somalia having the highest prevalence (71%)(Melese *et al.*, 2019).

2.2 Risk Factors for *H. pylori*

Hollander and colleagues found in their study that an individual's ethnic group strongly predicts *H. pylori* infection in pregnant women (den Hollander *et al.*, 2013). Similarly Nguyen T. and colleagues reported high prevalence (53.3%) in black males, 48.1% in Hispanic males and lowest (8.2%) in non-Hispanic white males (Nguyen *et al.*, 2015). The study indicated that genetic predisposition might play a critical role in acquiring infection irrespective of the socioeconomic status as earlier on reported.

Whereas evidence has shown that most *H. pylori* infection is acquired when one is a child, the infection varies across the young population globally. Among the European population *H. pylori* infection is reported to be rare before 10 years and this gradually increases with advance in age (Pounder *et al.*, 1995). However in countries where *H. pylori* prevalence is high in the general population like in Iran and Portugal, high incidence rate have also been reported in children (Peleteiro *et al.*, 2014).

The trend completely changes when looking at a similar situation in developing countries especially Africa, where more than half of the children are reported to be infected by the age of 10 years. A study conducted in

South-western Uganda showed that 24.3% of the children between the ages of 1-15 were infected and that infection prevalence increased with increase in ageas observed in children aged 6 to 10 who recorded11% higher prevalence compared to their 1 to 5 years counterparts (Aitila *et al.*, 2019).

A study conducted amongst the Peruvian patients reported a significantly lower age-specific rate of infection amongst Peruvian women from higher socioeconomic backgrounds than men from both high and low socioeconomic status (Hunt & Tytgat, 2012). On the other hand, Ozaydin and colleagues reported high prevalence of *H. pylori* infection amongst men than in women (Ozaydin *et al.*, 2013). Interestingly, a comprehensive meta-analysis also indicated a higher prevalence in males compared to females (Hooi *et al.*, 2017). Other risk factors have been published in different studies for example, asystematic review including 37 studies reported that patients who presented with gastrointestinal infection (GIT) symptoms were more at risk of infection when compared to those without any symptoms (OR = 2.23; 95% CI: 1.59–3.14)(Melese *et al.*, 2019). Moreover, alcohol use and abuse behavior have as well been linked to *H. pylori* infection risk (Melese *et al.*, 2019; Zhang *et al.*, 2010). Poor hygiene, living in poor sanitary conditions and consumption of unsafe food and water haveto a great extent been reported to predispose the population to *H. pylori* infection (Hunt *et al.*, 2011; Melese *et al.*, 2019).

2.3 Transmission of *Helicobacter pylori* Infection

Humans are considered to be the main reservoir hosts of *H. pylori* although *H. pylori*-like organisms have been isolated from several other non-human species such as pigs, macaques' monkeys and cats (Taylor *et al.*, 1995). Nevertheless, there is no evidence of zoonotic transmission that has been reported.

The modes of transmission still remain poorly understood with no single route clearly defined. Nonetheless, the fecal–oral route is considered to be the most common route. This has been supported by study reports that associated focally contaminated water with *H. pylori* infection (Aziz *et al.*, 2015). In another study, Yvonne and colleagues in Chile found that individuals who ate raw vegetables which were grown by irrigation were more at risk for infection. In the study, it was found that the water used for the irrigation had been contaminated with untreated sewage (van Duynhoven & Jonge, 2001).

Oral–oral route of transmission has also been suggested.This is seemingly supported by a study done in Accra Ghana by Richard H. Asmah and colleagues where *H. pylori* DNA was amplified in 90% of saliva samples and a 33.3% *H. pylori* prevalence in saliva of dyspeptic patients (Asmah *et al.*, 2014). Other study findings indicated high incidence amongst some ethnic groups who pre-masticate food, share spoons between mothers and children and mouth to mouth intimacy (Mégraud, 1995).

Person-to-person contact has been suggested as a likely transmission route. Koffi and colleagues noted in their study that spouses and children of infected individuals were more frequently sero-positive than those of non-infected individuals (Mégraud, 1995). Similarly, Urita and colleagues in Japan confirmed mother-to-child and guardian-to-child as a vehicle for *H. pylori* infection transmission (Urita *et al.*, 2013).

2.4 Pathogenesis of *H. pylori*

*Helicobacter pylori i*sthought to be the commonest cause of a number of gastroduodenal complications across the globe. Such infections have been documented to exist from the gastric antrum extending till the corpus following the damage of the mucosa (Akada *et al.*, 2003). Other conditions associated with *H. pylori* include gastritis autoimmune and hypersensitivity reactions. Abeit, such gastritis results from bacterial colonization of the stomach which consequently results into inflammation of gastric mucosa (which extends downwards from the corpus till the mucosa) due to irritation by bacterial endotoxins. It can be acute or chronic gastritis. A number of transient, nonspecific dyspeptic symptoms have been reported to accompany acute gastritis, and these include vomiting,abdominal fullness, and nausea, which may be coupled with the mucosa of the proximal and distal stomachs being inflamed. In more severe conditions, there is bleeding with hematemesis. Gastritis that occurs acutely most times limits itself and is followed by the mucosa completely regenerating itself and being healed just after few days (Tanih *et al.*, 2010).
An inflamed mucosa that is as result of chronic occurrence of *H. pylori* is thought to cause structural deformation ofthe gastric gland which eventually becomes fibrotic. In about half of *H. pylori* infected individuals, studies have indicated that atrophic gastritis associated severe inflammation occurs along with intestinal metaplasia. Tsukanov and colleagues reported that there's an increased gastric cancer risk brought about by an asymptomatic gradual increase in gland loss areas along with metaplasia of the intestinal tract (Tsukanov *et al.*, 2013). Several factors inclusive of the nature of the colonizing strain, the host's gene characteristics, immune response, the host's diet and amount of acid produced have been said to predict how severe the chronic inflammation process can be (Makola *et al.*, 2007).

Helicobacter pylori related mucosal inflammation causes damage to the mucous coating, exposing sensitive stomach lining to stomach acid. As such, the commonest peptic ulcersymptom isusually navel pain as well as around the area of the breastbone, and this happens due to stomach emptiness. When the host has eaten some food and ingested some antacids, there is a significant reduction in pain (Momtaz *et al.*, 2012). Lymphoma pathogen is of the mucosa and is a complex process that includes many gene alterations that result in cancer appearance. It's characterized by a slow multiplication of B lymphocyte cells in the stomach lining due to constant stimulation of the immune system by *H. pylori* infection or an autoimmune process (O'Rourke, 2008).

2.5 *Helicobacter pylori*related Gastric Cancer

Like previously mentioned in this chapter, the risk of gastric cancer is 10 times more among patients who develop the *H. pylori* infection (Hooi *et al.*, 2017). However it takes a series of decades from acquiring *H. pylori* infection to progression to gastric cancer. Some literature has suggested that whereas the *H. pylori* usually occur in childhood, gastric cancer may develop in adulthood. It's suggested that *H. pylori* infection plays an important role during the initiation steps of gastric cancer development (Asaka *et al.*, 2001).

2.6 Diagnosis and Treatment of *Helicobacter pylori*

2.6.1 Diagnosis

Diagnostic tests for *Helicobacter pylori* comprise of invasive (endoscopic) and non-invasive (non-endoscopic) methods. The invasive method involves endoscopic removal of a gastric biopsy from the upper gastrointestinal tract. Other diagnosis methods are histology, rapid urease test, culture and urea breath testing.In routine practice, culture, urea breath testing and histology are the common tests performed. On the other hand, patients undergoing care in hospitals are subjected to endoscopy and the biopsy obtained is subjected to histological testing to demonstrate in histopathological changes of the gastric mucosa that would reveal the different stagesof cancer if present.

Rapid urease test on the hand is the most widely used biopsy-based test. *Helicobacter pylori* is a strong urease enzyme producer and this forms the basic principle of the rapid urease test. The test is considered positive when the test reagent turns from yellow to a red color (Uotani & Graham, 2015).

Bacterial culture is also frequently used in the diagnosis of *H. pylori* infection. However, culture of *H. pylori* bacteria is not commonly done due to the high cost and the fastidious nature of this pathogen (Patel *et al.*, 2014).

Other non-invasive tests include serology, urea breath tests and *H. pylori* stool antigen which are in common practice. Serological tests actas a guide for primary care health workers when making treatment related decisions,although the reliability of the test is questionable. On the other hand,urea breath test requires that the patient drinks urea solution containing labelled carbon atom (13^{C}) which is later detected in the patient's breathing cases of *H. pylori* infection. Stool is recommended sample particularly when investigating for an active infection, determining treatment outcome and for children who may not undergo endoscopy or vene puncture (Iranikhah *et al.*, 2013).

2.6.2 *H. pylori* Treatment

In recent years, first line treatment for *H. pylori* infection fell below the recommended 80% threshold (Udoh & Obaseki, 2012). Non-compliance and evolution of *H. pylori* resistant strains are thought to be the major contributing factors to treatment failures (Graham *et al.*, 1991). As such, standard empirical triple therapy can now be used only in the parts of the world that have not experienced resistance to clarithromycin or where the resistance occurs in just less than 15% of the population. Parts that have high known clarithromycin resistance rates are recommended to use bismuth quadruple therapy (McColl, 2010).

2.7 Vaccines for *H. pylori*

Vaccination would reduce *H. pylori* associated complication and enhance control and eradication of the disease. Conversely, there are no vaccines against *H. pylori* available in the market. Vaccine candidates under clinical trial are in Phase I and Phase III (Sutton & Boag, 2019). Another randomized trial in China of an oral recombinant *H. pylori* vaccine is ongoing (Zeng *et al.*, 2015).

CHAPTER THREE: METHODS

3.1 Study Site

The present study was conducted at Kalkaal Hospital, Somalia. The hospital is located in the Hodan district of Mogadishu. It is a referral hospital that admits an average of 500 patients per day. The hospital offers all kinds of medical services, including inpatient and outpatient. It also has a wide range of specialists, plus the medical microbiology department, Infectious diseases department and a fully equipped laboratory are available for routine diagnostic services.

3.2 Study Design

This study was a cross-sectional study that utilized quantitative data collection methods.

3.3 Study Population

All patients who were present at Kalkaal Hospital, Somalia, presenting with dyspepsia complaints during the study periodand who met the eligibility criteria were included in this study.

3.4 Eligibility Criteria

3.4.1 Inclusion Criteria

- Inpatients and outpatients who sought health services at Kalkaal Hospital, Somalia,during the study period
- Presenting with dyspepsia
- Patients who provided informed consent or assent
- Patients above 6 months

3.4.2 Exclusion Criteria

- Patients who were unable to adhere to study procedures such as not being able to respond to questions in the questionnaire.
- Patients who were not able to provide a fresh stool sample.
- Patients on treatment of *H. pylori* infection, in less than seven days to this study

3.5 Sample Size Estimation

The formula for cross-sectional studies by Fishers et al. *(1998)* was used to determine the sample size for objective one:

$$n = \frac{Z^2_{\propto/2}\, P(1-P)}{d^2}$$

Where:

n= the required sample size

$Z^2_{\propto/2}$ is the standard normal value corresponding to the 95% level of confidence (1.96)

ZB

d (tolerable sampling error (precision) is 0.05

P is the proportion of patients with *H. pylori* infection. Using the results from the study conducted by Omar Hussein at Aden Abdulle Hospital in 2016, P = 0.43 (42.7% prevalence) (Omar Hussein, 2016). On substitution in the formula, a P of 0.43 yields a sample size of 376.6 participants. On adjusting for a 10% non-response rate:

Adjusted sample size = 414patients.

3.6 Sampling Method

Systematic sampling was used in this study. The sample size (*n* =414) of the target population was patients who had dyspepsia. It was estimated that 20 out of 500 patients seek medical services each day at Kalkaal Hospital had dyspepsia. With the assumption that the hospital operated 5 days a week for 2 months (which is approximately 8 weeks), the total number of patients with dyspepsia that were expected to be available during the study period was: 20 patients/day x 5 days/week x 8 weeks = 800 patients. Thus, with the desired sample size is 414; systematic sampling was used to select the representative sample. Since it was anticipated that 800 patients with dyspepsia would visit the hospital for eight weeks, the sampling interval was: 800 patients / 414 sample size = 1.93 (rounded to 2). Therefore, every second patient with dyspepsia was given a chance to be included in the study sample.

3.7 Study Variables

3.7.1 Dependent Variable

The dependent/outcome variable of the study was*H. pylori* infection. The *H. pylori* infection was based on a positive stool antigen test result.

3.7.2 Independent Variables

Information on socio-demographic characteristics, patient characteristics, and clinical characteristics was obtained. Socio-demographic characteristics, which included: age, marital status determined as either married or not married, level of education, employment status determinedas not working, farming, professional or manual labor, and number of living children. Social-economic factors included wealth status, mass media exposure and distance to the health facility,was estimated depending on whether patients coveredless than 5km or more to the hospital. Clinical characteristics included signs and symptoms such as malabsorption disorders, dyspeptic symptoms, heartburn, poor appetite, early satiety, vomiting, abdominal bloating, and a family history of peptic disease or gastric cancer.

3.8 Data Collection

The questionnaire was translated into the local Somali language and underwent pretesting. Data collection spanned a period of eight weeks. When selecting a participant, either the principal investigator or the research assistant introduced themselves, clarified the study's objective, and guided the participants through the information sheet. Following this, participants were given written informed consent and assent forms and asked to provide a fresh stool specimen before proceeding to answer the questionnaire. The interview itself lasted between 15 and 25 minutes.

3.8.1 Study Tool

A questionnaire was developed for data collection. The questionnaire contained sections on social demographic characteristics, patient factors and clinical factors. (Appendix 1)

3.8.2 Laboratory Investigation

The One-Step *H. pylori* stool antigen test was used for to detect infection with *H. pylori*.

Sample collection: To perform the test the patient picked up a collection container and instruction from the doctor's office. Patients collected stool samples at home and delivered the same to the lab within 24hrs. Stool samples were not to be less than 1 gram to obtain maximum antigens (if present).

Sample processing: The *Helicobacter pylori* antigen rapid test kit (stool) has been developed as a visual immunoassay with rapid results. Its primary function is to detect the presence of *Helicobacter pylori* antigen in human stool specimens, aiding in the diagnosis of *H. pylori* infection. This test kit utilizes an enzyme immunoassay to detect *H. pylori* antigens in stool samples. To ensure optimal performance, the test kit should be stored and maintained at temperatures between 15-30 degrees Celsius prior to testing.

For solid specimen: The solid stool was randomly stabbed at least at three different sites to collect approximately 50 mg of stool.For the **liquid specimen**: Two drops of 80ul of stool was transferred to the specimen collection tube. The cap was tightened securely and shaken vigorously to mix the specimen. The tube was left to settle uninterrupted for 2 minutes. This was followed by labeling the kit with the patient's information. Afterwards, 2-3 full drops of the extracted specimen were transferred to the specimen well of the test cassette. Results were recorded afterten minutes

The stool sample diffused through the nitrocellulose membrane by capillary action across immobilized *H. pylori* specific antibodies in the column. In the presence of *H. pylori* an antigen-antibody complex is formed, by capillary action, moves onto the nitrocellulose membrane towards the test line region on which the *H. pylori* specific antibodies become immobilized. A second control red line was also expected to appear in the result window indicating correct performance of the test Kit. In cases of absence of *H. pylori* antigen in the test samples then only the control line appeared in the test line region, A positive result was evidenced by development of two lines and negative results by a single control line.

3.9 Quality Assurance and Control

Experienced research assistants (RA) were selected on their basis of the qualifications and competences. Formal training was offered to these RA before being deployed to gather information from the field.The data collection tool was pre-tested among the first 30 participants. Where necessary, adjustments were effected. The data collected during the pre-testing of the data collection tool was not included in the final analysis. Several laboratory quality control procedures were ensured to obtain reproducible findings. The laboratory procedures were run by two experienced laboratory technicians who were selected depending on their previous experience in using the test kit. The tests were run following General Laboratory Practice guidelines and according to the manufacturer's instructions. The precautions ranged from the timing of the test, to the required temperature and procedures on sample processing.

3.10 Data Management

Data was collected using Open Data Kit (ODK). Data was cleaned and stored in password protected data base. Backup of stored data was available using flash discs. Only the study team could access documents in their hard copy form. Following data collection, excel file containing the data was downloaded from ODK and then transferredto R Language version4.2.2 for analysis.

3.11 Data Analysis

Descriptive statistics were computed for the socio-demographic, patient and clinical characteristics computed. Descriptive statistics were adopted foranalyzing continuous variables and presented incharts. Categorical variables were presented as frequencies and percentages. Cross tabulations and chi statistics were conducted to establish if there were any significant measures of associations between the dependent variable (*H. pylori*) and the independent variables. Any independent variable which recorded significant measures of associations with the dependent variable were later on subjected to multivariate analysis using hierarchical binary logistic regression.

For objective one, the frequencies and respective percentages of *H. pylori* infection were computed. The prevalence was determined by computing the proportion of patients who had positive *H. pylori* test out of all the patients included in the study. This prevalence was presented along with its 95% confidence interval. Differences between levels of categorical variables were explored using chi-square tests.

Objective two aimed to identify potentialrisk factors associated with the study participants. These factors encompassed individual characteristics, dietary patterns, lifestyle choices, history of digestive diseases, *H. pylori* test results, and general symptoms related to *H. pylori* infection.

Bivariate analysis was performed by comparing each independent variable with *H. pylori* infection. To ensure that the final model does not suffer from overfitting issues, only variables with chi-statistic significant values less than 0.05 and 0% of the expected cells having values less than 5 were included in the final logistic model, and vice versa. Probability values of < 0.05 were considered to be significant.

The data analysis plan that was used in the current study is presented in the table below:

Objectives	**Dependent outcome variable**	**Statistical procedure**	**Outcome of statistical measure**
To determine the prevalence of *H. pylori* infection among patients with dyspepsia attending Kalkaal Hospital, Somalia	Test Results for *H. pylori*	2-way cross-tabulation Prevalence = No. of cases/population size (sample size)	Odds ratio and Relative Risk.
To establish the risk factors associated with *H. pylori* infection among patients with dyspepsia Kalkaal Hospital, Somalia	The interaction of the predictors (socio-demographic, patient and clinical characteristic) on the outcome (*H. pylori*).	Chi-square statistic	The proportion of socio-demographics and *H. pylori* measured using significant *p*-values
		Hierarchical binary logistic and probit regression using maximum likelihood estimation	The odds and adjusted odds ratios are estimated at both 95% and 99% confidence levels.

3.12 Ethical Considerations

Ethical approval was sought form KNH-UoN ERC and Ministry of Health Somalia was allocated approval (P909/11/2021). The participants were not under any obligation to reveal their names on the questionnaire. Clarification of the study purpose to the participants was made. All the information reviewed was acknowledged and cited to counter plagiarism.

CHAPTER FOUR: RESULTS

4.0 Introduction

Evidence after processing data is presented in this chapter as per the following objectives; 1.To determine the prevalence of *H. pylori* infection and 2. To establish the risk factors associated with *H. pylori* infection among patients presenting with dyspepsia at Kalkaal Hospital in Mogadishu, Somalia. Between 20th January and 20th March 2023, 417 patients who met the eligibility criteria were enrolled in the study. A research assistant administered the questionnaire immediately after enrollment. To test for *H. pylori* infection, study participants were required to collect the stool samples at home and deliver to the hospital laboratory within 24hrs as per the study procedures.

4.1 Response Rate

In this study, the researcher administered questionnaires to all 417 participants recruited. However, for the questionnaire to be complete, *H. pylori* test results were required, as it was the main outcome of the study. Four (4) study participants failed to return stool samples to the laboratory for test of *H. pylori* infection and were therefore excluded from the analysis of the study objectives. This represents a response rate of 99.0%, which is strongly acceptable, as shown in Table 1

Table1: Response rate

Questionnaires	Number	Percentage
Complete	413	99.0
Incomplete	4	1.0
Total	**417**	**100**

4.2 Characteristics of Study participants

Of the 413 respondents studied, 54.7% were male. More than three quarters of the study participants were adults 78.3% while Children (>6month-12 years) at 9.9%. Most of the study participants had a tertiary level of education at 50.7% while those with the least percent (5.1%) had a primary level of education. The study had measured the weight and the heights of the study participants in terms of kilograms and meters respectively. The two attributes were converted into the body mass index (BMI). Most of the participants (44.1%) had normal BMI with the underweight representing 4.8%. The results also indicated that most of the study participants were married 47.3% while 2.9 percent were widowed.

The dietary intake of the study participants was investigated with the intention of trying to link the consumption of certain food with *H. pylori* infection. Foods consumed mostly once a day, included: vegetables (45.3%), fruits (41.5%), beans (42.0%), onions and garlic (42.6%), pickled foods (43.3%), fried foods (44.9%) and spicy foods (41.0%). Eggs, meat and milk were also consumed by the majority of the participants (44.2%). More descriptives on dietary consumption are shown in Table 2.The participant's lifestyle was estimated by asking if they were smokers (12.7%), drunk bottled water (52.6%) or had a family history of peptic disease/gastric cancer (26.3%). Additional characteristics of study participants are show in Table 2.

Table 2: Characteristics of study participants

Variables	Categories	Frequency
Demographics		
Age (*n*=412)	Child (>6months-12 years)	41(9.9%)
	Adolescence (13-18 years)	18(4.4%)
	Adult (19-59 years)	323(78.3%)
	Senior Adult (60 years & above)	30(7.3%)
Gender (*n*=409)	Female	183(44.3%)
	Male	226(54.7%)
BMI Adult & Senior Adults (*n*=367)	Underweight	17 (4.6%)
	Normal weight	162 (44.1%)
	Overweight	111 (30.2%)
	Obese	62 (16.8%)
	Morbidly obese	15 (4.1%)
Education level (*n*=412)	No formal education	70 (17%)
	Primary	21 (5.1%)
	Secondary	112 (27.2%)
	Tertiary	209 (50.7%)
Marital status (*n*=409)	Married/living together	194 (47.3%)
	Divorced/separated	32 (7.8%)
	Widowed	12 (2.9%)
	Never married	171 (42%)
Have children (*n*=412)	No	194 (47.1%)
	Yes	218 (52.9%)
Residence (*n*=412)	Rural	80 (19.4%)
	Urban	332 (80.6%)
Employed (*n*=413)	No	229 (55.4%)
	Yes	184 (44.6%)
Life style		

Smoking (*n*=410)	No	358 (87.3%)
	Yes	52 (12.7%)
Drink bottled water (*n*=411)	No	195 (47.4%)
	Yes	216 (52.6%)
***H. pylori* general symptoms**		
Nausea with vomiting (*n*=413)	No	224(54.2%)
	Yes	189(45.8%)
Heartburn (*n*=413)	No	180(43.6%)
	Yes	233(56.4%)
Diatery intake		
Beans (n=412)	Every other day once a day	152 (36.9%)
	Every other day	87 (21.1%)
	Once a day	173 (42%)
Fried foods (n=412)	Every other day once a day	141 (34.2%)
	Every other day	86 (20.9%)
	Once a day	185 (44.9%)
Fruits (n=412)	Every other day once a day	155 (37.6%)
	Every other day	86 (20.9%)
	Once a day	171 (41.5%)
Milk, eggs and meat (n=412)	Every other day once a day	182 (44.2%)
	Every other day	67 (16.3%)
	Once a day	163 (39.6%)
Onions &garlic (n=413)	Every other day once a day	151 (36.6%)
	Every other day	86 (20.8%)
	Once a day	176 (42.6%)
Pickled Foods (n=413)	Every other day once a day	147 (35.6%)
	Every other day	87 (21.1%)
	Once a day	179 (43.3%)
Spicy Foods (n=412)	Every other day once a day	145 (35.2%)
	Every other day	98 (23.8%)
	Once a day	169 (41%)
Vegetables (n=411)	Every other day once a day	130 (31.6%)
	Every other day	95 (23.1%)
	Once a day	186 (45.3%)
History of digestive disease		
Gastroenteritis (n=412)	No	269 (65.3%)
	Yes	143 (34.7%)
Hepatitis (n=412)	No	313 (76%)
	Yes	99 (24%)
Esophagitis (n=413)	No	308 (74.6%)
	Yes	105 (25.4%)
Peptic ulcers (n=411)	No	268 (65.2%)

	Yes	143 (34.8%)
Peptic / gastric cancer (n=410)	No	302 (73.7%)
	Yes	108 (26.3%)

4.3 Prevalence of *H. pylori*

Figure 1 shows the prevalence of *H. pylori* among patients who presented with dyspepsia at Kalkaal hospital in Mogadishu Somalia. Out of the 413 study participants, 232 (56.2%) tested positive while 181(43.8%) tested negative.

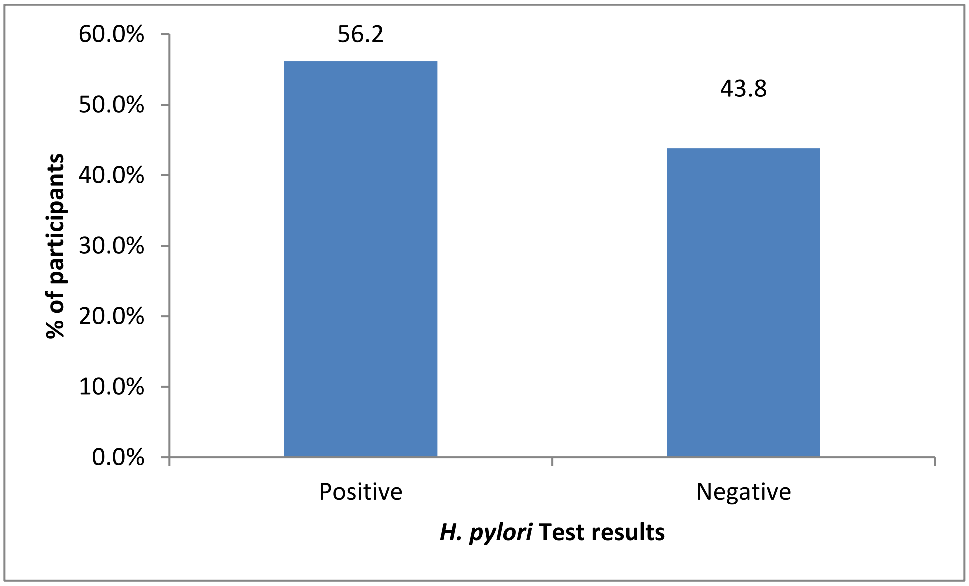


Figure 1: Prevalence of *H. pylori*

4.4 Risk factors associated with *H. pylori* infection

4.4.1 Univariable analysis

Table 3 shows chi-squaretest of association between socio-demographics, patient symptoms, history of digestive diseases, dietary intake and life style characteristics and *H. pylori* infection status among patients presenting with dyspepsia. Socio-demographic characteristics, including age (p=0.002), marital status (p < 0.001), whether has children (p < 0.001), residence (p = 0.005) and whether in employment (p = 0.003), were found to be significantly associated with *H. pylori* infection status at p < 0.05.

With regard to patient symptoms, life style and dietary intake, characteristics that were found to be associated with *H. pylori* infection at p < 0.05 included complaint of nausea with vomiting (p < 0.001), complaint of heartburn (p < 0.001), smoking status (p =0.001), use of bottled water for drinking (p <0.001), family history of peptic disease/gastric cancer (p < 0.001), frequency of eating pickled foods (p =0.008), and frequency of eating spicy foods (p < 0.001).

The study also investigated patient history of digestive disease and *H. pylori* infection. All characteristics of interest that included history of gastroenteritis ($p < 0.001$), peptic ulcers ($p < 0.001$), esophagitis ($p < 0.001$), and hepatitis ($p < 0.001$), were highly associated with status of *H. pylori* infection.

Table 3: Association between patient characteristics and *H. pylori* infection

Characteristics	Category	*H.pylori* positive	*H.pylori n*egative	*p-value*
Demographics				
Age (n=412)	Child (>6months-12 years)	13 (3.1%)	28 (6.8%)	0.002
	Adolescence (13-18 years)	10 (2.4%)	8 (1.9%)	
	Adult (19-59 years)	185 (45%)	138 (33.4%)	
	Senior Adult (60 years & above)	23 (5.6%)	7 (1.7%)	
Gender (n=409)	Male	125 (30.6%)	101 (24.7%)	0.675
	Female	105 (25.7%)	78 (19.1%)	
BMI status (n=367)	Underweight	11 (3%)	6 (1.6%)	0.001
	Normal weight	79 (21.5%)	83 (22.6%)	
	Overweight	71 (19.3%	40 (10.9%)	
	Obese	47 (12.8%)	15 (4.1%)	
	Morbidity Obese	6 (1.6%)	9 (2.5%)	
Education (n=412)	No formal education	37 (9%)	33 (8%)	0.525
	Primary	9 (2.2%)	12 (2.9%)	
	Secondary	63 (15.3%)	49 (11.9%)	
	Tertiary	122 (29.6%)	87 (21.1%)	
Marital status (n=409)	Married/living together	122 (29.8%)	72 (17.6%)	< 0.001
	Divorced/separated	25 (6.1%)	7 (1.7%)	
	Widowed	9 (2.2%)	3 (0.7%)	
	Never married	76 (18.5%)	95 (23.4%)	
Children (n=412)	Yes	145 (35.2%)	73 (17.7%)	< 0.001
	No	87 (21.1%)	107 (26%)	
Residence (n=412)	Urban	175 (42.5%)	157 (38.1%)	0.005
	Rural	56 (13.6%)	24 (5.8%)	
Employment (n=413)	Yes	118 (28.6%)	66 (16%)	0.003
	No	114 (27.6%)	115 (27.8%)	
Lifes style				
Smoking (n=410)	Yes	40 (9.7%)	12 (2.9%)	0.001
	No	190 (46.2%)	168 (41.1%)	
Bottled water (n=411)	Yes	145 (35.3%)	71 (17.3%)	< 0.001
	No	86 (20.9%)	109 (26.5%)	
Diatery Intake				
Pickled foods (n=413)	Everyday other day	46 (11.1%)	41 (9.9%)	0.008
	Every other day-once a day	50 (12.1%)	97 (23.5%)	
	Once a day	85 (20.6%)	94 (22.8%)	
Spicy foods (n=412)	Everyday other day	47 (11.4%)	51 (12.4%)	

	Every other day-once a day	46 (11.2%)	99 (24%)	<0.001
	Once a day	88 (21.4%)	81 (19.7%)	
Beans (n=412)	Every other day	36(8.7%)	51 (12.4%)	0.552
	Every other day-once a day	63 (15.3%)	89 (21.6%)	
	Once a day	81 (19.7%)	92 (22.3%)	
Onion & garlic (n=413)	Every other day	43 (10.4%)	43 (10.4%)	0.190
	Every other day-once a day	58 (14%)	93 (22.5%)	
	Once a day	80 (19.4%)	96 (23.2%)	
Fried foods (n=412)	Every other day	43 (10.4%)	43 (10.4%)	0.143
	Every other day-once a day	53 (12.9%)	88 (21.4%)	
	Once a day	85 (20.6%)	100 (24.3%)	
Vegetables (n=411)	Every other day	41 (10%)	54 (13.1%)	0.046
	Every other day-once a day	46 (11.2%)	84 (20.4%)	
	Once a day	92 (22.4%)	94 (22.9%)	
Fruits (n=412)	Every other day	44 (10.7%)	42 (10.2%)	0.067
	Every other day-once a day	57 (13.8%)	98 (23.8%)	
	Once a day	79 (19.2%)	92 (22.3%)	
Milk, eggs &meat (n=412)	Every other day	26 (6.3%)	41 (10%)	0.298
	Every other day-once a day	76 (18.4%)	106 (25.7%)	
	Once a day	79 (19.2%)	84 (20.8%)	
History of digestive diseases		Positive	Negative	
Gastroenteritis (n=412)	Yes	110 (26.7%)	33 (8%)	< 0.001
	No	122 (29.6%)	147 (35.7%)	
Peptic ulcer (n=411)	Yes	117 (28.5%)	26 (6.3%)	< 0.001
	No	114 (27.7%)	154 (37.5%)	
Esophagitis (n=412)	Yes	79 (19.2%)	20 (4.9%)	< 0.001
	No	153 (37.1%)	160 (38.8%)	
Hepatitis (n=413)	Yes	81 (19.6%)	24 (5.8%)	< 0.001
	No	151 (36.6%)	157 (38%)	
Family history of Peptic disease/gastric cancer (n=410)	Yes	79 (19.3%)	29 (7.1%)	< 0.001
	No	152 (37.1%)	150 (36.4%)	

Patient symptoms				
Nausea with vomiting (n=413)	Yes	165 (40%)	24 (5.8%)	< 0.001
	No	67 (16.2%)	157 (38%)	
Heartburn (n=413)	Yes	200 (48.4%)	33 (8%)	< 0.001
	No	32 (7.7%)	148 (35.8%)	

4.5 Multivariate Logistic Regression

Table 4 shows the results of the binary logistics regression modelling of the individual characteristics with status of *H. pylori* infection among patient presenting with dyspepsia at Kalkaal hospital in Mogadishu, Somalia. From the findings after controlling for other factors; Children (>6months-12years) are 90% (AOR = 0.097; 95% C. I: 0.014-0.688) less likely to have *H. pylori* infection compared to senior adults. Patients who are obese are 4.5 times (AOR = 4.547; 95% C. I: (1.250-16.54) more likely to have the infection. Those who use bottled water are 2.5 times (AOR = 2.532; 95% C. I: 1.686–3.803) more likely to have *H. pylori* infection compared to those who does not. Regarding patients with digestive disease, those with history of peptic ulcers are 3.8 times (AOR = 3.770; 95% C. I: 2.095–6.86) had greater risk of having *H. pylori* as opposed to those who lacked. Those with the history of gastroenteritis are 2.3 times (AOR = 2.282; 95% C. I: 1.350–3.858) had greater risk of having *H. pylori* as opposed to those who lacked. By contrast, there is no relationship between history of gastric cancer and *H. pylori* infection.

Patient smoking status significantly predicted *H. pylori* infection status. Patients who reported to be smoker were 3.1 times (AOR = 3.072; 95% C. I: 1.507–6.264) more likely to test positive for *H. pylori* than the non-smokers. Regarding symptoms that patient presented with, those who complained of nausea with vomitting were almost 6 times (AOR = 5.890; 95% C. I: 3.230–10.430) more likely to be infected with *H. pylori* compared to those who didn’t. Likewise, those who complained of heartburn were 14.1 times (AOR = 14.13; 95% C. I: 7,991–24.92) had greater risk of having *H. pylori* as opposed to those who lacked.

TABLE 4: Risk factors associated with *H. pylori* infection

Characteristic	Category	COR	95% CI (COR)	*p*-value	AOR	95% CI (AOR)	*p*-value
Socio-demographics							
Age	Child (>6months-12 years)	0.116	**(0.038,0.352)**	<0.001	0.097	**(0.014,0.688)**	0.020
	Adolescence (13-18 years)	0.313	(0.086,1.135)	0.077	0.466	(0.090,2.407)	0.362
	Adult (19-59 years)	0.335	**(0.133,0.842)**	0.020	0.420	(0.156,1.135)	0.087
	Senior Adult (60 years & above) ref	1			1		

BMI	Underweight	2.750	(0.655,11.52)	0.167	3.558	(0.731,17.325)	0.116
	Normal weight	1.428	(0.486,4.196)	0.517	1.415	(0.443, 4.522)	0.559
	Overweight	2.662	(0.883,8.025)	0.082	2.126	(0.649, 6.962)	0.213
	Obese	4.700	**(1.437,15.38)**	0.010	4.547	**(1.250,16.54)**	0.022
	Morbidity obese (ref)	1			1		
Marital status	Never married (ref)	1			1		
	Married	2.118	**(1.393,3.221)**	<0.001	0.862	(0.312, 2.382)	0.775
	Divorced	4.464	**(1.832,10.88)**	0.001	1.889	(0.524, 6.810)	0.331
	Widowed	3.750	(0.981,14.34)	0.053	1.879	(0.391, 9.033)	0.431
Children	Yes (ref)	1			1		
	No	0.413	**(0.277,0.616)**	<0.001	0.614	(0.237, 1.589)	0.315
Residence	Urban	1			1		
	Rural	2.184	**(1.284,3.715)**	0.004	1.418	(0.787, 2.557)	0.245
Employment	Yes (ref)	1			1		
	No	0.546	**(0.367,0.813)**	0.003	0.781	(0.466, 1.308)	0.347
Dietary choice							
Vegetables	Once a day (ref)	1			1		
	Every other day	1.275	(0.775,2.098)	0.339	1.357	(0.755, 2.436)	0.307
	Every other day-once a day	1.768	**(1.115,2.803)**	0.015	1.225	(0.719, 2.086)	0.455
Pickled foods	Once a day (ref)	1			1		
	Every other day	0.806	(0.482,1.346)	0.410	0.507	(0.255, 1.006)	0.052
	Every other day-once a day	1.790	**(1.139,2.812)**	0.012	1.043	(0.586, 1.856)	0.886
Spicy foods	Once a day (ref)	1			1		
	Every other day	1.179	(0.716,1.940)	0.517	1.472	(0.759,2.855)	0.253
	Every other day -once a day	2.390	**(1.503,3.801)**	<0.001	2.230	**(1.224,4.061)**	0.009
Life style							
Smoking	Yes	2.947	**(1.497, 5.804)**	0.002	3.072	**(1.507, 6.264)**	0.002
	No(ref)						
Drinking bottled water	Yes	2.625	**(1.757, 3.923)**	<0.001	2.532	**(1.686, 3.803)**	<0.001
	No(ref)	1				1	
History of digestive diseases							
Gastric cancer	Yes	2.670	**(1.649, 4.323)**	<0.001	0.930	(0.507, 1.703)	0.813
	No(ref)	1			1		
Gastroenteritis	Yes	4.142	**(2.612, 6.568)**	<0.001	2.282	**(1.350, 3.858)**	0.002
	No(ref)	1			1		
Peptic ulcers	Yes	6.039	**(3.703, 9.851)**	<0.001	3.770	**(2.095, 6.786)**	<0.001

	No(ref)	1			1		
Oesophagitis	Yes	4.105	**(2.396, 7.033)**	<0.001	1.674	(0.896, 3.129)	0.106
	No(ref)	1			1		
Hepatitis	Yes	3.487	**(2.099, 5.792)**	<0.001	1.765	(0.991, 3.142)	0.054
	No(ref)	1			1		
Symptoms							
Nausea with vomiting	Yes	16.01	**(9.56, 26.793)**	<0.001	5.89	**(3.230,10.43)**	<0.001
	No (Ref)	1			1		
Heartburn	Yes	28.9	**(16.95, 49.31)**	<0.001	14.13	**(7.991, 24.92)**	<0.001
	No (Ref)						

Final model

The current study modeled an equation that medical practitioners could use to diagnose cases of *H. pylori* among patients seeking medical services in any hospital in Mogadishu. The study used a multivariable logistic regression model:

$Logit\ (Li) = B_0 + B_1X_1 + \ldots + B_KX_K$

*Logit (Li) = -2.368 +2.132(smoking) + 0.660(Bottled water) +1.147 (peptic ulcers) +0.9471(*Gastroenteritis*) 1.587 (nausea with vomiting) + 2.420 (Heartburn)*

Logit (Li) = 3.45

Therefore *natural logarithm* $e^{Li} = 31.5$. The conditional probability of a patient testing positive for *H. pylori* assuming all the above conditions have been met becomes

$p_1 = e^{Li}/(1+ e^{Li}) = 0.969.$

Table 5:Final logit model with significant variables and *H. pylori* test

Characteristic	Category	AOR	95% CI	*p*-value
Smoking	Yes	3.072	(1.507, 6.264)	0.002
	No	1		
Bottled water	Yes	2.532	(1.686, 3.803)	<0.001
	No (ref)	1		
Gastroenteritis	Yes	2.282	(1.350, 3.858)	0.001
	No (ref)	1		
Peptic ulcers	Yes	3.770	(2.095, 6.786)	<0.001
	No (ref)	1		
Nausea with vomiting	Yes	5.89	(3.230, 10.43)	<0.001
	No (ref)	1		

Heartburn	Yes	14.13	(7.991, 24.92)	<0.001
	No (ref)	1		
Age	Child (>6months-12 years)	0.097	(0.014,0.688)	0.020
BMI	Obese	4.547	(1.250,16.54)	0.022
Spicy food	Every other dayonce a day	2.230	(1.224,4.061)	0.009

CHAPTER FIVE

DISCUSSIONS, CONCLUSION AND RECOMMENDATIONS

5.1 Introduction

This chapter gives a discussion and a conclusion of the study based on the survey findings that have been addressed in the previous chapter. It goes ahead to give recommendations on the lessons learned.

5.2 Discussion

The study recorded an *H. pylori* infection prevalence of 56.2% (positive tests *n*=232; negative tests *n*=181). This finding revealed a significantly higher prevalence compared to the 42.7% reported in the Abdirahman (2016) study conducted at Aden Abdulle Hospital in Mogadishu, Somalia. Insurgents are known for targeting and destroying water sources, including wells, in various parts of Somalia, including Mogadishu. By targeting critical infrastructure such as wells, the group aims to disrupt essential services, cause hardship for the local population, and undermine the stability of the region. The destruction of water sources has severe humanitarian consequences, affecting access to clean water for drinking, sanitation, and agriculture, thus exacerbating the already challenging living conditions in conflict-affected areas. This has resulted in an increase in the prevalence of *H. pylori* in Mogadishu from 2016 to 2022.

In comparison to other countries within the UN Africa Region, the prevalence of *H. pylori* (56.2%) in Somalia was lower than that of Benin (74.1%), the Democratic Republic of Congo (77.4%), Libya (76.4%), Nigeria (87.7%), South Africa (77.6%), and Tunisia (72.8%). However, the prevalence observed in the current study in Somalia closely aligned with the reported rates in China (55.8%), Japan (51.7%), Korea (54%), Taiwan (53.9%), Finland (56.8%), Albania (53.5%), Croatia (52.7%), Greece (52.1%), Italy (56.2%), Spain (54.9%), Lebanon (52%), Iran (59%), Mexico (52.5%), Panama (54.1%), and the Bahamas (57.8%) (Hooi *et al.*, 2017). It is intriguing to note that despite the aforementioned countries' higher levels of accessibility to clean water and urbanization and varied socioeconomic status compared to Somalia, they exhibit similar or even higher prevalence levels.

The second objective of the current study focused on the risk factors that would be associated with *H. pylori*. Several risk factors were investigated: individual characteristics, dietary intake, lifestyle, history of digestive diseases, and symptoms of *H. pylori*.The overall final logistic model in Table 5 shows that use of bottled water (p = 0.024); gastroenteritis (p = 0.045); peptic ulcers (p=0.006); nausea with vomiting (p<0.001) and heartburn (p< 0.001) were associated with *H. pylori* among patients with dyspepsia seeking treatment at Kalkaal Hospital. It is noteworthy that both the current study and Abdirahman's (2016) study reported no association between age, gender, residence, smoking, and *H. pylori* infection. Similarly, a study conducted by Monno *et al.* (2019) in Italy revealed no significant correlation between age, gender, employment, residence, level of educationand *H. pylori* infection. However, Monno's study differed from the present study as it indicated a significant association between cigarette smoking and *H. pylori* infection, which was not observed in the current study.

Several dietary intakes were investigated as potential risk factors for *H. pylori* infection. The following dietary factors were investigated in the current study: consumption of vegetables, fruits, animal proteins (milk, eggs, and meat), onions and garlic, pickled foods, fried foods and spicy foods. The logit model on dietary intakes did not find any significant association between the consumption of fruits, spicy foods, pickled food, beans, milkand meatwith *H. pylori* infections. These findings are similar to what was reported by (Monno *et al.*, 2019) study in Apulia in Italy where there was no significant association between *H. pylori* and the consumption of fruits, meat, and milk products.

Smoking, drinking bottled water and family history of peptic disease/gastric cancer patients were investigated as potential predictors. Smoking and drinking water had a significant association with *H. pylori*. However, only the consumption of bottled water had a significant association with *H. pylori* in the final logit model. This meant consumers of bottled water were 1.9 times more likely to test positive for *H. pylori*. This finding among the consumers of bottled water causes a worrying trend because drinking contaminated water has been associated with *H. pylori* infections (Khan *et al.*, 2012; Mladenova & Durazzo, 2018). This means that there could be a problem with the source of water that is being used to pack the bottled water or a potential faecal transmission to the bottles while handling the water bottles. Studies such as Khan *et al.* (2012); Mladenova and Durazzo (2018) have documented the association between contaminated drinking water and *H. pylori* infections. A study in Iran by Gholami-Borujeni *et al.* (2022) associated the prevalence of *H. pylori* with the source of contamination from the municipal drinking water supply. Since the fall of the government of Siad-Barre and the protracted war, there is no clean piped water distributed by the Government of Somalia. Drilled borehole water is supplied in Mogadishu by private companies and thus its cleanliness and purity are not guaranteed. Equally, there is no guarantee that the consumed bottled water is clean and where it comes from. Sanitation is very poor in Mogadishu and there is no facility to test and treat drinking water for the municipality. All these factors contribute to water being a risk factor for contracting

H. pylori in Mogadishu, Somalia. It is very difficult to find clean piped water has and this might have contributed to the persistence of high level of *H. pylori*. Genotyping and antimicrobial studies conducted elsewhere have shown bottled mineral water to be the source of virulent and resistant strains of *H. pylori*. For example, the Ranjbar *et al*. (2016) study on genotyping and antimicrobial isolates conducted in Iran found samples of mineral bottled water contaminated with *H. pylori* and it was concluded that careful monitoring of bottled mineral water was necessary to reduce the risk of transmission into the human population. Bahrami *et al*. (2013) study on city water, dental units' water and bottled mineral water in Isfanhan, Iran, tested positive of *H. pylori* isolates. The contamination of water was probably due to the contamination of the main river (Zayande-Rood River) which was the main source of drinking water for companies producing bottled water and lack of treatment of water via radiation. Therefore, this is a gap that further studies involving laboratory investigations need to focus on to ascertain if bottled water being sold in Mogadishu and Somalia at large is clean and safer for human consumption.

A significant odds ratio was observed in children compared to senior adults and other age categories, indicating a lower risk of *H. pylori* infection. This conform with findings from a study conducted in Europe that established that infection by *H. pylori* among children is generally rare before are age of 10 years (Sahil, 2009).

The study revealed that the odds of testing positive for *H. pylori* infection were 4.6 times higher among individuals with obesity-related comorbidities. However, no statistically significant association was found between *H. pylori* infection and other BMI categories. It is important to note that this study was not designed to establish a causal relationship between *H. pylori* and obesity. The computation of BMI was solely conducted because the study had collected weight and height information from the patients. Therefore, this study confirms that there is no direct causal relationship between obesity and *H. pylori* infection. Obesity is a multifactorial condition influenced by genetic, environmental, and lifestyle factors. While obesity itself does not directly elevate the risk of *H. pylori* infection, certain lifestyle factors commonly observed in obese individuals, such as dietary patterns and socioeconomic status, may indirectly contribute to a higher prevalence of *H. pylori* infection. It should be emphasized that the relationship between obesity and *H. pylori* is complex and influenced by multiple factors. Further research is warranted to gain a deeper understanding of the potential associations and interactions between these two variables.

Consuming spicy foods once daily increases the likelihood of testing positive for *H. pylori* by 2.2 times compared to those who consume spicy foods only once a day. Spicy foods contain antibacterial compounds like capsaicin, suggesting a potential association with *H. pylori* infection. However, evidence on the relationship between spicy food consumption and *H. pylori* infection remains inconclusive. Some studies propose that the antimicrobial properties of spicy foods may inhibit the growth of *H. pylori* bacteria, leading to a lower prevalence of *H. pylori* infection. However, conflicting results or no significant association have been observed in other studies.

The logistic regression analysis revealed a statistically significant association between smoking and *H. pylori* infection. Smokers had approximately three times higher odds of testing positive for *H. pylori* compared to non-smokers, as indicated by the adjusted odds ratio of 3.072 ($p = 0.002$). These findings demonstrate a strong positive association between smoking and *H. pylori* infection. A study conducted in Nigeria by Bello *et al.* (2018) explored the relationship between smoking and *H. pylori* infection, reporting smoking as a significant risk factor for *H. pylori* infection. Smoking has been shown to increase the risk of acquiring an *H. pylori* infection and may also contribute to the persistence of the infection. Smoking can affect the gastric mucosa and impair the immune response, making individuals more susceptible to *H. pylori* colonization. Additionally, smokers are known to share a lit cigarette among themselves, which increases the transmission rate of *H. pylori* infection. However, it is worth noting that smoking rates in Somalia have traditionally been relatively low compared to many other countries. This can be attributed, in part, to cultural factors and the prevalence of other forms of tobacco use, such as chewing khat (a stimulant plant), which is more common in the region.

The logistic regression analysis found that individuals with gastroenteritis had approximately 2.3 times higher odds of testing positive for *H. pylori* compared to those without gastroenteritis (adjusted odds ratio, $p = 0.002$). This significant positive association suggests that gastroenteritis may serve as a risk factor for acquiring *H. pylori* infection.

In the logistic regression analysis, individuals with peptic ulcers had approximately 3.8 times higher odds of testing positive for *H. pylori* compared to those without peptic ulcers (adjusted odds ratio, $p < 0.001$). These findings strongly support a positive association between peptic ulcers and *H. pylori* infection, suggesting that *H. pylori* may play a significant role in the development of peptic ulcers.

The logistic regression analysis revealed that individuals experiencing nausea had approximately 5.9 times higher odds of testing positive for *H. pylori* compared to those without nausea (adjusted odds ratio, $p <0.001$). This robust positive association suggests that nausea may be a significant symptom associated with *H. pylori* presence or its related effects.

The logistic regression analysis demonstrated a strong positive association between heartburn and *H. pylori* infection. Individuals with heartburn had approximately 14 times higher odds of testing positive for *H. pylori* compared to those without heartburn (adjusted odds ratio, $p < 0.001$). These findings indicate that heartburn may serve as a significant symptom or indicator of *H. pylori* presence or its associated effects.

The final logit model of possible statistically significant risk factors of *H. pylori* in Mogadishu, Somalia, comprised of drinking bottled water, family history of peptic ulcers, nausea with vomiting, and heartburn. This means the probability of a patient testing positive for *H. pylori* in Mogadishu having fully met the above criteria is 97%.

5.3 Socio-economic factors (future studies)

The study findings on *H. pylori* infection among patients with dyspepsia in Mogadishu, Somalia, presents an opportunity to explore socio-economic determinants and their impact on infection prevalence, adding valuable depth to the research:

1. Economic Status and Water Source:
    1) Investigate the correlation between economic status and the choice of water source, focusing on whether individuals with lower economic status are more likely to rely on bottled water, as identified in the study.
    2) Explore the socio-economic factors influencing access to safe drinking water, considering potential disparities in water quality and sanitation practices among different economic groups.
2. Educational Attainment and Awareness:
    1) Examine the relationship between educational attainment and awareness of *H. pylori* infection, investigating whether individuals with lower levels of education are less informed about the associated risk factors and symptoms.
    2) Explore how educational programs and interventions targeting different socio-economic groups could enhance awareness and contribute to preventive behaviours.
3. Occupational Exposures:
    1) Assess occupational exposures as potential socio-economic determinants, particularly in contexts where certain occupations may involve increased contact with contaminated environments or contribute to stressors that could impact *H. pylori* infection rates.
    2) Explore the relationship between occupation, workplace conditions, and the likelihood of developing dyspepsia symptoms associated with *H. pylori* infection.

4. Healthcare Access and Utilization:

1) Investigate the socio-economic factors influencing access to healthcare services, including disparities in seeking medical attention for dyspeptic symptoms.
2) Explore how economic constraints, education levels, and awareness impact healthcare-seeking behaviour and subsequent *H. pylori* diagnosis and management.

5. Living Conditions and Hygiene Practices:

1) Examine living conditions, sanitation facilities, and hygiene practices among different socio-economic groups to identify potential factors contributing to *H. pylori* transmission.
2) Explore the influence of socio-economic status on adherence to recommended hygiene practices that could mitigate the risk of *H. pylori* infection.

By delving into these socio-economic determinants, the study can provide a more nuanced understanding of the contextual factors influencing *H. pylori* infection prevalence in Mogadishu and similar settings. This exploration can inform targeted interventions and public health strategies that address socio-economic disparities, ultimately contributing to more effective and equitable approaches to *H. pylori* control and management.

5.4 Public health implications

The study findings on *H. pylori* infection among patients with dyspepsia in Mogadishu, Somalia, offer valuable insights that can influence policy and practice in Somalia and similar contexts, particularly in the realm of public health. To leverage these results effectively:

1. Policy Development:

1) Advocate for the integration of water safety and hygiene promotion campaigns into public health policies, aiming to reduce *H. pylori* infection rates associated with drinking bottled water.
2) Encourage policy formulation that prioritizes screening and management of individuals with a history of peptic ulcers, addressing a significant risk factor for *H. pylori* infection.

2. Healthcare Practices:

1) Propose guidelines for healthcare providers in Somalia to include targeted screening and management protocols for dyspeptic patients with identified risk factors, aligning with the study's findings.

2) Promote awareness among healthcare professionals regarding the strong association between specific symptoms (nausea with vomiting and heartburn) and *H. pylori* infection, leading to improved diagnostic and treatment strategies.

3. Public Health Education:

1) Develop public health education programs aimed at raising awareness about *H. pylori* infection, its symptoms, and associated risk factors in the local population.
2) Emphasize the importance of seeking medical attention for dyspeptic symptoms, especially in light of the study's finding that patients in Mogadishu with dyspepsia have a high likelihood of testing positive for *H. pylori*.

4. Research Support:

1) Advocate for increased research funding to conduct further studies, including longitudinal research, to monitor the effectiveness of interventions and refine policies over time.
2) Collaborate with national and international research entities to broaden the evidence base and strengthen the foundation for informed decision-making in *H. pylori* control and management.

By incorporating these recommendations into policy and practice, the study's findings can contribute to more effective public health measures tailored to the specific needs of Somalia and similar contexts, ultimately improving the prevention, diagnosis, and management of *H. pylori* infections in these populations.

5.5 Addressing global health perspectives

The findings of the study on H. pylori infection in patients with dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia, hold relevance for global health perspectives. To address *H. pylori* infection on a broader scale, global health initiatives can draw insights from the identified risk factors and prevalence rates. Public health campaigns targeting safe water practices and hygiene, as indicated by the association between drinking bottled water and *H. pylori* infection, could be implemented globally to reduce the burden of this bacterial infection. Strategies for screening and managing individuals with a history of peptic ulcers, identified as a significant risk factor, could be integrated into international health guidelines for dyspepsia management.

Moreover, the strong association between specific symptoms (nausea with vomiting and heartburn) and *H. pylori* infection suggests the need for a global re-evaluation of diagnostic and treatment protocols for individuals presenting with these symptoms. Collaborative efforts in research and intervention strategies, informed by the study's findings, can contribute to a more comprehensive and standardized approach to *H. pylori* control and management worldwide. By incorporating these insights into global health frameworks, the study's implications extend beyond Mogadishu, providing valuable contributions to the broader efforts in controlling and managing *H. pylori* infections on a global scale.

5.6 Study limitations

Future studies should include questions pertaining to water sanitation and hygiene (WASH) practices to directly assess the relationship between poor WASH activities and *H. pylori*. Although the present study identified a potential link between drinking bottled water and *H. pylori* contamination, it did not conduct specific testing for *H. pylori* bacteria in the bottled water samples. Therefore, future studies can focus on the laboratory analysis of bottled water to confirm its safety for human consumption.

Height for most of the children was missing in the medical records. This affected the computation of BMI for children. As a result, children category was under-represented in the analysis of BMI with *H. pylori* infection. Future researchers should design studies with a large sample size to increase the chance of getting height in children medical records.

Conducted solely at Kalkaal Hospital in Mogadishu, the study's limited sample size of 414 participants poses challenges for generalizability. Recognizing this constraint is crucial for a comprehensive understanding of the findings. The modest sample size may affect the study's external validity, warranting caution in interpreting and applying the results to broader populations. Future research should consider expanding the sample size and including multiple locations to enhance the study's representativeness and reliability.

5.7 Conclusion

In conclusion, the current study aimed to determine the prevalence of *H. pylori* infection and identify associated risk factors among patients seeking medical services at Kalkaal Hospital in Mogadishu, Somalia. The findings reveal a prevalence rate of 56.2% for *H. pylori* infection in Mogadishu. Furthermore, the study identified several significant risk factors, including the consumption of bottled water, a family history of peptic ulcers, and the presence of symptoms such as nausea with vomiting and heartburn. Notably, when patients reported consuming bottled water, having a family history of peptic ulcers, experiencing nausea with vomiting, and suffering from heartburn, there was a high probability (97%) of testing positive for *H. pylori* infection in their stool samples. These findings emphasize the importance of understanding the prevalence and risk factors associated with *H. pylori* infection in Mogadishu, contributing to the knowledge base for effective prevention and management strategies in this context.

5.8 Recommendations

i. The following study has statistically identified bottled water aspotential sources for *H. pylori*. Therefore, future confirmatory laboratory tests involving isolation and genotyping of *H. pylori*from sample bottled water will be necessary.

ii. There is a need for more health awareness campaigns among residents residing inMogadishu on the consumption of bottled water, family history of peptic ulcers, complaints of nausea with vomiting and heartburn as a risk factor associated with *H. pylori* infection. This will encourage persons exposed tothese risk factors to seek medical attention.

iii. More awareness campaigns among these residents should focus on encouraging early testing and treatment of *H. pylori* which is averagely prevalent in Mogadishu.

iv. Boreholes are the main sources of drinking water in Mogadishu. This means that companies that sell drinking water also use borehole water for their bottling purposes.Therefore, companies focus on treating borehole water before packaging andusinghygienic practices while handling bottled water since contamination may occur even during packaging.

v. Based on the study findings, it is recommended that public health interventions in Mogadishu, Somalia, focus on addressing the high prevalence of *H. pylori* infection among patients with dyspepsia. Considering the statistically significant risk factors identified, strategies could include public health campaigns promoting safe drinking water practices.

vi. Additionally, healthcare providers should prioritize screening and management for individuals with a history of peptic ulcers, as they demonstrated a higher risk of *H. pylori* infection.

vii. Longitudinal studies exploring the effectiveness of interventions aimed at reducing *H. pylori* prevalence in the population would provide valuable insights. Furthermore, given the strong association between specific symptoms (nausea with vomiting and heartburn) and *H. pylori* infection, future research could investigate tailored treatment approaches for individuals presenting with these symptoms. These recommendations aim to inform targeted interventions and contribute to the development of more effective healthcare strategies in the region.

REFERENCES

Abdirahman, O. H. (2016). *Prevalence and factors associated with Helicobacter pylori Infection in adults attending medical outpatient unit at Aden Abdulle Hospital, Mogadishu, Somalia*

Adedoyin, J. J., & David, I. (2020). Seroprevalence of Helicobacter pylori infection among children selected hospitals in Keffi, Nigeria. *Adv Pub Health Com Trop Med*. (APCTM-120)

Adlekha, S., Chadha, T., Krishnan, P., Sumangala, B. J. A. o. m., & research, h. s. (2013). Prevalence of Helicobacter pylori infection among patients undergoing upper gastrointestinal endoscopy in a medical college hospital in Kerala, India. *3*(4), 559-563.

Aitila, P., Mutyaba, M., Okeny, S., Ndawula Kasule, M., Kasule, R., Ssedyabane, F., Okongo, B., Onyuthi Apecu, R., Muwanguzi, E., & Oyet, C. J. J. o. t. m. (2019). Prevalence and risk factors of Helicobacter pylori infection among children aged 1 to 15 years at holy innocents children's hospital, Mbarara, South Western Uganda. *2019*.

Akada, J. K., Ogura, K., Dailidiene, D., Dailide, G., Cheverud, J. M., & Berg, D. E. (2003). Helicobacter pylori tissue tropism: mouse-colonizing strains can target different gastric niches. *Microbiology (Reading)*, *149*(Pt 7), 1901-1909. https://doi.org/10.1099/mic.0.26129-0

Archampong, T. N., Asmah, R. H., Aidoo, E. K., Wiredu, E. K., Gyasi, R. K., Adjei, D. N., Beleza, S., Bayliss, C. D., & Krogfelt, K. (2017). Helicobacter pylori cagA and vacA genes in dyspeptic Ghanaian patients. *BMC Research Notes*, *10*(1), 231. https://doi.org/10.1186/s13104-017-2542-8

Asaka, M., Sugiyama, T., Nobuta, A., Kato, M., Takeda, H., & Graham, D. Y. (2001). Atrophic gastritis and intestinal metaplasia in Japan: results of a large multicenter study. *Helicobacter*, *6*(4), 294-299. https://doi.org/10.1046/j.1523-5378.2001.00042.x

Asfaw, T. (2018). Prevalence and emergence of drug resistance in Helicobacter pylori. *Int Res J Microbial*, *7*(2), 56-66.

Asmah, R., Archampong, T., Brown, C., Ntiamoah, S., Aidoo, E., Gyasi, R., & Wiredu, E. (2014). Helicobacter Pylori vacA Gene Detection in Saliva of Patients with Upper Gastrointestinal Disorders in Accra, Ghana. *International Journal of Genetics and Genomics*, *2*. https://doi.org/10.11648/j.ijgg.20140205.11

Asrat, D., Nilsson, I., Mengistu, Y., Ashenafi, S., Ayenew, K., Al-Soud, W. A., Wadström, T., & Kassa, E. (2004). Prevalence of Helicobacter pylori infection among adult dyspeptic patients in Ethiopia. *Annals of Tropical Medicine and Parasitology*, *98*(2), 181-189. https://doi.org/10.1179/000349804225003190

Awuku, Y. A., Simpong, D. L., Alhassan, I. K., Tuoyire, D. A., Afaa, T., & Adu, P. (2017). Prevalence of helicobacter pylori infection among children living in a rural setting in Sub-Saharan Africa. *BMC Public Health*, *17*, 1-6.

Ayele, B., & Molla, E. J. C. M. (2017). Dyspepsia and Associated Risk Factors at Yirga Cheffe Primary Hospital, Southern Ethiopia. *6*, 3.

Aziz, R. K., Khalifa, M. M., & Sharaf, R. R. (2015). Contaminated water as a source of Helicobacter pylori infection: A review. *Journal of Advanced Research*, *6*(4), 539-547. https://doi.org/https://doi.org/10.1016/j.jare.2013.07.007

Bahrami, A. R., Rahimi, E., & Ghasemian Safaei, H. (2013). Detection of Helicobacter pylori in city water, dental units' water, and bottled mineral water in Isfahan, Iran. *The Scientific World Journal*, *2013*.

Bastos, J., Peleteiro, B., Barros, R., Alves, L., Severo, M., de Fátima Pina, M., Pinto, H., Carvalho, S., Marinho, A., & Guimarães, J. T. J. H. (2013). Sociodemographic determinants of prevalence and incidence of Helicobacter pylori infection in Portuguese adults. *18*(6), 413-422.

Bello, A., Umar, A., & Borodo, M. (2018). Prevalence and risk factors for Helicobacter pylori infection in gastroduodenal diseases in Kano, Nigeria. *African Journal of Medical and Health Sciences*, *17*(1), 41-41.

Benajah, D. A., Lahbabi, M., Alaoui, S., El Rhazi, K., El Abkari, M., Nejjari, C., Amarti, A., Bennani, B., Mahmoud, M., Ibrahimi, S. A. J. C., hepatology, r. i., & gastroenterology. (2013). Prevalence of Helicobacter pylori and its recurrence after successful eradication in a developing nation (Morocco). *37*(5), 519-526.

Benberin, V., Bektayeva, R., Karabayeva, R., Lebedev, A., Akemeyeva, K., Paloheimo, L., & Syrjänen, K. J. A. r. (2013). Prevalence of H. pylori infection and atrophic gastritis among symptomatic and dyspeptic adults in Kazakhstan. A hospital-based screening study using a panel of serum biomarkers. *33*(10), 4595-4602.

Bertrant, E. B., Epesse, S. F. E. B., Stephane, M. B., Clementine, F., Roger, K., & Brigitte, K. J. (2020). Prevalence of Helicobacter pylori infection and relevant endoscopic features among patients with gastro-duodenal disorders. A three years cross sectional study in the Littoral region of Cameroon. *Eur J Sci Res*, *15*(3), 247-257.

Bossali, F., Deby, G., Ahoui-Apendi, C., Ndolo, D., Ndziessi, G., & Atipo-Ibara, B. (2017). Study of the management of Helicobacter pylori infection in the cities of pointe-noire and Bbrazzaville in 2015. *Ann Univ M Ngouabi*, *17*(1), 1-9.

Chukwuma, O., Chukwuma, G., Manafa, P., Akulue, J., & Jeremiah, Z. (2020). Prevalence and possible risk factors for Helicobacter pylori seropositivity among peptic ulcerative individuals in Nnewi Nigeria. *BioMed Research Journal*, *4*(1), 166-172.

Correa, P., & Piazuelo, M. B. (2011). Helicobacter pylori Infection and Gastric Adenocarcinoma. *US Gastroenterol Hepatol Rev*, *7*(1), 59-64.

Correa, P., Piazuelo, M. B. J. U. g., & review, h. (2011). Helicobacter pylori infection and gastric adenocarcinoma. *7*(1), 59.

Cover, T. L., & Blaser, M. J. (2009). Helicobacter pylori in health and disease. *Gastroenterology*, *136*(6), 1863-1873. https://doi.org/10.1053/j.gastro.2009.01.073

Daniyan Olapeju, W., Ibe Chidozie, C., Ezeonu Thecla, C., Anyanwu Onyinye, U., Ezeanosike Obumneme, B., & Omeje Kenneth, N. (2020). Seroprevalence and risk factors of Helicobacter pylori among children in South East Nigeria. *J Gastroentrol Hepatol Res*, *9*, 1.

Das, J. C., & Paul, N. J. T. I. J. o. P. (2007). Epidemiology and pathophysiology of Helicobacter pylori infection in children. *74*(3), 287-290.

den Hollander, W. J., Sostres, C., Kuipers, E. J., & Lanas, A. J. H. (2013). Helicobacter pylori and nonmalignant diseases. *18*, 24-27.

Dube, C., Nkosi, T., Clarke, A., Mkwetshana, N., Green, E., & Ndip, R. J. R. o. e. h. (2009). Helicobacter pylori antigenemia in an asymptomatic population of Eastern Cape Province, South Africa: public health implications. *24*(3), 249-255.

Fischbach, W., & Malfertheiner, P. (2018). Helicobacter Pylori infection: when to eradicate, how to diagnose and treat. *Deutsches Ärzteblatt International*, *115*(25), 429.

Fuccio, L., Eusebi, L. H., & Bazzoli, F. (2010). Gastric cancer, Helicobacter pylori infection and other risk factors. *World Journal of Gastrointestinal Oncology*, *2*(9), 342-347. https://doi.org/10.4251/wjgo.v2.i9.342

Galal, Y. S., Ghobrial, C. M., Labib, J. R., & Abou-Zekri, M. E. (2019). Helicobacter pylori among symptomatic Egyptian children: prevalence, risk factors, and effect on growth. *Journal of the Egyptian Public Health Association*, *94*, 1-8.

Gholami-Borujeni, F., Rahimi, H., & Sajjadi, S. M. (2022). Detection of Helicobacter pylori genes (CagA and VacA) in municipal drinking water. *Environmental Health Engineering And Management Journal*, 0-0.

Graham, D. Y., Malaty, H. M., Evans, D. G., Evans, D. J., Jr., Klein, P. D., & Adam, E. (1991). Epidemiology of Helicobacter pylori in an asymptomatic population in the United States. Effect of age, race, and socioeconomic status. *Gastroenterology*, *100*(6), 1495-1501. https://doi.org/10.1016/0016-5085(91)90644-z

Hanafi, M. I., & Mohamed, A. M. J. T. J. o. t. E. P. H. A. (2013). Helicobacter pylori infection: seroprevalence and predictors among healthy individuals in Al Madinah, Saudi Arabia. *88*(1), 40-45.

Holcombe, C. J. G. (1992). Helicobacter pylori: the African enigma. *33*(4), 429.

Hooi, J. K., Lai, W. Y., Ng, W. K., Suen, M. M., Underwood, F. E., Tanyingoh, D., Malfertheiner, P., Graham, D. Y., Wong, V. W., & Wu, J. C. (2017). Global prevalence of Helicobacter pylori infection: systematic review and meta-analysis. *Gastroenterology*, *153*(2), 420-429.

Hunt, R. H., & Tytgat, G. (2012). *Helicobacter pylori: basic mechanisms to clinical cure*. Springer Science & Business Media.

Hunt, R. H., Xiao, S. D., Megraud, F., Leon-Barua, R., Bazzoli, F., van der Merwe, S., Vaz Coelho, L. G., Fock, M., Fedail, S., Cohen, H., Malfertheiner, P., Vakil, N., Hamid, S., Goh, K. L., Wong, B. C., Krabshuis, J., & Le Mair, A. (2011). Helicobacter pylori in developing countries. World Gastroenterology Organisation Global Guideline. *Journal of Gastrointestinal and Liver Diseases*, *20*(3), 299-304.

Iranikhah, A., Ghadir, M.-R., Sarkeshikian, S., Saneian, H., Heiari, A., & Mahvari, M. (2013). Stool antigen tests for the detection of Helicobacter pylori in children. *Iranian journal of pediatrics*, *23*(2), 138-142. https://pubmed.ncbi.nlm.nih.gov/23724172
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3663302/

Jemilohun, A. C., Otegbayo, J. A., Ola, S. O., Oluwasola, O. A., & Akere, A. (2010). Prevalence of Helicobacter pylori among Nigerian patients with dyspepsia in Ibadan. *The Pan African Medical Journal*, *6*, 18-18. https://pubmed.ncbi.nlm.nih.gov/21734925
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3120989/

Kasmi, H., Doukani, K., Ali, A., Tabak, S., & Bouhenni, H. (2020). Epidemiological profile of Helicobacter pylori infection in patients with digestive symptoms in Algeria. *Journal of Epidemiology and Global Health*, *10*(4), 293.

Khalifa, M. M., Sharaf, R. R., & Aziz, R. K. J. G. p. (2010). Helicobacter pylori: a poor man's gut pathogen? , *2*(1), 1-12.

Khan, A., Farooqui, A., & Kazmi, S. U. (2012). Presence of Helicobacter pylori in drinking water of Karachi, Pakistan. *The Journal of Infection in Developing Countries*, *6*(03), 251-255.

Kimang'a, A. N., Revathi, G., Kariuki, S., Sayed, S., & Devani, S. J. S. A. M. J. (2010). Helicobacter pylori: prevalence and antibiotic susceptibility among Kenyans. *100*(1).

Kodaman, N., Pazos, A., Schneider, B. G., Piazuelo, M. B., Mera, R., Sobota, R. S., Sicinschi, L. A., Shaffer, C. L., Romero-Gallo, J., de Sablet, T., Harder, R. H., Bravo, L. E., Peek, R. M., Jr., Wilson, K. T., Cover, T. L., Williams, S. M., & Correa, P. (2014). Human and Helicobacter pylori coevolution shapes the risk of gastric disease. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(4), 1455-1460. https://doi.org/10.1073/pnas.1318093111

Kpossou, A. R., Kouwakanou, B., Séidou, F., Alassane, K., Vignon, R. K., & Sokpon, C., et al. (2020). Prevalence of Helicobacter pylori infection and gastric preneoplastic lesions in patients admitted for upper gastro-intestinal endoscopy in Cotonou (Benin Republic). *Gastroenterología y Hepatologia*, *11*(6), 208-213.

Kumar, S., Metz, D. C., Ellenberg, S., Kaplan, D. E., & Goldberg, D. S. J. G. (2020). Risk factors and incidence of gastric cancer after detection of Helicobacter pylori infection: a large cohort study. *158*(3), 527-536. e527.

Lawson–Ananissoh, L., Bouglouga, O., Bagny, A., Kaaga, L., & Redah, D. (2015). Epidemiological profile of peptic ulcers at the Lomé campus hospital and university center (Togo). *J Afr Hépatol Gastroentérol*, *9*(3), 99-103.

Leja, M., Grinberga-Derica, I., Bilgilier, C., & Steininger, C. J. H. (2019). Epidemiology of Helicobacter pylori infection. *24*, e12635.

Li, Z., Zou, D., Ma, X., Chen, J., Shi, X., Gong, Y., Man, X., Gao, L., Zhao, Y., Wang, R. J. O. j. o. t. A. C. o. G., & ACG. (2010). Epidemiology of peptic ulcer disease: endoscopic results of the systematic investigation of gastrointestinal disease in China. *105*(12), 2570-2577.

Makola, D., Peura, D. A., & Crowe, S. E. (2007). Helicobacter pylori infection and related gastrointestinal diseases. *Journal of Clinical Gastroenterology*, *41*(6), 548-558. https://doi.org/10.1097/MCG.0b013e318030e3c3

Malcolm, C., MacKay, W., Shepherd, A., & Weaver, L. J. S. m. j. (2004). Helicobacter pylori in children is strongly associated with poverty. *49*(4), 136-138.

Marshall, B., & Warren, J. R. J. T. l. (1984). Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *323*(8390), 1311-1315.

Mathewos, B., Moges, B., & Dagnew, M. J. B. R. N. (2013). Seroprevalence and trend of Helicobacter pylori infection in Gondar University Hospital among dyspeptic patients, Gondar, North West Ethiopia. *6*(1), 1-4.

McColl, K. E. (2010). Clinical practice. Helicobacter pylori infection. *New England Journal of Medicine*, *362*(17), 1597-1604. https://doi.org/10.1056/NEJMcp1001110

Mégraud, F. (1995). Transmission of Helicobacter pylori: faecal-oral versus oral-oral route. *Alimentary Pharmacology and Therapeutics, 9 Suppl 2*, 85-91.

Melese, A., Genet, C., Zeleke, B., & Andualem, T. (2019). Helicobacter pylori infections in Ethiopia; prevalence and associated factors: a systematic review and meta-analysis. *BMC Gastroenterology*, *19*(1), 8. https://doi.org/10.1186/s12876-018-0927-3

Mitchell, H. J. G. D., & pylori, H. (1999). The epidemiology of Helicobacter pylori. 11-30.

Mladenova, I., & Durazzo, M. (2018). Transmission of Helicobacter pylori. *Minerva Gastroenterologica e Dietologica*, *64*(3), 251-254.

Momtaz, H., Souod, N., Dabiri, H., & Sarshar, M. (2012). Study of Helicobacter pylori genotype status in saliva, dental plaques, stool and gastric biopsy samples. *World Journal of Gastroenterology*, *18*(17), 2105-2111. https://doi.org/10.3748/wjg.v18.i17.2105

Monno, R., De Laurentiis, V., Trerotoli, P., Roselli, A. M., Ierardi, E., & Portincasa, P. (2019). Helicobacter pylori infection: association with dietary habits and socioeconomic conditions. *Clinics and research in hepatology and gastroenterology*, *43*(5), 603-607.

Muhammad, J. S., Sugiyama, T., & Zaidi, S. F. (2013). Gastric pathophysiological ins and outs of helicobacter pylori: a review. *Journal of the Pakistan Medical Association*, *63*(12), 1528-1533.

Muhammad, J. S., Zaidi, S. F., Saeed, S. A., & Ishaq, M. (2017). Current status of Helicobacter pylori association with haematological and cardiovascular diseases: A mini review. *Journal of the Pakistan Medical Association*, *67*(6), 907-911.

Naja, F., Kreiger, N., & Sullivan, T. (2007a). Helicobacter pylori infection in Ontario: prevalence and risk factors. *Canadian Journal of Gastroenterology and Hepatology*, *21*, 501-506.

Naja, F., Kreiger, N., & Sullivan, T. J. C. J. o. G. (2007b). Helicobacter pylori infection in Ontario: prevalence and risk factors. *21*(8), 501-506.

Nguyen, T., Ramsey, D., Graham, D., Shaib, Y., Shiota, S., Velez, M., Cole, R., Anand, B., Vela, M., & El-Serag, H. B. J. H. (2015). The Prevalence of H elicobacter pylori Remains High in African American and Hispanic Veterans. *20*(4), 305-315.

Nguyen, T. V. H. (2017). Prevalence and risk factors of Helicobacter pylori infection in Muong children in Vietnam. *Annals of Clinical and Laboratory Research*, *5*(01).

Ntagirabiri, R., Harerimana, S., Makuraza, F., Ndirahisha, E., Kaze, H., & Moibeni, A. (2014). Helicobacter pylori in Burundi: first assessment of prevalence in endoscopy and eradication. *Journal Africain d'Hépato-Gastroentérologie*, *8*, 217-222.

O'Rourke, J. L. (2008). Gene expression profiling in Helicobacter-induced MALT lymphoma with reference to antigen drive and protective immunization. *Journal of Gastroenterology and Hepatology, 23 Suppl 2*, S151-156. https://doi.org/10.1111/j.1440-1746.2008.05553.x

Olokoba, A., Gashau, W., Bwala, S., Adamu, A., & Salawu, F. J. G. M. J. (2013). Helicabacter pylori Infection in Nigerians with Dyspepsia. *47*(2), 79-81.

Omar Hussein, A. (2016). *Prevalence and factors associated with Helicobacter pylori Infection in adults attending medical outpatient unit at Aden Abdulle Hospital, Mogadishu, Somalia*

Ozaydin, N., Turkyilmaz, S. A., & Cali, S. J. B. p. h. (2013). Prevalence and risk factors of helicobacter pylori in Turkey: a nationally-representative, cross-sectional, screening with the 13 C-Urea breath test. *13*(1), 1-12.

Patel, S. K., Pratap, C. B., Jain, A. K., Gulati, A. K., & Nath, G. (2014). Diagnosis of Helicobacter pylori: what should be the gold standard? *World Journal of Gastroenterology*, *20*(36), 12847-12859. https://doi.org/10.3748/wjg.v20.i36.12847

Peek, R. M., & Blaser, M. J. J. N. R. C. (2002). Helicobacter pylori and gastrointestinal tract adenocarcinomas. *2*(1), 28-37.

Peleteiro, B., Bastos, A., Ferro, A., Lunet, N. J. D. d., & sciences. (2014). Prevalence of Helicobacter pylori infection worldwide: a systematic review of studies with national coverage. *59*(8), 1698-1709.

Pounder, R., Ng, D. J. A. p., & therapeutics. (1995). The prevalence of Helicobacter pylori infection in different countries. *9*, 33-39.

Ranjbar, R., Khamesipour, F., Jonaidi-Jafari, N., & Rahimi, E. (2016). Helicobacter pylori in bottled mineral water: genotyping and antimicrobial resistance properties. *BMC Microbiology*, *16*, 1-10.

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Salih, B. A. J. S. j. o. g. o. j. o. t. S. G. A. (2009). Helicobacter pylori infection in developing countries: the burden for how long? , *15*(3), 201.

Schwarz, S., Morelli, G., Kusecek, B., Manica, A., Balloux, F., Owen, R. J., Graham, D. Y., van der Merwe, S., Achtman, M., & Suerbaum, S. (2008). Horizontal versus familial transmission of Helicobacter pylori. *PLoS Pathogens*, *4*(10), e1000180. https://doi.org/10.1371/journal.ppat.1000180

Segal, I., Ally, R., & Mitchell, H. J. Q. (2001). Helicobacter pylori—an African perspective. *94*(10), 561-565.

Sethi, A., Chaudhuri, M., Kelly, L., & Hopman, W. J. C. F. P. (2013). Prevalence of Helicobacter pylori in a First Nations population in northwestern Ontario. *59*(4), e182-e187.

Shmuely, H., Obure, S., Passaro, D. J., Abuksis, G., Yahav, J., Fraser, G., Pitlik, S., & Niv, Y. (2003). Dyspepsia symptoms and Helicobacter pylori infection, Nakuru, Kenya. *Emerging Infectious Diseases*, *9*(9), 1103-1107. https://doi.org/10.3201/eid0909.020374

Smith, S. I., Ajayi, A., Jolaiya, T., Onyekwere, C., Setshedi, M., Schulz, C., Otegbayo, J. A., Ndip, R., Dieye, Y., & Alboraie, M. (2022). Helicobacter pylori infection in Africa: update of the current situation and challenges. *Digestive Diseases*, *40*(4), 535-544.

Sokpon, M., Salihoun, M., Lahlou, L., Acharki, M., Razine, R., & Kabbaj, N. (2016). Factors predictive of Helicobacter pylori infection in chronic gastritis: about a Moroccan study. *Journal Africain d'Hépato-Gastroentérologie*, *10*, 203-207.

Somali National Bureau of Statistics. (2021). *Economic Characteristics Of The Somali People https://www.nbs.gov.so/volume-4-economic-characteristics-of-the-somali-people/*. Retrieved 4th October 2021 from

Sutton, P., & Boag, J. M. (2019). Status of vaccine research and development for Helicobacter pylori. *Vaccine*, *37*(50), 7295-7299. https://doi.org/10.1016/j.vaccine.2018.01.001

Tanih, N., Ndip, L., Clarke, A., & Ndip, R. (2010). An overview of pathogenesis and epidemiology of Helicobacter pylori infection. *African Journal of Microbiology Research*, *4*.

Taylor, N., Fox, J., & Yan, L. J. J. o. m. m. (1995). In-vitro hepatotoxic factor in Helicobacter hepaticus, H. pylori and other Helicobacter species. *42*(1), 48-52.

The EUROGAST Study Group. (1993). Epidemiology of, and risk factors for, Helicobacter pylori infection among 3194 asymptomatic subjects in 17 populations. . *Gut*, *34*(12), 1672-1676. https://doi.org/10.1136/gut.34.12.1672

Tsukanov, V. V., Amel, #039, chugova, O. S., Butorin, N. N., Tret, #039, iakova, O. V., & Vasiutin, A. V. (2013). Helicobacter pylori eradication: Current aspects. *Terapevticheskii Arkhiv*, *85*(2), 73-75 %J TA. https://ter-arkhiv.ru/0040-3660/article/view/31227

Udoh, M. O., & Obaseki, D. E. (2012). HISTOPATHOLOGICAL EVALUATION OF H. PYLORI ASSOCIATED GASTRIC LESIONS IN BENIN CITY, NIGERIA. *East African Medical Journal*, *89*(12), 408-413.

Uotani, T., & Graham, D. Y. (2015). Diagnosis of Helicobacter pylori using the rapid urease test. *Ann Transl Med*, *3*(1), 9. https://doi.org/10.3978/j.issn.2305-5839.2014.12.04

Urita, Y., Watanabe, T., Kawagoe, N., Takemoto, I., Tanaka, H., Kijima, S., Kido, H., Maeda, T., Sugasawa, Y., Miyazaki, T., Honda, Y., Nakanishi, K., Shimada, N., Nakajima, H., Sugimoto, M., & Urita, C. (2013). Role of infected grandmothers in transmission of Helicobacter pylori to children in a Japanese rural town. *Journal of Paediatrics and Child Health*, *49*(5), 394-398. https://doi.org/10.1111/jpc.12191

van Duynhoven, Y. T., & Jonge, R. d. J. B. o. t. W. H. O. (2001). Transmission of Helicobacter pylori: a role for food? , *79*, 455-460.

Venneman, K., Huybrechts, I., Gunter, M. J., Vandendaele, L., Herrero, R., & Van Herck, K. (2018). The epidemiology of Helicobacter pylori infection in Europe and the impact of lifestyle on its natural evolution toward stomach cancer after infection: A systematic review. *Helicobacter*, *23*(3), e12483.

Walker, T. D., Karemera, M., Ngabonziza, F., & Kyamanywa, P. (2014). Helicobacter pylori status and associated gastroscopic diagnoses in a tertiary hospital endoscopy population in Rwanda. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, *108*(5), 305-307. https://doi.org/10.1093/trstmh/tru029

Yamaoka, Y. J. C. M., & Infection. (2009). Helicobacter pylori typing as a tool for tracking human migration. *15*(9), 829-834.

Yim, J. Y., Kim, N., Choi, S. H., Kim, Y. S., Cho, K. R., Kim, S. S., Seo, G. S., Kim, H. U., Baik, G. H., & Sin, C. S. J. H. (2007). Seroprevalence of Helicobacter pylori in south Korea. *12*(4), 333-340.

Zeng, M., Mao, X. H., Li, J. X., Tong, W. D., Wang, B., Zhang, Y. J., Guo, G., Zhao, Z. J., Li, L., Wu, D. L., Lu, D. S., Tan, Z. M., Liang, H. Y., Wu, C., Li, D. H., Luo, P., Zeng, H., Zhang, W. J., Zhang, J. Y., Guo, B. T., Zhu, F. C., & Zou, Q. M. (2015). Efficacy, safety, and immunogenicity of an oral recombinant Helicobacter pylori vaccine in children in China: a randomised, double-blind, placebo-controlled, phase 3 trial. *Lancet*, *386*(10002), 1457-1464. https://doi.org/10.1016/s0140-6736(15)60310-5

Zhang, L., Eslick, G. D., Xia, H. H., Wu, C., Phung, N., & Talley, N. J. (2010). Relationship between alcohol consumption and active Helicobacter pylori infection. *Alcohol and Alcoholism*, *45*(1), 89-94. https://doi.org/10.1093/alcalc/agp068

Zhu, Y., Zhou, X., Wu, J., Su, J., Zhang, G. J. G. r., & practice. (2014). Risk factors and prevalence of Helicobacter pylori infection in persistent high incidence area of gastric carcinoma in Yangzhong city. *2014*.

APPENDICES

Appendix I: Data Collection Tool

Study ID: ---------------------------------

Patient Initials: ----------------------------

Date of data collection: ---------------------------

Part A: Individual Characteristics

1. Age in years: …………………………………..

2. Sex: Male ☐ Female ☐

3. Weight (kgs) -----------------------------

4. Height (cm) -------------------------------

5. Highest level of education: 1. No formal education ☐ 3. Secondary ☐

   2. Primary ☐ 4. Tertiary ☐

6. Number of family members………………

7. What is your marital status? 1. Married/livingtogether ☐ 2. Divorced/separated ☐

   3. Widowed ☐ 4. Never married ☐

8. Do you have children? 1. Yes ☐ 2. No ☐

9. How many children do you have currently? ---

10. Where do you live? 1. Rural ☐ 2. Urban ☐

11. What is your household head's level of education? 1. Primary ☐ 2. Secondary ☐

    3. University ☐ 4. None ☐ 5.Others(specify)________________

12. Are you employed? Yes ☐ ☐

13. If yes, what is your occupation? ---------------------------------------

14. What is your monthly income? ……………………………

Part B: Dietary characteristics and lifestyle

15. How often do you eat vegetables?

    1. <Every other day ☐ 2. Every other day-once a day ☐ 3.>Once a day ☐

16. Frequency of eating fruits

☐ ☐ ☐

1. <Every other day 2. Every other day-once a day 3.>Once a day

17. Frequency of eating milk, egg, and meat

1. <Every other day ☐ 2. Every other day-once a day ☐ 3.>Once a day ☐

18. Frequency of eating beans

1. <Every other day ☐ 2. Every other day-once a day ☐ 3.>Once a day ☐

19. Frequency of eating onion and garlic

1. <Every other day ☐ 2. Every other day-once a day ☐ 3.>Once a day ☐

20. Frequency of eating pickled foods

1. <Every other day ☐ 2. Every other day-once a day ☐ 3.>Once a day ☐

21. Frequency of eating fried foods

1. <Every other day ☐ 2. Every other day-once a day ☐ 3.>Once a day ☐

22. Frequency of eating spicy foods

1. <Every other day ☐ 2. Every other day-once a day ☐ 3.>Once a day ☐

23. Do you smoke?

1. Yes ☐ 2. No ☐

24. No. of cigarettes smoked per day ---

25. The period of smoking (years) --

26. Do you drink bottled water?

☐ ☐

1. Yes 2. No

27. The period of drinking of bottled water (years) ------------------------------------

28. Family history of peptic disease/gastric cancer 1. Yes ☐ 2. No ☐

Part C: History of digestive diseases

29. Gastroenteritis 1. Yes ☐ 2. No ☐

30. Peptic ulcer 1. Yes ☐ 2. No ☐

31. Esophagitis 1. Yes ☐ 2. No ☐

32. Hepatitis 1. Yes ☐ 2. No ☐

Part D: *H. pylori* test results

1. Positive ☐ 0. Negative ☐

Part E: *H. pylori*general symptoms

1. Nausea with vomiting 1. Yes ☐ 2. ☐
2. Heart burn 2. Yes ☐ 2. ☐

Appendix II: Informed Consent Form

Title of Study: Prevalence and Risk Factors for *Helicobacter pylori* Infection among Patients with Dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia

Principal Investigator\and institutional affiliation: Dr. Mustaf Ibrahim / University of Nairobi

Co-Investigators and institutional affiliation: University of Nairobi

Introduction:

I would like to tell you about a study being conducted by the above listed researchers. The purpose of this consent form is to give you the information you will need to help you decide whether or not to be a participant in the study. Feel free to ask any questions about the purpose of the research, what happens if you participate in the study, the possible risks and benefits, your rights as a volunteer, and anything else about the research or this form that is not clear. When we have answered all your questions to your satisfaction, you may decide to be in the study or not. This process is called 'informed consent'. Once you understand and agree to be in the study, I will request you to sign your name on this form. You should understand the general principles which apply to all participants in medical research: i) Your decision to participate is entirely voluntary ii) You may withdraw from the study at any time without necessarily giving a reason for your withdrawal iii) Refusal to participate in the research will not affect the services you are entitled to in this health facility or other facilities. We will give you a copy of this form for your records.

May I continue? YES / NO

This study has approval by The Kenyatta National Hospital-University of Nairobi Ethics and Research Committee protocol no. ______________________________

WHAT IS THIS STUDY ABOUT?

The researchers listed above are interviewing individuals who present to Kalkaal Hospital in Mogadishu, Somalia with Dyspepsia. The purpose of the interview is to find out the number of patients who have *H. pylori* infection and the reason why some patients have it and others don't. Participants in this research study will be asked questions about their socio-demographic characteristics, their life style, their feeding characteristics, house hold and previous history of some infections. Participants will also have the choice to undergo test such as *H. pylori* stool antigen testing. There will be approximately 414 participants in this study randomly chosen. We are asking for your consent to consider participating in this study.

WHAT WILL HAPPEN IF YOU DECIDE TO BE IN THIS RESEARCH STUDY?

If you agree to participate in this study, the following things will happen: You'll be required to provide a stool sample which will be used for the test. If you decline to join the study, it will not affect your access to medical care in any way. After providing the sample, you will be interviewed by a trained interviewer in a private area where you feel comfortable answering questions. The interview will last approximately interview 15 minutes. We will ask for a telephone number where we can contact you if necessary. If you agree to provide your contact information, it will be used only by people working for this study and will never be shared with others. The reasons why we may need to contact you include: to refer you for treatment in case the test turns out positive and also to let you know of your test results.

ARE THERE ANY RISKS, HARMS DISCOMFORTS ASSOCIATED WITH THIS STUDY?

Medical research has the potential to introduce psychological, social, emotional and physical risks. Effort should always be put in place to minimize the risks. One potential risk of being in the study is loss of privacy. We will keep everything you tell us as confidential as possible. We will use a code number to identify you in a password-protected computer database and will keep all of our paper records in a locked file cabinet. However, no system of protecting your confidentiality can be absolutely secure, so it is still possible that someone could find out you were in this study and could find out information about you. Also, answering questions in the interview may be uncomfortable for you. If there are any questions you do not want to answer, you can skip them. You have the right to refuse the interview or any questions asked during the interview. We will do everything we can to ensure that this is done in private. Furthermore, all study staff and interviewers are professionals with special training in these examinations/interviews. You may feel some discomfort during the process of providing a stool sample. In case of an injury, illness or complications related to this study, contact the study staff right away at the number provided at the end of this document. The study staff will treat you for minor conditions or refer you when necessary.

ARE THERE ANY BENEFITS BEING IN THIS STUDY?

You may benefit by receiving free *H. pylori* testing, counseling, health information etc. We will refer you to a hospital for care and support where necessary. Also, the information you provide will help us better understand the risk factors for the infection and thereby implement public health measures to prevent the infection. This information is a contribution to science and public health.

WILL BEING IN THIS STUDY COST YOU ANYTHING?

Participation in this research is free of charge.

WILL YOU GET REFUND FOR ANY MONEY SPENT AS PART OF THIS STUDY?

You will not receive any payment for participating in this study.

WHAT IF YOU HAVE QUESTIONS IN FUTURE?

If you have further questions or concerns about participating in this study, please call or send a text message to the study staff at the number provided at the bottom of this page.

For more information about your rights as a research participant you may contact the Secretary/Chairperson, Kenyatta National Hospital-University of Nairobi Ethics and Research Committee Telephone No. 2726300 Ext. 44102 email uonknh_erc@uonbi.ac.ke.

The study staff will pay you back for your charges to these numbers if the call is for study-related communication.

WHAT ARE YOUR OTHER CHOICES?

Your decision to participate in research is voluntary. You are free to decline participation in the study and you can withdraw from the study at any time without injustice or loss of any benefits.

CONSENT FORM (STATEMENT OF CONSENT)

Participant's statement

I have read this consent form or had the information read to me. I have had the chance to discuss this research study with a study counselor. I have had my questions answered in a language that I understand. The risks and benefits have been explained to me. I understand that my participation in this study is voluntary and that I may choose to withdraw any time. I freely agree to participate in this research study.

I understand that all efforts will be made to keep information regarding my personal identity confidential.

By signing this consent form, I have not given up any of the legal rights that I have as a participant in a research study.

I agree to participate in this research study:	**Yes**	**No**
I agree to have (define specimen) preserved for later study:	Yes	No
I agree to provide contact information for follow-up:	Yes	No

Participant printed name:____

Participant signature /Thumb stamp________________________ Date________________

Researcher's statement

I, the undersigned, have fully explained the relevant details of this research study to the participant named above and believe that the participant has understood and has willingly and freely given his/her consent.

Researcher's Name: Mustaf Ibrahim Haji Habibullah____________ **Date**: ________________

Signature ____

Role in the study: To know the prevalence of *H. pylori* infection among all age group ______*[i.e. study staff who explained informed consent form.]*

For more information contact ______________________at____________________from ___________________________to__________________________

APPENDIX III CHILD ASSENT FORM

Project Title: Prevalence and Factors Associated with Helicobacter Pylori Infection among Patients with Dyspepsia attending Kalkaal Hospital in Mogadishu, Somalia

Investigator(s): Dr. Mustaf Ibrahim

We are doing a research study about patients have dyspepsia and we wish to find out the number of patients who have *H. pylori* infection and the reason why some patients have it and others don't.

Permission has been granted to undertake this study by the Kenyatta National Hospital-University of Nairobi Ethics and Research Committee (KNH-UoN ERC Protocol No. **(P909/11/2021)**)

This research study is a way to learn more about people. At least 417patients will be participating in this research study with you.

If you decide that you want to be part of this study, you will be asked to provide a stool sample and answer some few questions about you, how you live and your environment**.**

There are some things about this study you should know. As you participate, you may feel some discomfort when providing the stool sample and also when answering the questions. We shall try to make sure that we don't make you feel uncomfortable throughout the process. We shall also make sure that whatever you tell us is kept private and no one else can access this information except those on the study team. If there are any questions you do not want to answer, you can skip them.

Much as this research may not benefit you directly, it will help us to know what we can do to stop other people from catching stomach ulcers. If you are found to have *H. pylori* infection, we shall give you treatment.

When we are finished with this study we will write a report about what was learned. This report will not include your name or that you were in the study.

You do not have to be in this study if you do not want to be. If you decide to stop after we begin, that's okay too. Your parents know about the study too.

If you decide you want to be in this study, please sign your name.

I,____, want to be in this research study.

__________________________________ ____________________

(Signature/Thumb stamp) **(Date)**