Scientific Research Publishing

Open Journal of Gastroenterology, 2025, 15(10), 581-589
https://www.scirp.org/journal/ojgas
ISSN Online: 2163-9469
ISSN Print: 2163-9450

Epidemiological and Diagnostic Profile of Peptic Ulcer Disease in Upper Gastrointestinal Endoscopy in Libreville

Patrice Emery Itoudi Bignoumba, Francine Tchoffo, Inès Flore Maganga Moussavou, Patrick Dieudonné Nzouto, Agnès Angéla Engoang, Arlette Nsegué Mezuie, Monique Mbounja, Jean Baptiste Moussavou Kombila

Hepato-Gastroenterology Department, CHU Libreville, Libreville, Gabon
Email: Itoudi_emery@hotmail.com

How to cite this paper: Itoudi Bignoumba, P.E., Tchoffo, F., Maganga Moussavou, I.F., Nzouto, P.D., Engoang, A.A., Nsegué Mezuie, A., Mbounja, M. and Moussavou Kombila, J.B. (2025) Epidemiological and Diagnostic Profile of Peptic Ulcer Disease in Upper Gastrointestinal Endoscopy in Libreville. *Open Journal of Gastroenterology*, **15**, 581-589.
https://doi.org/10.4236/ojgas.2025.1510053

Received: September 2, 2025
Accepted: September 20, 2025
Published: September 23, 2025

Abstract

Introduction: Peptic ulcer disease (PUD) results from an imbalance between aggressive and protective factors of the gastric and duodenal mucosa. Helicobacter pylori (*H. pylori*) is a common etiology. However, the African paradox exists, where the WHO-estimated 80% prevalence of *H. pylori* is not statistically associated with a high frequency of PUD. This study aimed to investigate the relationship between *H. pylori* and PUD in Libreville. **Methodology:** We conducted a 3-year prospective study (2017-2019) at the Libreville University Teaching Hospital (CHUL), including 167 patients with PUD confirmed by endoscopy and biopsies. Epidemiological, clinical, therapeutic, and histological data were analyzed using Epi Info 7.2, with statistical tests (Chi-square, Fisher). **Result:** *H. pylori* was present in 79.3% of patients. PUD predominantly occurred in patients with low socioeconomic status (66.5%). Regarding patients with negative *H. pylori*, there was a significant association with NSAID use ($p = 0.0001$), stress ($p = 0.0108$), and alcohol consumption ($p = 0.0242$). Gastric ulcers (61.1%) were most frequent, with chronic gastritis (89.2%) as the predominant histological lesion. **Conclusion:** While *H. pylori* remains a major factor in peptic ulcer in Libreville, our study highlights the important role of NSAIDs, stress, and alcohol. These findings underscore an epidemiological shift and call for integrated management strategies addressing both infectious and non-infectious risk factors.

Keywords

Gastric Ulcer, *Helicobacter pylori*, Libreville

1. Introduction

Peptic ulcer is a localized loss of mucosa in the gastric and/or duodenal wall, reaching deep into the muscularis [1]. It results from an imbalance between aggressive and protective factors of the gastroduodenal wall [1]-[3]. Of the several etiologies of PUD, the most frequent is chronic *H. pylori* infection, as shown by the study of Marshall and Warren [4]. However, *H. pylori* seems to decrease with the level of industrialization of the country [5] [6] and highlights the African paradox where the prevalence of *H. pylori,* estimated at 80% by the World Health Organization (WHO), is not associated with a higher frequency of peptic ulcer [7] [8]. It is in this light that we question the impact of *H. pylori* on the occurrence of PUD in Libreville.

The aim of this study was to establish the epidemiological and etiological profile of patients with PUD and investigate the relationship between *H. pylori* and PUD in Libreville.

2. Patients and Method

This is a prospective cross-sectional study carried out in the Hepato-Gastro-Enterology (HGE) Department of the Libreville University Teaching Hospital. The study was conducted between 1 January 2017 and 31 December 2019. The study population involved all patients undergoing oesogastroduodenal endoscopy (OGDE). We included patients with gastric and/or duodenal ulcers who gave their consent and had undergone at least two antral and two fundic biopsies for *H. pylori*, and for whom we had pathological results. We excluded patients who had previously received antibiotic therapy less than 1 month and those who had received a proton pump inhibitor (PPI) less than 2 weeks previously. Using a standardized form, we collected epidemiological data (age, sex, socio-economic level), clinical data (pain characteristics), indication for OGDE, personal history (PUD, notion of *H. pylori* gastritis, upper digestive hemorrhage, gastro-esophageal reflux disease (GORD), stroke, diabetes, sickle cell anemia, HIV infection, hepatitis B, hepatitis C, cirrhosis), family history (history of PUD, history of stomach cancer), history of taking gastrotoxic substances (anti-inflammatories, anti-platelet aggregates, anti-coagulants, traditional concoctions, tobacco, alcohol), notion of stress, results of OGDE (number of PUD, location and site, Forrest classification, associated endoscopic lesions), anatomopathological results of biopsies (presence or absence of *H. pylori,* acute gastritis, chronic gastritis, dysplasia, metaplasia, cancer). Data were entered and analyzed using Epi Info 7.2 statistical software. Qualitative variables were expressed as percentages and quantitative variables as means and standard deviation. Comparisons or associations between categorical variables were analyzed using Pearson's chi-square test corrected according to Yates and Fisher to account for the number of patients. Confidence intervals were expressed at the alpha error threshold equal to 0.05. Variables whose association with the expected event had a p-value of less than 0.05 were considered statistically significant.

3. Results

3.1. Epidemiological Data on Gastric and/or Duodenal Ulcers

During the study period, 1407 patients underwent OGDE and 227 developed gastric and/or duodenal ulcers, representing a hospital frequency of 16.1%. Of the 227 patients, 60 were excluded because biopsies were absent, or they took antibiotics or PPIs (Figure 1).

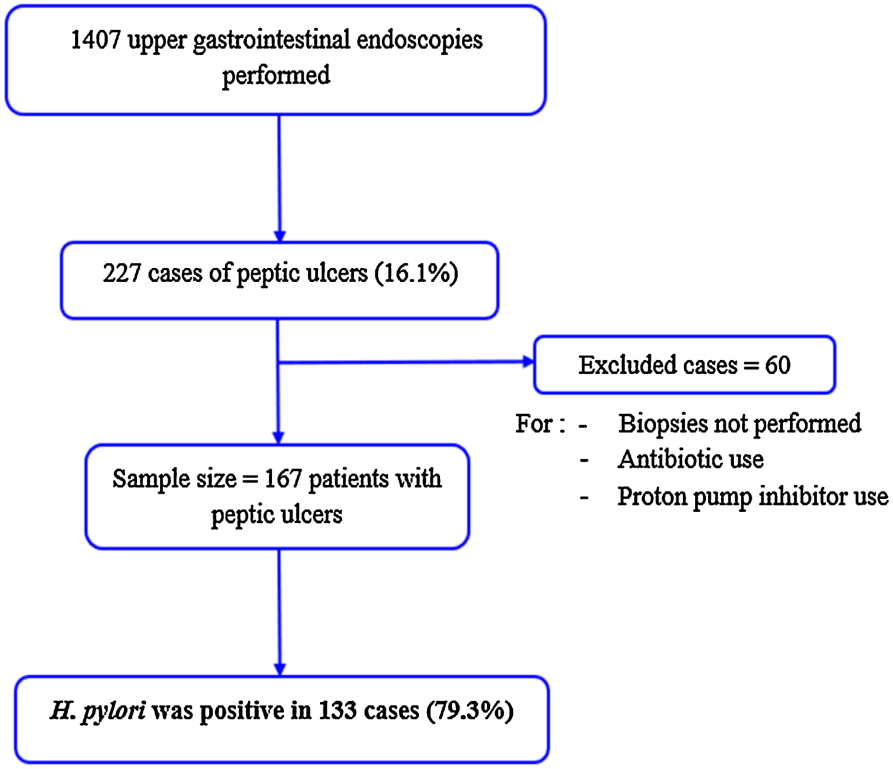

Figure 1. Flow chart.

The analysis included 167 patients with PUD. There were 88 men and 79 women, giving a sex ratio of approximately 1.1. The mean age of the patients was 48.7 (±17.3) years. *H. pylori* testing was positive in 133 of the 167 patients included, representing a hospital frequency of 79.3%. The mean age of patients positive for *H. pylori* was 48.4 (±17.2) years compared to 50.0 (±18.0) years for patients negative for *H. pylori.* This difference was not statistically significant (p = 0.697). Both groups were predominantly male.

3.2. Diagnostic Data

Typical ulcer pain was present in 69.2% of patients in the *H. pylori* positive group and 79.4% in the *H. pylori* negative group. This difference was not statistically significant (p = 0.2919).

Cardiovascular and hepatic pathologies were present to a similar extent in both groups (p = 0.1984). Regarding drug consumption, 6.8% of the *H. pylori* positive group and 38.2% of the *H. pylori* negative group reported taking NSAIDs. This

difference was statistically significant (p = 0.00014) (Table 1).

Table 1. Distribution of patients with gastric and/or duodenal ulcers according to *H. pylori* status and medication taken.

	Number of cases		*H. pylori* positive		*H. pylori* negative		p
	n	%	n	%	n	%	
Non-steroidal anti-inflammatory drugs							0.00014
Yes	22	13.2	9	6.8	13	38.2	
No	145	86.8	124	93.2	21	61.8	
Total	167	100.0	133	100.0	34	100.0	
Antiretrovirals							1.0000
Yes	6	3.6	5	3.8	1	2.9	
No	161	96.4	128	96.2	33	97.1	
Total	167	100.0	133	100.0	34	100.0	
Oral hypoglycemics							0.6889
Yes	10	6.0	9	6.8	1	2.9	
No	157	94.0	124	93.2	33	97.1	
Total	167	100.0	133	100.0	34	100.0	
Anti-hypertensive							0.4683
Yes	32	19.2	24	18.0	8	2.9	
No	135	80.8	109	82.0	26	97.1	
Total	167	100.0	133	100.0	34	100.0	

Stress was found in 15.8% and 32.3% of patients with positive and negative *H. pylori* testing, respectively. This difference was statistically significant (p = 0.0108). Table 2 shows that work-related stress was the most frequent and had a higher frequency in the *H. pylori* negative group. This difference was statistically significant (p = 0.003). Among patients who did not experience stress, 84.2% were *H. pylori* positive and 64.7% were *H. pylori* negative. This difference was also statistically significant (p = 0.0108).

Table 2. Distribution of patients with gastric and/or duodenal ulcers according to stress and *H. pylori* status.

	Number of cases		*H. pylori* positive		*H. pylori* negative		p
	n	%	n	%	n	%	
Professional	12	7.2	5	3.8	7	20.6	0.003
Family	10	6.0	6	4.5	4	11.8	0.121
Professional + Family	4	2.4	4	3.0	0	0.0	0.583
Social	3	1.8	3	2.3	0	0.0	1.000

Continued

Social + Family	2	1.2	2	1.5	0	0.0	1.000
Professional + Social + Family	2	1.2	1	0.8	1	2.9	0.367
No stress	134	80.2	112	84.2	22	64.7	0.011
Total	*167*	*100.0*	*133*	*100.0*	*34*	*100.0*	

The proportion of patients reporting active alcohol consumption was 11.3% in the *H. pylori* positive group and 26.5% in the *H. pylori* negative group. This difference was statistically significant (p = 0.0242) as shown in Table 3.

Table 3. Distribution of patients with gastric and/or duodenal ulcers according to *H. pylori* status and alcohol and tobacco consumption.

	Number of cases		*H. pylori* positive		*H. pylori* negative		p
	n	%	n	%	n	%	
Alcohol							0.0242
Yes	24	14.4	15	11.3	9	26.5	
No	143	85.6	118	88.7	25	73.5	
Total	167	100.0	133	100.0	34	100.0	
Tobacco							0.1844
Yes	4	2.4	2	1.5	2	5.9	
No	163	97.6	131	98.5	32	94.1	
Total	167	100.0	133	100.0	34	100.0	

Based on endoscopic evaluations, the location of the ulcer was gastric in 102 patients (61.1%), duodenal in 32 patients (19.2%), and gastroduodenal in 34 patients (20.3%). Gastric ulcers were present in 61.6% of patients positive for *H. pylori* and 55.9% of patients negative for *H. pylori*. This difference was not statistically significant (p = 0.489). In order of frequency, the site of the ulcer was antral (33.5%), fundic (21%), bulbar (19.2%), antral and bulbar (12%), antral and fundic (6%), fundic and antro-bulbar (4.8%), and fundic and bulbar (3.6%). According to *H. pylori* status, there was a statistically significant difference between the antral site (38.3%) and the fundal site (13.5%), with 22.6% and 13.5% respectively (p = 0.019 and p = 0.020). According to *H. pylori* status, the antral site with 38.3% and 13.5% in patients with positive and negative *H. pylori*, respectively, as well as the fundic site with 22.6% and 13.5%, showed a statistically significant difference (p = 0.019 and p = 0.020) (Table 4).

Forrest classification was, in order of frequency, stage III (65.3%), stage IIb (16.8%), stage IIc (15.0%), stage IIa (1.8%) and stage Ib (1.2%). There was no statistical difference according to *H. pylori* status.

Table 4. Distribution of patients according to site of gastric and/or duodenal ulcer and *H. pylori* status.

	Number of cases		*H. pylori positive*		*H. pylori negative*		p
	n	%	n	%	n	%	
Antrum	56	33.5	51	38.3	5	13.5	0.019
Fundus	35	21.0	30	22.6	5	13.5	0.020
Bulb	32	19.2	20	15.0	12	32.4	0.779
Antrum + fundus	10	6.0	8	6.0	2	5.4	1.000
Antrum + bulb	20	12.0	14	10.5	6	16.2	1.000
Fundus + bulb	6	3.6	2	1.5	4	10.8	0.575
Fundus + antrum + bulb	8	4.8	8	6.0	0	0.0	0.115
Total	167	100.0	133	100.0	37	100.0	

In terms of pathology, all patients had a histological abnormality. The most frequent histological lesion was chronic gastritis with 90.2% in the *H. pylori* positive group and 79.4% in the *H. pylori* negative group (p = 0.371). Acute gastritis lesions were 6.8% in the *H. pylori* positive group and 17.6% in the *H. pylori* negative group. This difference was statistically significant (p = 0.047). For precancerous lesions, dysplasia represented 1.8% and metaplasia 9.6% with no statistically significant difference between the two groups (p = 0.384 and p = 0.023). We observed one gastric adenocarcinoma in the *H. pylori* positive group and none in the other group.

4. Discussion

Study limitations

The fact that immunohistochemistry is not routinely used to detect *H. pylori* infection means that the incidence of this condition may be underestimated. However, pathological analysis remains the gold standard for diagnosing *H. pylori* infection [9].

Epidemiology

The hospital incidence of PUD was 16.1% in upper GI endoscopy at the Libreville University Teaching Hospital. This frequency, similar to that found in Togo (15.53%) and Cameroon (17.1%), was higher than that reported by Perret *et al.* in Gabon (8.75%) and Diouf *et al.* in Senegal [10]-[13]. These differences support the observation by Archampong *et al.* in Ghana that the development of digestive endoscopy in Africa has led to a significant increase in the number of cases in recent publications [14]. The mean age was 48.7 ± 17.3 years, similar to that found in all African countries [10]-[15]. However, it remains lower than in studies conducted in the Western countries, where it is 15 years to 20 years higher [16] [17]. The male predominance of patients with PUD (52.7%) is consistent with the

literature [10]-[18]. Analysis of the socioeconomic profile reveals a predominance of the disease in participants with low socioeconomic status (65.9%). This same observation was made in Cameroon, including 64.9% of patients with PUD by Ankouane *et al.* [15]. Diagnosis Clinically, the typical ulcer syndrome was present in 71.3% of patients. It was also observed in 64.3% of patients in Congo by Ontsira *et al.* and in 64.52% of patients in Togo by Lawson *et al.* [10] [19]. The most common location of the ulcer was gastric (55.1%), whereas it was duodenal in the other African studies [10] [11] [13]-[15]. This gastric location of PUD in Gabon was already highlighted by Perret *et al.* more than 30 years ago [12].

In terms of etiology, *H. pylori* infection was present in 79.6% of patients. Although this prevalence is close to that found in Cameroon (72.5%), Congo (72%), Togo (70.41%), and Senegal (83.6%), it contrasts with previous Gabonese data which found a prevalence of *H pylori* of 37.5% in endoscopy and 62% in pediatrics [10]-[13] [15] [21]-[23]. This difference with previous Gabonese data is probably a consequence of methodological differences. We excluded patients who had taken antibiotics and PPIs, which was not the case in previous studies. In addition, comparative analysis of other known risk factors for PUD revealed a statistically significant difference between the absence of *H. pylori* infection and the use of NSAIDs ($p = 0.00014$), as well as alcohol consumption ($p = 0.0242$) and occupational stress ($p = 0.003$). These other risk factors, although poorly identified in African studies, are among the most common causes of PUD in the West [10]-[20]. Similarly, the predominant gastric location of positive *H. pylori* PUD and the predominant duodenal location of PUD without *H. pylori*, reveals a decrease in gastric protective factors during *H. pylori* infection and an increase in aggressive factors when taking NSAIDs, alcohol, and occupational stress [16]-[18].

5. Conclusion

PUD is a frequent pathology in upper digestive endoscopy in Libreville, preferentially affecting men of low socioeconomic status aged under 50. It is revealed by a typical PUD syndrome with a common endoscopic site in the stomach. *H. pylori* infection is the main etiology, although the use of NSAIDs, alcohol, and occupational stress are risk factors that need to be clarified in our context.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Pioche, M., Jacques, J., Saurin, J.C. and Varon, C. (2019) Gastric and Duodenal Ulcers, Gastritis. *La Revue du Praticien*, **69**, 147-154.

[2] Holle, G.E. (2010) Pathophysiology and Modern Treatment of Ulcer Disease (Review). *International Journal of Molecular Medicine*, **25**, 483-491. https://doi.org/10.3892/ijmm_00000368

[3] Søreide, K. (2016) Current Insight into Pathophysiology of Gastroduodenal Ulcers:

Why Do Only Some Ulcers Perforate? *Journal of Trauma and Acute Care Surgery*, **80**, 1045-1048. https://doi.org/10.1097/ta.0000000000001035

[4] Marshall, B. and Warren, J.R. (1984) Unidentified Curved Bacilli in the Stomach of Patients with Gastritis and Peptic Ulceration. *The Lancet*, **323**, 1311-1315. https://doi.org/10.1016/s0140-6736(84)91816-6

[5] Hooi, J.K.Y., Lai, W.Y., Ng, W.K., Suen, M.M.Y., Underwood, F.E., Tanyingoh, D., *et al.* (2017) Global Prevalence of *Helicobacter pylori* Infection: Systematic Review and Meta-Analysis. *Gastroenterology*, **153**, 420-429. https://doi.org/10.1053/j.gastro.2017.04.022

[6] Nagy, P., Johansson, S. and Molloy-Bland, M. (2016) Systematic Review of Time Trends in the Prevalence of *Helicobacter pylori* Infection in China and the Usa. *Gut Pathogens*, **8**, Article No. 8. https://doi.org/10.1186/s13099-016-0091-7

[7] Smith, S., Fowora, M. and Pellicano, R. (2019) Infections with *Helicobacter pylori* and Challenges Encountered in Africa. *World Journal of Gastroenterology*, **25**, 3183-3195. https://doi.org/10.3748/wjg.v25.i25.3183

[8] Graham, D.Y., Lu, H. and Yamaoka, Y. (2009) African, Asian or Indian Enigma, the East Asian *Helicobacter pylori*: Facts or Medical Myths. *Journal of Digestive Diseases*, **10**, 77-84. https://doi.org/10.1111/j.1751-2980.2009.00368.x

[9] Pohl, D., Keller, P.M., Bordier, V. and Wagner, K. (2019) Review of Current Diagnostic Methods and Advances in *Helicobacter pylori* Diagnostics in the Era of Next Generation Sequencing. *World Journal of Gastroenterology*, **25**, 4629-4660. https://doi.org/10.3748/wjg.v25.i32.4629

[10] Lawson-Ananissoh, L.M., Bouglouga, O., Bagny, A., El-Hadj Yakoubou, R., Kaaga, L. and Redah, D. (2015) Epidemiological Profile of Peptic Ulcers at the Campus University Teaching Hospital in Lomé (Togo). *Journal Africain d'Hépato-Gastroentérologie*, **9**, 99-103. https://doi.org/10.1007/s12157-015-0597-5

[11] Eloumou Bagnaka, S.A.F., Luma Namme, H., Noah Noah, D., Essomba, N.E., Malongue, A., Manga, A., *et al.* (2016) Risk Factors Associated with Gastroduodenal Lesions in a Douala Referral Hospital (Cameroon). *Médecine et Santé Tropicales*, **26**, 104-109. https://doi.org/10.1684/mst.2015.0521

[12] Perret, J.L. and Nguemby-Mbina, C. (1991) Epidemiological, Clinical, and Evolutionary Aspects of Peptic Ulcers in Gabonese Subjects. *Medecine d'Afrique Noire*, **38**, 828-832.

[13] Diouf, M., Ondélé-Ipongo, A., Dia, D., Bassène, M., Mbengue, M. and Seck, A. (2010) Évolution de la prévalence des ulcères gastroduodénaux dans le centre d'endoscopie digestive de l'hôpital Aristide-Le-Dantec de Dakar. *Journal Africain d'Hépato-Gastroentérologie*, **5**, 23-27. https://doi.org/10.1007/s12157-010-0236-4

[14] Archampong, T.N., Asmah, R.H., Richards, C.J., Martin, V.J., Bayliss, C.D., Botão, E., *et al.* (2019) Gastro-Duodenal Disease in Africa: Literature Review and Clinical Data from Accra, Ghana. *World Journal of Gastroenterology*, **25**, 3344-3358. https://doi.org/10.3748/wjg.v25.i26.3344

[15] Andoulo, F.A., Noah Noah, D., Tagni-Sartre, M., Ndjitoyap, E.C. and Ngu Blackett, K. (2013) Epidemiology of *Helicobacter pylori* Infection in Yaoundé: From Peculiarity to African Enigma. *Pan African Medical Journal*, **16**, 115-120. https://doi.org/10.11604/pamj.2013.16.115.3007

[16] Graham, D.Y. (2014) History of *Helicobacter pylori*, Duodenal Ulcer, Gastric Ulcer and Gastric Cancer. *World Journal of Gastroenterology*, **20**, 5191-5204. https://doi.org/10.3748/wjg.v20.i18.5191

[17] Dunlap, J.J. and Patterson, S. (2019) Peptic Ulcer Disease. *Gastroenterology Nursing*, **42**, 451-454. https://doi.org/10.1097/sga.0000000000000478

[18] Katelaris, P., Hunt, R., Bazzoli, F., Cohen, H., Fock, K.M., Gemilyan, M., *et al.* (2022) *Helicobacter pylori* World Gastroenterology Organization Global Guideline. *Journal of Clinical Gastroenterology*, **57**, 111-126. https://doi.org/10.1097/mcg.0000000000001719

[19] Ontsira Ngoyi, E.N., Atipo Ibara, B.I., Moyen, R., Ahoui Apendi, P.C., Ibara, J.R., Obengui, O., *et al.* (2015) Molecular Detection of *Helicobacter pylori* and Its Antimicrobial Resistance in Brazzaville, Congo. *Helicobacter*, **20**, 316-320. https://doi.org/10.1111/hel.12204

[20] Graham, D.Y., Dang, B.N. and El-Serag, H.B. (2019) *Helicobacter pylori* Infection. *The New England Journal of Medicine*, **381**, 587-588.

[21] Itoua-Ngaporo, N.A., Mimiesse Monamou, J.F., Ahoui Apendi, C., Mongo Onkouo, A., Atipo Ibara Ollandzobo, L.C., Ngalessami Mouakosso, M., *et al.* (2018) Epidemiological, Clinical and Endoscopic Profile of *Helicobacter pylori* Infection in Two Digestive Endoscopy Centers in Brazzaville. *Gastroenterology, Hepatology & Digestive Disorders*, **1**, 1-5.

[22] Itoudi Bignoumba, P.E., Maganga Moussavou, I.F., Ziza, N., Nzouto, P.D., Saibou, M. and Moussavou Kombila, J.B. (2019) *Hélicobacter pylori* au Centre Hospitalier de Libreville: Aspect épidémiologique et clinique à propos de 728 cas. *Health Sciences and Diseases*, **20**, 64-68.

[23] Ategbo, S., Rogombe, S.M., Ngoungou, E.B., Midili, T.L. and Moussavou, A. (2013) Epidemiology of *Helicobacter pylori* Infection in Children Aged 6 Months to 7 Years in Libreville, Gabon. *Clinics in Mother and Child Health*, **10**, 1-5. https://doi.org/10.4303/cmch/c120901