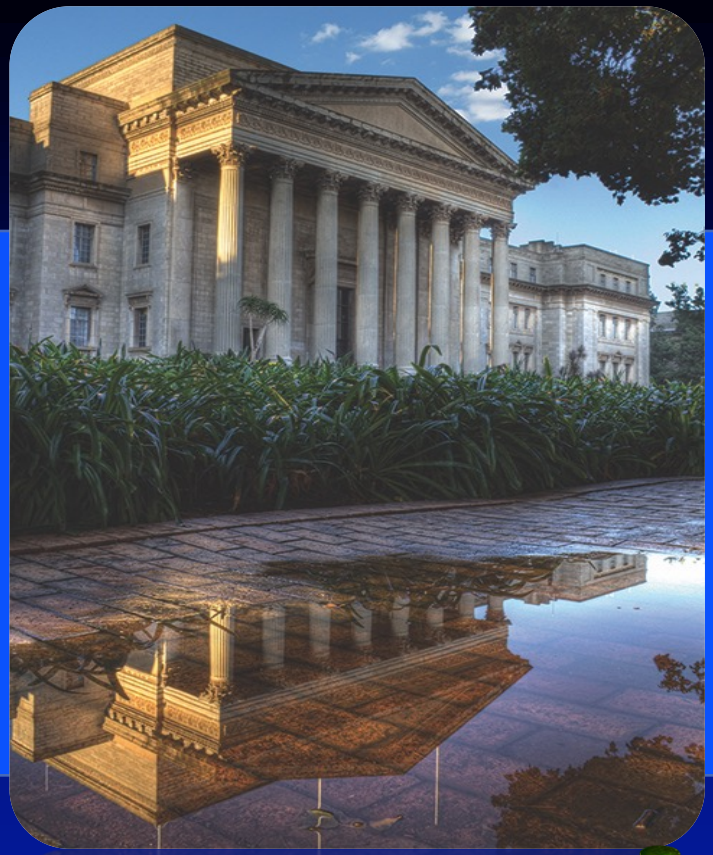
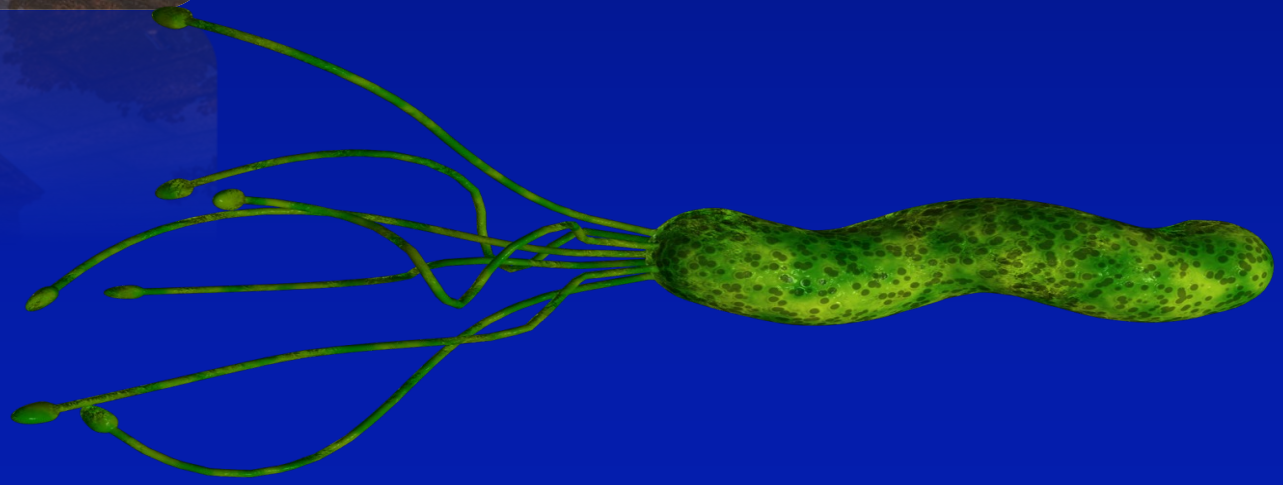
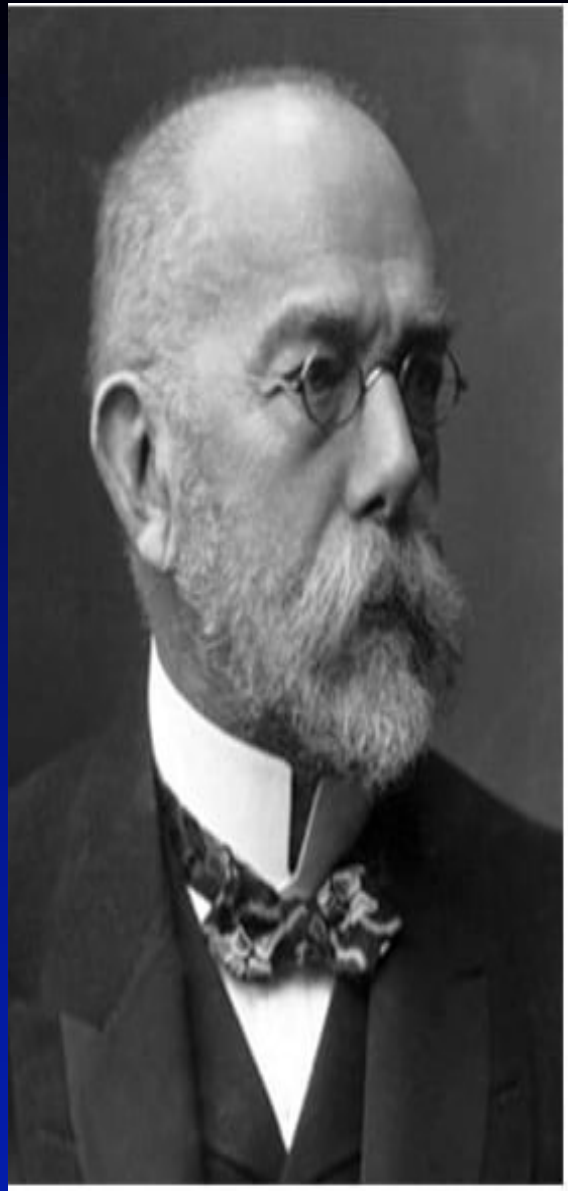


Helicobacter Pylori

The Soweto Experience





Robert Koch (1843 - 1910)

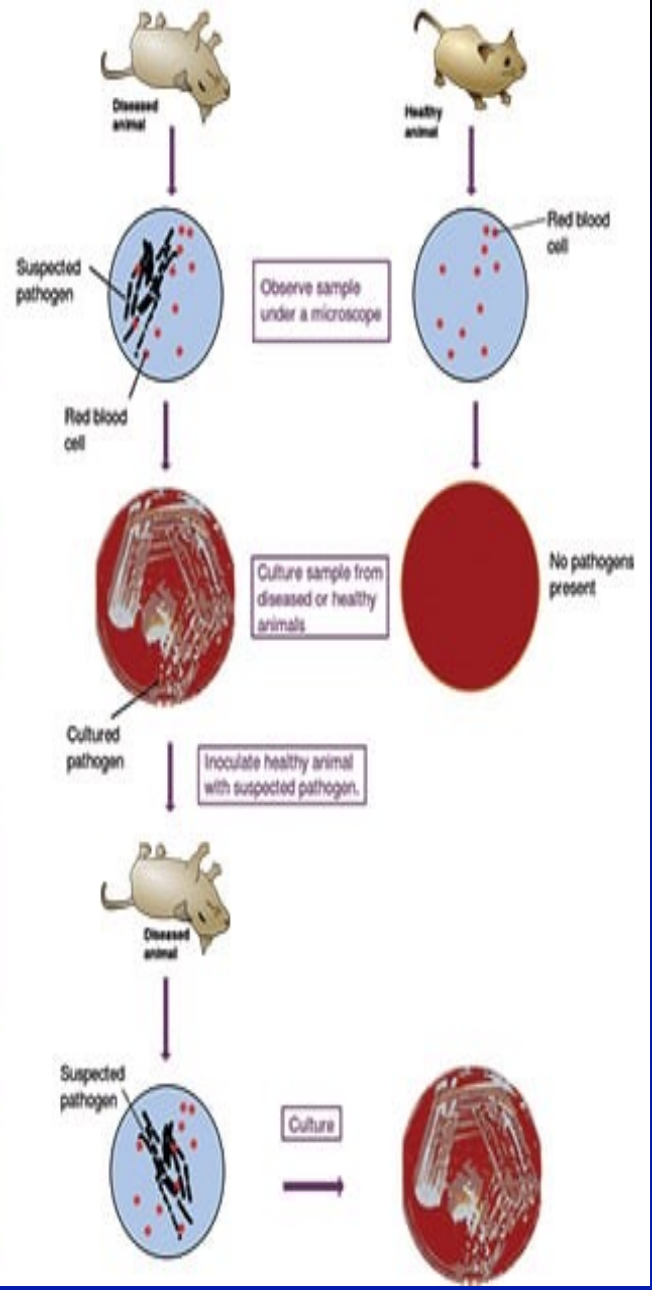
Koch's Postulates:

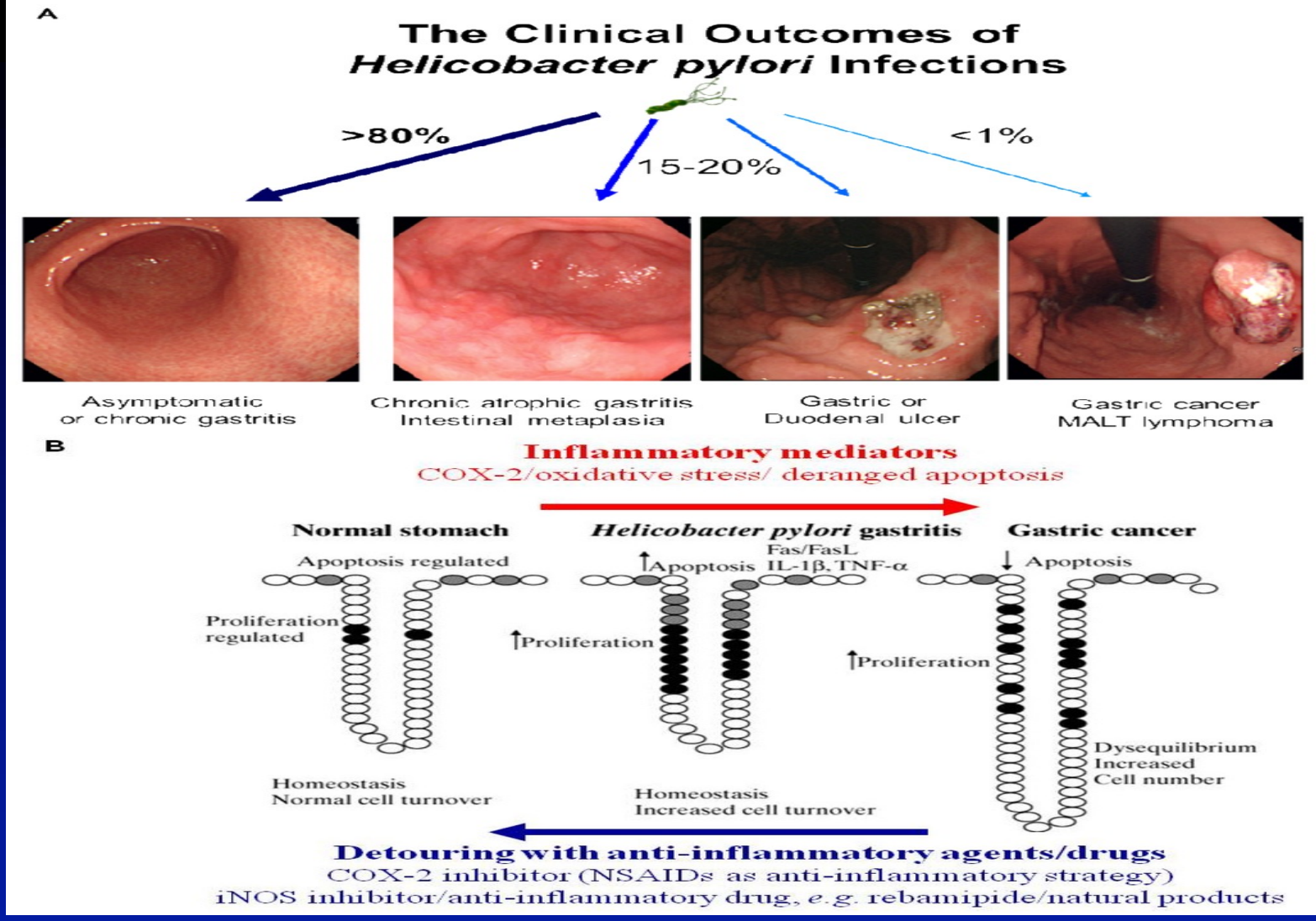
① The microorganism must be found in abundance in all organisms suffering from the disease, but should not be found in healthy organisms.

② The microorganism must be isolated from a diseased organism and grown in pure culture.

③ The cultured microorganism should cause disease when introduced into a healthy organism.

④ The microorganism must be reisolated from the inoculated, diseased experimental host and identified as being identical to the original specific causative agent.





Cancers **2011**, *3*, 3018-3028; doi:10.3390/cancers3033018

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cancers

ISSN 2072-6694

www.mdpi.com/journal/cancers

Review

Detouring the Undesired Route of *Helicobacter pylori*-Induced Gastric Carcinogenesis

Eun-Hee Kim [1], Kyung-Sook Hong [1], Hua Hong [1] and Ki Baik Hahm [1,2,*]

Original research

Mass eradication of *Helicobacter pylori* to reduce gastric cancer incidence and mortality: a long-term cohort study on Matsu Islands

Tsung-Hsien Chiang,[1,2,3] Wei-Jung Chang,[4] Sam Li-Sheng Chen,[5]

2020

Design Mass eradication of *H. pylori* infection was launched in 2004 and continued until 2018 for a high-risk Taiwanese population aged 30 years or older dwelling on Matsu Islands with prevalent *H. pylori* infection. Test positives for the ^{13}C-urea breath test

National Institutes of Health (NIH) (.gov)
https://www.ncbi.nlm.nih.gov › articles › PMC7815911

Mass eradication of Helicobacter pylori to reduce gastric ...

by TH Chiang · 2021 · Cited by 201 — Although mass eradication of Helicobacter pylori has been proposed as a means to **eliminate gastric cancer**, its long-term effects remain unclear.

control period from 1995 to 2003, the effectiveness in reducing gastric cancer incidence and mortality during the chemoprevention period was 53% (95% CI 30% to 69%, p<0.001) and 25% (95% CI −14% to 51%, p=0.18), respectively. No significant changes were noted in the incidence

postulated that it may increase acid reflux to the oesophagus and even lead to an increase in oesophageal adenocarcinoma.[14] Antibiotic treatment also has the potential to disrupt the gut microbiota, which might produce unknown consequences.[15] Finally, there is concern regarding its effect on global antimicrobial resistance or emergence of antibiotic-resistant strains of *H. pylori*.[16 17] These potentially collateral effects of *H. pylori* eradication are largely theoretical and real-world evidence is urgently needed to throw light on the benefits and harms.

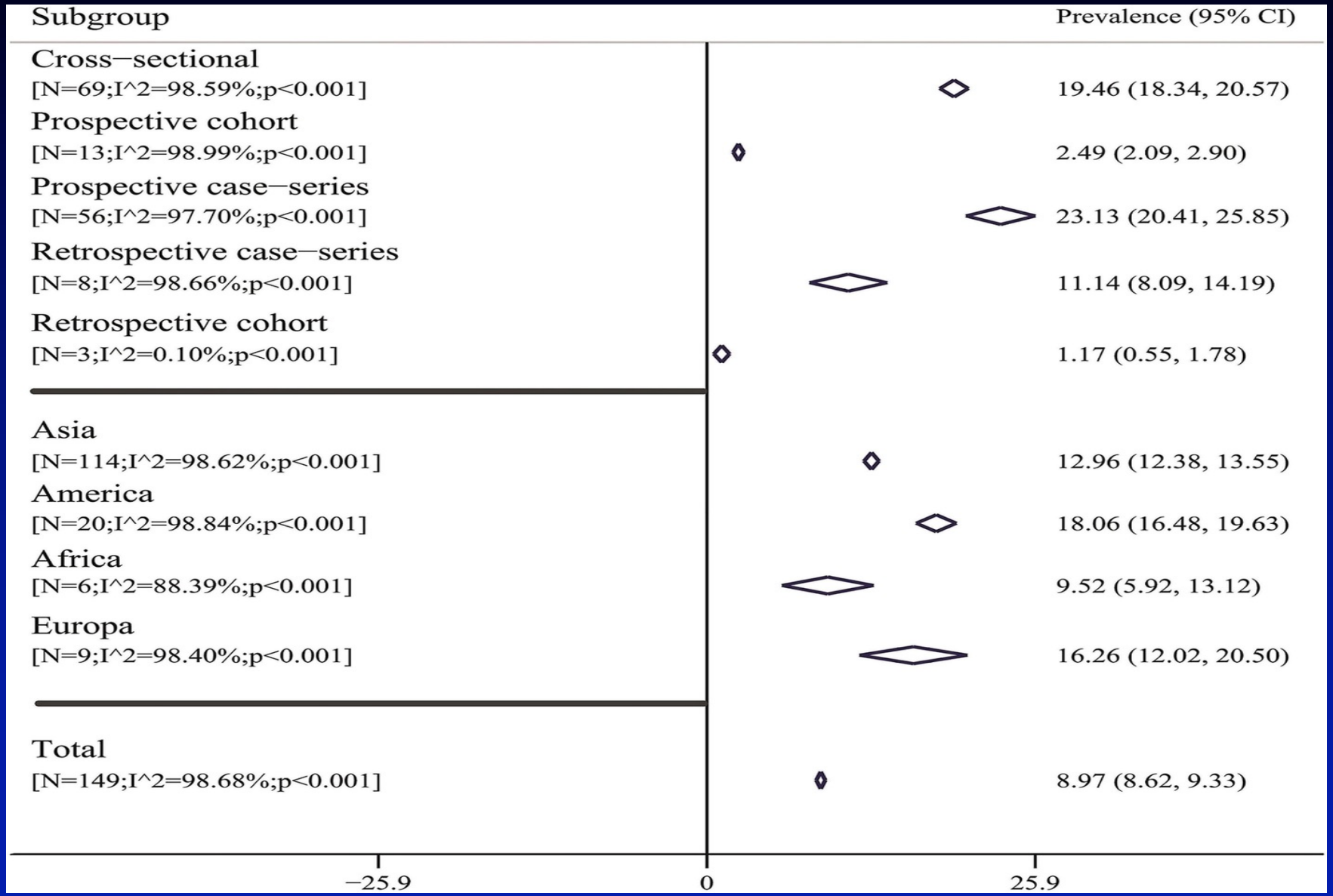
Shirani et al. BMC Infectious Diseases (2023) 23:543
https://doi.org/10.1186/s12879-023-08504-5

BMC Infectious Diseases

RESEARCH Open Access

The global prevalence of gastric cancer in *Helicobacter pylori*-infected individuals: a systematic review and meta-analysis

Maryam Shirani[1†], Reza Pakzad[2,3†], Mohammad Hossein Haddadi[4], Sousan Akrami[5,6], Arezoo Asadi[7], Hossein Kazemian[4], Melika Moradi[6], Vahab Hassan Kaviar[8], Abolfazl Rafati Zomorodi[9], Saeed Khoshnood[3,4], Mahnaz Shafieian[10], Ronia Tavasolian[11], Mohsen Heidary[12,13*] and Morteza Saki[6*]


Subgroup
Prevalence (95% CI)
Cross−sectional
[N=69;I^2=98.59%;p<0.001]
19.46 (18.34, 20.57)
Prospective cohort
[N=13;I^2=98.99%;p<0.001]
2.49 (2.09, 2.90)
Prospective case−series
[N=56;I^2=97.70%;p<0.001]
23.13 (20.41, 25.85)
Retrospective case−series
[N=8;I^2=98.66%;p<0.001]
11.14 (8.09, 14.19)
Retrospective cohort
[N=3;I^2=0.10%;p<0.001]
1.17 (0.55, 1.78)
Asia
[N=114;I^2=98.62%;p<0.001]
12.96 (12.38, 13.55)
America
[N=20;I^2=98.84%;p<0.001]
18.06 (16.48, 19.63)
Africa
[N=6;I^2=88.39%;p<0.001]
9.52 (5.92, 13.12)
Europa
[N=9;I^2=98.40%;p<0.001]
16.26 (12.02, 20.50)
Total
[N=149;I^2=98.68%;p<0.001]
8.97 (8.62, 9.33)
−25.9
0
25.9

hygiene

MDPI

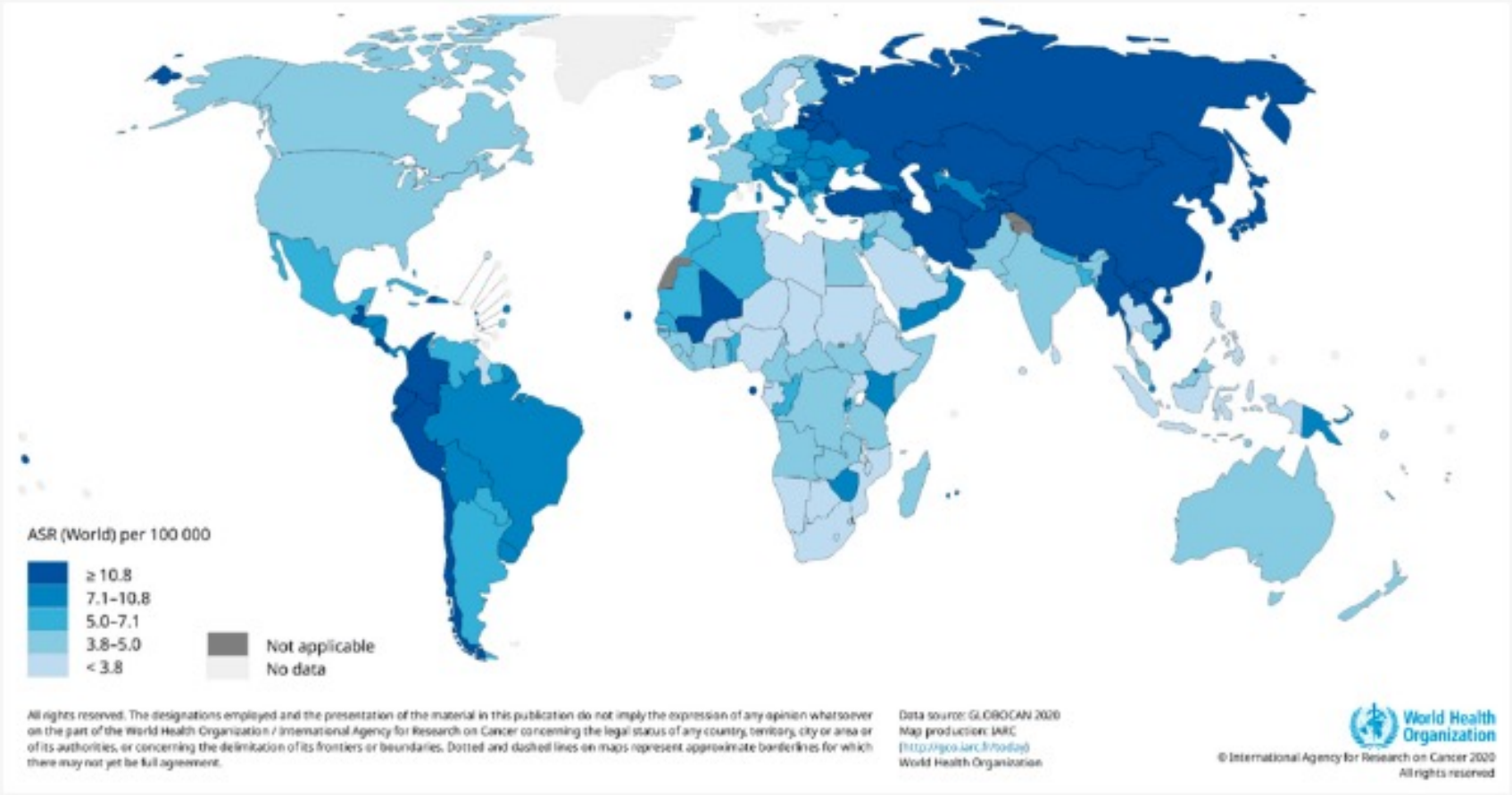
Review

Gastric Cancer Epidemiology: Current Trend and Future Direction

Chidozie Declan Iwu [1,*] and Chinwe Juliana Iwu-Jaja [2]

1 School of Health Systems and Public Health, Faculty of Health Sciences, University of Pretoria, Pretoria 0001, South Africa
2 Department of Global Health, Stellenbosch University, Stellenbosch 7602, South Africa
* Correspondence: chidoziedelan@gmail.com

Figure 1. Estimated age-standardized global incidence rates of GC in 2020. Source: [9].



J Gastroenterol (2020) 55:281–288
https://doi.org/10.1007/s00535-019-01639-w

The Japanese of Gastroente

ORIGINAL ARTICLE—ALIMENTARY TRACT

Risk of gastric cancer in the second decade of follow-up after *Helicobacter pylori* eradication

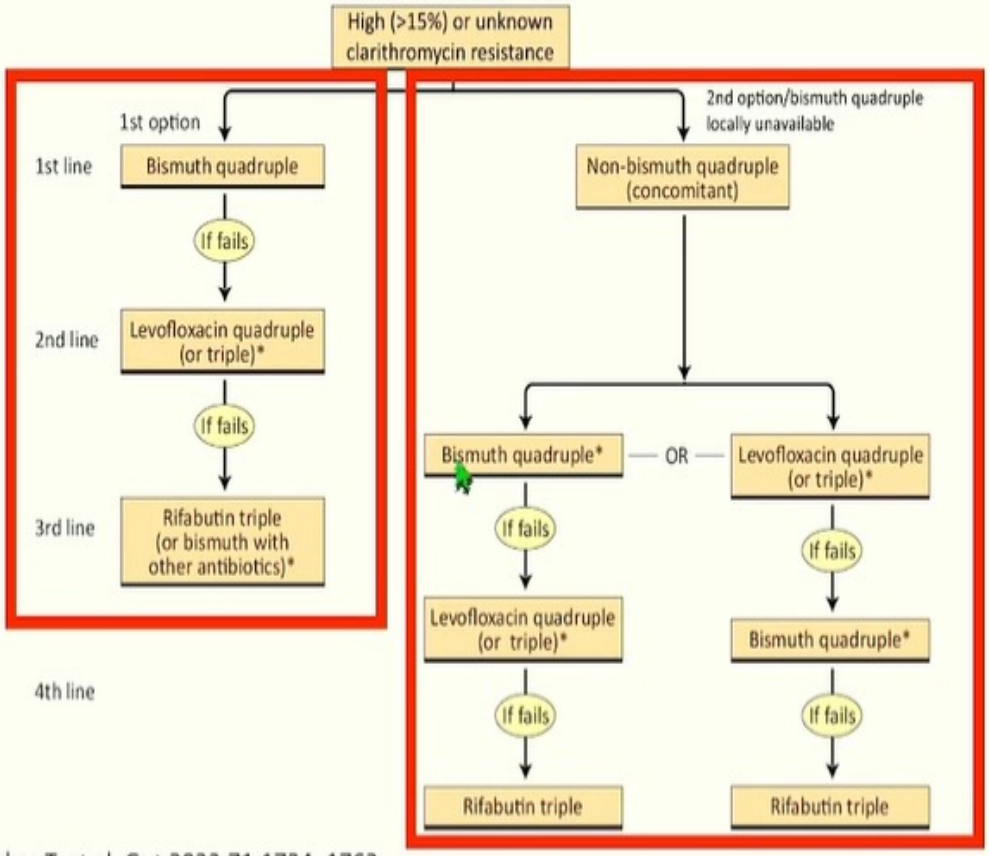
Susumu Take[1,3] · Motowo Mizuno[2] · Kuniharu Ishiki[3] · Chiaki Kusumoto[3] · Takayuki Imada[3] · Fumihiro Hamada[4] · Tomowo Yoshida[3] · Kenji Yokota[5] · Toshiharu Mitsuhashi[6] · Hiroyuki Okada[7]

Conclusions

The longer the follow-up, the greater the risk of developing diffuse-type gastric cancer becomes in patients with mild-to-moderate gastric atrophy at baseline. Endoscopic surveillance should be continued beyond 10 years after cure of *H. pylori* irrespective of the severity of gastric atrophy.

ueg week

Management of Helicobacter Pylori Infection



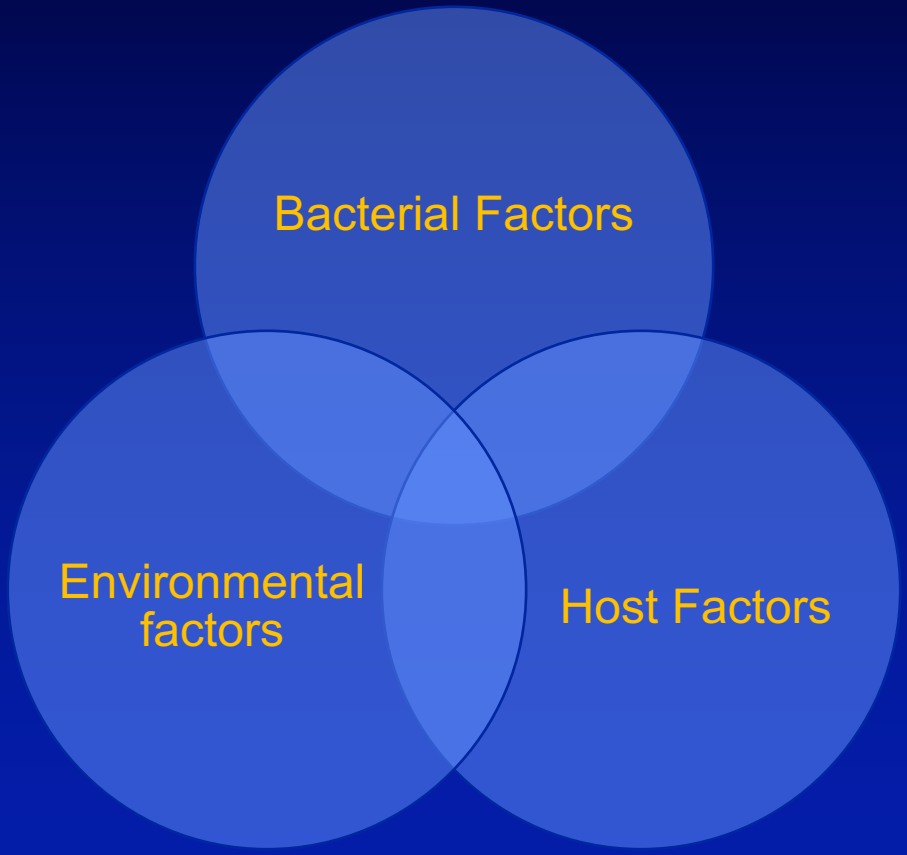
ueg.eu

Malfertheiner P, Megraud F, Rokkas T, et al. Gut 2022;71:1724–1762.

Laboratory Techniques

- 1) PCR
- 2) Culture , Colonies , AB sensitivity
- 3) Tissue RNA extraction/ recognition

The outcome of infection by *H. pylori* reflects an interaction between:



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8. Ally R, Morton D, Bardhan KD, Bukofzer S, Ahmed R, Bhaga H, Isaacs R, Mistry R, Hassan H, Segal I. Helicobacter pylori (Hp) in Soweto, South Africa: I) prevalence in asymptomatic children, ii) prevalence and Cag A status in adults undergoing endoscopy. (1998) SAGES 36th Congress Proceedings.
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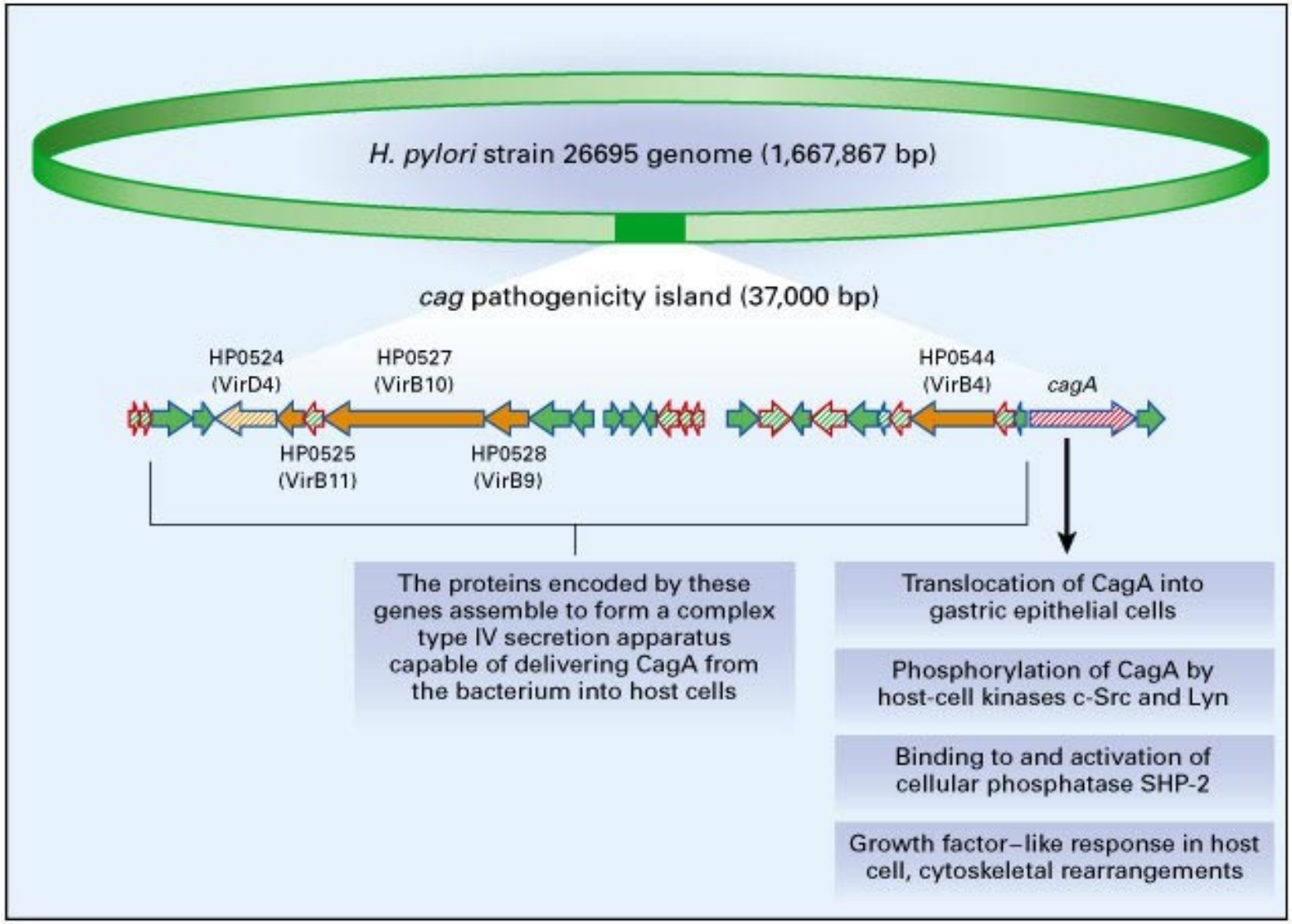
A . BACTERIAL FACTORS

Medical Progress

HELICOBACTER PYLORI INFECTION

SEBASTIAN SUERBAUM, M.D., AND PIERRE MICHETTI, M.D.

N Engl J Med, Vol. 347, No. 15 · October 10, 2002 · www.nejm.org · 1175



NEJM 2002;347:1175-86

AMERICAN SOCIETY FOR MICROBIOLOGY | Journal of Bacteriology

POPULATION GENETICS AND EVOLUTION
1 June 2000 Volume 182 Issue 11
https://doi.org/10.1128/jb.182.11.3210-3218.2000

Differences in Genotypes of *Helicobacter pylori* from Different Human Populations

Dangeruta Kersulyte[1], Asish K. Mukhopadhyay[1], Billie Velapatiño[1,2], WanWen Su[1], ZhiJun Pan[1], Claudia Garcia[1,3], Virginia Hernandez[1], Yanet Valdez[1,2], Rajesh S. Mistry[1,4], Robert H. Gilman[2], Yuan Yuan[1,5], Hua Gao[1,5], Teresa Alarcón[6], Manuel López-Brea[6], G. Balakrish Nair[7], Abhijit Chowdhury[7], Simanti Datta[7], Mutsunori Shirai[8], Teruko Nakazawa[8], Reidwaan Ally[4], Isidore Segal[4], Benjamin C. Y. Wong[9], S. K. Lam[9], Farzad O. Olfat[10,11], Thomas Borén[10], Lars Engstrand[11], Olga Torres[3], Roberto Schneider[3], Julian E. Thomas[12], Steven Czinn[13], Douglas E. Berg[1,*]

5 STRAINS

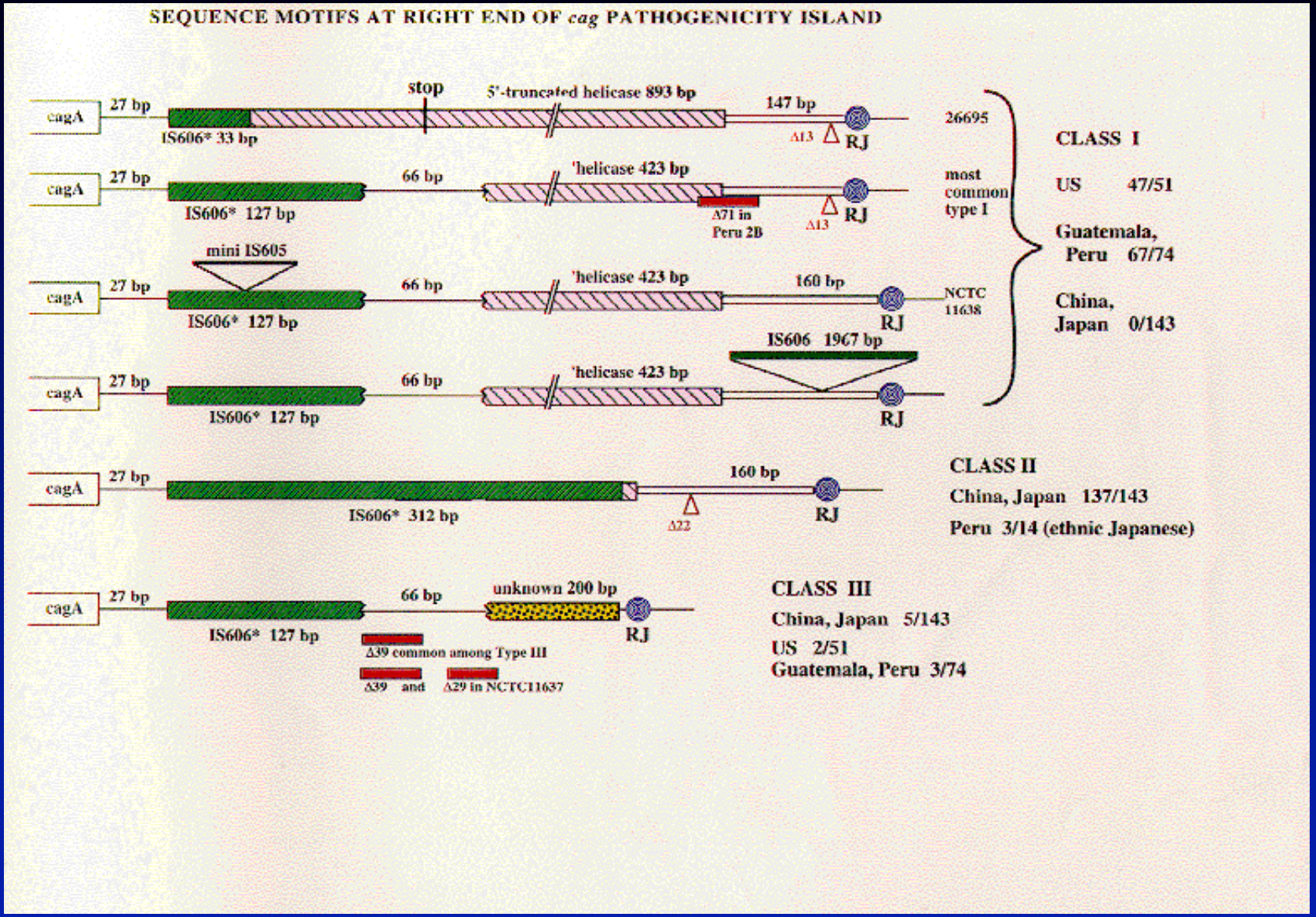
TABLE 1

Distribution of *cag* right-junction motifs among major types of *H. pylori* strains

Geographic region	No. (%) of strains				
	Total	Type I	Type II	Type III	Other
South Europe	36	33 (92%)	1 (3%)	2 (6%)	0
Spain	36	33	1	2	0
Latin America	96	89 (93%)	0	6 (6%)	1 (1%)
Peru	68	62	0	6	0
Guatemala	28	27	0	0	1[a]
Africa	40	40 (100%)	0	0	0
Gambia	8	8	0	0	0
South Africa	32	32	0	0	0
North America	51	45 (88%)	0	2 (4%)	4 (8%)
Louisiana	16	15	0	0	1[a]
Missouri	7	7	0	0	0
Ohio	13	11	0	1	1[a]
Tennessee	5	5	0	0	0
West Virginia	10	7	0	1	2[b]
East Asia	204	1 (0.5%)	194 (95%)	8 (4%)	1 (0.5%)

Distribution of subtypes of type I strains

Geographic region	No. (%) of strains of subtype:			
	Total	Ia	Ib[a]	Ic[b]
Latin America	89	64 (82%)	25 (28%)	0
Guatemala	27	21	6	0
Peru	62	43	19	0
Europe	43	28 (65%)	15 (35%)	0
Spain	33	23	10	0
Sweden	7	2	5	0
Lithuania	3	3	0	0
Africa	40	32 (80%)	8 (20%)	0
Gambia	8	8	0	0
South Africa	32	24	8	0
United States	45	36 (80%)	3 (7%)	6 (13%)
Tennessee	5	4	1	0
Ohio	11	5	1	5
West Virginia	7	6	1	0
Missouri	7	7	0	0


SEQUENCE MOTIFS AT RIGHT END OF cag PATHOGENICITY ISLAND
cagA
27 bp
stop
5'-truncated helicase 893 bp
147 bp
IS606* 33 bp
Δ13
RJ
26695
CLASS I
US 47/51
Guatemala, Peru 67/74
China, Japan 0/143
66 bp
'helicase 423 bp
IS606* 127 bp
Δ71 in Peru 2B
most common type I
mini IS605
160 bp
NCTC 11638
IS606 1967 bp
CLASS II
China, Japan 137/143
Peru 3/14 (ethnic Japanese)
IS606* 312 bp
Δ22
unknown 200 bp
CLASS III
China, Japan 5/143
US 2/51
Guatemala, Peru 3/74
Δ39 common among Type III
Δ39 and Δ29 in NCTC11637

26

encourage further analyses of strains from relatively understudied geographic regions and human ethnic groups. Such "geographic genomics" may uncover new genes that affect human infection,

nature communications

Article

https://doi.org/10.1038/s41467-023-43562-y

The *Helicobacter pylori* Genome Project: insights into *H. pylori* population structure from analysis of a worldwide collection of complete genomes

Received: 5 September 2023

Accepted: 13 November 2023

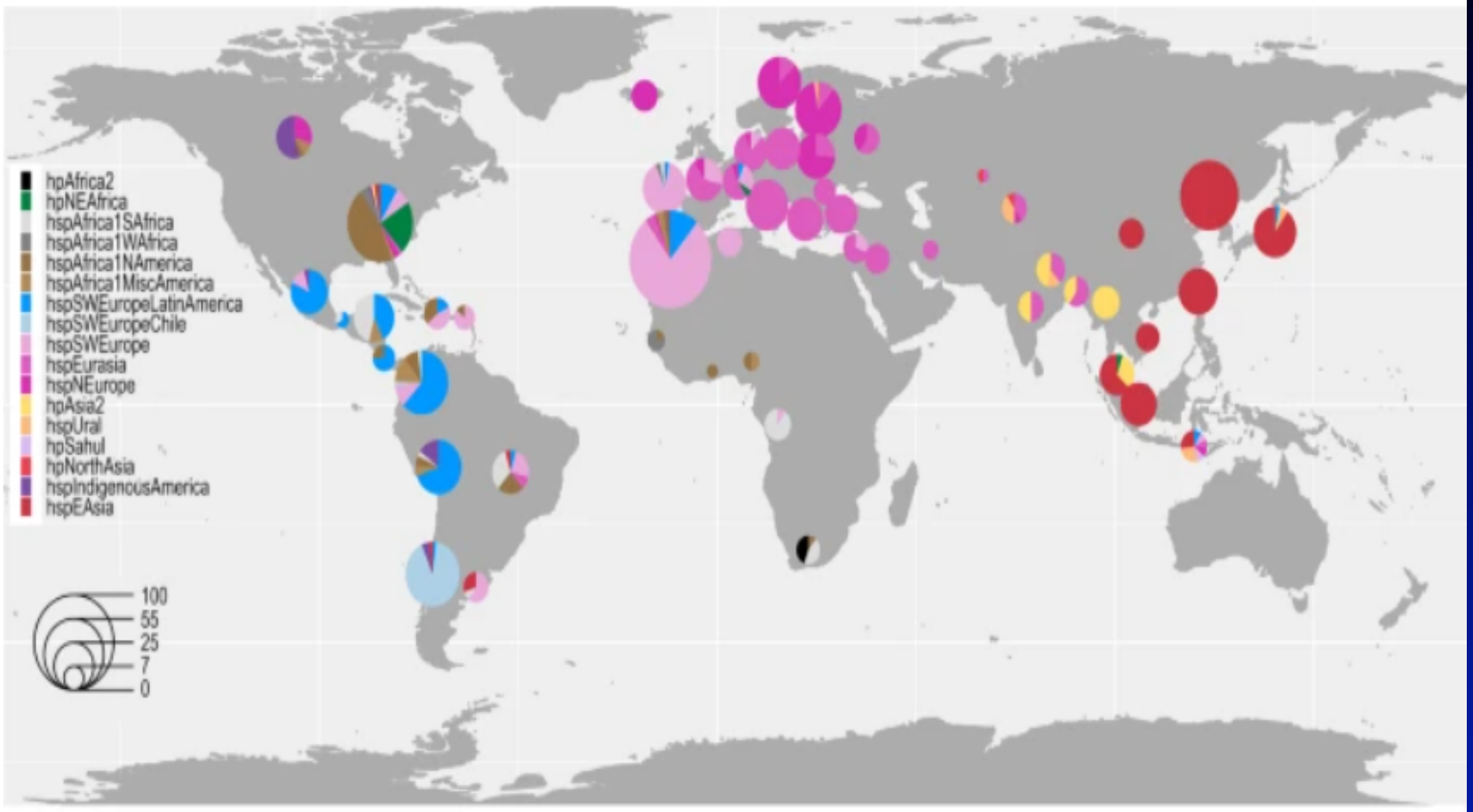
Published online: 11 December 2023

Check for updates

Kaisa Thorell[1,2,04], Zilia Y. Muñoz-Ramírez[2,2,04], Difei Wang[3,4], Santiago Sandoval-Motta[5,6,7], Rajiv Boscolo Agostini[8], Silvia Ghirotto[8], Roberto C. Torres[9], HpGP Research Network*, Daniel Falush[9], M. Constanza Camargo[4,2,05] & Charles S. Rabkin[4,2,05]

Helicobacter pylori, a dominant member of the gastric microbiota, shares co-

Fig. 1: World map of *Hp*GP strain origins and population assignments.



The fineSTRUCTURE global analysis revealed four main *H. pylori* population clusters:

Fig. 2: Distance network analyses of the core genome of the *H. pylori* strains studied.

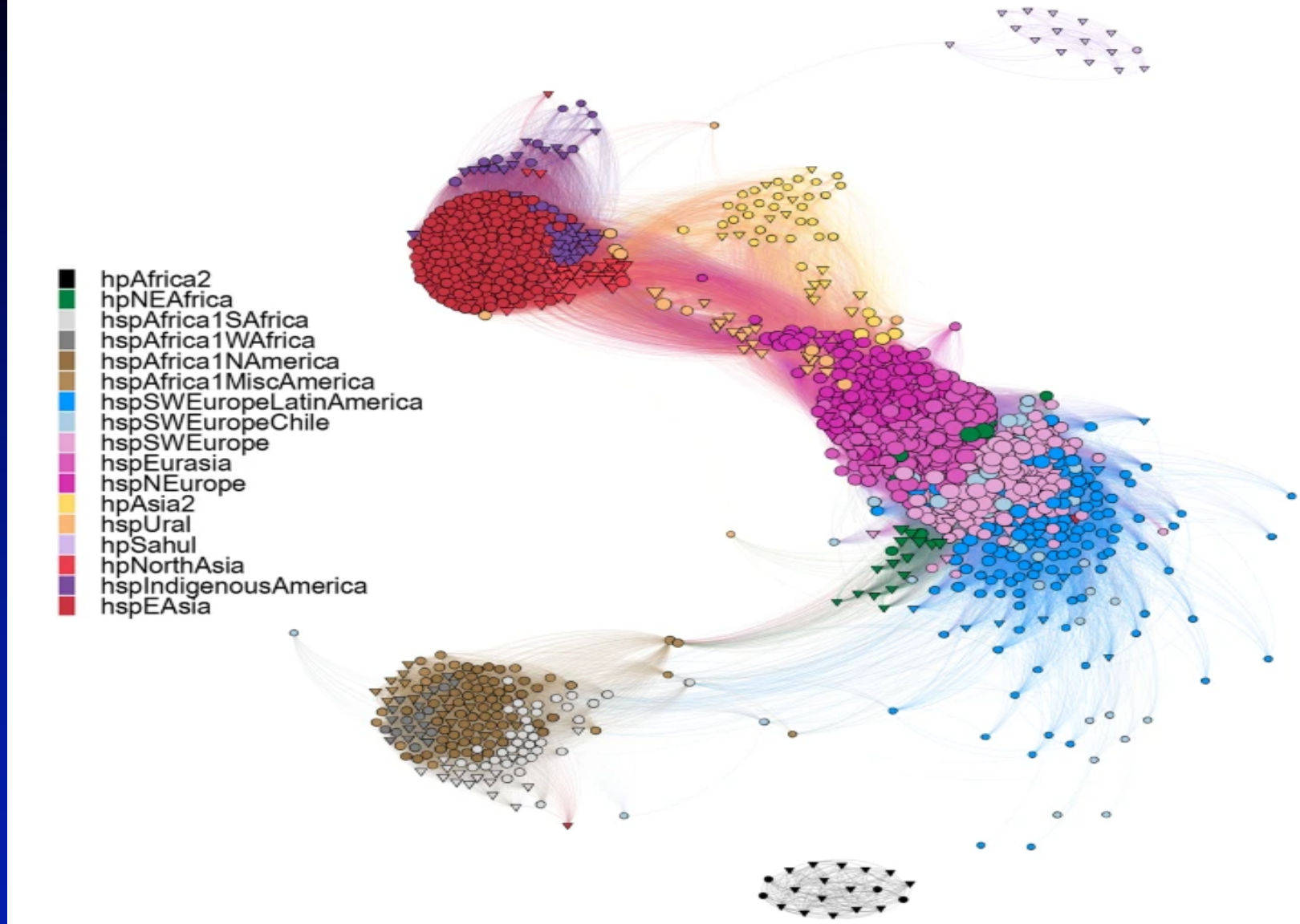
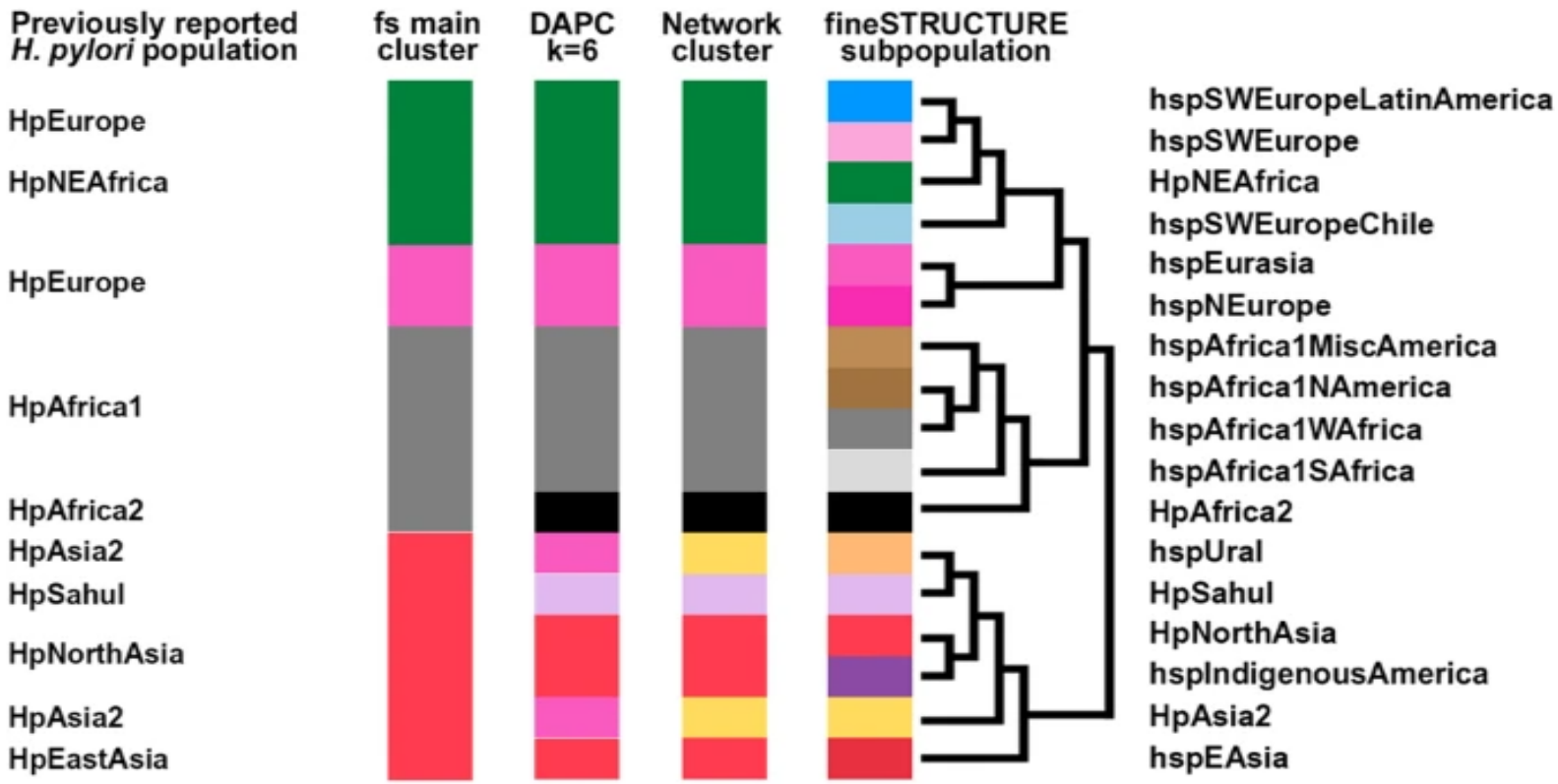


Fig. 5: Summary of population classifications.



Summary of the clustering results using the respective analyses in relation to previously reported MLST and whole genome-based *H. pylori* populations (Hp) and subpopulations (hsp). Colors are based on classifications from the fineSTRUCTURE (fs) analyses visualized in Supplementary Fig. 1, on the $K = 6$ discriminant analysis of principal components, DAPC (Supplementary Fig. 3), and the network clusters (Fig. 2). The topology of the dendrogram to the left is based on the fineSTRUCTURE hierarchical clustering of Supplementary Fig. 1.

Summary of the *Hp*GP strain collection

Country	Total number	Non-atrophic gastritis (%)	Intestinal metaplasia (%)	Gastric cancer (%)
Algeria[a]	10	100		
Argentina	10	100		
Bangladesh	10	100		
Brazil	21	48	38	14
Bulgaria[a]	8	100		
Canada	20	35	65	
Chile	46	54	46	
China	10			100
DR Congo[a]	11	91		9
Colombia	45	78	16	7
Costa Rica	8	100		
Dominican Republic[a]	11	91		9
France	21	48		52
Germany	17	59		41
Ghana[a]	2	100		
The Gambia	5	100		
Greece	21	48		52
Guatemala	3	100		
Honduras	26	35	38	27
Indonesia	11	91		9
India	10	100		

Iceland	11	91	9	
Israel	10	70	30	
Italy	29	34	34	31
Japan	29	38	21	41
Jordan[a]	10	100		
Kazakhstan[a]	2	100		
Kyrgyzstan[a]	10	100		
Korea	54	19	19	63
Latvia[a]	34	29	24	47
Lithuania	23	43	35	22
Malaysia	19	47	53	
Mexico	22	45		55
Myanmar[a]	12	83		17
Nepal	13	77		23
Nigeria	4	100		
Peru	33	30	24	45
Poland[a]	20	100		
Portugal	30	57	27	17
Russia	10	60		40
Singapore	21	38	33	29
South Africa	9	100		
Spain	106	72	13	15
Sweden	30	33	33	33
Switzerland	15	60	40	
Taiwan	24	42		58

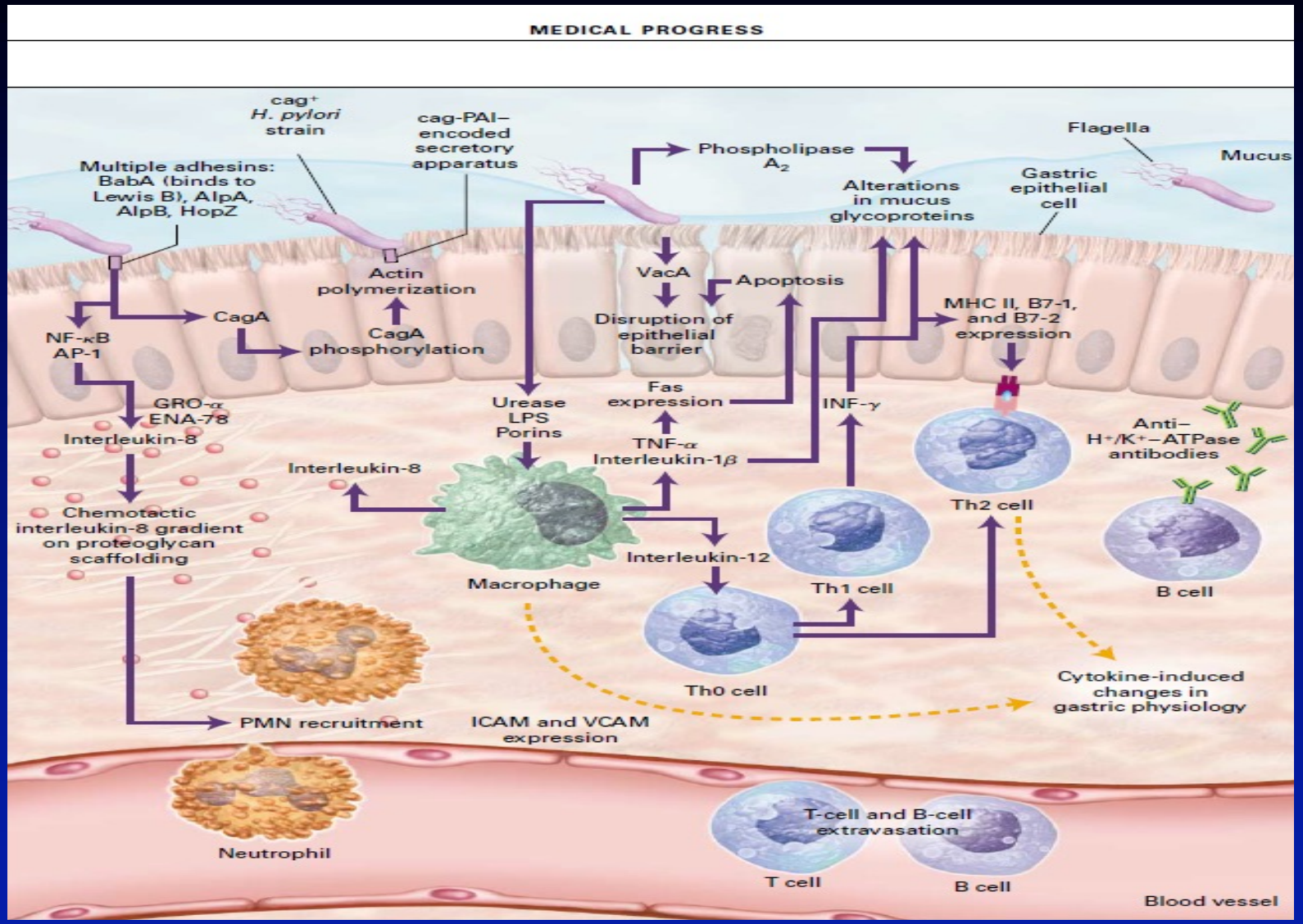
[a]Geographical areas from which no *H. pylori* whole-genome sequences were previously available in GenBank

analysis of only a handful of genes rather than whole genomes[3,4]. The risk of developing disease from *H. pylori* infection varies greatly by geography[7] and genomic studies of both humans and *H. pylori* are required to identify the factors that modify this risk.

B . HOST FACTORS

- Immunology
- Pathobiology
- Stomach Microbiome
- Acid Secretion

1. Immunology/ Pathobiology

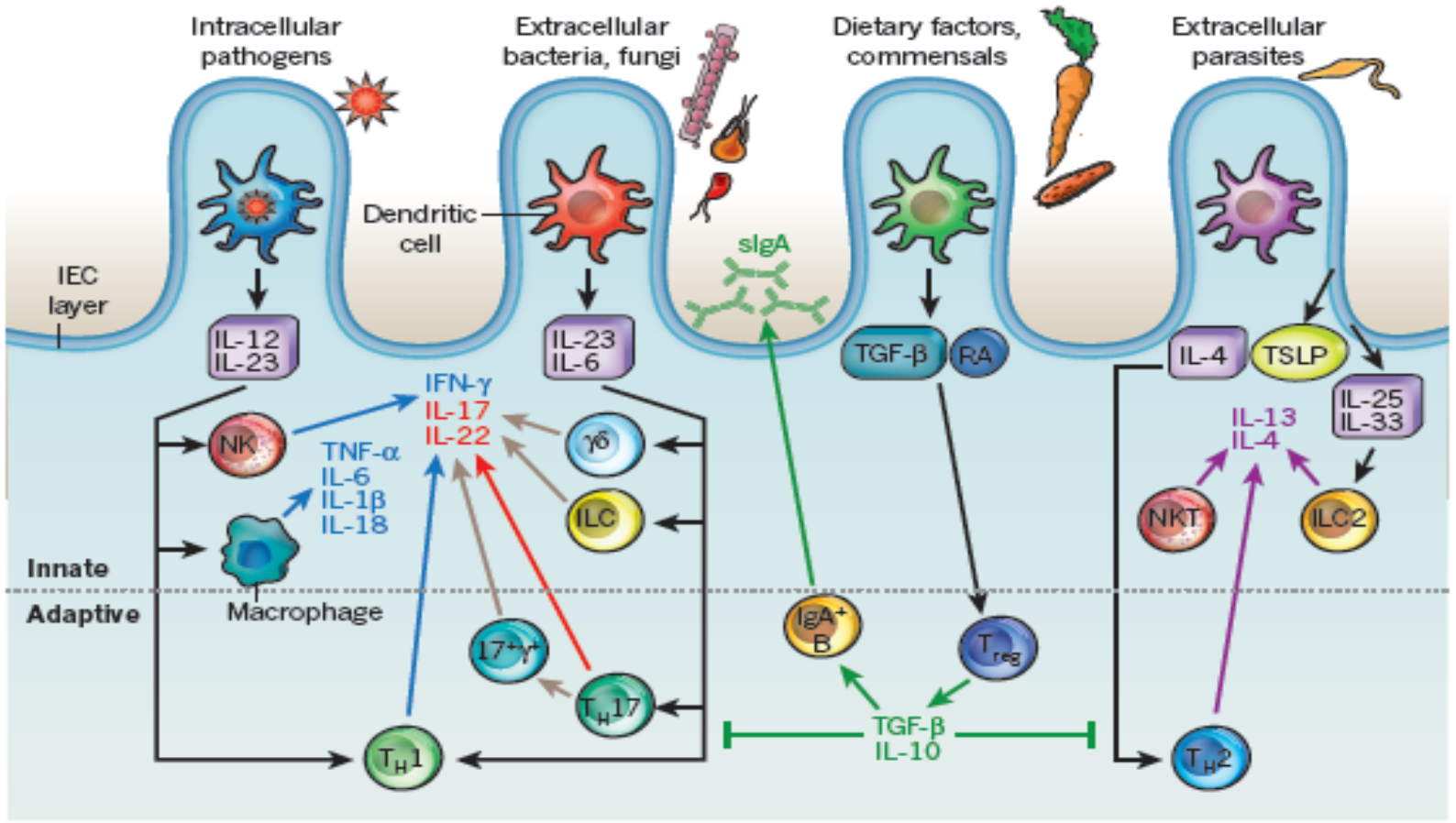

MEDICAL PROGRESS
cag+ H. pylori strain
cag-PAI–encoded secretory apparatus
Multiple adhesins: BabA (binds to Lewis B), AlpA, AlpB, HopZ
Phospholipase A2
Flagella
Mucus
Alterations in mucus glycoproteins
Gastric epithelial cell
Actin polymerization
VacA
Apoptosis
CagA
CagA phosphorylation
Disruption of epithelial barrier
MHC II, B7-1, and B7-2 expression
NF-κB AP-1
Urease LPS Porins
Fas expression
INF-γ
GRO-α ENA-78 Interleukin-8
TNF-α Interleukin-1β
Anti–H+/K+–ATPase antibodies
Interleukin-8
Th2 cell
Chemotactic interleukin-8 gradient on proteoglycan scaffolding
Macrophage
Interleukin-12
Th1 cell
B cell
Th0 cell
Cytokine-induced changes in gastric physiology
PMN recruitment
ICAM and VCAM expression
Neutrophil
T-cell and B-cell extravasation
T cell
B cell
Blood vessel

NEJM 2002

Concurrent enteric helminth infection modulates inflammation and gastric immune responses and reduces helicobacter-induced gastric atrophy

JAMES G. FOX[1], PAUL BECK[3], CHARLES A. DANGLER[1], MARK T. WHARY[1], TIMOTHY C. WANG[3], HAI NING SHI[2] & CATHRYN NAGLER-ANDERSON[2]

[1]Division of Comparative Medicine, Massachusetts Institute of Technology Cambridge, Massachusetts, 02139, USA
[2]Mucosal Immunology Laboratory, Pediatric Gastroenterology and [3]Gastroenterology Unit Department of Medicine, Massachusetts General Hospital, Boston, Massachusetts 02114, USA
Address correspondence to J.G.F.; email: jgfox@mit.edu


Intracellular pathogens
Extracellular bacteria, fungi
Dietary factors, commensals
Extracellular parasites
Dendritic cell
IEC layer
sIgA
IL-12
IL-23
IL-23
IL-6
TGF-β
RA
IL-4
TSLP
IL-25
IL-33
IL-13
IL-4
IFN-γ
IL-17
IL-22
NK
TNF-α
IL-6
IL-1β
IL-18
γδ
ILC
NKT
ILC2
Innate
Adaptive
Macrophage
IgA+ B
Treg
17+γ+
TH17
TH1
TH2
TGF-β
IL-10

J Egypt Soc Parasitol. 2008 Apr;38(1):73-84.

Impact of coinfection with Schistosoma mansoni on Helicobacter pylori induced disease

Sahar A Abou Holw [1], Medhat M Anwar, Rasha B Bassiouni, Neveen A Hussen, Hend A Eltaweel

Affiliations + expand

PMID: 19143122

Acta Parasitol. 2021 Sep;66(3):857-862. doi: 10.1007/s11686-020-00330-y. Epub 2021 Feb 17.

Impact of Coinfection with Schistosoma mansoni on the Antibody Response to Helicobacter pylori

Ashraf Fawzy Mosa Ahmed [1], Mona Hassan El-Sayad [1], Hala Shehata Ali [1], Hend Aly El-Taweel [2]

Affiliations + expand

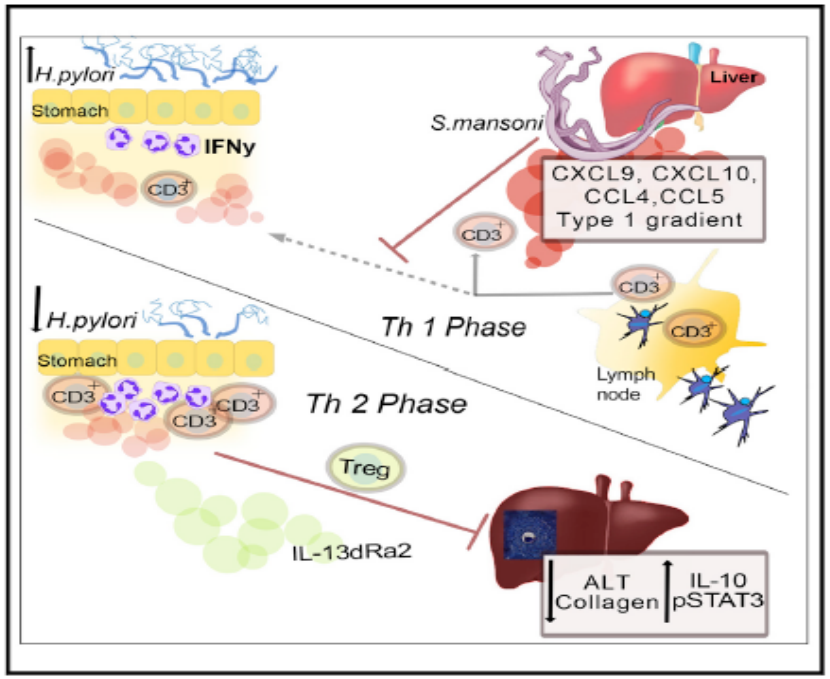
PMID: 33598776 DOI: 10.1007/s11686-020-00330-y

Article

Cell Reports

Concomitant Infection of *S. mansoni* and *H. pylori* Promotes Promiscuity of Antigen-Experienced Cells and Primes the Liver for a Lower Fibrotic Response

Graphical Abstract



Authors

Sonakshi Bhattacharjee, Raquel Mejías-Luque, Eva Loffredo-Verde, Albulena Toska, Michael Flossdorf, Markus Gerhard, Clarissa Prazeres da Costa

Correspondence

markus.gerhard@tum.de (M.G.), clarissa.dacosta@tum.de (C.P.d.C.)

In Brief

Co-infection is ubiquitous in human populations and is yet not the most widely studied experimental topic. Bhattacharjee et al. demonstrate that the immunological interaction of two prominent, anatomically isolated human pathogens, *H. pylori* and *S. mansoni*, eventually results in an unusual, mutually ameliorating effect on the detrimental course of both infections.

Highlights

- Co-infection of *H. pylori* and *S. mansoni* results in altered disease-specific pathology

The IgG subclass response to infection is considered to be a biomarker of the T helper cell response, IFNγ having been shown in humans to promote the production of IgG2 subclass antibodies while significantly suppressing the production of IgG1 subclass antibodies (20, 21). Measurement of the relative levels of anti-*H. pylori* IgG1 and IgG2 subclass antibodies in *H. pylori* positive individuals from a developed country has shown the predominant IgG subclass response to be IgG2, a finding that is consistent with a Th1 predominant response (18, 22).

Scandinavian Journal of Gastroenterology ›
Volume 37, 2002 - Issue 5

Research Article

Major Differences in the IgG Subclass Response to Helicobacter pylori in the First and Third Worlds

H. M. Mitchell, R. Ally, A. Wadee, M. Wiseman & I. Segal

Pages 517-522 | Published online: 08 Jul 2009

Cite this article https://doi.org/10.1080/00365520252903044

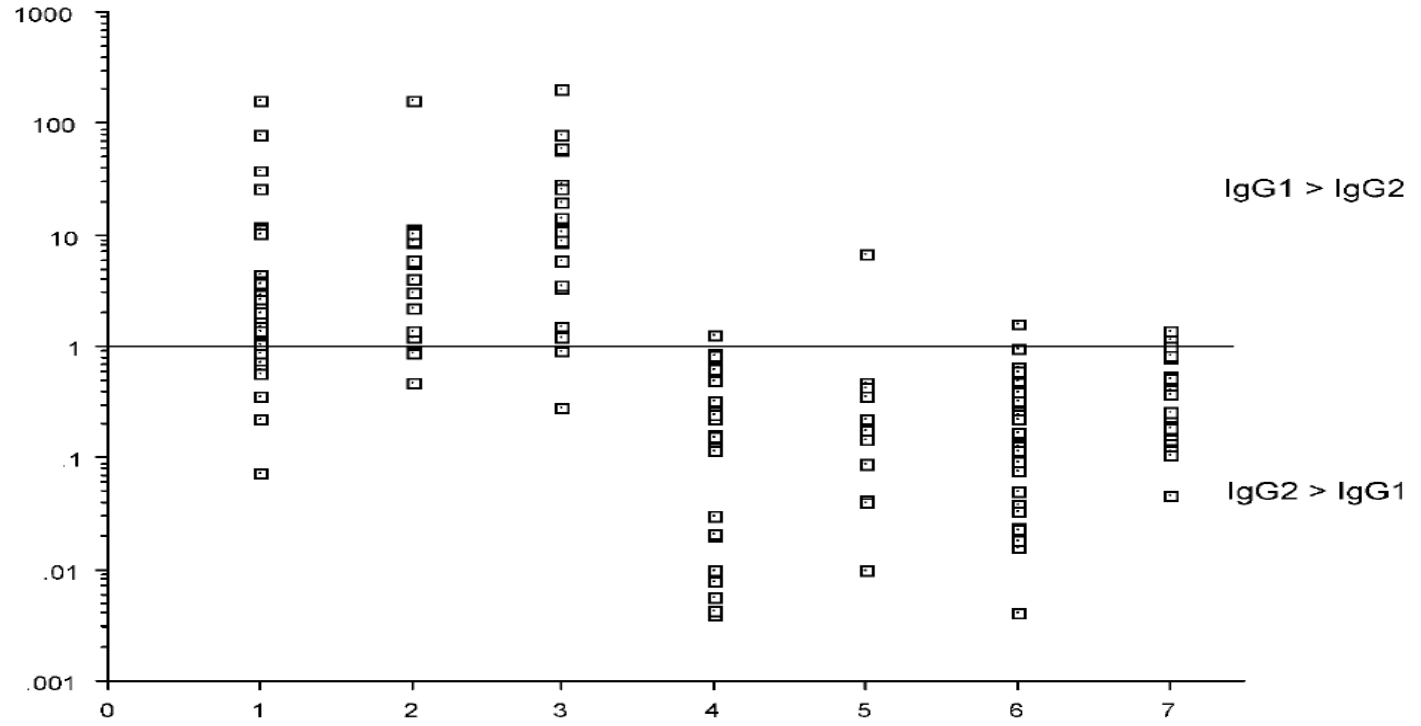


Fig. 1. The IgG1/IgG2 subclass ratio in subjects from Soweto, Australia and Germany. (1 = Sowetan adults with disease (GU, DU and GC); 2 = Sowetan adults, NUD; 3 = Symptomatic Sowetan children with NUD; 4 = German adults with DU; 5 = German adults, NUD; 6 = Australian adults with DU; 7 = Australian adults with NUD.)

In conclusion, the results of the present study provide the first evidence that the host immune response to *H. pylori* infection in an African population differs from that observed in subjects from developed countries. The reason for this

BIOMEDICAL JOURNAL 39 (2016) 14–23

Available online at www.sciencedirect.com

ScienceDirect

Biomedical Journal

journal homepage: www.elsevier.com/locate/bj

Review Article

Helicobacter pylori infection: An overview of bacterial virulence factors and pathogenesis

Cheng-Yen Kao [a], *Bor-Shyang Sheu* [b], *Jiunn-Jong Wu* [a,c,d,*]

[a] *Department of Medical Laboratory Science and Biotechnology, College of Medicine, National Cheng Kung University, Tainan, Taiwan*

[b] *Department of Internal Medicine, College of Medicine, National Cheng Kung University, Tainan, Taiwan*

[c] *Center of Infectious Disease and Signaling Research, National Cheng Kung University, Tainan, Taiwan*

[d] *Department of Biotechnology and Laboratory Science in Medicine, School of Biomedical Science and Engineering, National Yang-Ming University, Taipei, Taiwan*

Prof. Jiunn-Jong Wu

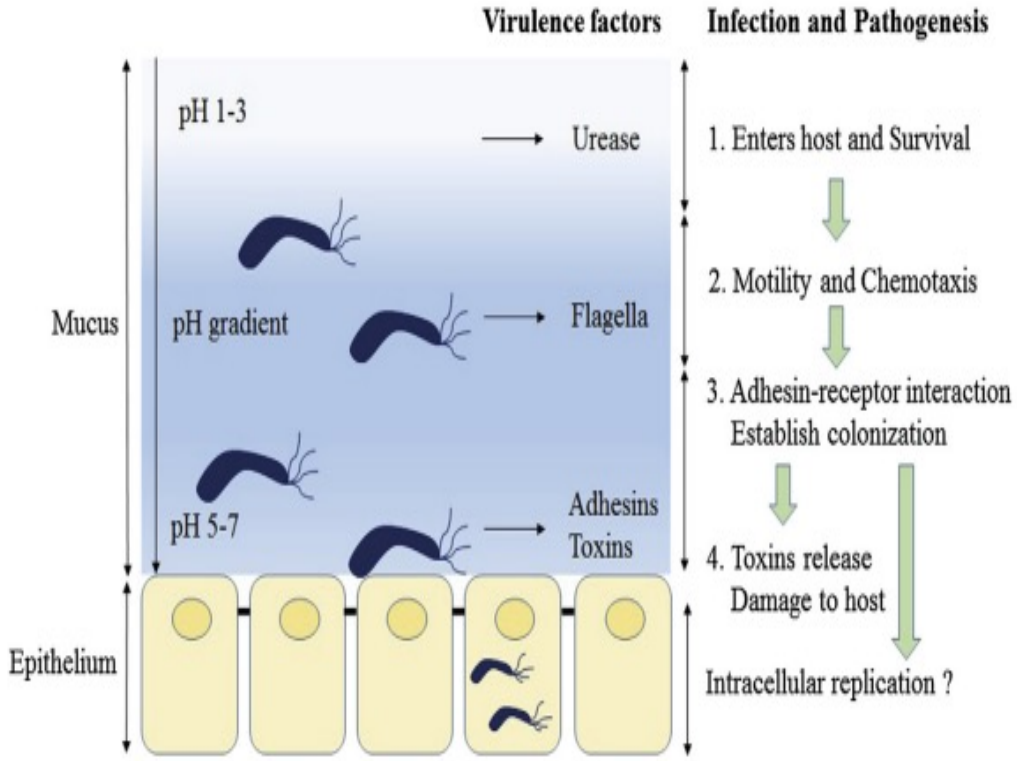


Fig. 1 – Schematic diagram of *Helicobacter pylori* infection and pathogenesis. The urease activity and flagella-mediated motility of *H. pylori* facilitate its survival and movement toward the lower mucus gel above the epithelium, followed by several adhesins, including blood-antigen binding protein A, sialic acid-binding adhesin, and other outer membrane proteins interacting with receptors on the host epithelium cells. After successful colonization, toxins, including cytotoxin-associated gene A, and vacuolating cytotoxin A, are involved in damage of host tissue and intracellular replication.

Microb Health Dis 2022; 4 (3): e717

European Helicobacter & Microbiota Study Group

REVIEW: PATHOGENESIS OF *HELICOBACTER PYLORI* INFECTION

S. Freisberg[1], C. Schulz[2], J. Bornschein[3]

[1]Department of Internal Medicine and Gastroenterology, Helios-Klinikum Berlin-Buch, Berlin, Germany
[2]Medical Department II, University Hospital, LMU, Munich, Germany
DZIF Deutsches Zentrum für Infektionsforschung, Partner Site Munich, Munich, Germany
[3]Translational Gastroenterology Unit, John Radcliffe Hospital, Oxford University Hospitals, Headington, Oxford, United Kingdom and NIHR Oxford Biomedical Research Centre, Oxford, UK

Minireview

Am J Physiol Gastrointest Liver Physiol 311: G514–G520, 2016.
First published July 28, 2016; doi:10.1152/ajpgi.00146.2016.

How host regulation of *Helicobacter pylori*-induced gastritis protects against peptic ulcer disease and gastric cancer

Poshmaal Dhar,[1,2] Garrett Z. Ng,[1,2] and Philip Sutton[1,2,3]

[1]*Murdoch Childrens Research Institute, Royal Children's Hospital, Parkville, Victoria, Australia;* [2]*Centre for Animal Biotechnology, School of Veterinary and Agricultural Science, University of Melbourne, Parkville, Victoria, Australia; and* [3]*Department of Paediatrics, University of Melbourne, Parkville, Victoria, Australia*

Submitted 12 April 2016; accepted in final form 26 July 2016

inflammatory response to this pathogen is regulated and disease does not ensue; in most cases this is successful. From the literature presented here, it is evident that most known innate

1.4.1 The interleukin (IL)-1 gene

The IL-1 gene family on chromosome 2q includes 3 related genes IL-1A, IL-1B and IL-1RN that encodes the pro-inflammatory cytokines IL-1α, IL-1β and IL-1ra (receptor antagonist) respectively (13, 23, 45). IL-1β is a pro-inflammatory cytokine that inhibits the acid secretion by interfering with the acid secretion of the parietal cells in the stomach. It is estimated that IL-1β is 100 times more potent than a proton pump inhibitor (eg. Omeprazole)(60). IL-1ra is a naturally occurring anti-inflammatory cytokine that competitively binds to IL-1 receptors.

1.4.2 The TNFα cytokine (Tumor necrosis factor alpha)

The TNFA gene maps to chromosome 6p21.3 and spans about 3kb and contains 4exons. TNFα is a pro-inflammatory cytokine involved in systemic inflammation and is a member of a group of cytokines that stimulate an acute phase reaction. TNFα has also been associated with inhibiting acid secretion however to a much lesser extent that IL-1β.

1.4.3 The interleukin (IL)-10 gene

In humans, the IL-10 gene is located on chromosome 1 and consists of 5 exons. IL-10 is an anti-inflammatory cytokine capable of inhibiting the synthesis of pro-inflammatory cytokines as TNFα or IL-1.

1.4.4 The role of Cytokine Polymorphisms

RAPPORT DE STAGE D'OPTION SCIENTIFIQUE

Titre du Rapport

Study of the cytokine gene polymorphisms in the host and its influence in H.pylori related diseases in a South African population

NON CONFIDENTIEL

Option : Biologie
Champ de l'option : Biologie cellulaire
Directeur de l'option : Monsieur Blanquet Sylvain
Directeur de stage : Professeur Mitchell Hazel
Dates du stage : 14 Avril 2007 au 14 juillet 2007
Adresse de l'organisme :

School of Biotechnology & Biomolecular Sciences
The University of New South Wales
Sydney, NSW 2052
Australia

between these polymorphisms and *H. pylori* related disease. Gastric biopsy samples were collected at the time of endoscopic examination from 81 patients attending the Chris Hani Baragwanath Hospital in Soweto. Of these patients 14 were diagnosed with cancer GC, 3 with duodenal ulcer (DU), 31 with gastric ulcer (GU) and 30 with functional dyspepsia (FD) (control group)

Allele/Country group	Number of studies	Mean value (%)	Std. Deviation	P (2 tailed test for equality of means)
IL-1B-511 T allele frequency				
East Asia	16	49.4	3.1	<0.001
Western	8	32.4	3.0	
South Africa	1	60		
IL-1RN 2 allele frequency				
East Asia	12	5.9	3.0	<0.001
Western	9	26.3	2.7	
South Africa	1	23		
IL-10-1082 A allele frequency				
East Asia	5	94.2	2.3	<0.001
Western	9	51.8	6.0	
South Africa	1	70		
IL-10-592 A allele frequency				
East Asia	5	69.4	2.6	<0.001
Western	7	22.0	2.1	
South Africa	1	35		
TNFA-308 A allele frequency				
East Asia	6	7.2	3.1	<0.001
Western	9	16.3	3.6	
South Africa	1	30		

Table 7: Comparison of the mean values of the allele frequencies of the IL-1, IL-10 and TNF-A cytokine polymorphisms between East-Asian populations, Western populations and the South African population.

2.3.1 Frequency of IL-1 genotypes

The frequency of IL-1 genotypes is shown in Table 4.1 for the GC group, in Table 4.2 for the GU group and in Table 4.3 for the DU disease group.

Locus	Genotype	G.C. n (%)	Controls n (%)	Odds ratio	P
IL-1B-511	C/C	4 (10)	4(14)	1	/
	C/T	20(51)	16(53)	1.2 (0.3-4.2)	0.7
	T/T	15(39)	10(33)	1.3 (0.4-4.5)	1
IL-1RN	1.1	28(71)	20(67)	1	/
	1.2	8(20)	7(23)	0.9 (0.7-1.2)	0.8
	1.3	0(0)	2(7)	0.9 (0.7-1.0)	0.3
	1.4	1(3)	1(3)		
	2.2	1(3)	0(0)	1.04 (1-1.10)	1
	2.4	1(3)	0(0)	1.04 (1-1.10)	1

Table 4.1: Distribution of the IL-1B and IL-1RN genotypes in the gastric cancer disease group

Locus	Genotype	G.U. n (%)	Controls n (%)	Odds ratio	P
IL-1B-511	C/C	12(21)	4(14)	1	/
	C/T	18(31)	16(53)	0.5 (0.2-1.3)	0.2
	T/T	28(48)	10(33)	0.9 (0.4-2.5)	1
IL-1RN	1.1	41(70)	20(67)	1	/
	1.2	14(24)	7(23)	1 (0.8-1.3)	0.8
	1.3	1(2)	2(7)	0.9 (0.8-1.1)	0.3
	1.4	1(2)	1(3)		
	2.2	0(0)	0(0)	/	/
	3.2	1(2)	0(0)	1.02 (1-1.1)	1

Table 4.2: Distribution of the IL-1B and IL-1RN genotypes in the gastric ulcer disease group

		n (%)	n (%)		
IL-1B-511	C/C	9(19)	4(14)	1	/
	C/T	21(45)	16(53)	0.8 (0.3-2.2)	1
	T/T	17(36)	10(33)	0.7 (0.2-1.9)	0.5
IL-1RN	1.1	39(83)	20(67)	1	/
	1.2	3(6.5)	7(23)	0.8 (0.6-1)	0.04
	1.3	1(2)	2(7)	1 (0.8-1.2)	1
	1.4	3(6.5)	1(3)		
	1.5	1(2)	0(0)	/	/
	2.2	0(0)	0(0)	/	/

Table 4.3: Distribution of the IL-1B and IL-1RN genotypes in the duodenum ulcer disease group

polymorphisms in IL-1B, IL-1RN, IL-10 and TNF-A and GU or GC. In contrast we found that those with DU had a significantly lower frequency (p=0.04) of the IL-1RN 1/2 genotype than controls, resulting in an OR of 0.8 (CI, 0.6-1). The finding that the frequency of the IL-1RN1/2 genotype was lower in the DU patients is consistent with previous findings in the literature (18) that have shown that IL-1RN allele 2 is an independent protective factor for duodenal ulcer disease. An explanation for the

To date, there are no published studies regarding the association between host cytokine polymorphisms and *H. pylori* related diseases in South Africa. While this study will therefore provide important data for future studies in the African population, given the small number of samples analysed and the absence of other published analysis of the South African population, the conclusions drawn from the current study must be confirmed by larger scale studies.

3. Stomach Microbiome

Review > Adv Exp Med Biol. 2016:908:393-408. doi: 10.1007/978-3-319-41388-4_19.

Helicobacter pylori, Cancer, and the Gastric Microbiota

Lydia E Wroblewski [1], Richard M Peek Jr [2]

Affiliations + expand

PMID: 27573782 DOI: 10.1007/978-3-319-41388-4_19

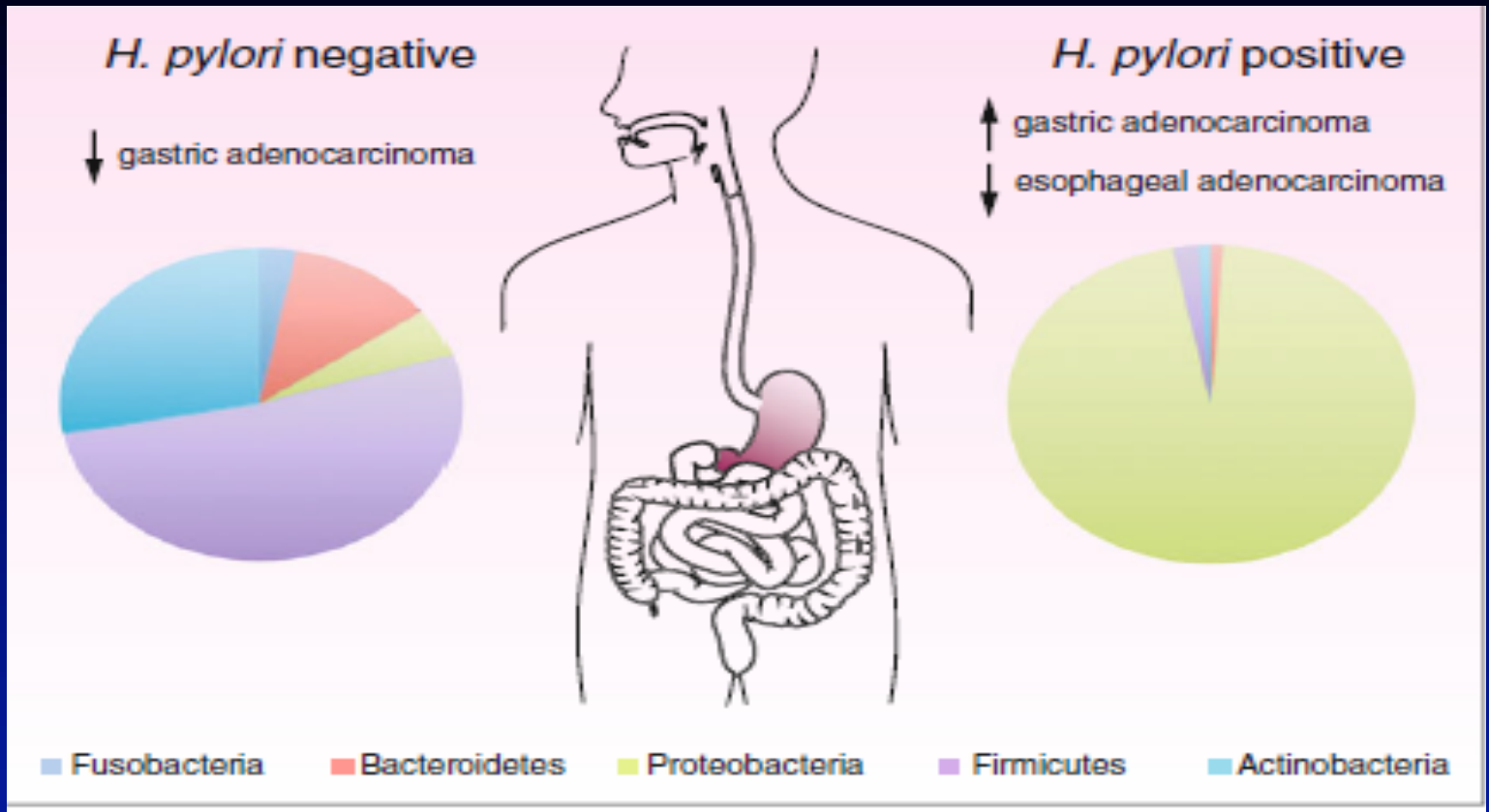
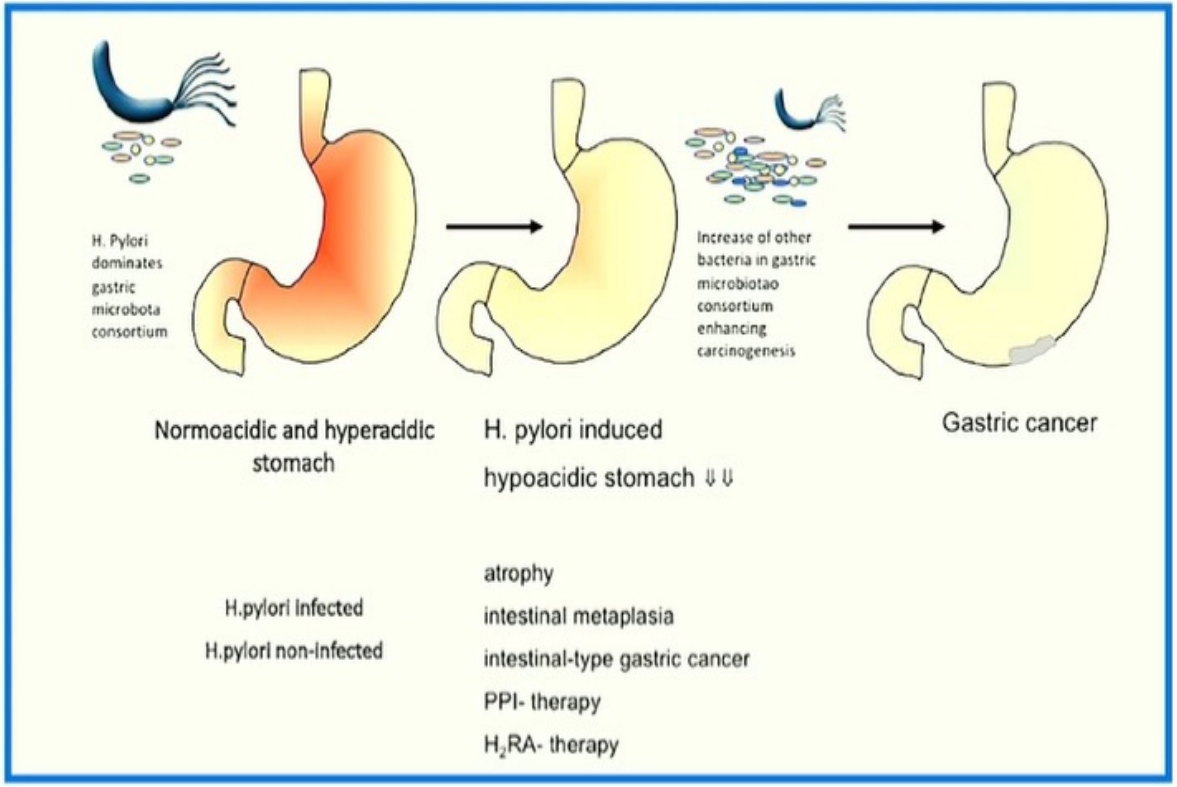


Fig. 19.1 Schematic representation showing the differences in the composition of the human gastric microbiota based on *H. pylori* status. *H. pylori*-negative individuals possess a highly diverse gastric microbiota and exhibit decreased risk of developing gastric adenocarcinoma when compared to *H. pylori* positive individuals who harbor a less diverse microbiota, possess an increased risk for developing gastric adenocarcinoma and concomitant decreased risk for developing esophageal adenocarcinoma

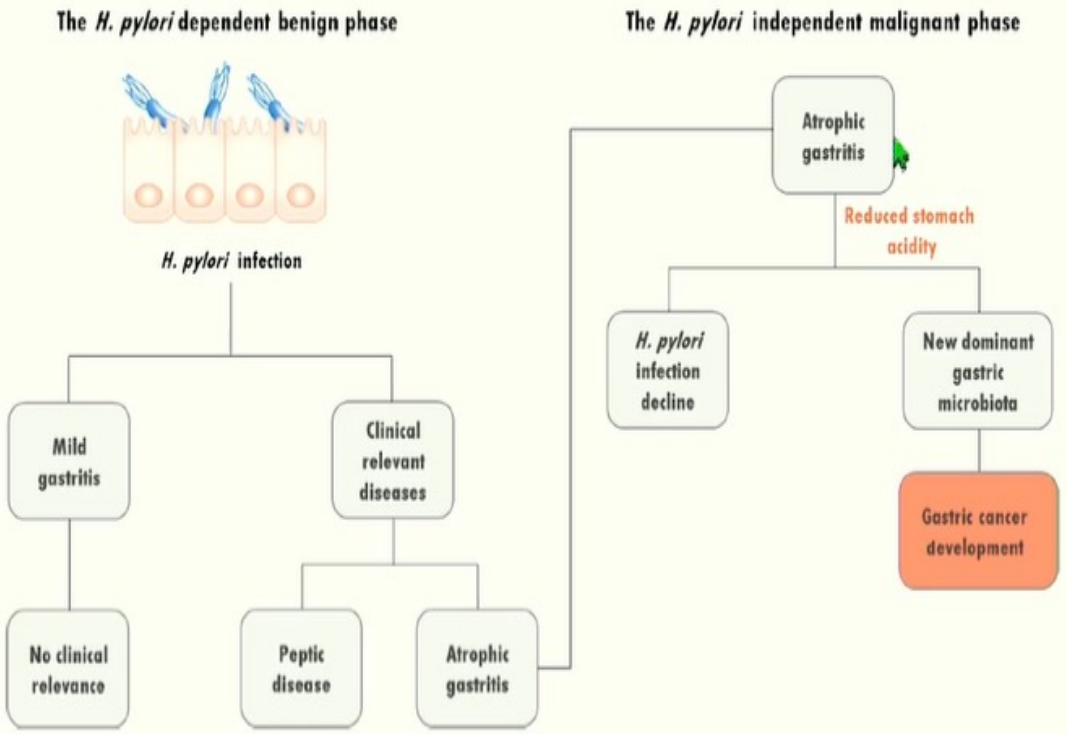
genes can predict obesity with 90 % accuracy [99]. It is tempting to speculate that in the future, it may be possible to identify groups of bacterial taxa present in the stomach that are predictive of gastric disease outcome at specific stages along the Correa cascade. Indeed, it may also be possible to manipulate an individual's specific microbiota to proffer more favorable outcomes following infection with *H. pylori*.

Hypothesis on the impact of other gastric microbiota on gastric cancer development



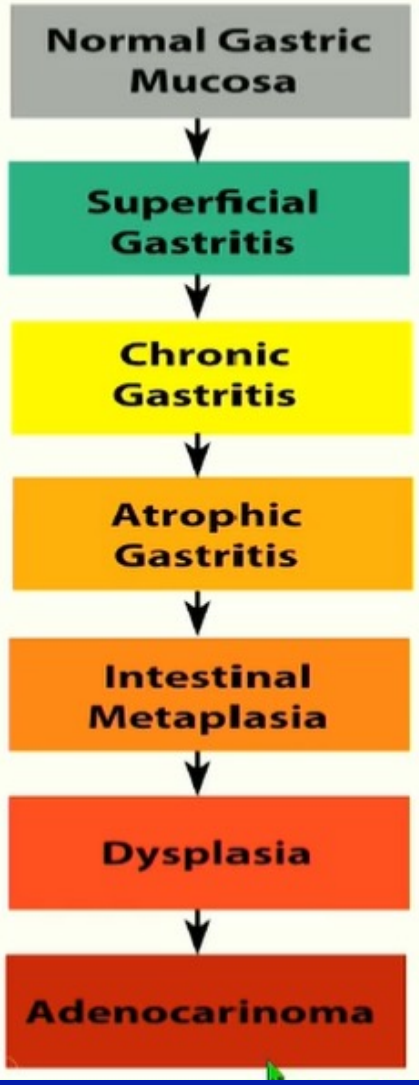
Schulz, Ther Adv Gastroenterol; 2019

The H. pylori dependent and independent phases



Pimentel de Assumpção; Eur J Clin Microbiol Infect Dis; 2019

GASTRIC MICROBIOTA AND GASTRIC DISEASE PROGRESSION



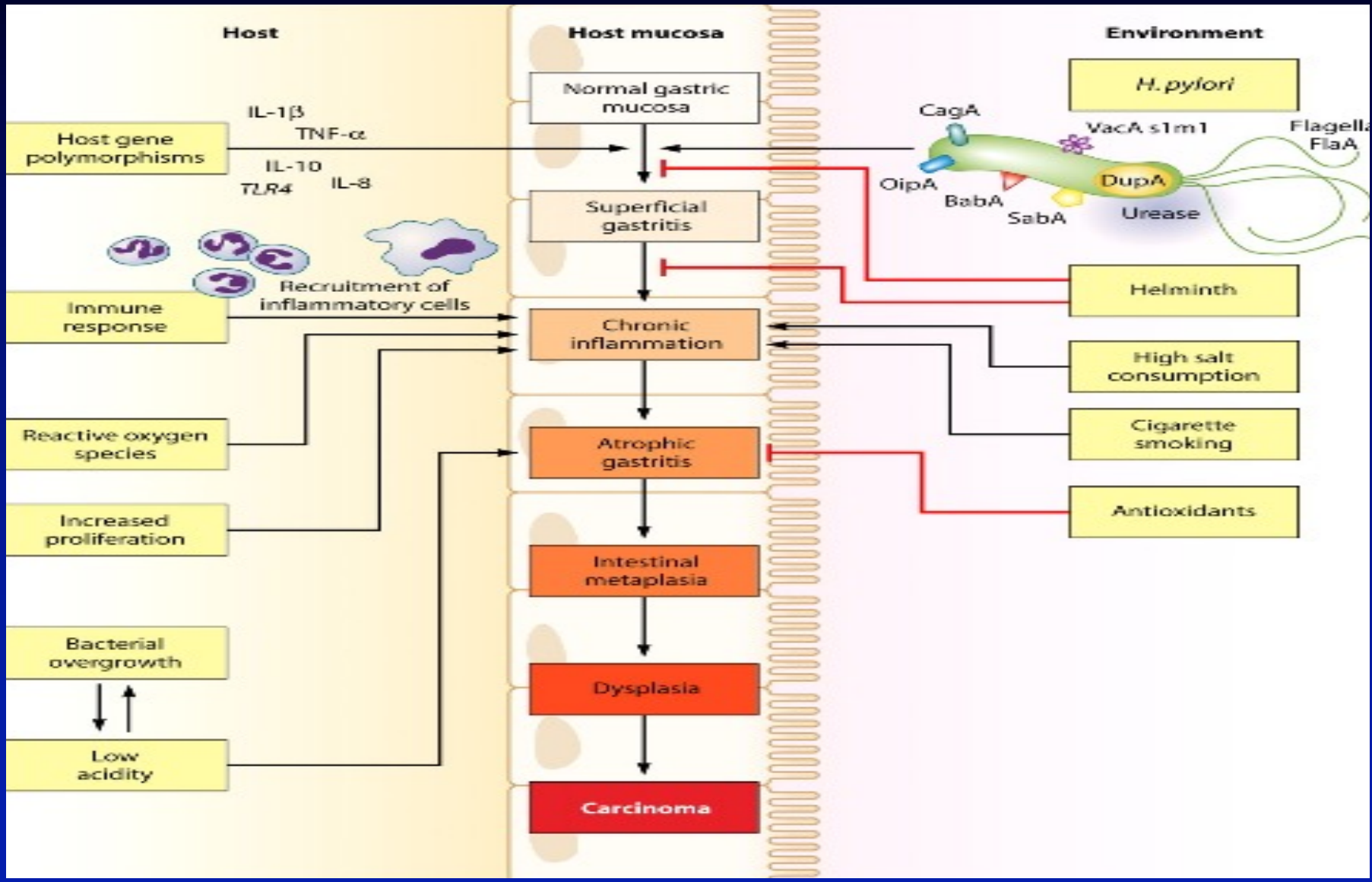
Helicobacter pylori

Enriched Genera
Proteobacteria:
Escherichia-Shigella
Burkholderia
Firmicutes:
Lactobacillus
Lachnospiraceae
Streptococcus
Veillonella
Bacteroidetes:
Prevotella

Noto, PLoS Pathog 2017

AMERICAN SOCIETY FOR MICROBIOLOGY | Clinical Microbiology Reviews®

PMC full text:
Clin Microbiol Rev. 2010 Oct; 23(4): 713–739.
doi: 10.1128/CMR.00011-10



Published in final edited form as:
S Afr Med J. ; 103(4): 255–259.

Gastric adenocarcinoma in Zambia: a case-control study of HIV, lifestyle risk factors, and biomarkers of pathogenesis

Violet Kayamba[1], **Akwi W Asombang**[1,2], **Victor Mudenda**[3], **Mpala Mwanza Lisulo**[1], **Edford Sinkala**[1], **Stayner Mwanamakondo**[1], **Isaac Mweemba**[3], and **Paul Kelly**[1,4]

Conclusions—HIV was not associated with gastric cancer and does not explain the apparent change in age distribution in Zambia. Atrophy was common and was not essential for the development of intestinal metaplasia, suggesting that gastric carcinogenesis in Africa does not always follow the Correa pathway.

ORIGINAL RESEARCH

Serum antibodies to selected *Helicobacter pylori* antigens are associated with active gastritis in patients seen at the University Teaching Hospital in Lusaka, Zambia

Violet Kayamba[1,2], Julia Butt[3]. Matthew Gordon Varga[4], Aaron Shibemba[5], Maria Blanca Piazuelo[6], Keith Tucker Wilson[6,7], Kanekwa Zyambo[1], Simutanyi Mwakamui[1], Chola Mulenga[1], Tim Waterboer[3], Meira Epplein[8], Douglas Corbett Heimburger[9], Masharip Atadzhanov[2], Paul Kelly[1,2,10]

Conclusions

Among Zambian patients seen at a single center, antibodies to *H. pylori* (CagA, VacA, Omp, HcpC, HP0305 and HpaA) were associated with active gastritis.

RESEARCH ARTICLE

Helicobacter pylori infection and hypochlorhydria in Zambian adults and children: A secondary data analysis

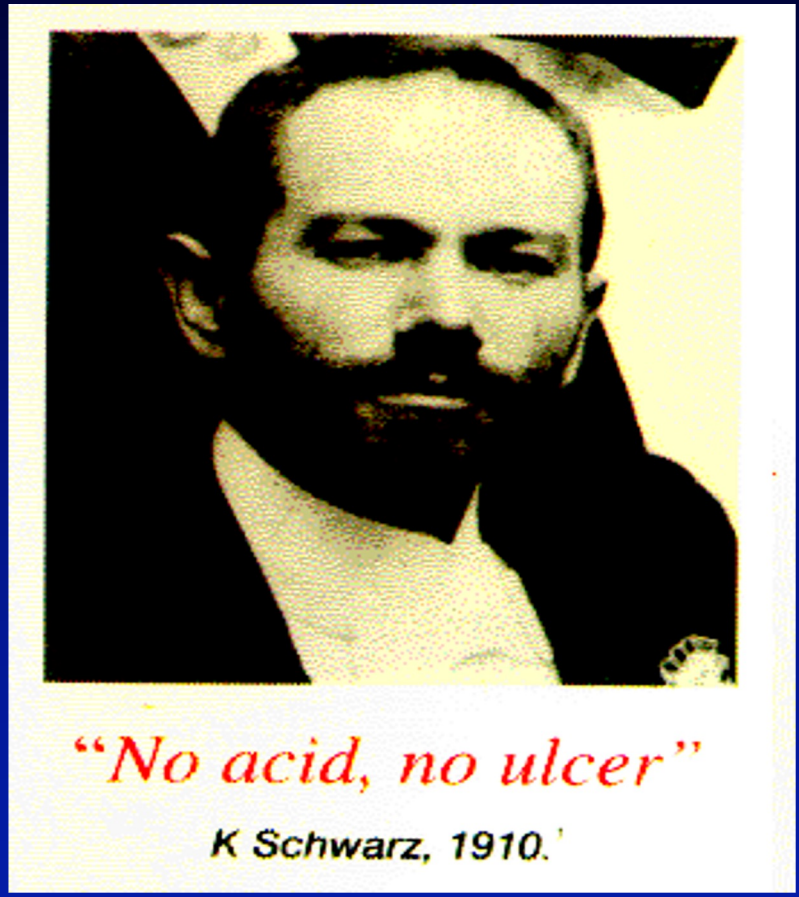
Phoebe Hodges[1,2], **Paul Kelly**[1,2], **Violet Kayamba**[1]*

1 Tropical Gastroenterology & Nutrition group, University of Zambia School of Medicine Department of Internal Medicine, Lusaka, Zambia, **2** Blizard Institute, Barts & The London School of Medicine and Dentistry, Queen Mary University of London, London, United Kingdom

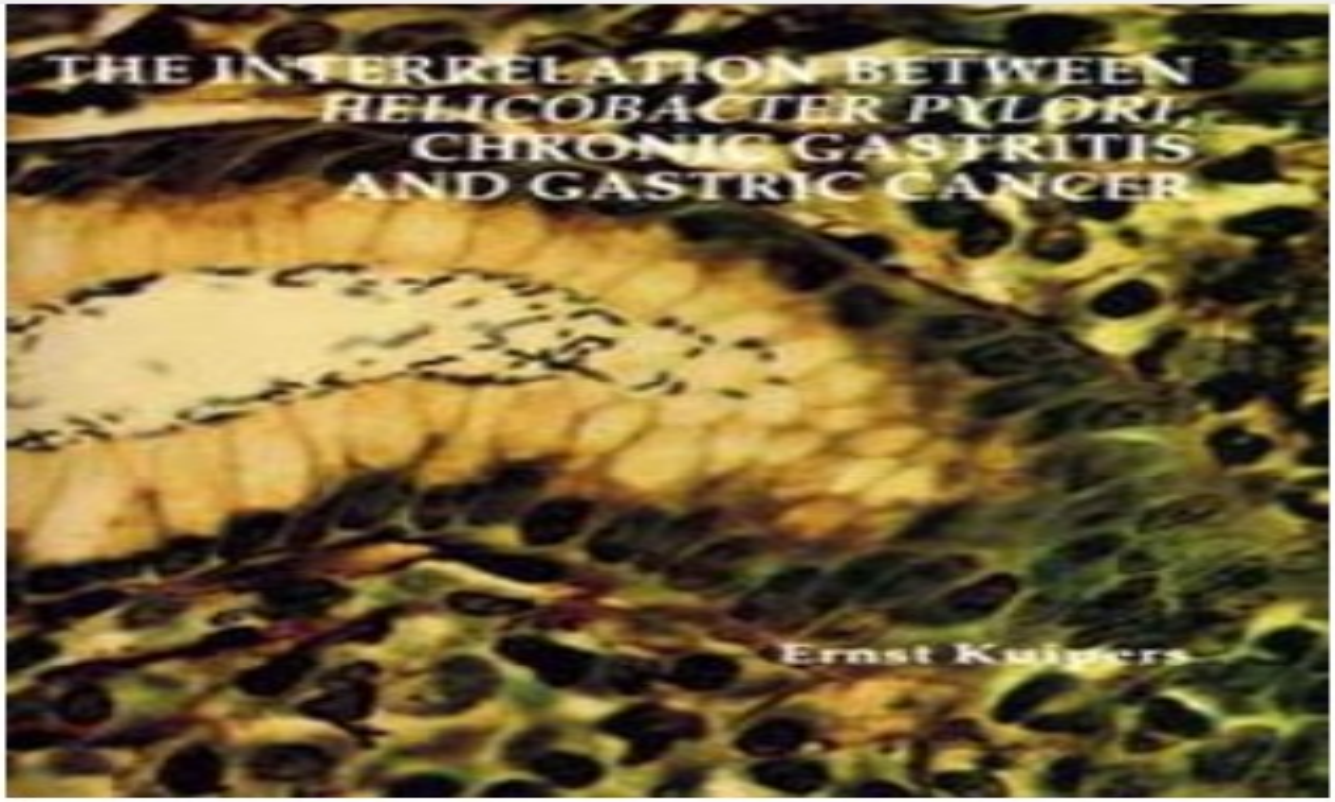
4. Acid Secretion

Dictum - “NO ACID
NO ULCER”

K. SWARZ 1910



76



Ernst Kuipers **1995**

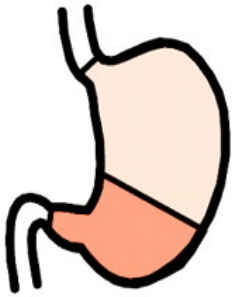
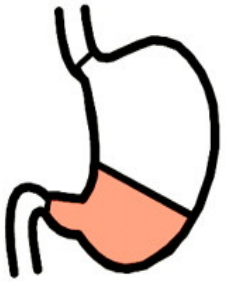
The Interrelation between Helicobacter pylori, chronic gastritis and gastric cancer.

CLINICAL MICROBIOLOGY REVIEWS, July 2006, p. 449–490 Vol. 19, No. 3
0893-8512/06/$08.00+0 doi:10.1128/CMR.00054-05

Pathogenesis of *Helicobacter pylori* Infection

Johannes G. Kusters,* Arnoud H. M. van Vliet, and Ernst J. Kuipers

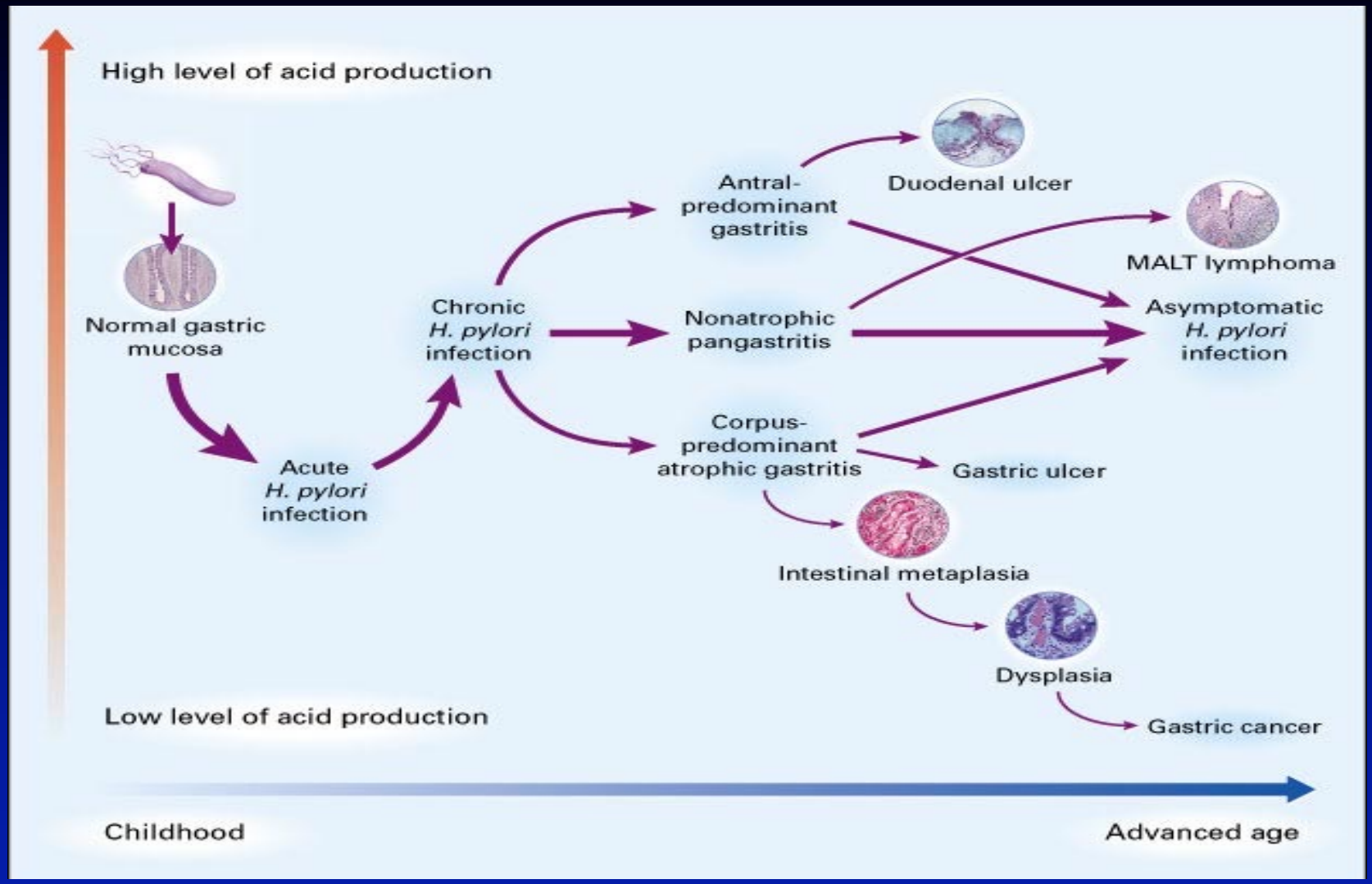
Department of Gastroenterology and Hepatology, Erasmus MC—University Medical Center, Rotterdam, The Netherlands

Pattern of gastritis	Gastric histology	Duodenal histology	Acid secretion	Clinical condition
 Pan-gastritis	• Chronic inflammation • Atrophy • Intestinal metaplasia	• Normal	• Reduced	• Gastric ulcer • Gastric cancer
 Antral-predominant	• Chronic inflammation • Polymorph activity	• Gastric metaplasia • Active chronic inflammation	• Increased	• Duodenal ulcer

Review | 01 July 2006

Pathogenesis of *Helicobacter pylori* Infection

Authors: Johannes G. Kusters, Arnoud H. M. van Vliet, Ernst J. Kuipers


High level of acid production
Normal gastric mucosa
Acute H. pylori infection
Chronic H. pylori infection
Antral-predominant gastritis
Duodenal ulcer
Nonatrophic pangastritis
MALT lymphoma
Asymptomatic H. pylori infection
Corpus-predominant atrophic gastritis
Gastric ulcer
Intestinal metaplasia
Dysplasia
Gastric cancer
Low level of acid production
Childhood
Advanced age

NEJM 2002

Substitute marker for acid secretion

- **PEPSINOGENS**

Pentti Ilmari SIPPONEN

Accuracy of the GastroPanel test in the detection of atrophic gastritis

Semi Korpela

2015, European journal of gastroenterology & hepatology

	Lot nr 16GC0702	Lot nr 17HC0702	Lot nr 16PA0702	Lot nr 23PB0703
Sample	**Gastrin 17**	**H.pylori**	**Pepsinogen I**	**Pepsinogen II**
Unit of measure	pmol/l	EIU	ug	ug/l
Conrtol range	8 to 11	40 - 60	33 - 49	23 - 31

Prof. Alley study, Sowetho, South Africa

South Africa Series 2007 / Pentti Sipponen

Case No.	Antrum Chr.infl.	Activity	Atrophy	IM	Hp	Corpus Chr.infl.	Activity	Atrophy	IM	Hp	Hp Positivity	Remarks	Antrum/Corpus	Histology: Panel-type Classifi-cation	Name	Date of birth	S	Age	Collected		PGI	PGII	PGI/II	G17b	G17s	HPAB	HP	Pentti Sipponen Histology: Panel-type Classifi-cation	Gastr SoftD 27050
																dd/mm/yy		y			µg/l	µg/l		pmol	pmol	EIU	+/-		

Prof. Alley study, Sowetho, South Africa

South Africa Series 2007 / Pentti Sipponen

Case No.	Antrum Chr.infl.	Activity	Atrophy	IM	Hp	Corpus Chr.infl.	Activity	Atrophy	IM	Hp	Hp Positivity	Remarks	Antrum/Corpus	Histology: Panel-type Classification
111	2	1	1	1	0	2	1	1	0	1	1	on PPI?	A1/A1	S
112	1	0	0	0	1	2	0	1	1	1	1		S/A1	S
113	2	0	0	0	1	2	0	0	0	1	1		S/S	S
114	2	0	0	0	2	2	0	0	0	3	1		S/S	S
115	1	0	0	0	1	2	0	0	0	0	1		S/S	S
116	3	0	0	0	3	2	0	0	0	3	1		S/S	S
117	1	0	0	0	2	2	0	0	0	2	1		S/S	S
118	1	0	0	0	2	2	1	0	0	3	1		S/S	S
119	1	0	0	0	2	1	0	0	0	3	1		S/S	S
120	1	0	0	0	3	2	0	0	0	3	1		S/S	S
121	2	0	0	0	3	2	0	0	0	3	1		S/S	S
122	2	1	0	0	1	2	1	0	0	0	1		S/S	S
123	2	0	1	0	1	2	1	1	0	1	1		A1/A1	S
200	CARCINOMA OF INTESTINAL S					1	3	3	3	0	0		?/A3	C

Pentti Sipponen

Name	Date of birth	S	Age	Illectaly		PGI	PGII	PGI/II	G17b	G17s	HPAB	HP	Histology: Panel-type Classification	Gastr SoftD 27050
	dd/mm/yy		y			µg/l	µg/l		pmol	pmol	EIU	+/-		
PRO111	20/02/45	S	59	###	##	129,1	38,5	3,4	2,8		97,8	+	S	S
PRO112	20/02/45	S	59	###	##	69,3	11,1	6,2	0,8		55,7	+	S	S
PRO113	20/02/45	S	59	###	##	133,9	31,9	4,2	13,2		2732,6	+	S	S
PRO114	20/02/45	S	59	###	##	92,9	15,3	6,1	4,7		107,7	+	S	S
PRO115	20/02/45	S	59	###	##	78,1	12,7	6,1	2		269,7	+	S	S
PRO116	20/02/45	S	59	###	##	84,9	24,3	3,5	94,8		18633	+	S	S
PRO117	20/02/45	S	59	###	##	81,8	15,9	5,1	7,4		101,6	+	S	S
PRO118	20/02/45	S	59	###	##	56,8	13,4	4,2	6,8		48,4	+	S	S
PRO119	20/02/45	S	59	###	##	81	9,5	8,5	16,3		29,3	-	S	N
PRO120	20/02/45	S	59	###	##	124,7	18,4	6,8	2		33	+	S	S
PRO121	20/02/45	S	59	###	##	72,5	13,4	5,4	5		177,9	+	S	S
PRO122	20/02/45	S	59	###	##	48,2	29,3	1,6	3,5		146	+	S	C
PRO123	20/02/45	S	59	###	##	104,6	14	7,5	3,1		190,1	+	S	S
PRO200	20/02/45	S	59	###	##	5,9	5,2	1,1	134,7		16,1	-	C	C

-----Original Message-----
From: Pentti Sipponen [mailto:pentti.sipponen@sll.fimnet.fi]
Sent: 28. toukokuuta 2008 10:16
To: Paloheimo, Lea
Cc: AletP@ScientificGroup.com; VicusV@ScientificGroup.com; Rao, Venkat
Subject: Re: Prof Alley study in Soweto . Final results with the newGastroSoftD100 version

Anyhow, I believe the result is very good. Especiallly high specificity of 94,6% and high negative predictive value of 86,9% indicate that GastroPanel can find the healthy stomachs with high percentage. In this sence the results are excellent. Congratulations.

Please do not hesitate to contact us

Best regards,

C . ENVIRONMENTAL FACTORS

- Altitude
- Diet - SALT

WJGO World Journal of Gastrointestinal Oncology

Submit a Manuscript: http://www.wjgnet.com/esps/
Help Desk: http://www.wjgnet.com/esps/helpdesk.aspx
DOI: 10.4251/wjgo.v8.i4.341

World J Gastrointest Oncol 2016 April 15; 8(4): 341-350
ISSN 1948-5204 (online)

TOPIC HIGHLIGHT

2016 *Helicobacter pylori*: Global view

Helicobacter pylori associated Asian enigma: Does diet deserve distinction?

Syed Faisal Zaidi

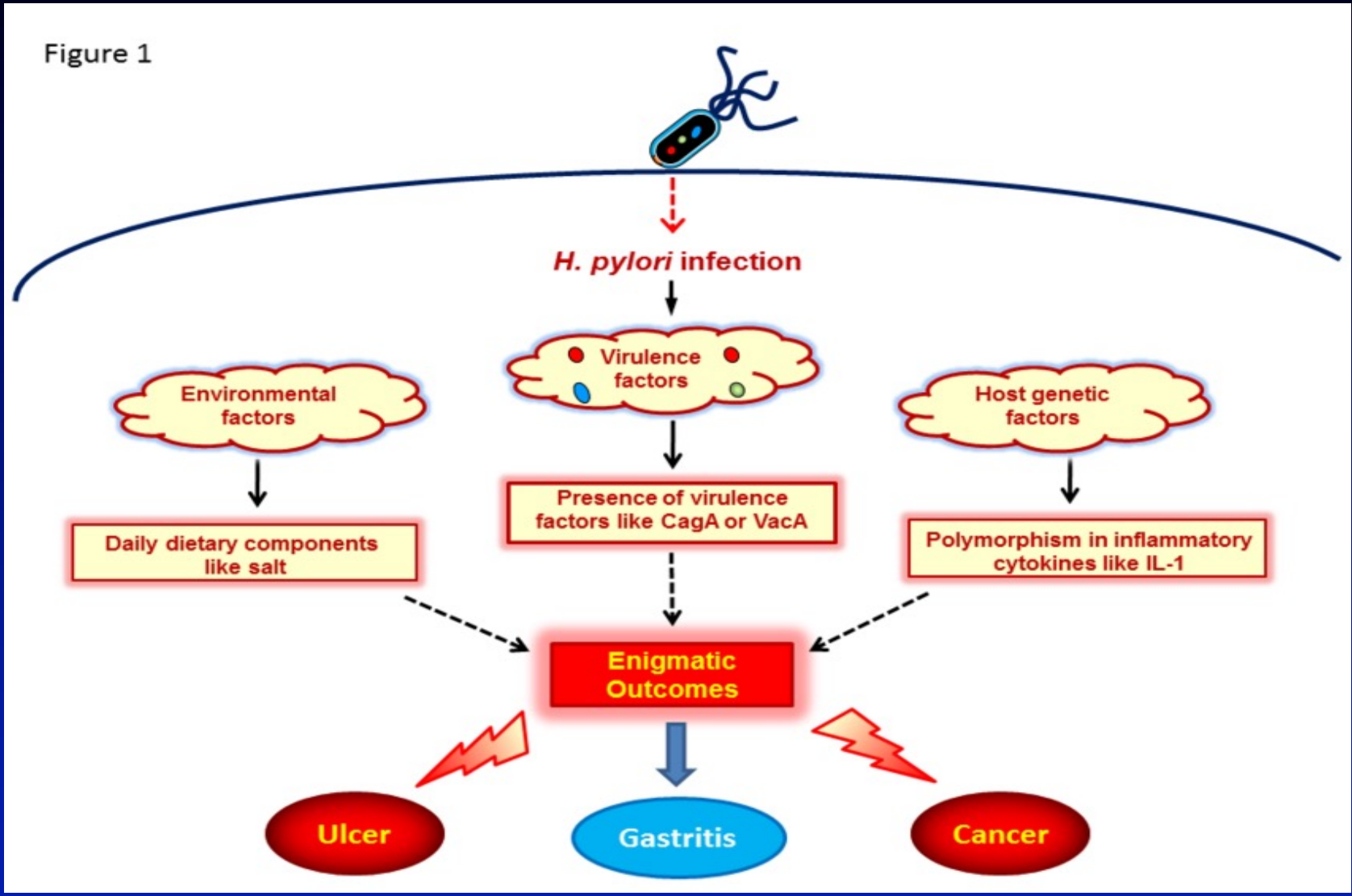


Figure 1
H. pylori infection
Virulence factors
Environmental factors
Host genetic factors
Presence of virulence factors like CagA or VacA
Daily dietary components like salt
Polymorphism in inflammatory cytokines like IL-1
Enigmatic Outcomes
Ulcer
Gastritis
Cancer

PRO STUDY

- 371 Consecutive Patients
- Questionnare
- Endoscopy
- Biopsies : 5 sets (Antrum / Corpus)
  1 – histology
  2 – Snap Frozen – PCR (Bacteriology)
  1 – Snap Frozen - RNA extraction
  1 – Culture (Antibiotic sensitivity)
- Bloods : Gastropanel , Ig's

354	Goodman Boxana	09427793	21-09-06	O	1536FredomChartersq		75	M	OGJ ?Ca
355	William Ngwenya	09445352	26-09-06	O	1554 Zone1 diepkloof	C	53	M	? Ca Stomach
356	Dudu Ann Tsotsetsi	gp08818282	05-10-06	O	236 Zone3 natalspruit Lcn	C	56	F	>pre Pyl GU
357	Rudha Ndlovu	gt09446594	11-10-06	O	2467 kgolaSt Wolmstadt		54	F	GU=bilr reflux
358	Carel de Jager	gp08816853	06-10-06	O	16Ohara st W-turfontein		60		>Gas Ulcer
359	Eric Matha	gt09447343	10-10-06	O			56	M	> GU D1 Ulcer
360	Phumrlele Ngcobo	gt09460621	03-11-06	O	71B zone2 Meadowlnds		36	F	GU+dU
361	Eunice masale	gt09461313	09-11-06	O	5392 Orlando east	9351643	45	F	Multiple GU
3602	S.Myeni	gt0946555	20-11-06	In-P	355 Siphwe Vlg Dobsnvl	C	60	F	Antral GU
3603	Cecelia Shifango	gt09454152	12-12-06	In-P	11907 Zone 9 pimville	C	69	F	? Ca?
3604	Chicoane Doris	gp08830304	02-01-07	In-P	44A White City Jabavu		73	F	mass in stomach?
3605	Evelyn Mothebyowe	08788914	02-01-07	In-P			48	F	???stomach
3606	JohannesMolepo		04-01-07	O	640 White City Jabuvu		46	M	????
3607	Emily Sonti	gp08641921	08-01-07	O	1212MaggulaStMoletsane		77	F	>Gu?Ca
3608	Johannes Soetland	08832759	17-01-07	O	14HtherlndStX14EldoPrk		61	M	Ca stomach
3609	Catrherine Samara	00454923	30-01-07	O			78		>GU-
3610	Edward Nanzini	gp08720254	19-01-07	O	952 Mkhonza St Dube		78	M	> GU
3611	Catherine Cauara	gt09455225	30-01-07	O	421 Fox st Jeppestown		58	F	GU

DISASTER

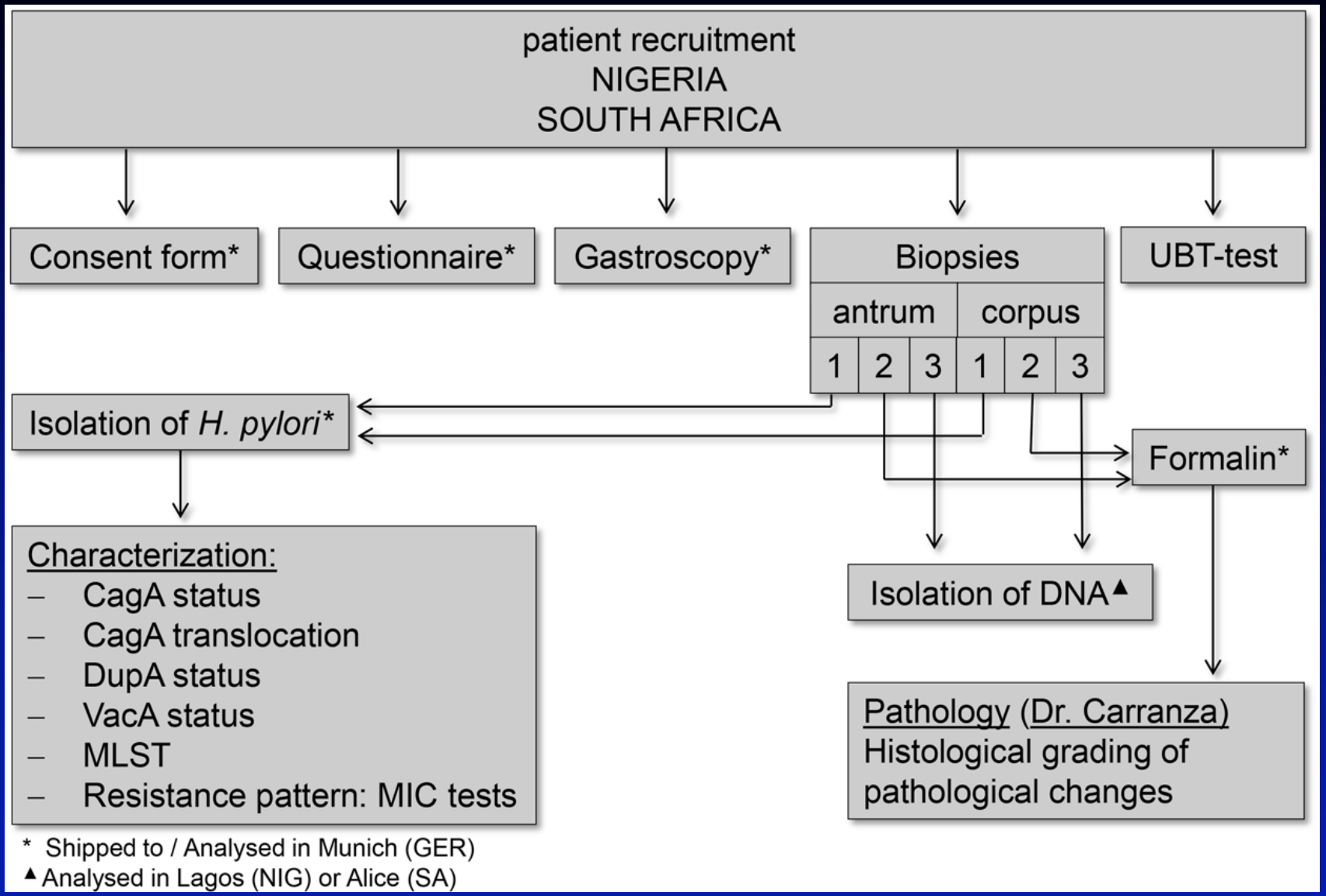
- Quantas Flight – delayed for 7 hours
- They did not place the box in – 70
- Arrived in Sydney – all denatured

RECENT

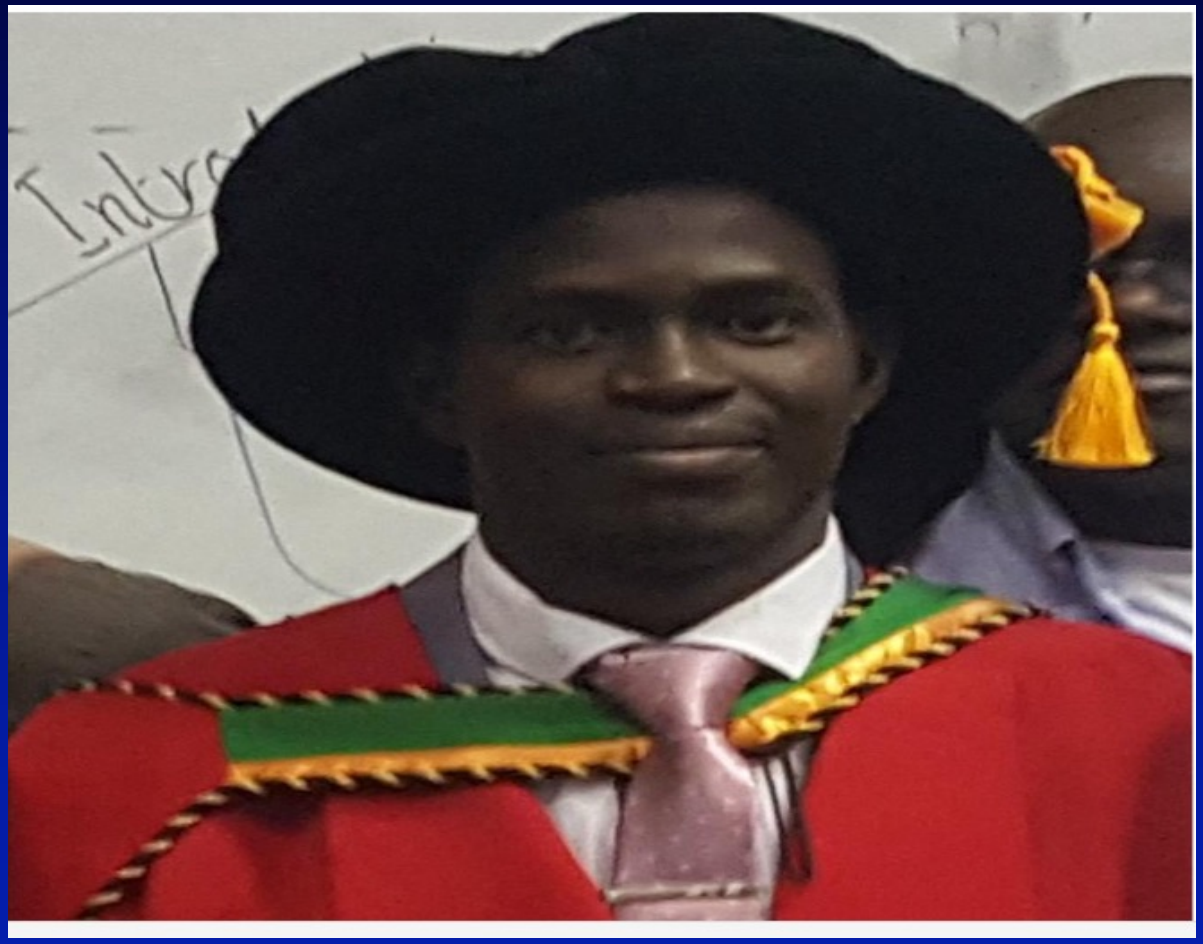
DFG form 54.012 – 04/14

Project Description – Project Proposals

Rainer Haas, München, Germany
Stella I. Smith, Lagos, Nigeria
Anna M. Clarke, Alice, South Africa


patient recruitment
NIGERIA
SOUTH AFRICA
Consent form*
Questionnaire*
Gastroscopy*
Biopsies
antrum
corpus
1
2
3
1
2
3
UBT-test
Isolation of H. pylori*
Formalin*
Characterization:
– CagA status
– CagA translocation
– DupA status
– VacA status
– MLST
– Resistance pattern: MIC tests
Isolation of DNA▲
Pathology (Dr. Carranza)
Histological grading of pathological changes
* Shipped to / Analysed in Munich (GER)
▲ Analysed in Lagos (NIG) or Alice (SA)

Ayodeji (James) Idowu



University of Fort Hare
Together in Excellence

Prevalence, diagnostic accuracy and molecular characterization of *Helicobacter Pylori* strains from patients with gastroduodenal pathologies in Chris Hani Baragwanath Academic Hospital, Soweto, South Africa

By

AYODEJI AKINDELE IDOWU (201516917)

A thesis submitted in fulfilment of the requirements for the degree of

Doctor of Philosophy

SCIENTIFIC REPORTS

nature research

Check for updates

Helicobacter pylori patient isolates from South Africa and Nigeria differ in virulence factor pathogenicity profile and associated gastric disease outcome

Pia Palamides[1] ✉, Tolulope Jolaiya[2], Ayodeji Idowu[3], Eva Loell[1], Charles Onyekwere[4], Rose Ugiagbe[5], Ifeanyi Agbo[5], Olufunmilayo Lesi[6], Dennis Ndububa[7], Olusegun Adekanle[7], Manuel Carranza[8], Reidwaan Ally[9], Henry Njom[3], Isaac A. Adeleye[2], Ute Harrison[1], Anna Clarke[3], Wolfgang Fischer[1,11], Stella Smith[10] & Rainer Haas[1,11] ✉

Idowu *et al. BMC Gastroenterology* (2019) 19:73
https://doi.org/10.1186/s12876-019-0986-0

RESEARCH ARTICLE

Detection of *Helicobacter pylori* and its virulence genes (*cag*A, *dup*A, and *vac*A) among patients with gastroduodenal diseases in Chris Hani Baragwanath Academic Hospital, South Africa

Ayodeji Idowu[1*], Asisipho Mzukwa[1], Ute Harrison[2], Pia Palamides[2], Rainer Haas[2], Melvin Mbao[3], Razinah Mamdoo[3], Jonathan Bolon[3], Tolulope Jolaiya[4], Stella Smith[5], Reidwaan Ally[3], Anna Clarke[1] a
Henry Njom[1]

Topic: Evaluation of biofilm production of *Helicobacter pylori* strains isolated from gastric biopsies: An *in vitro* study in Chris Hani Baragwanath Academic Hospital South Africa.

Authors' list: Ayodeji Idowu[1,2*], Hafeez Mohamed[2], Priyamvada Pradeep[2], Razinah Mamdoo[1], Siyanda Mahlasela[1], Nazeer Chopdat[1], Hitendrakumar Bhaga[1], Reidwaan Ally[1]

Authors' affiliations

1. African Institute of Digestive Diseases, Division of Gastroenterology, Chris Hani Baragwanath Academic Hospital (CHBAH), Soweto, Johannesburg 2013, South Africa.
2. Austell Pharmaceuticals (Pty) Ltd., 1 Sherborne Road, Parktown 2193, Johannesburg South Africa.

*Corresponding author

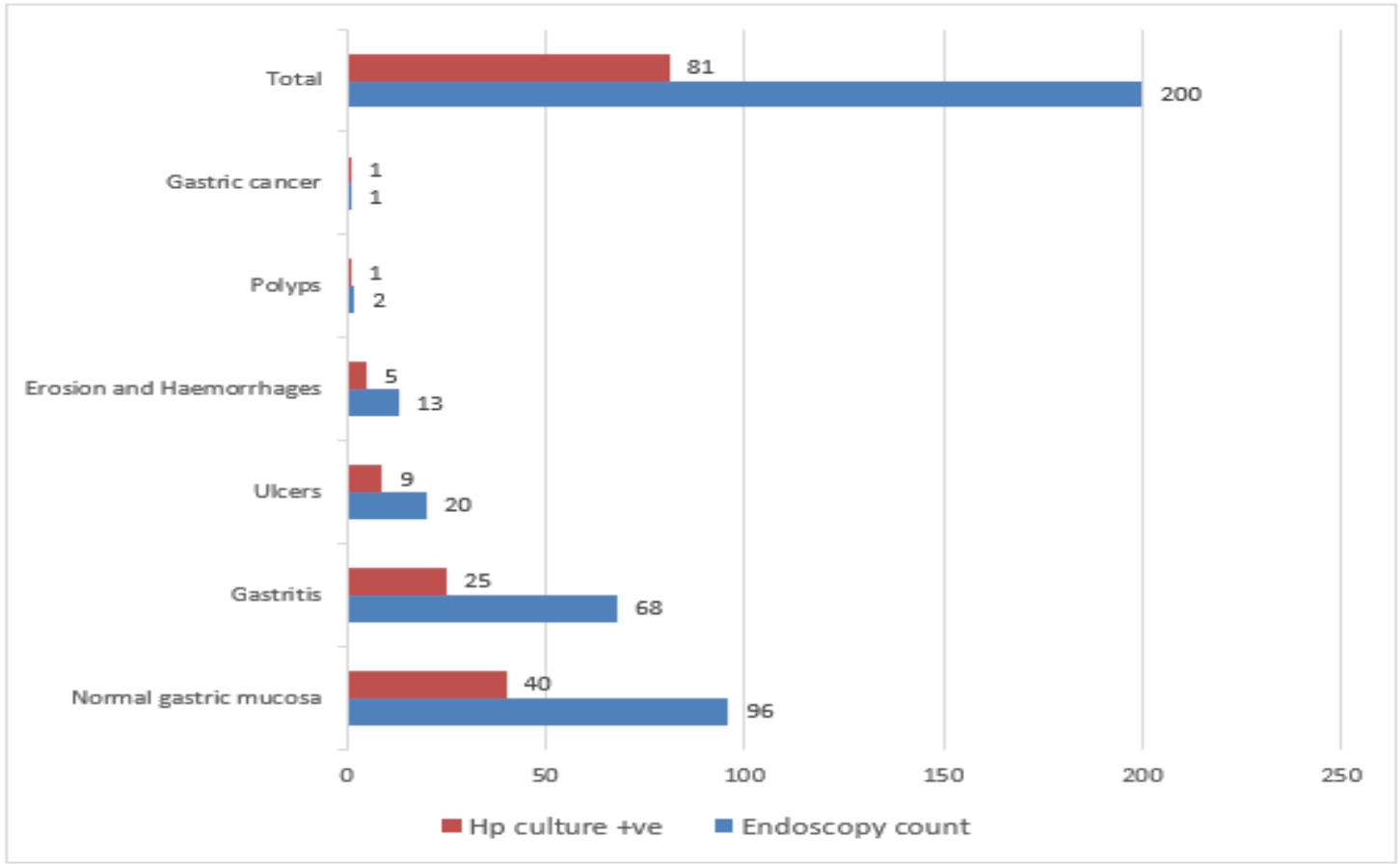


Figure 1. Endoscopy findings and *Hp* culture results

Table 2. Comparison of *Hp* biofilm results between PCR vs TCP and CRA

PCR genes K747_10375/*homD*	**TCP**					**CRA**				
	N-BF	W-BF	M-BF	S-BF	**Total**	N-BF	W-BF	M-BF	S-BF	**Total**
Positive	3	13	18	39	**73**	6	13	16	38	**73**
Negative	8	0	0	0	**8**	8	0	0	0	**8**
Total	11	**13**	**18**	**39**	**81**	14	**13**	**16**	**38**	**81**

Abbreviations: N-BF (Non-biofilm formers); W-BF (Weak biofilm formers); M-BF (Moderate biofilm formers); S-BF (Strong biofilm formers).

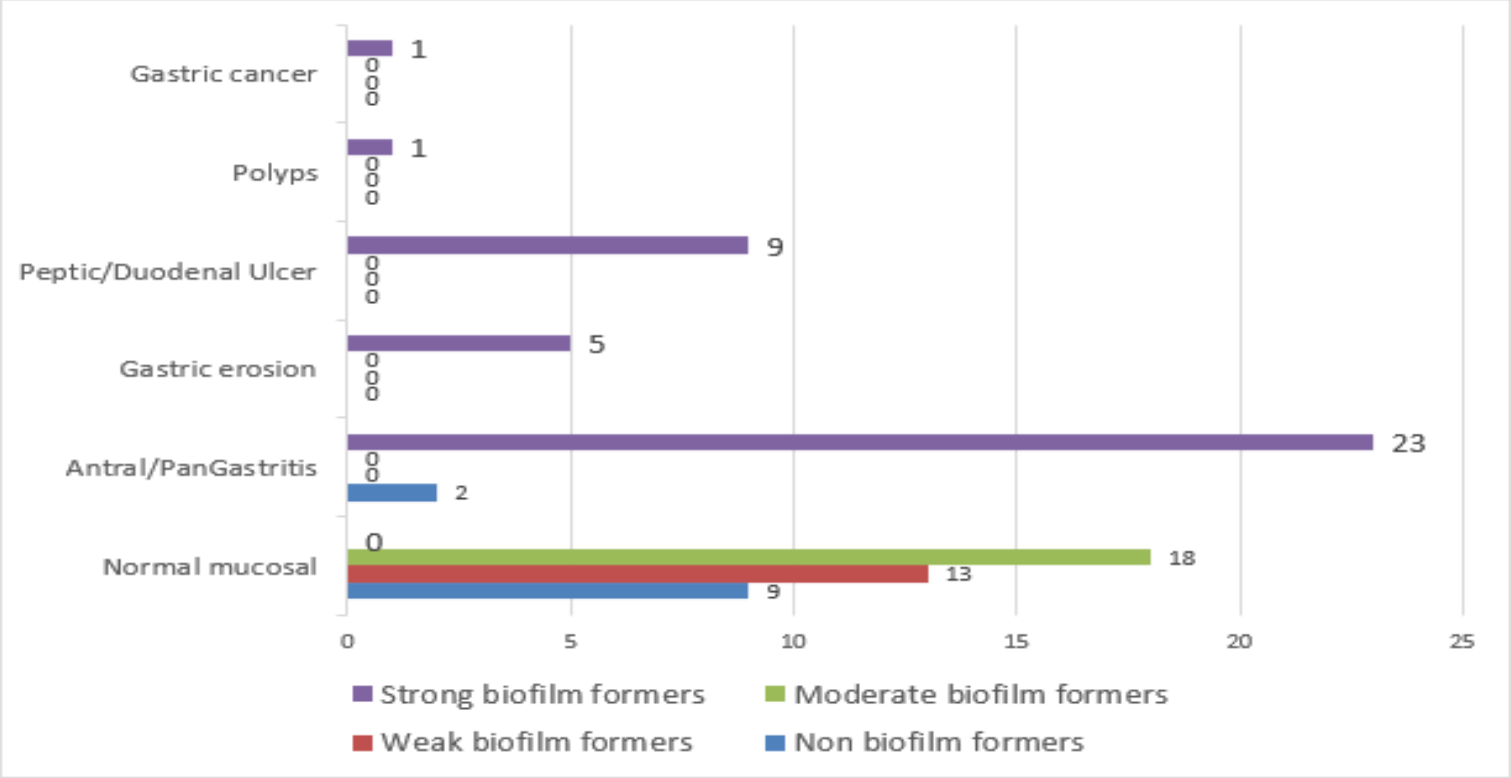
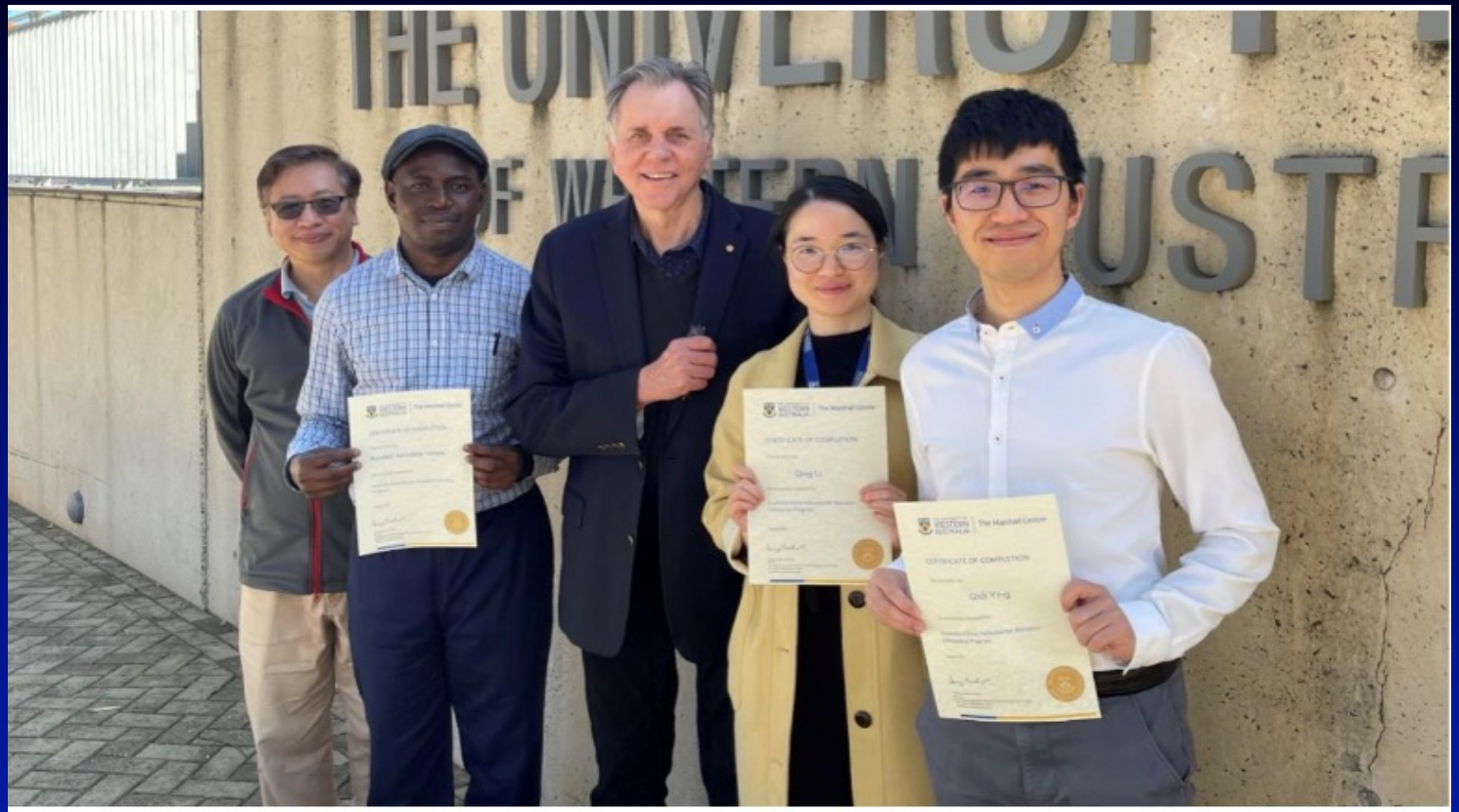


Figure 4. Distribution of *Hp* biofilm production and gastric disease pathology of patients

Conclusion

This study showed good performance and synergy among the three methods used in detecting *Hp* biofilm production. It revealed that most *Hp* strains from South Africa are strong biofilm formers and are associated with gastric diseases. Furthermore, the present study has demonstrated that for effective management of *Hp* in the clinical settings, the factor of biofilm formation by the pathogen, besides the burden of antibiotic drug resistance, must also be considered. Further investigations are encouraged to assess the pattern of resistance of *Hp* biofilm against commonly prescribed antibiotic drugs in South Africa. This knowledge will help in better understanding of the disease pathogen and appropriate treatment strategies will be employed against the infections.


THE UNIVERSITY OF WESTERN AUSTRALIA
The Marshall Centre
CERTIFICATE OF COMPLETION
Ayodeji Akindele Idowu
Australia Helicobacter Research Fellowship Program



With Prof. Barry Marshall (middle), two other students (right front) and H. pylori lab manager (far left).

SUMMARY - Soweto

- Bacterium – virulent
- Host response – Anti inflammatory
  Reduces Inflammation
  Mitigation of pathways – cancer
- Acid secretion – Pepsinogens - surrogate
- Gastrtopanel – ideal test for Africa