



# Draft Genome Sequences of 13 Colombian *Helicobacter pylori* Strains Isolated from Pacific Coast and Andean Residents

Alvaro Pazos,<sup>a\*</sup> Nuri Kodaman,<sup>b</sup> M. Blanca Piazuolo,<sup>a</sup> Judith Romero-Gallo,<sup>a</sup> Rafal S. Sobota,<sup>c</sup> Dawn A. Israel,<sup>a</sup> Luis E. Bravo,<sup>d</sup> Douglas R. Morgan,<sup>a</sup> Keith T. Wilson,<sup>a,e</sup> Pelayo Correa,<sup>a</sup> Richard M. Peek, Jr.,<sup>a</sup> Scott M. Williams,<sup>b</sup> Barbara G. Schneider<sup>a</sup>

Division of Gastroenterology, Department of Medicine, Vanderbilt University Medical Center, Nashville, Tennessee, USA<sup>a</sup>; Department of Epidemiology and Biostatistics, Case Western Reserve University, Cleveland, Ohio, USA<sup>b</sup>; Vanderbilt Genetics Institute, Vanderbilt University Medical Center, Nashville, Tennessee, USA<sup>c</sup>; Department of Pathology, School of Medicine, Universidad del Valle, Cali, Colombia<sup>d</sup>; Department of Veterans Affairs, Veterans Affairs Tennessee Valley Healthcare System and Office of Medical Research, Nashville, Tennessee, USA<sup>e</sup>

**ABSTRACT** We present here the draft genomes of 13 *Helicobacter pylori* strains isolated from Colombian residents on the Pacific coast ( $n = 6$ ) and in the Andes mountains ( $n = 7$ ), locations that differ in gastric cancer risk. These 13 strains were obtained from individuals with diagnosed gastric lesions.

Infection of human gastric mucosae with *Helicobacter pylori* is the major known risk factor for gastric cancer (1, 2), a disease that killed an estimated 723,000 people worldwide in 2012 and is the third most common cause of cancer deaths (3). Infection is typically acquired in childhood, and about half of the world's population is infected. Although most infected persons have mild symptoms and no serious sequelae, a small proportion (1 to 3%) of those infected may develop gastric cancer. The series of lesions that may lead to the intestinal type of gastric cancer include nonatrophic gastritis, multifocal atrophic gastritis, intestinal metaplasia, and dysplasia (4, 5). Previously we reported that the disrupted coevolution of human hosts and *H. pylori* genomes is associated with more advanced gastric lesions in Colombian populations (6).

Here, we present draft genomes of 13 *H. pylori* strains isolated from residents of Tumaco ( $n = 6$ ) on the Pacific coast, where incidence of gastric cancer is low, and from residents of Túquerres ( $n = 7$ ) in the Andes mountains, where incidence is high. All participants provided informed consent; the study was approved by the institutional review boards of Vanderbilt University Medical Center and of the local hospitals. Participants were 40 years of age or older and were genotyped using the ImmunoChip (7), as previously described (6), to estimate ancestry (Table 1). Diagnosis of gastric biopsies and cultures of one antral gastric biopsy per subject were performed as previously described (6). DNA from the *H. pylori* pellet was isolated with DNAzol (Thermo Fisher Scientific) and then sheared and used to prepare a library for 250-bp paired-end sequencing with an Illumina MiSeq instrument. Sequencing reads were assembled *de novo* into contigs using CLC Genomics Workbench version 8.5 (CLC bio, Aarhus, Denmark). Calculated depth of coverage ranged from 36 $\times$  to 142 $\times$ . All genomes contained the *cag* pathogenicity island (8). Statistics for the assemblies are shown in Table 1. Draft sequences were annotated using the NCBI Prokaryotic Genome Annotation Pipeline.

Received 3 February 2017 Accepted 16 February 2017 Published 13 April 2017

**Citation** Pazos A, Kodaman N, Piazuolo MB, Romero-Gallo J, Sobota RS, Israel DA, Bravo LE, Morgan DR, Wilson KT, Correa P, Peek RM, Jr, Williams SM, Schneider BG. 2017. Draft genome sequences of 13 Colombian *Helicobacter pylori* strains isolated from Pacific coast and Andean residents. *Genome Announc* 5:e00113-17. <https://doi.org/10.1128/genomeA.00113-17>.

**Copyright** © 2017 Pazos et al. This is an open-access article distributed under the terms of the [Creative Commons Attribution 4.0 International license](https://creativecommons.org/licenses/by/4.0/).

Address correspondence to Barbara G. Schneider, [barbara.schneider@vanderbilt.edu](mailto:barbara.schneider@vanderbilt.edu).

\* Present address: Alvaro Pazos, Department of Biology, University of Nariño, Pasto, Colombia.

**TABLE 1** Statistics for Colombian *Helicobacter pylori* strains

Strain ID	Accession no.	Host residence	No. of contigs >200 bp	Coverage (×)	Genome size (bp)	N <sub>50</sub> (bp)	Diagnosis	Age of host [yr(s)]	Estimated ancestry of host (%)		
									European	African	Amerind
PZ5005_3A3	MTWJ00000000	LR <sup>a</sup>	51	65	1,672,956	77,172	NAG <sup>c</sup>	53	16.4	76.5	17.1
PZ5006_3A3	MTWK00000000	LR	53	57	1,643,170	101,990	NAG	45	10	79.6	10.4
PZ5009_3A2	MSY00000000	LR	53	101	1,677,035	96,381	NAG	53	22.7	60.8	16.5
PZ5016_3A3	MTWL00000000	LR	44	38	1,644,424	80,627	MAG <sup>d</sup>	40	19.6	49.1	31.3
PZ5019_3A3	MTWM00000000	LR	44	44	1,681,561	103,042	IM	47	23.4	29.7	46.9
PZ5033_3A2	MTWN00000000	LR	60	142	1,656,908	130,584	IM	57	11.8	72.6	15.5
SV328_2	MTWO00000000	HR <sup>b</sup>	56	37	1,645,479	95,218	Dys <sup>e</sup>	54	44.8	4.3	50.9
SV340_2	MTWP00000000	HR	53	41	1,633,298	128,372	NAG	45	16.6	0.8	82.6
SV355_2	MTWQ00000000	HR	39	41	1,635,304	125,154	IM <sup>f</sup>	45	13.1	1.8	85.1
SV376_1	MTWR00000000	HR	207	43	1,691,791	30,293	IM	55	45.6	7.6	46.8
SV380_1	MTWU00000000	HR	40	41	1,631,819	136,108	IM	43	36.9	0.7	62.5
SV397_2	MTWS00000000	HR	47	38	1,668,205	100,030	NAG <sup>g</sup>	45	32.8	1.4	65.8
SV449_1	MTWT00000000	HR	41	36	1,654,884	104,133	IM	42	28.3	9.2	62.5

<sup>a</sup>LR, host is resident of area where risk for gastric cancer is low.

<sup>b</sup>HR, host is resident of area where risk for gastric cancer is high.

<sup>c</sup>NAG, nonatrophic gastritis.

<sup>d</sup>MAG, multifocal atrophic gastritis.

<sup>e</sup>Dys, dysplasia.

<sup>f</sup>IM, intestinal metaplasia.

<sup>g</sup>Diagnosis from corpus biopsy only. Severity of lesions may be underestimated due to lack of incisura and antrum biopsies.

**Accession number(s).** The draft genome sequence projects presented here have been deposited at DDBJ/ENA/GenBank under the accession numbers shown in Table 1. The versions described in this paper are the first versions (e.g., MTWJ01000000 to MTWT01000000).

## ACKNOWLEDGMENTS

This study was supported by U.S. National Institutes of Health grants P01 CA028842 (awarded to K.T.W. and P.C.), 2 R01 DK 58587, 6 R01 CA 77955, and 5 P01 CA 116087 (awarded to R.M.P.), and 5 R01 CA190612 (awarded to K.T.W.), Prevent Cancer Foundation (awarded to B.G.S.), and by the National Center for Advancing Translational Sciences grant 5UL1TR000445 (awarded to P.C.; principal investigator, Gordon Bernard).

We appreciate the assistance of Chelsea R. O'Hara, Mary E. Aakre, and Ivo C. Violich from the VANTAGE Laboratory at Vanderbilt University Medical Center, as well as helpful advice from Travis A. Clark and Holli Dilks regarding whole-genome sequencing.

## REFERENCES

- International Agency for Research on Cancer. 1994. Schistosomes, liver flukes and *Helicobacter pylori*. Monogr Eval Carcinog Risks Hum 61: 218–220.
- Peek RM, Jr, Blaser MJ. 2002. *Helicobacter pylori* and gastrointestinal tract adenocarcinomas. Nat Rev Cancer 2:28–37. <https://doi.org/10.1038/nrc703>.
- Ferlay J, Soerjomataram I, Ervik M, Dikshit R, Eser S, Mathers C, Rebelo M, Parkin DM, Forman D, Bray F. 2013. GLOBOCAN 2012 v1.0, Cancer incidence and mortality worldwide. IARC CancerBase 11. International Agency for Research on Cancer, Lyon, France. <http://globocan.iarc.fr>.
- Correa P. 1988. A human model of gastric carcinogenesis. Cancer Res 48:3554–3560.
- Correa P. 1983. The gastric precancerous process. Cancer Surv 2:437–450.
- Kodaman N, Pazos A, Schneider BG, Piazuolo MB, Mera R, Sobota RS, Sicinschi LA, Shaffer CL, Romero-Gallo J, de Sablet T, Harder RH, Bravo LE, Peek RM, Wilson KT, Cover TL, Williams SM, Correa P. 2014. Human and *Helicobacter pylori* coevolution shapes the risk of gastric disease. Proc Natl Acad Sci U S A 111:1455–1460. <https://doi.org/10.1073/pnas.1318093111>.
- Cortes A, Brown MA. 2011. Promise and pitfalls of the Immunochip. Arthritis Res Ther 13:101. <https://doi.org/10.1186/ar3204>.
- Blaser MJ, Perez-Perez GI, Kleanthous H, Cover TL, Peek RM, Chyou PH, Stemmermann GN, Nomura A. 1995. Infection with *Helicobacter pylori* strains possessing *cagA* is associated with an increased risk of developing adenocarcinoma of the stomach. Cancer Res 55:2111–2115.