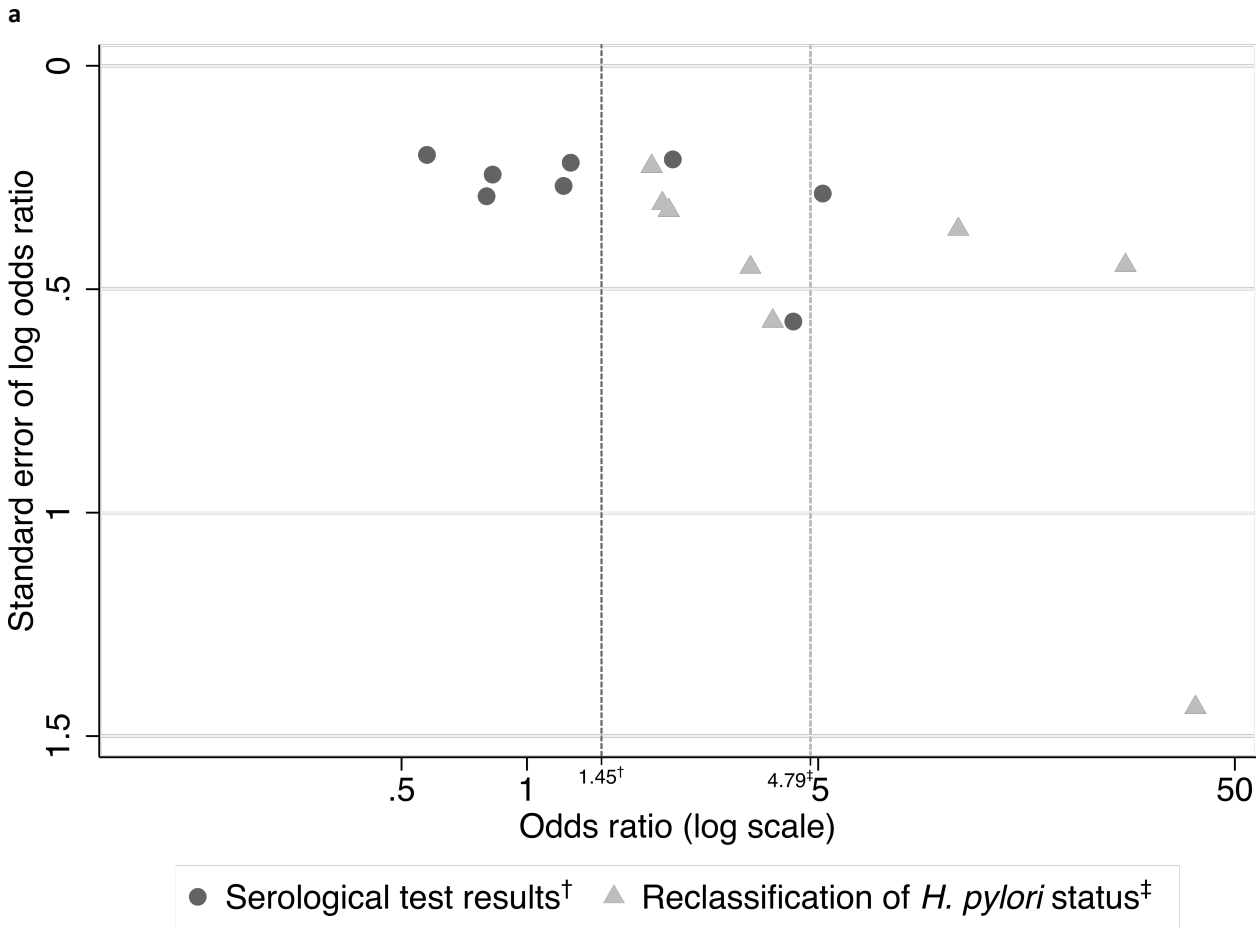
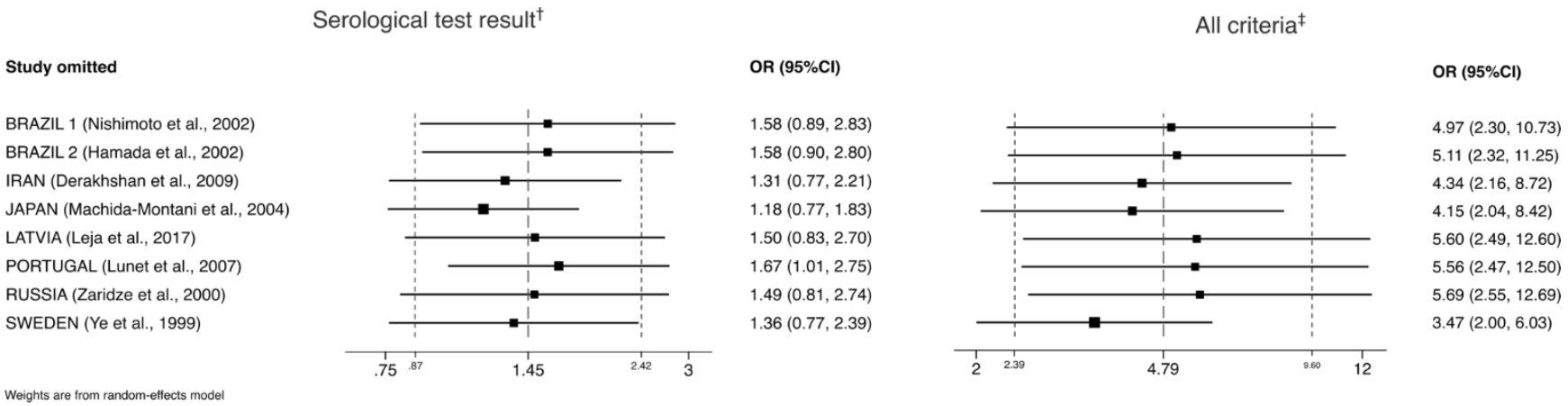


Supplementary Figure 1. Funnel plot of the Stomach Cancer Pooling (StoP) Project studies evaluating the association between *H. pylori* infection status, considering serological test results† and additionally reclassifying as positive the *H. pylori* infection status of those likely to correspond to false-negative results of the serological test‡, and non-cardia gastric cancer.

† *H. pylori* infection status was defined considering serological tests using the same criteria applied in each original study. Egger's regression asymmetry test p-value=0.319. Pooled estimate using random-effects analysis: OR=1.45, 95%CI:0.87-2.42.

‡ Additional criteria were used to reclassify *H.* pylori infection status: a negative serological test result for *H. pylori* infection was reclassified as positive if a positive result was obtained for cytotoxin-associated gene A (CagA) status (cases and controls when available) or gastric atrophy was present (cases and controls when available) or tumour stage at diagnosis was advanced (cases only when available). Egger's regression asymmetry test p-value=0.125. Pooled estimate using random-effects analysis: OR=4.79, 95%CI:2.39-9.60.

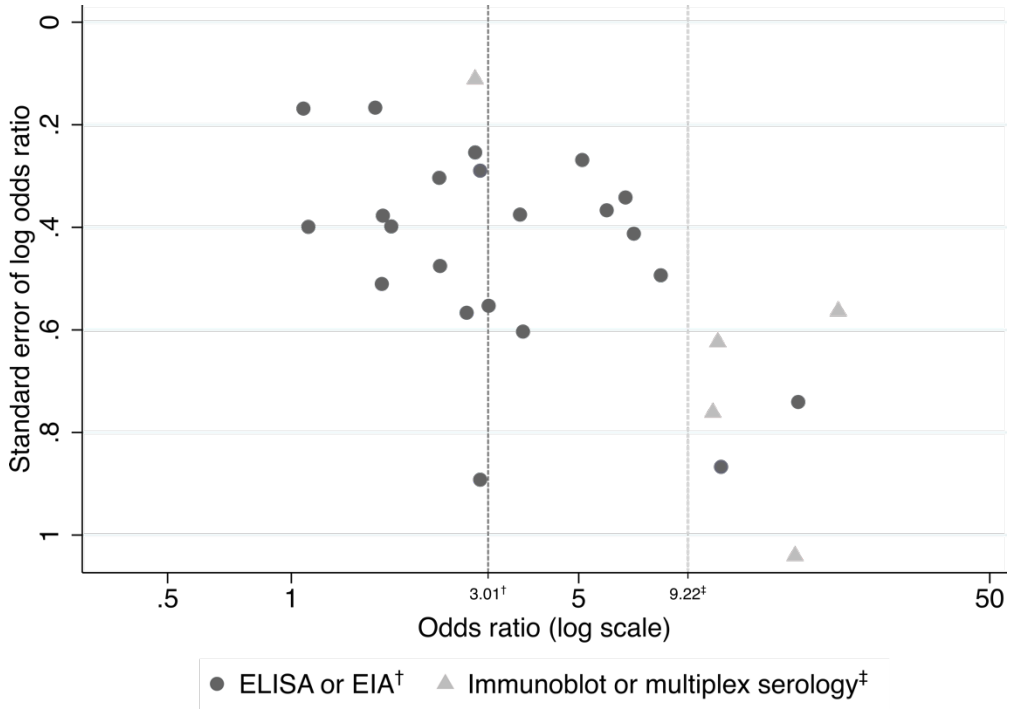


Supplementary Figure 2. Leave-one-out meta-analysis describing the association between *H. pylori* infection status, considering serological test results[†] and additionally reclassifying as positive the *H. pylori* infection status of those likely to correspond to false-negative results of the serological test[‡], and non-cardia gastric cancer using estimates from the Stomach Cancer Pooling (StoP) Project database.

95%CI – 95% confidence interval; OR – odds ratio.

[†] *H. pylori* infection status was defined considering serological tests using the same criteria applied in each original study. Pooled estimate using random-effects analysis: OR=1.45, 95%CI:0.87-2.42.

[‡] Additional criteria were used to reclassify *H.* pylori infection status: a negative serological test result for *H. pylori* infection was reclassified as positive if a positive result was obtained for cytotoxin-associated gene A (CagA) status (cases and controls when available) or gastric atrophy was present (cases and controls when available) or tumour stage at diagnosis was advanced (cases only when available). Pooled estimate using random-effects analysis: OR=4.79, 95%CI:2.39-9.60.

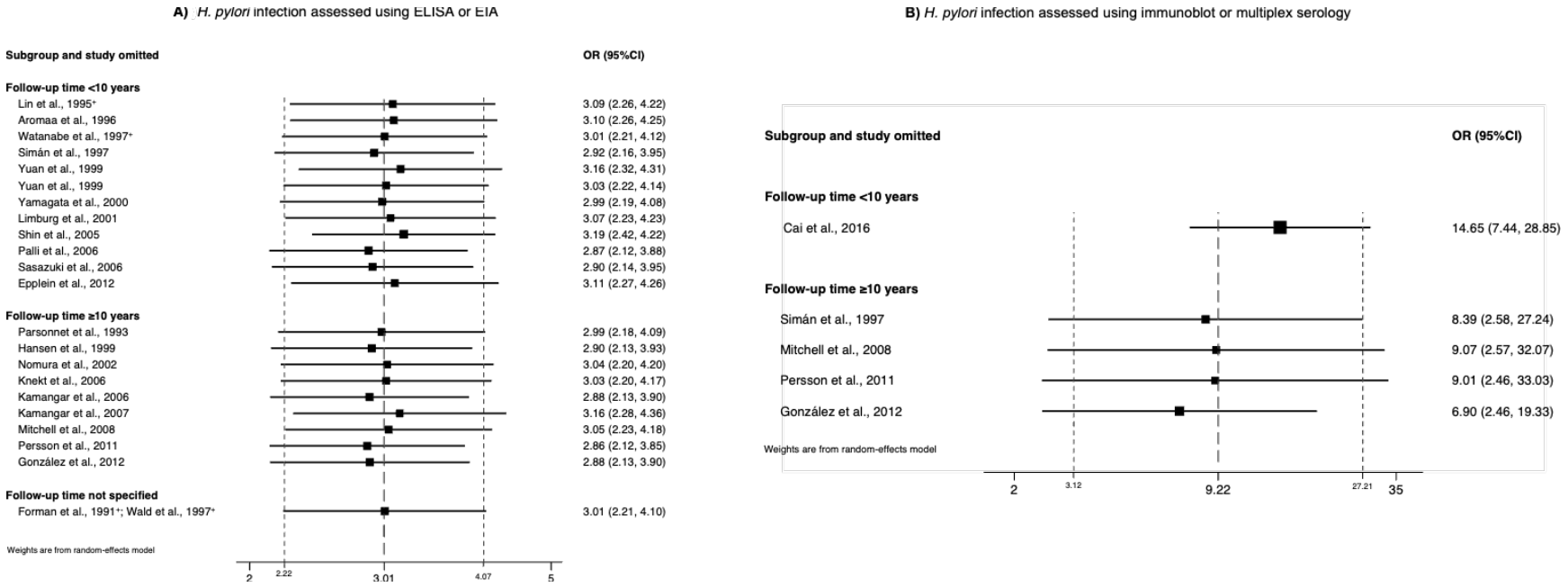


Supplementary Figure 3. Funnel plot of literature review of prospective studies of non-cardia gastric cancer quantifying the association with *H. pylori* infection considering the method of *H. pylori* infection assessment [A) ELISA or EIA†, or B) immunoblot or multiplex serology‡].

EIA – enzyme immunoassay; ELISA – enzyme-linked immunosorbent assay.

† *H. pylori* infection status was defined using ELISA or EIA. Egger's regression asymmetry test p-value=0.006 (follow-up time <10 years: p-value=0.126; follow-up time ≥10 years: p-value=0.002). Pooled estimate using random-effects analysis: OR=3.01, 95%CI:2.22-4.07.

‡ *H. pylori* infection status was defined using immunoblot or multiplex serology. Egger's regression asymmetry test p-value=0.021 (follow-up time <10 years: p-value=--; follow-up time ≥10 years: p-value=0.759). Pooled estimate using random-effects analysis: OR=9.22, 95%CI:3.12-27.21.



Supplementary Figure 4. Leave-one-out meta-analysis of the literature review of prospective studies of non-cardia gastric cancer quantifying the association with *H. pylori* infection considering the method of *H. pylori* infection assessment [A) ELISA or EIA, or B) immunoblot or multiplex serology] and follow-up time in years (<10, ≥10, not specified).

95%CI – 95% confidence interval; EIA – enzyme immunoassay; ELISA – enzyme-linked immunosorbent assay.

† Data obtained from the *Helicobacter* and Cancer Collaborative Group.[1]

Supplementary Table 1. Specific variables available for each study.

Study	Variables available
BRAZIL 1 Nishimoto et al., 2002[2]	CagA serostatus[†] (cases and controls), Gastric atrophy (cases and controls) Sex, Age, Social class, Histological type, Family history of gastric cancer in first-degree relatives, Smoking status, Alcohol drinking, Fruit and vegetables intake, Salt intake Multicentre study
BRAZIL 2 Hamada et al., 2002[3]	CagA serostatus[†] (cases and controls), Gastric atrophy (cases and controls) Sex, Age, Social class, Histological type, Family history of gastric cancer in first-degree relatives, Smoking status, Alcohol drinking, Fruit and vegetables intake, Salt intake Multicentre study
IRAN Derakhshan et al., 2009[4]	Gastric atrophy (cases only) Sex, Age, Histological type
JAPAN Machida-Montani et al., 2004[5]	Gastric atrophy (cases and controls) Sex, Age, Social class, Family history of gastric cancer in first-degree relatives, Smoking status, Alcohol drinking, Fruit and vegetables intake, Salt intake Multicentre study
LATVIA Leja et al., 2017[6]	Gastric atrophy (cases and controls), Tumour stage (cases only) Sex, Age, Histological type, Family history of gastric cancer in first-degree relatives, Smoking status, Alcohol drinking, Fruit and vegetables intake, Salt intake
PORTUGAL Lunet et al., 2007[7]	CagA serostatus[‡] (cases and controls), Gastric atrophy (cases only), Tumour stage (cases only) Sex, Age, Social class, Histological type, Family history of gastric cancer in first-degree relatives, Smoking status, Alcohol drinking, Fruit and vegetables intake, Salt intake
RUSSIA Zaridze et al., 2000[8]	Tumour stage (cases only) Sex, Age, Social class, Histological type, Family history of gastric cancer in first-degree relatives, Smoking status, Alcohol drinking, Fruit and vegetables intake, Salt intake
SWEDEN Ye et al., 1999[9]	CagA serostatus[§] (cases and controls) Sex, Age, Social class, Histological type, Family history of gastric cancer in first-degree relatives, Smoking status, Alcohol drinking, Fruit and vegetables intake, Salt intake

CagA – Cytotoxin-associated gene A.

[†] Serum samples were tested for CagA (RADIM S.A., Parc Scientifique du Sart-Til-man, Italy) and determined by optical density reading in relation to a standard curve, which was obtained through a calibrator using a kit. A value of 10 units per milliliter (U/ml) or more was considered a positive test in CagA-Ab; this criterion was determined based on the manufacturer's recommendation.[10]

[‡] The assay was conducted as proposed by the manufacturer, and the results were interpreted following the manufacturer's recommended criteria for CagA status as the presence of the CagA band at 116kD with one or more of the following bands: 89kD (VacA), 37kD, 35kD, 30kD (UreA) and 19.5kD. The presence of the CagA band was also considered regardless of the remaining criteria to define infection.[11]

[§] CagA antibodies were detected with immunoblot using an antigen from *H. pylori* strain NCTC 11637, a previously described and evaluated method.[12]

Supplementary Table 2. Distribution of non-cardia gastric cancer cases and controls according to sex, age and other selected covariates.

	Cases		Controls	
	N	%	N	%
Total	1325	100.0	3121	100.0
Sex				
Males	809	60.6	1694	54.3
Females	516	39.4	1427	45.7
Age (years)				
≤65	724	54.6	2083	66.7
>65	601	45.4	1038	33.3
Geographic region				
Americas	734	62.2	2062	71.3
Asia	182	15.4	419	14.5
Europe	264	22.4	412	14.2
Socioeconomic status†				
Low	653	59.4	1008	36.6
Intermediate	341	31.0	1087	39.4
High	106	9.6	663	24.0
Missing	*225*		*363*	
Family history of cancer‡				
No	987	82.1	1944	89.0
Yes	215	17.9	239	11.0
Missing	*123*		*938*	
Smoking status				
Never	614	49.4	1592	54.4
Ever	629	50.6	1336	45.6
Missing	*82*		*193*	
Alcohol drinking‡				
Never	378	35.4	819	34.5
Ever	691	64.6	1782	68.5
Missing	*256*		*520*	
Fruit and vegetable intake‡				
Low	240	22.3	451	16.5
Intermediate/High	834	77.7	2275	83.5
Missing	*251*		*395*	
Salt intake‡				
Low	615	71.0	1572	73.0
Intermediate/High	251	29.0	582	27.0
Missing	*459*		*967*	
Histological type§				
Intestinal	544	50.3	--	
Diffuse	364	33.6	--	
Unspecified	174	16.1	--	
Missing	*243*		--	

Percentages may not add to 100 due to rounding.

† As defined in each original study based on education, income or occupation. No information available for study: LATVIA (Leja et al., 2017).[6]

‡ No information available for study: IRAN (Derakhshan et al., 2009).[4]

§ No information available for study: JAPAN (Machida-Montani et al., 2004).[5]

Supplementary Table 3 Number of *Helicobacter pylori* negative and positive non-cardia gastric cancer cases and controls by study, considering serological test results[†] and additionally reclassifying as positive the *H. pylori* infection status of cases and controls likely to correspond to false-negative results of the serological test[‡].

	CASES					CONTROLS				
	TOTAL (N)	Serological test results[†]		Reclassification of *H. pylori* status[‡]		TOTAL (N)	Serological test results[†]		Reclassification of *H. pylori* status[‡]	
		H. pylori +	*H. pylori* -	*H. pylori* +	*H. pylori* -		*H. pylori* +	*H. pylori* -	*H. pylori* +	*H. pylori* -
TOTAL	1325	1049	276	1228	97	3121	2304	817	2386	735
BRAZIL 1 Nishimoto et al., 2002[2]	183	136	47	181	2	226	178	48	206	20
BRAZIL 2 Hamada et al., 2002[3]	81	51	30	74	7	186	127	59	145	41
IRAN Derakhshan et al., 2009[4]	56	52	4	56	0	119	88	31	88	31
JAPAN Machida-Montani et al., 2004[5]	126	107	19	116	10	300	165	135	169	131
LATVIA Leja et al., 2017[6]	164	134	30	146	18	228	180	48	183	45
PORTUGAL Lunet et al., 2007[7]	364	315	49	352	12	1376	1215	161	1233	143
RUSSIA Zaridze et al., 1999[8]	126	69	57	81	45	441	215	226	215	226
SWEDEN Ye et al., 1999[9]	225	185	40	222	3	245	136	109	147	98

[†] *H. pylori* infection status was defined considering serological tests using the same criteria applied in each original study.

[‡] Additional criteria were used to reclassify *H.* pylori infection status: a negative serological test result for *H. pylori* infection was reclassified as positive if a positive result was obtained for cytotoxin associated-gene A (CagA) status or gastric atrophy was present or tumour stage at diagnosis was advanced.

Supplementary Table 4. Assessing the quality of StoP Project studies included in the meta-analyses.

StoP Project Studies	Selection	Comparability	Exposure
BRAZIL 1 Nishimoto et al., 2002[2]	★★★	★★	★★
BRAZIL 2 Hamada et al., 2002[3]	★★★	★★	★★
IRAN Derakhshan et al., 2009[4]	★★★	★★	★★
JAPAN Machida-Montani et al., 2004[5]	★★★★	★★	★★
LATVIA Leja et al., 2017[6]	★★★	★★	★★
PORTUGAL Lunet et al., 2007[7]	★★★★	★★	★★
RUSSIA Zaridze et al., 1999[8]	★★★	★★	★★★
SWEDEN Ye et al., 1999[9]	★★★★	★★	★★

Supplementary Table 5. Pooled estimates for the association between *H. pylori* infection status, considering serological test results[†] and additionally reclassifying as positive the *H. pylori* infection status of those likely to correspond to false-negative results of the serological test[‡], and non-cardia gastric cancer using estimates from the Stomach Cancer Pooling (StoP) Project database.

Criterion	Serological test results[†]			Reclassification of *H. pylori* status[‡]	
	aOR[§] (95%CI)	I^2 (%)		aOR[§,¶] (95%CI)	I^2 (%)
CagA[††]	0.96 (0.51-1.83)	87.3		3.18 (0.88-11.44)	93.1
Atrophy[‡‡]	1.41 (0.71-2.80)	88.4		**2.64 (1.21-5.75)**	85.7
Stage[§§]	0.94 (0.53-1.65)	77.5		1.24 (0.68-2.25)	80.2
All[¶¶]	1.45 (0.87-2.42)	87.8		**4.79 (2.39-9.60)**	84.3

95%CI – 95% confidence interval; aOR – adjusted odds ratio.

[†] *H. pylori* infection status was defined considering serological tests using the same criteria applied in each original study.

[‡] Additional criteria were used to reclassify *H.* pylori infection status: a negative serological test result for *H. pylori* infection was reclassified as positive if a positive result was obtained for cytotoxin-associated gene A (CagA) status (cases and controls when available) or gastric atrophy was present (cases and controls when available) or tumour stage at diagnosis was advanced (cases only when available).

[§] Pooled ORs were computed using random-effects models. Study-specific ORs were adjusted, when available and applicable, for sex, age (five-year age groups: <40; 40-44; …; 70-74; ≥75), socioeconomic status (low, intermediate, or high, as defined in each original study based on education, income or occupation) and study center (for multicenter studies).

[¶] The crude OR and 95%CI for the study from IRAN (Derakhshan et al., 2009)[4] was calculated by adding 0.5 to each cell as all cases were *H. pylori* positive following the reclassification of infection status.

[††] Including studies: BRAZIL 1 (Nishimoto et al., 2002);[2] BRAZIL 2 (Hamada et al., 2002);[3] PORTUGAL (Lunet et al., 2007);[7] SWEDEN (Ye et al., 1999).[9]

[‡‡] Including studies: BRAZIL 1 (Nishimoto et al., 2002);[2] BRAZIL 2 (Hamada et al., 2002);[3] IRAN (Derakhshan et al., 2009);[4] JAPAN (Machida-Montani et al., 2004);[5] LATVIA (Leja et al., 2017);[6] PORTUGAL (Lunet et al., 2007).[7]

[§§] Including studies: LATVIA (Leja et al., 2017);[6] PORTUGAL (Lunet et al., 2007);[7] RUSSIA (Zaridze et al., 2000).[8]

[¶¶] Including studies: BRAZIL 1 (Nishimoto et al., 2002);[2] BRAZIL 2 (Hamada et al., 2002);[3] IRAN (Derakhshan et al., 2009);[4] JAPAN (Machida-Montani et al., 2004);[5] LATVIA (Leja et al., 2017);[6] PORTUGAL (Lunet et al., 2007);[7] RUSSIA (Zaridze et al., 2000);[8] SWEDEN (Ye et al., 1999).[9]

Supplementary Table 6. Literature review of prospective studies of non-cardia gastric cancer quantifying the association with *Helicobacter pylori* infection.

Study	Country	Follow-up (years)	N cases	% cases positive	OR (95%CI)	Assessment of *H. pylori* infection status
Forman et al., 1991[†, 13] Wald et al., 1997[†, 14]	United Kingdom (British United Provident Study and Caerphilly Collaborative Heart Disease)	8.7 (median – all gastric cancer cases)	9	66.7	2.88 (0.50-16.5)	ELISA (IgG antibodies against whole-cell antigens)
Parsonnet et al., 1993[15]	United States of America (California)	14.2 (mean)	98	85.7	3.6 (1.7-7.4)	ELISA (IgG antibodies against whole-cell antigens)
Lin et al., 1995[†, 16]	Taiwan	3.1 (mean)	21	71.4	1.66 (0.61-4.51) *Males only*	ELISA (IgG antibodies against whole-cell antigens)
Watanabe et al., 1997[†, 17]	Japan (two rural towns in Kyoto)	3.2 (mean)	38	89.5	3.02 (1.02-8.91)	EIA (IgG antibodies against whole-cell antigens)
Hansen et al., 1999[18]	Norway (Janus Cohort)	12.0 (mean)	132	Not specified	5.85 (2.85-12.0)	ELISA (IgG antibodies against whole-cell antigens)
Yuan et al., 1999[19]	China (Shanghai)	5.2 (mean)	114	87.7	Follow-up <5 years 1.10 (0.50-2.39) Follow-up ≥5 years 2.67 (0.88-8.11) *Males only*	ELISA (IgG antibodies against whole-cell antigens)
Yamagata et al., 2000[20]	Japan (Hisayama Study)	4.9 (mean)	40	Not specified	3.66 (1.12-11.92) *Males only*	EIA (IgG antibodies against whole-cell antigens)
Unpublished in 2001[†, ‡, 1]	Iceland	Not specified	35	77.1	1.52 (0.66-3.53)	Not specified
Unpublished in 2001[†, §, 1]	Finland	Not specified	93	93.5	4.72 (1.98-11.3)	Not specified
Nomura, et al., 2002[21]	United States of America (Japanese Americans in Hawaii)	12.7 (mean)	261	88.5	2.8 (1.7-4.6)	ELISA (IgG antibodies against whole-cell antigens)
				72.4	1.9 (1.3-2.8)	ELISA (IgG antibodies against CagA antigens)
Shin et al., 2005[22]	Korea (Korean Multicentre Cancer Cohort)	2.6 (mean)	70	85.7	1.07 (0.77-1.49)	ELISA (IgG antibodies against whole-cell antigens)
Kamangar et al., 2006[23]	Finland (Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study)	14 (maximum)	173	86.1	3.32 (1.72-6.42)	ELISA (IgG antibodies against whole-cell antigens)
				29.5	6.55 (2.31–18.53)	ELISA (IgG antibodies against whole-cell antigens positive and CagA antigens negative)
				63.8	8.93 (3.27-24.40)	ELISA (IgG antibodies against whole-cell and CagA antigens)
				93.1	7.92 (3.02-20.9)	ELISA (IgG antibodies against CagA antigens positive)
Aromaa et al., 1996[24]	Finland (Finnish Mobile Clinic Health Examination Survey)	9.5 (mean)	75	100.0	2.76 (1.11-6.87)	EIA (IgA antibodies against whole-cell antigens)
				97.3	1.75 (0.80-3.81)	EIA (IgG antibodies against whole-cell antigens)
Knekt et al., 2006[25]		24 (maximum)	193	82.4	3.12 (1.97-4.95)	EIA (IgA antibodies against whole-cell antigens)
				91.2	2.88 (1.63-5.07)	EIA (IgG antibodies against whole-cell antigens)
Sasazuki et al., 2006[26]	Japan (Japan Public Health Center Study)	9 (maximum)	368	93.5	5.1 (3.0-8.6)	ELISA (IgG antibodies against whole-cell antigens)
Simán et al., 1997[27]	Sweden (Malmö Preventive Medicine Cohort)	5.7	27	88.9	11.1 (2.4-71.8)	ELISA (IgG antibodies against whole-cell antigens)
Simán et al., 2007[28]		9.2-12.6 (mean)	67	97.0	17.8 (4.2-74.8)	Immunoblot (IgG antibodies against whole-cell antigens)
				98.5	16.8 (2.2-130)	Immunoblot (IgG antibodies against CagA antigens)
				97.0	9.7 (1.5-∞)	Immunoblot (IgG antibodies against whole-cell and CagA antigens)
Mitchell et al., 2008[29]	Australia (Melbourne Collaborative Cohort Study)	11.6 (mean)	34	79.4	2.3 (0.9-5.8)	ELISA (IgG antibodies against whole-cell antigens)
				94.1	10.6 (2.4-47.4)	Immunoblot (IgG antibodies against CagA, VacA and UreA antigens)

Study	Country	Follow-up (years)	N cases	% cases positive	OR (95%CI)	Assessment of *H. pylori* infection status
Persson et al., 2011[30]	Sweden (Swedish Institute for Infectious Disease Control Biobank and Malmö Microbiology Biobank)	16.5 (mean for all gastric adenocarcinomas)	41	85.4	17.1 (4.0-72.9)	ELISA (IgG antibodies against HP-CSAs)
				82.9	10.9 (3.2-36.9)	Immunoblot (IgG antibodies again CagA)
Epplein et al., 2012[31]	China (Shanghai Men's Health Study)	3.6 (median)	226	91.6	1.67 (0.80-3.51)	EIA (IgG antibodies against CagA antigens)
				62.4	2.08 (1.31-3.30)	Multiplex serology (UreA, Catalase, GroEL, NapA, CagA, CagM, Cagd, HP0231, VacA, HpaA, Cad, HyuA, Omp, HcpC, and HP0305; 4-5 vs. 0-3)
				24.8	3.49 (2.00-6.11)	Multiplex serology (UreA, Catalase, GroEL, NapA, CagA, CagM, Cagd, HP0231, VacA, HpaA, Cad, HyuA, Omp, HcpC, and HP0305; 6 vs. 0-3)
Palli et al., 2006[32]	10 European countries (EPIC-EURGAST): Denmark, France, Germany, Greece, Italy, Netherlands, Norway, Spain, Sweden, and the United Kingdom	6.1 (mean)	127	11.8	1.6 (0.7-3.8)	ELISA (IgG antibodies against whole-cell antigens positive and CagA antigens negative)
				78.7	6.5 (3.3-12.6)	ELISA (IgG antibodies against CagA antigens)
				90.6	4.7 (2.5-9.0)	ELISA (IgG antibodies against whole-cell and CagA antigens)
González et al., 2012[33]		10.65 (mean; update)	88	81.8	6.81 (3.0-15.1)	ELISA (IgG antibodies against whole-cell antigens)
				93.2	21.4 (7.1-64.6)	Immunoblot (IgG antibodies against whole-cell antigens)
Limburg et al., 2001[34]	China (Linxian General Population Trial)	6-6.25 (mean)	82	62.2	1.68 (0.96-2.95)	ELISA (IgG antibodies against whole-cell antigens)
				36.6	1.84 (1.01-3.34)	ELISA (IgG antibodies against CagA)
				72.0	2.29 (1.26-4.14)	ELISA (IgG antibodies against whole-cell and/or CagA antigens)
Kamangar et al., 2007[35]		16 (maximum)	343	80.5	1.60 (1.15-2.21)	ELISA (IgG antibodies against whole-cell or CagA antigens)
				58.9	1.58 (1.13-2.22)	ELISA (IgG antibodies against whole-cell and CagA antigens)
				22.9	1.62 (1.08-2.45)	ELISA (IgG antibodies against whole-cell antigens positive and CagA antigens negative)
Murphy et al., 2015[36]			330	94.8	3.44 (1.91-6.19)	Multiplex serology (UreA, Catalase, GroEL, Nap A, CagA, CagM, Cagδ, HP0231, VacA, HpaA, Cad, HyuA, Omp, HcpC, and HP0305)
Cai et al., 2016[37]	East Asia (Japan – Japan Public Health Center-based Prospective Study I, Japan Public Health Center-based Prospective Study II; Korea – Korean Cancer Prevention Study II, Korean Multicenter Cancer Cohort I, Korean National Cancer Screenee Cohort; China – Linxian Nutrition Intervention Trial, Shanghai Men's Health Study, Shanghai Women's Health Study)	5.3 (median)	1608	92.0	2.80 (2.25-3.48)	Multiplex serology (UreA, Catalase, GroEL, NapA, CagA, CagM, Cagd, HP0231, VacA, HpaA, Cad, HyuA, Omp, HcpC, and HP0305)

95%CI – 95% confidence interval; CagA – cytotoxin-associated gene A; EIA – enzyme immunoassay; ELISA – enzyme-linked immunosorbent assay; HpCSA – *H. pylori* cell surface antigens; IgA – immunoglobulin A; IgG – immunoglobulin G; OR – odds ratio; UreA – urease A; VacA – vacuolating cytotoxin A.

† Data obtained from the *Helicobacter* and Cancer Collaborative Group.[1]

‡ Contributors: H Tulinius, H Ogmundsdottir (Icelandic Cancer Society, Reykjavik, Iceland).[1]

§ Contributors: TU Kosunen, OP Heinonen, (University of Helsinki, Finland); J Virtamo (National Public Health Institute, Helsinki, Finland).[1]

Supplementary Table 7. Assessing the quality of prospective studies included in the meta-analyses following the literature review.

Studies	Selection	Comparability	Outcome
Forman et al., 1991[†, 13]	★★	★★	★★
Wald et al., 1997[†, 14]			
Parsonnet et al., 1993[15]	★★★	★★	★★★
Lin et al., 1995[†, 16]	★	★★	★★
Watanabe et al., 1997[†, 17]	★★★★	★★	★★
Hansen et al., 1999[18]	★★★★	★★	★★★
Yuan et al., 1999[19]	★★	★★	★★
Yamagata et al., 2000[20]	★	★	★★
Unpublished in 2001[†, ‡, 1]	Not specified	Not specified	Not specified
Unpublished in 2001[†, §, 1]	Not specified	Not specified	Not specified
Nomura, et al., 2002[21]	★★	★	★★★
Shin et al., 2005[22]	★★★★	★	★★
Kamangar et al., 2006[23]	★	★★	★★★
Aromaa et al., 1996[24]	★★★	★★	★★★
Knekt et al., 2006[25]			
Sasazuki et al., 2006[26]	★★★★	★★	★★
Simán et al., 1997[27]	★★★	★★	★★★
Simán et al., 2007[28]			
Mitchell et al., 2008[29]	★★★★	★★	★★★
Persson et al., 2011[30]	★★★★	★★	★★★
Epplein et al., 2012[31]	★★	★★	★★
Palli et al., 2006[32]	★★★★	★★	★★★
González et al., 2012[33]			
Limburg et al., 2001[34]			
Kamangar et al., 2007[35]	★★★★	★★	★★★
Murphy et al., 2015[36]			
Cai et al., 2016[37]	★★★★	★★	★★

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