

Helicobacter pylori infection as a trigger of ANA



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Introduction

- *Helicobacter pylori* has been considered a trigger of various autoimmune diseases
- Epidemiological, serological, immunological and microbiological studies have incriminated Hp infection as pivotal for the de novo induction or as an accelerator of SLA, SSc, SSc, PBC, AIH and other autoimmune diseases



Online Submissions: <http://www.wjgnet.com/esps/bpgoffice@wjgnet.com>
doi:10.3748/wjg.v20.i3.613

World J Gastroenterol 2014 January 21; 20(3): 613-629
ISSN 1007-9327 (print) ISSN 2219-2840 (online)
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TOPIC HIGHLIGHT

WJG 20th Anniversary Special Issues (6): *Helicobacter pylori*

***Helicobacter pylori* and autoimmune disease: Cause or bystander**

Daniel S Smyk, Andreas L Koutsoumpas, Maria G Mytilinaiou, Eirini I Rigopoulou, Lazaros I Sakkas, Dimitrios P Bogdanos

Table 1 Autoimmune diseases or autoimmune disease-related disorders which have been studied for their possible (direct or indirect) relation with *Helicobacter pylori* infection

AID or AID-related disorders linked to <i>H. pylori</i>		AID or AID-related disorders linked to <i>H. pylori</i>	
1	Alopecia areata	49	Juvenile diabetes (Type 1 diabetes)
2	Antiphospholipid syndrome	50	Kawasaki syndrome
3	Autoimmune angioedema	51	Leukocytoclastic vasculitis
4	Autoimmune hepatitis	52	Lichen planus
5	Autoimmune hyperlipidemia	53	Linear IgA disease
6	Autoimmune hemolytic anemia	54	Lupus (SLE)
7	Autoimmune myocarditis	55	Microscopic polyangiitis
8	Autoimmune oophoritis	56	Mixed connective tissue disease
9	Autoimmune pancreatitis	57	Mooren's ulcer
10	Autoimmune polyglandular syndromes	58	Multiple sclerosis
11	Autoimmune thrombocytopenic purpura	59	Myositis
12	Autoimmune thyroid disease	60	Narcolepsy
13	Autoimmune urticaria	61	Neuromyelitis optica (Devic's)
14	Axonal and neuronal neuropathies	62	Neutropenia
15	Behcet's disease	63	Ocular cicatricial pemphigoid
16	Bullous pemphigoid	64	Optic neuritis
17	Cardiomyopathy	65	Palindromic rheumatism
18	Celiac disease	66	Pars planitis (peripheral uveitis)
19	Chagas disease	67	Pemphigus
20	Chronic inflammatory demyelinating polyneuropathy	68	Peripheral neuropathy
21	Chronic recurrent multifocal osteomyelitis	69	Perivenous encephalomyelitis
22	Crohn's disease	70	Pernicious anemia
23	Cogans syndrome	71	Polyarteritis nodosa
24	Demyelinating neuropathies	72	Polymyalgia rheumatica
25	Dermatitis herpetiformis	73	Polymyositis
26	Dermatomyositis	74	Primary biliary cirrhosis
27	Devic's disease (neuromyelitis optica)	75	Primary sclerosing cholangitis
28	Eosinophilic esophagitis	76	Psoriasis
29	Eosinophilic fasciitis	77	(Idiopathic) pulmonary fibrosis
30	Erythema nodosum	78	Pyoderma gangrenosum
31	Experimental allergic encephalomyelitis	79	Raynaud's phenomenon
32	Fibromyalgia	80	Reactive Arthritis
33	Fibrosing alveolitis	81	Reiter's syndrome
34	Giant cell arteritis (temporal arteritis)	82	Relapsing polychondritis
35	Giant cell myocarditis	83	Rheumatoid arthritis
36	Glomerulonephritis	84	Sarcoidosis
37	Goodpasture's syndrome	85	Scleroderma (systemic sclerosis)
38	Graves' disease	86	Sjogren's syndrome
39	Guillain-Barre syndrome	87	Temporal arteritis/Giant cell arteritis
40	Hashimoto's thyroiditis	88	Thrombocytopenic purpura
41	Henoch-Schonlein purpura	89	Transverse myelitis
42	Hypogammaglobulinemia idiopathic thrombocytopenic purpura	90	Type 1 diabetes
43	IgA nephropathy	91	Ulcerative colitis
44	IgG4-related sclerosing disease	92	Undifferentiated connective tissue disease
45	Immunoregulatory lipoproteins	93	Uveitis
46	Inclusion body myositis	94	Vasculitis (other forms)
47	Interstitial cystitis	95	Vesiculobullous dermatosis
48	Juvenile arthritis		

Table 2 Autoimmune diseases or autoimmune diseases-related disorders which have not been studied for their possible (direct or indirect) relation with *Helicobacter pylori* infection

AID or AID-related disorders not linked to <i>H. pylori</i>	
1	Acute Disseminated Encephalomyelitis
2	Acute necrotizing hemorrhagic leukoencephalitis
3	Addison's disease
4	Agammaglobulinemia
5	Amyloidosis
6	Ankylosing spondylitis
7	Anti-GBM/ Anti-TBM nephritis
8	Autoimmune aplastic anemia
9	Autoimmune dysautonomia
10	Autoimmune immunodeficiency
11	Autoimmune inner ear disease
12	Autoimmune retinopathy
13	Balo disease
14	Castleman disease
15	Chronic fatigue syndrome
16	Churg-Strauss syndrome
17	Cicatrical pemphigoid/benign mucosal pemphigoid
18	Congenital heart block
19	Coxsackie myocarditis
20	CREST disease
21	Essential mixed cryoglobulinemia
22	Discoid lupus
23	Dressler's syndrome
24	Endometriosis
25	Evans syndrome
26	Granulomatosis with Polyangiitis (formerly called Wegener's Granulomatosis)
27	Hashimoto's encephalitis
28	Herpes gestationis
29	Juvenile myositis
30	Lambert-Eaton syndrome
31	Lichen sclerosis
32	Ligneous conjunctivitis
33	Lyme disease,
34	(Chronic) Meniere's disease
35	Mucha-Habermann disease
36	Myasthenia gravis
37	Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcus
38	Paraneoplastic cerebellar degeneration
39	Paroxysmal nocturnal hemoglobinuria
40	Parry Romberg syndrome
41	Parsonage-Turner syndrome
42	POEMS syndrome
43	Postmyocardial infarction syndrome
44	Postpericardiotomy syndrome
45	Progesterone dermatitis
46	Psoriatic arthritis
47	Pure red cell aplasia
48	Reflex sympathetic dystrophy
49	Restless legs syndrome
50	Retroperitoneal fibrosis
51	Rheumatic fever
52	Schmidt syndrome
53	Scleritis
54	Sperm and testicular autoimmunity
55	Stiff person syndrome
56	Subacute bacterial endocarditis
57	Susac's syndrome
58	Sympathetic ophthalmia
59	Takayasu's arteritis
60	Tolosa-Hunt syndrome
61	Vitiligo

The list includes diseases in alphabetical order as they have been deposited in the official website of AARDA (American Autoimmune Related Diseases Association) with minor revisions. Diseases with at least one study (Pubmed Search) investigating *Helicobacter pylori* (*H. pylori*) as a trigger have been included. AID: Autoimmune disease.

Table 3 Evidence in support or against the role of *Helicobacter pylori* in autoimmune disease

Autoimmune condition	Evidence in support and/or against the role of <i>H. pylori</i>	Overall opinion
SjS	Support: Oral cavity populated with <i>H. pylori</i> Higher level of anti- <i>H. pylori</i> antibodies in SjS patients Increased incidence of mucosal associated lymphoid tissue and lymphomas in parotid and lacrimal glands of SjS patients Against: Low levels of anti- <i>H. pylori</i> antibodies in SjS patients compared to controls	Inconclusive
SSc	Support: Higher incidence of <i>H. pylori</i> antibodies in SSc patients than controls <i>H. pylori</i> eradication improves Raynaud's in SSc patients Possible protective role against Barrett's esophagus Higher level of CagA strain <i>H. pylori</i> infected patients Against: Low incidence of anti- <i>H. pylori</i> antibodies compared to controls	Inconclusive
RA	Support: Increased rheumatoid factor IgM from B cells chronically stimulated with <i>H. pylori</i> urease Against: Low prevalence of anti- <i>H. pylori</i> in RA patients Unchanged clinical course or symptomatology after <i>H. pylori</i> eradication	Unlikely
SLE	Support: <i>H. pylori</i> urease exposure induced anti-ssDNA antibody production in an animal model of SLE Against: Low levels of anti- <i>H. pylori</i> found among SLE patients, at levels comparable to controls Negative association between <i>H. pylori</i> seropositivity and the development of SLE in African-American women	Unlikely
ITP	Support: Improvement of platelet counts following <i>H. pylori</i> eradication (CagA type <i>H. pylori</i> in particular) Anti-CagA antibodies cross-react with peptides on platelets of ITP patient Platelet associated IgGs declined following <i>H. pylori</i> eradication Found in high prevalence in some ITP cohorts Platelet eluates from ITP patients recognize <i>H. pylori</i> CagA Against: Low levels of <i>H. pylori</i> found in ITP patients	Probable
AiTD	Support: Higher seropositivity and positive stool cultures for <i>H. pylori</i> in Graves' disease patients CagA strain predominant among Graves' disease patients Amino acid similarities between CagA and thyroid peroxidase Reduction in anti-thyroid antibodies following <i>H. pylori</i> eradication Against: Low levels of infection among Hashimoto's thyroiditis patients	Probable in Graves' disease Unlikely in Hashimoto's thyroiditis
MS and NMO	Support: High rate of <i>H. pylori</i> infection among NMO patients Correlation between <i>H. pylori</i> infection and presence of aquaporin-4 antibodies Against: <i>H. pylori</i> infection rates in MS patients similar to or lower than control groups	Probable in NMO Unlikely in MS
Psoriasis	Support: Higher levels of anti- <i>H. pylori</i> antibodies in patients Appears to be correlation between <i>H. pylori</i> infection and disease severity Clinical improvement following <i>H. pylori</i> eradication Against: No difference in anti- <i>H. pylori</i> levels compared to controls No difference of CagA seropositivity between patients and controls	Probable
Behçet's disease	Support: Higher infection prevalence in patients Some clinical improvement noted after eradication Against: No difference between patients and controls	Unlikely
Alopecia areata	Support: Higher infection prevalence Against: No difference in infection prevalence between patients and controls	Unlikely

Table 3 Evidence in support or against the role of *Helicobacter pylori* in autoimmune disease

Autoimmune condition	Evidence in support and/or against the role of <i>H. pylori</i>	Overall opinion
SjS	Support: - Oral cavity populated with <i>H. pylori</i> - Higher level of anti-<i>H. pylori</i> antibodies in SjS patients - Increased incidence of mucosal associated lymphoid tissue and lymphomas in parotid and lacrimal glands of SjS patients Against: - Low levels of anti-<i>H. pylori</i> antibodies in SjS patients compared to controls	Inconclusive
SSc	Support: - Higher incidence of <i>H. pylori</i> antibodies in SSc patients than controls <i>H. pylori</i> eradication improves Raynaud's in SSc patients - Possible protective role against Barrett's esophagus - Higher level of CagA strain <i>H. pylori</i> infected patients Against: - Low incidence of anti-<i>H. pylori</i> antibodies compared to controls	Inconclusive
RA	Support: - Increased rheumatoid factor IgM from B cells chronically stimulated with <i>H. pylori</i> urease Against: - Low prevalence of anti-<i>H. pylori</i> in RA patients - Unchanged clinical course or symptomatology after <i>H. pylori</i> eradication	Unlikely



PBC	Support: - Higher prevalence of anti-<i>H. pylori</i> antibodies among PBC patients - Amino acid similarities between pyruvate dehydrogenase E2 (PDC-E2) and urease beta of <i>H. pylori</i> Against: - No differences of infection found between patients and controls - No immunological cross reactivities at the B or CD4 T-cell level - No crossreactivity between <i>H. pylori</i> VacA and PDC-E2	Unlikely
AIH	Support: - No current evidence Against: - No differences in anti-<i>H. pylori</i> antibodies between patients and controls - No significant difference between <i>H. pylori</i> in liver tissues in patients compared to controls	Unlikely
PSC	Support: - Detectable <i>H. pylori</i> DNA in PSC liver samples - CagA in samples from PSC patients - Concomitant ulcerative colitis may be related to <i>H. pylori</i> translocation from the gut to the liver Against: - No difference in <i>H. pylori</i> prevalence among pediatric or adult PSC patients compared to controls - No significant difference between <i>H. pylori</i> in liver tissues in patients compared to controls	Unlikely

> J Neuroimmunol. 2016 Sep 15:298:19–23. doi: 10.1016/j.jneuroim.2016.06.009.
Epub 2016 Jun 23.

Anti-hsp60 antibody responses based on *Helicobacter pylori* in patients with multiple sclerosis: (ir)Relevance to disease pathogenesis

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Lazaros I Sakkas⁶, Georgios Hadjigeorgiou², Dimitrios P Bogdanos⁷

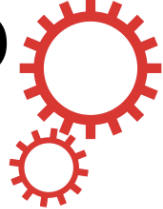
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PMID: 27609271 DOI: [10.1016/j.jneuroim.2016.06.009](https://doi.org/10.1016/j.jneuroim.2016.06.009)

Abstract

In view of published data suggesting that *Helicobacter pylori* (Hp) is a trigger of multiple sclerosis (MS), we assessed anti-heat shock protein 60 (hsp60)Hp antibody reactivity in 129 MS patients and 48 demographically-matched healthy controls (HCs). Anti-Hp antibodies by ELISA were more elevated in MS than HCs but did not differ between different MS phenotypes. All anti-Hp-positive MS sera, irrespectively of their clinical phenotype, were anti-anti-hsp60 positive. Anti-hsp60 Hp seropositivity correlated with age at disease onset. In conclusion, anti-hsp60 Hp antibodies are present in all anti-Hp positive MS patients, and their relevance to disease pathogenesis is questionable.

SCIENTIFIC REPORTS



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Immune responses against *Helicobacter pylori*-specific antigens differentiate relapsing remitting from secondary progressive multiple sclerosis

Received: 12 April 2017

Accepted: 29 June 2017

Published online: 11 August 2017

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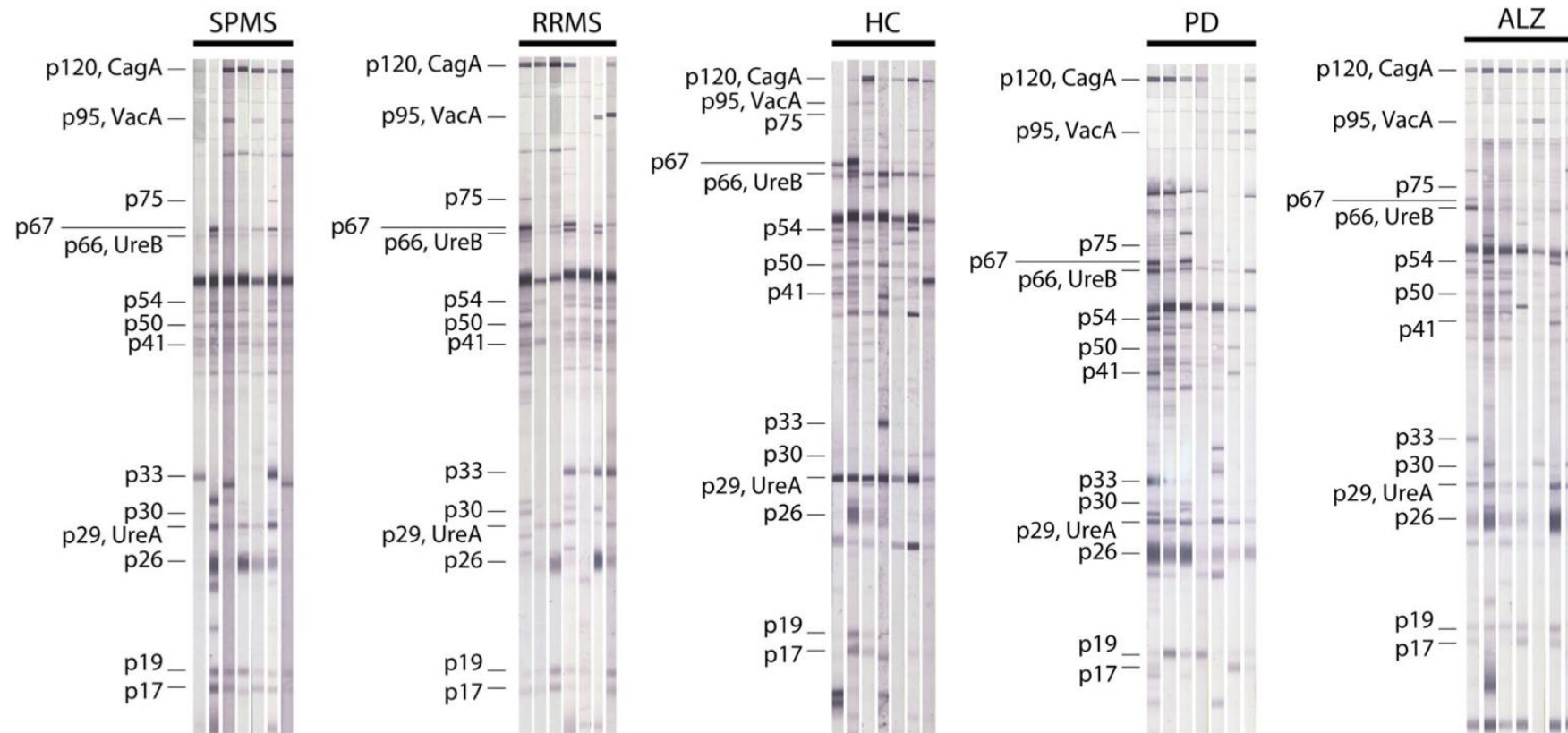


Figure 1. Antibody reactivity against *H. pylori* antigens by Western immunoblotting in representative RRMS (n = 7) patients, SPMS (n = 7) patients, healthy controls (HC) (n = 7), patients with Parkinson's disease (PD) (n = 7) and patients with Alzheimer's disease (ALZ) (n = 7). Abbreviations: CagA, protein from cytotoxin-associated gene A; UreA, urease A; UreB, urease B; VacA, vacuolating cytotoxin A.



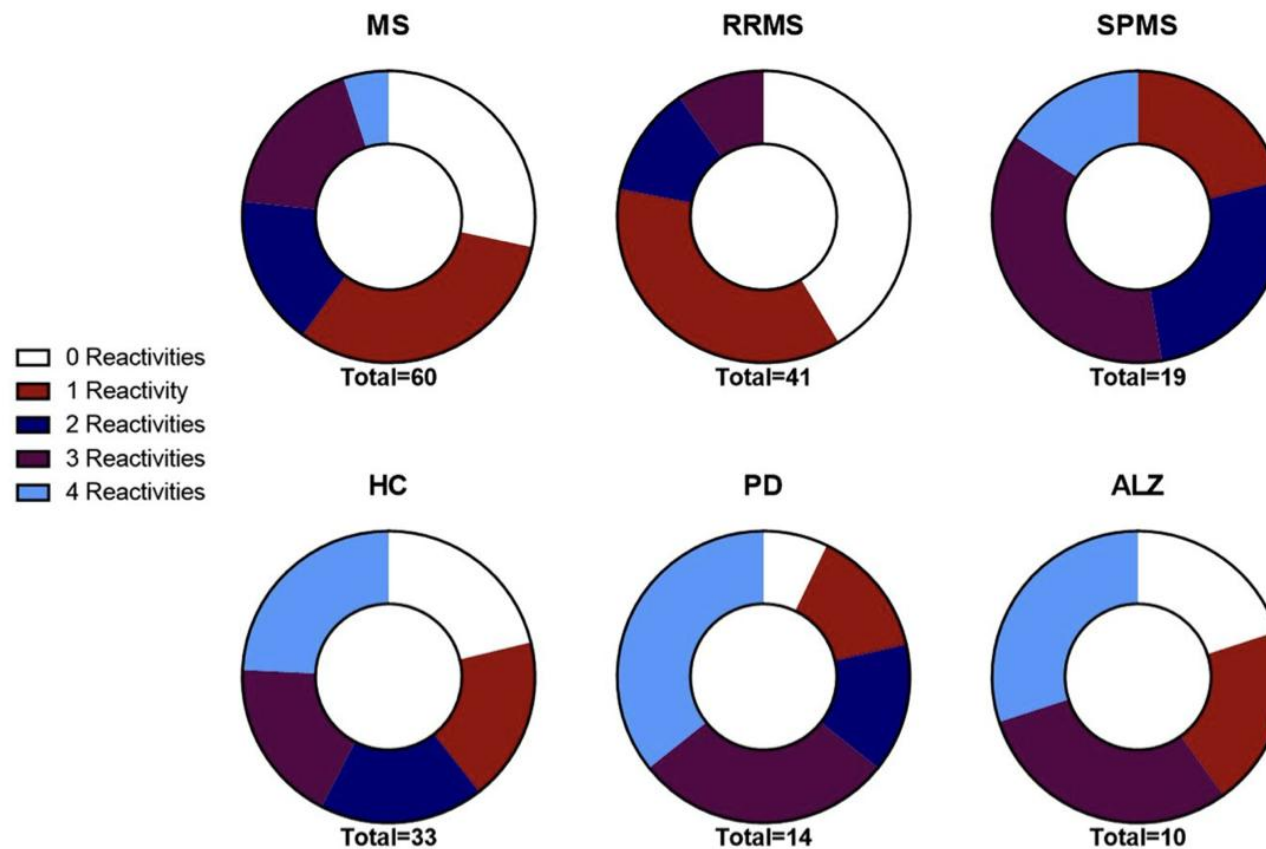


Figure 2. Doughnut charts indicating the proportion of serum samples of total multiple sclerosis (MS), relapse-remitting MS (RRMS), secondary progressive MS (SPMS), healthy controls (HC), Parkinson's disease (PD) and Alzheimer's disease (ALZ) patients that were reactive with multiple Hp antigens p54-flagellin, p50, p41 and p29-UreA.

Major Findings

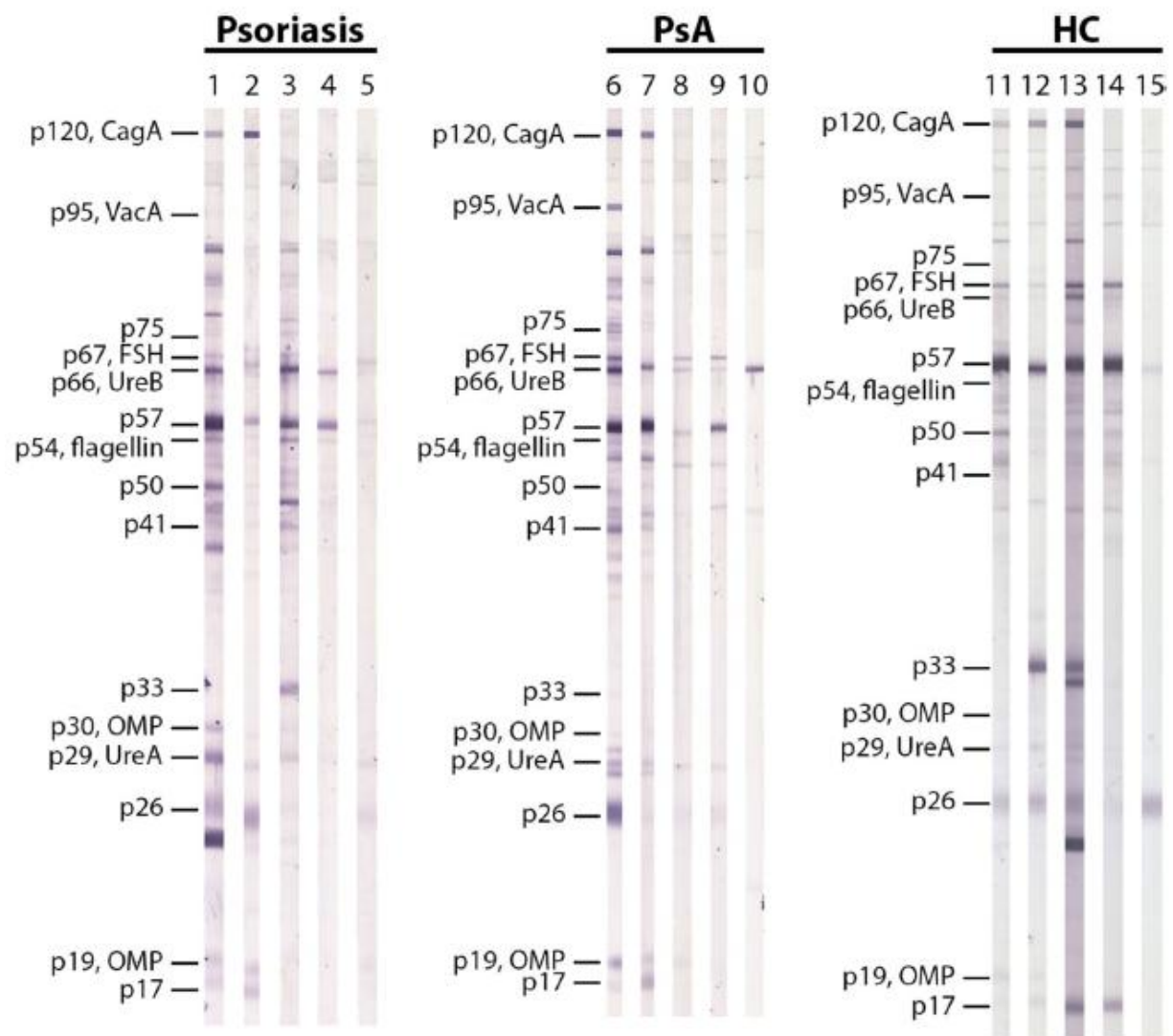
- We found that neither the frequency nor the magnitude of anti-Hp antibody differs between MS patients and HCs or between RRMS and SPMS when ELISA detecting antibodies against the whole Hp extract is used
- Reactivities against particular Hp immunodominant antigens by immunoblotting revealed several differences between MS and HCs, and more importantly between RRMS and SPMS.
- Anti-VacA antibodies were four times more frequent in SPMS than in HCs.

RESEARCH



Investigating the link between *Helicobacter pylori* infection and psoriatic disease: an immunological study

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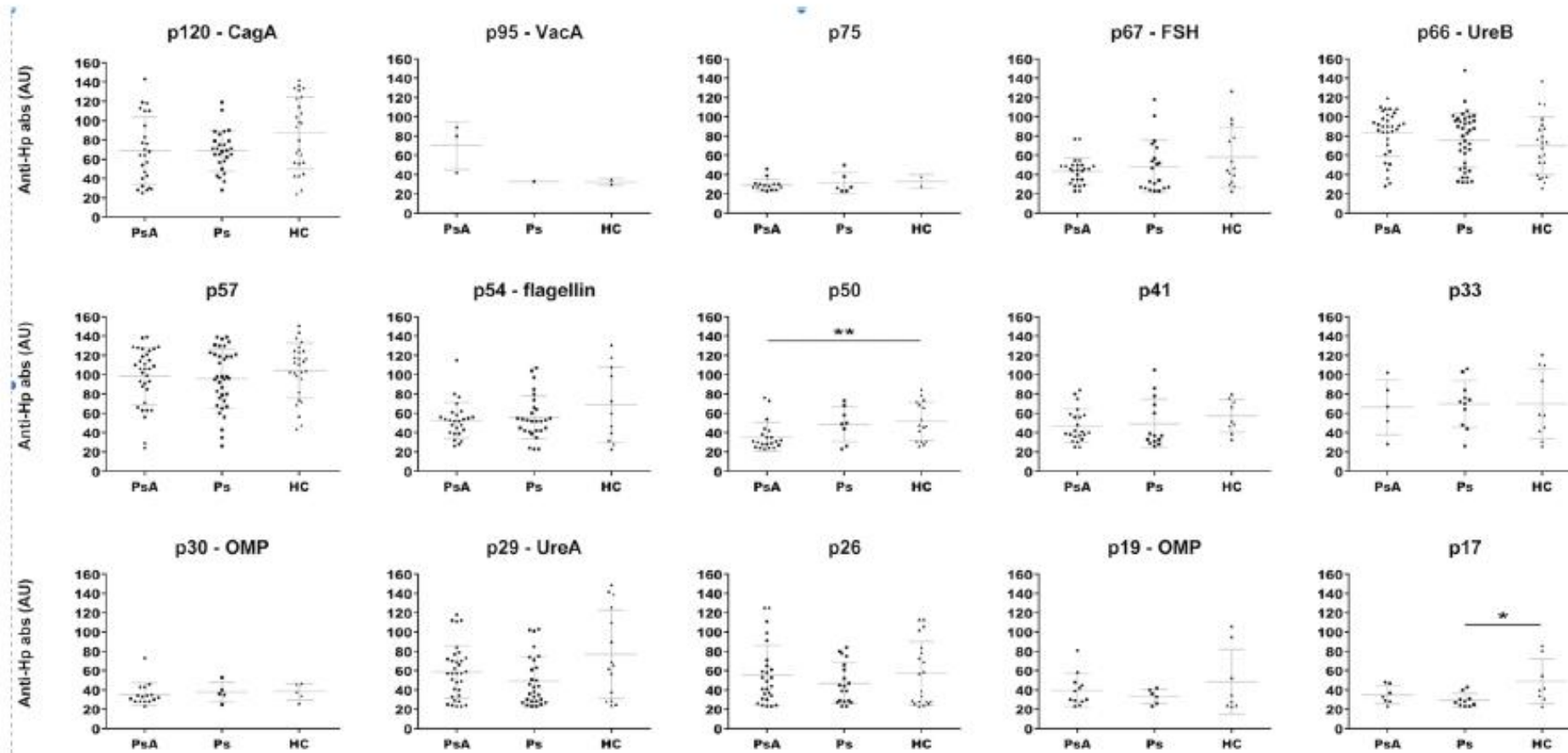
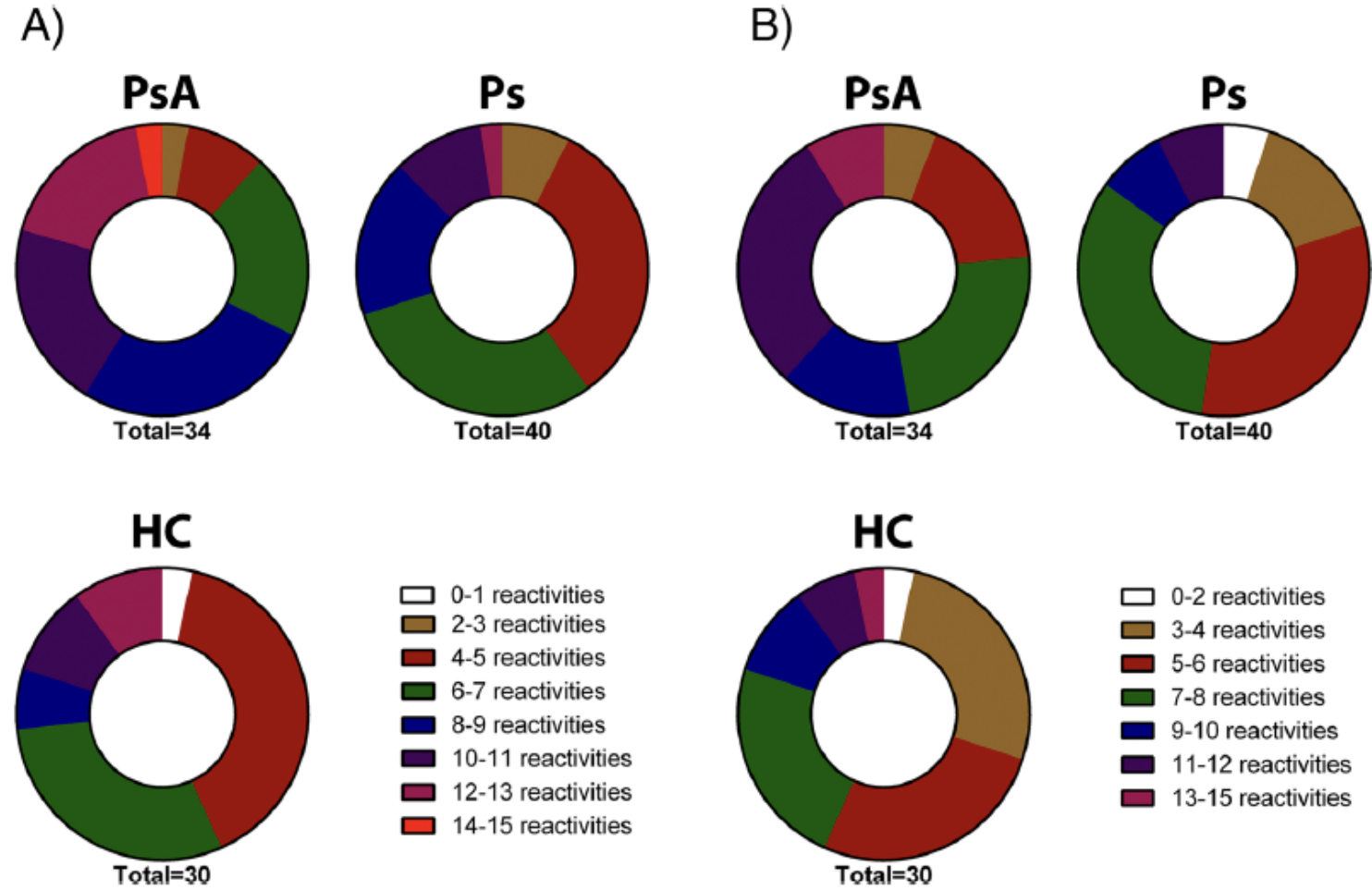


Fig. 2 Scatter plots illustrating antibody (Ab) levels against individual *Helicobacter pylori* antigens by Western immunoblotting in patients with psoriasis (Ps), psoriatic arthritis (PsA), and healthy con-

trols (HC). * $p < 0.05$. Abbreviations: CagA, protein from cytotoxin-associated gene A; UreA, urease A; UreB, urease B; VacA, vacuolating cytotoxin A



Fig. 3 (A): Doughnut charts indicating the proportion of *Helicobacter pylori* (Hp) (+) patients with psoriasis, Hp (+) patients with psoriatic arthritis (PsA) and Hp (+) healthy controls (HC) that were reactive with multiple Hp antigens (0–1 reactivities, 2–3, 4–5, 6–7, 8–9, 10–11, 12–13 reactivities, eight groups in total) **(B):** Doughnut charts indicating the proportion of *Helicobacter pylori* (Hp) (+) patients with psoriasis, Hp (+) patients with psoriatic arthritis (PsA) and Hp (+) healthy controls (HC) that were reactive with multiple Hp antigens (0–2 reactivities, 3–4, 5–6, 7–8, 9–10, 11–12, 13 reactivities, seven groups in total)



Major Findings

- *Much to our surprise, the central finding of our study is that ab responses to most Hp antigens in PsA and Ps did not differ and were reduced in prevalence and magnitude compared to controls.*
- *In our study, anti-UreA and anti-flagellin Hp were more frequent in PsA than HC. Similarly, anti-UreB and anti-p54- flagellin Hp antibodies were more prevalent in Ps than HC,*
- *while anti-p50 antibodies were less prevalent.*



Antigen-specific humoral responses against *Helicobacter pylori* in patients with systemic sclerosis

Georgios Efthymiou¹ • Christos Liaskos¹ • Theodora Simopoulou¹ • Emmanouela Marou¹ • Eleni Patrikiou¹ • Thomas Scheper² • Wolfgang Meyer² • Dimitrios Daoussis³ • Lazaros I. Sakkas¹ • Dimitrios P. Bogdanos¹

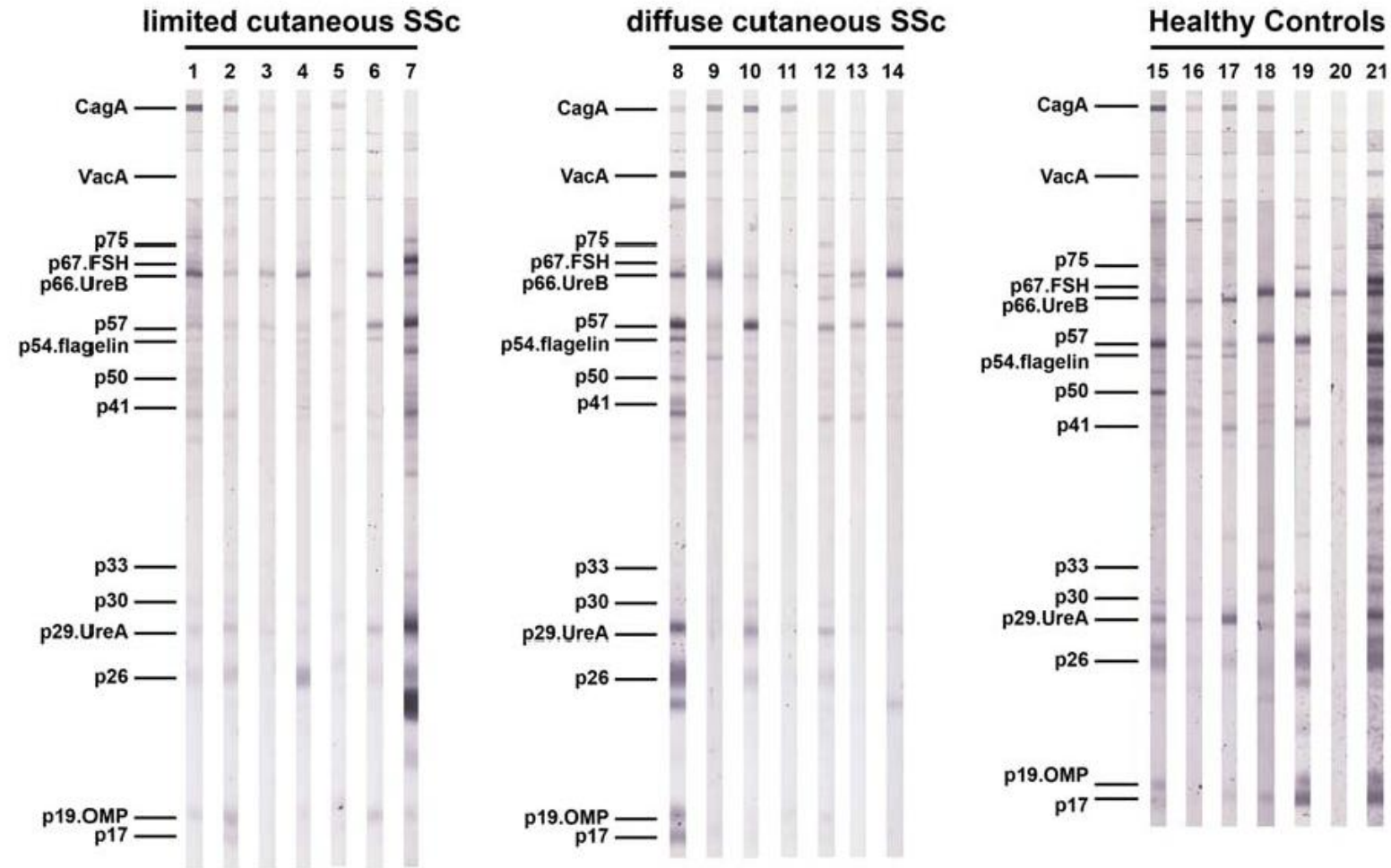


Fig. 1 Anti-*Helicobacter pylori* antibody reactivity by immunoblotting in representative patients with systemic sclerosis (SSc), including limited cutaneous SSc and healthy controls (HCs)

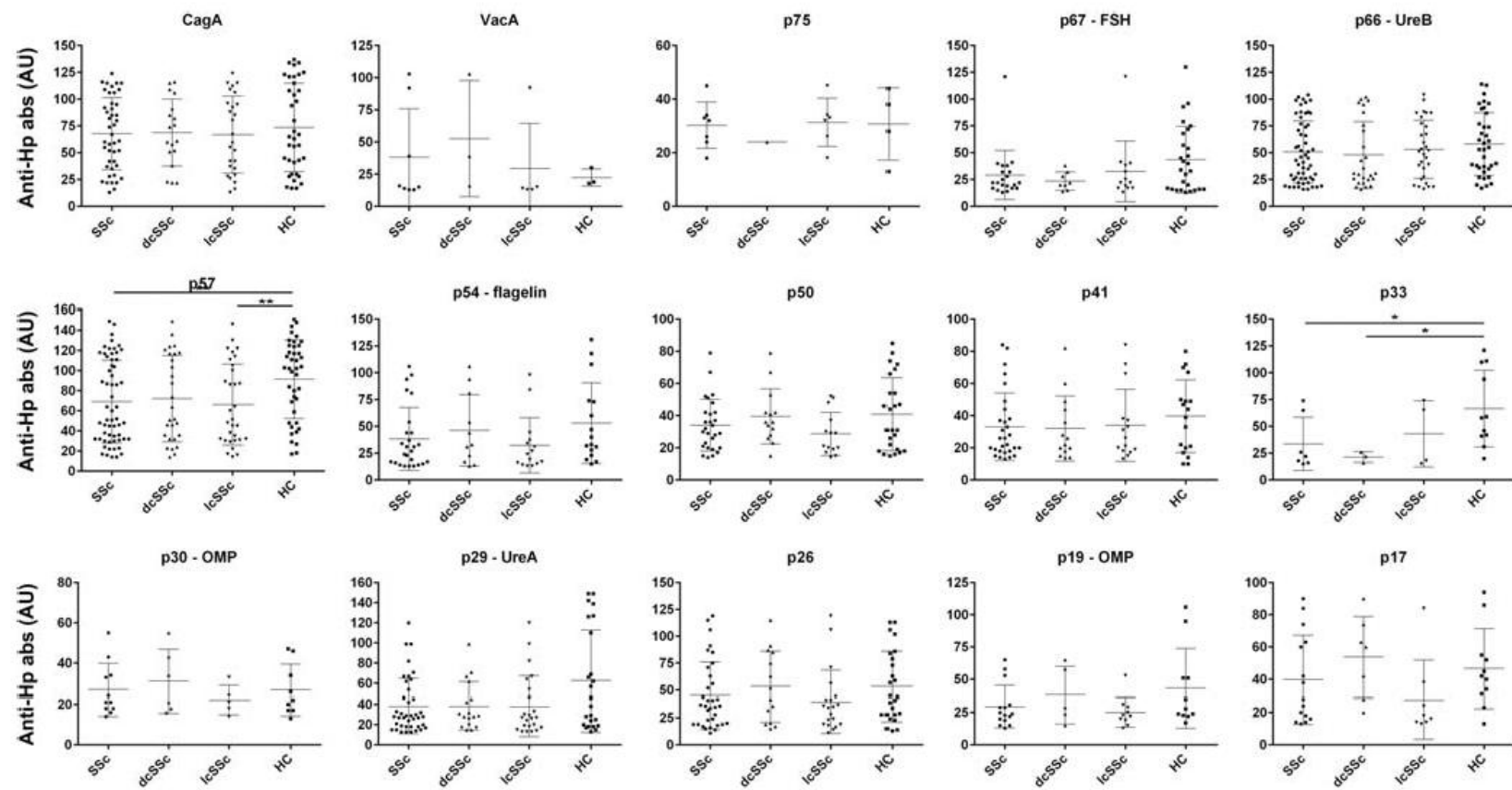


Fig. 2 Scatter plots illustrating antibody titers by western immunoblotting against individual Hp antigens in sera of systemic sclerosis (SSc) patients, including diffuse cutaneous SSc (dcSSc) and limited cutaneous SSc (lcSSc), and healthy controls (HC)

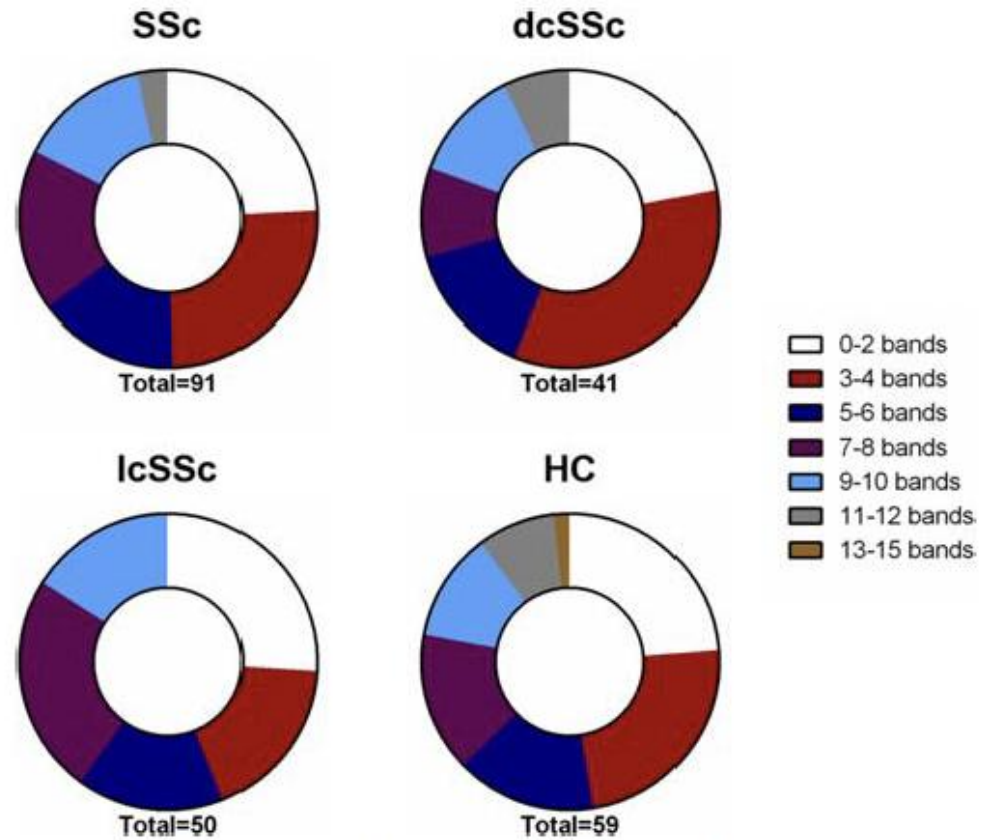


Fig. 3 Doughnut charts indicating the proportion of serum samples of total systemic sclerosis patients (SSc), limited cutaneous SSc (lcSSc), diffuse cutaneous SSc (dcSSc), and healthy controls (HCs) that were reactive with multiple *Hp* antigens



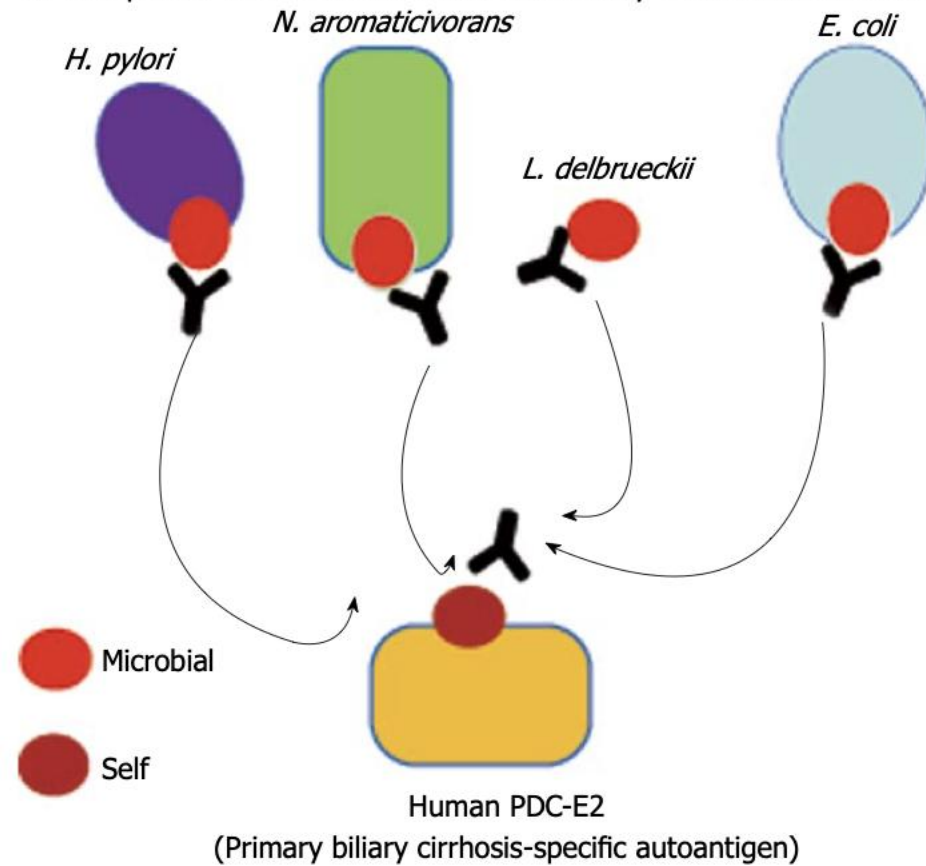
Major Findings

- Anti-Hp Abs are neither more frequent nor elevated in SSc compared with healthy population,
- The only exception is the higher frequency and levels of anti-VacA Abs in pulmonary hypertension and lung fibrosis, respectively.
- These results suggest that Hp is unlikely to be involved in the development of SSc.

Mechanistical explanation for the likely link

Molecular mimicry is always to blame

A "multiple hit" mechanism of molecular mimicry-induced autoimmunity



Extensive homology between the major immunodominant mitochondrial antigen in primary biliary cirrhosis and *Helicobacter pylori* does not lead to immunological cross-reactivity

D.-P. Bogdanos, H. Baum, F. Gunsar, D. Arioli, D. Polymeros, Y. Ma, ...show all

Pages 981-987 | Received 27 Feb 2004, Accepted 26 Apr 2004, Published online: 08 Jul 2009

 Cite this article  <https://doi.org/10.1080/00365520410003236>

Background: Primary biliary cirrhosis (PBC) is an immune-mediated chronic cholestatic disease characterized by the presence of antibodies directed predominantly against the E2 subunit of the pyruvate dehydrogenase complex (PDC-E2). What provokes tolerance breakdown in PBC remains to be established, though there is evidence to indicate that microbes may induce anti-mitochondrial antibodies (AMA) through a mechanism of molecular mimicry. **Methods:** Having found that urease beta (UREB)₂₂₋₃₆ antigen of *Helicobacter pylori* (HELPHY) shares extensive (87%) similarity with PDC-E2₂₁₂₋₂₂₆, the major mitochondrial autoepitope, it was hypothesized that this would also lead to cross-reactivity. The UREB/PDC-E2 mimics were thus constructed and tested by ELISA in 112 PBC patients and 114 controls. **Results:** Reactivity to PDC-E2₂₁₂₋₂₂₆ was found in 104 patients but to UREB₂₂₋₃₆ in only 2. In these two patients, the double reactivity was not cross-reactive. The lack of surface antibody accessibility to UREB₂₂₋₃₆, as demonstrated through three-dimensional model prediction analysis, may explain this unexpected finding. There was some speculation on whether HELPHY UREB₂₂₋₃₆ might act as a cross-reactive CD4 T-cell epitope. All seven PBC patients, tested in a standard proliferation assay against PDC-E2₂₁₂₋₂₂₆, gave a positive response. All seven were unresponsive to HELPHY UREB₂₂₋₃₆. The pattern of reactivity to HELPHY antigens by immunoblot was similar between anti-PDC-E2-positive and negative PBC cases, as well as between PBC patients and controls. **Conclusion:** Contrary to common belief, extensive sequence homology (molecular mimicry) between self and microbe does not necessarily result in cross-reactivity. It is therefore likely that, when present, cross-reactivity between self and microbes is of biological importance.

Mechanistical explanation for the likely link

Molecular mimicry at the B cell and T cell level is not present in Primary Biliary Cholangitis

Aim

- To assess whether Hp is a trigger of disease-specific ANA
- To assess the role of molecular mimicry

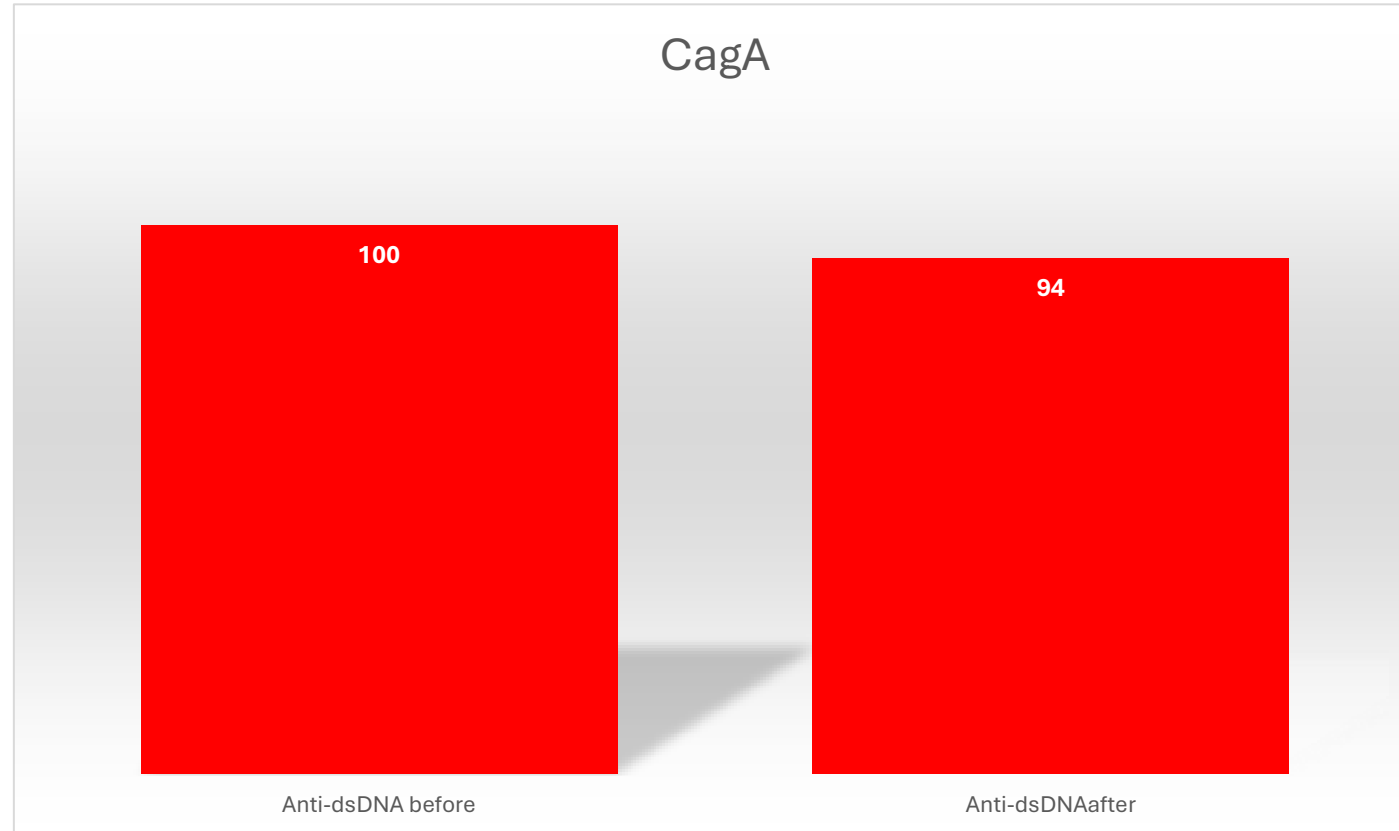
Purified serum samples from Disease-specific AIRDs were incubated with Hp specific antigens and ANA reactivity was determined before and after incubation

Hp as a trigger of SSc-ANA

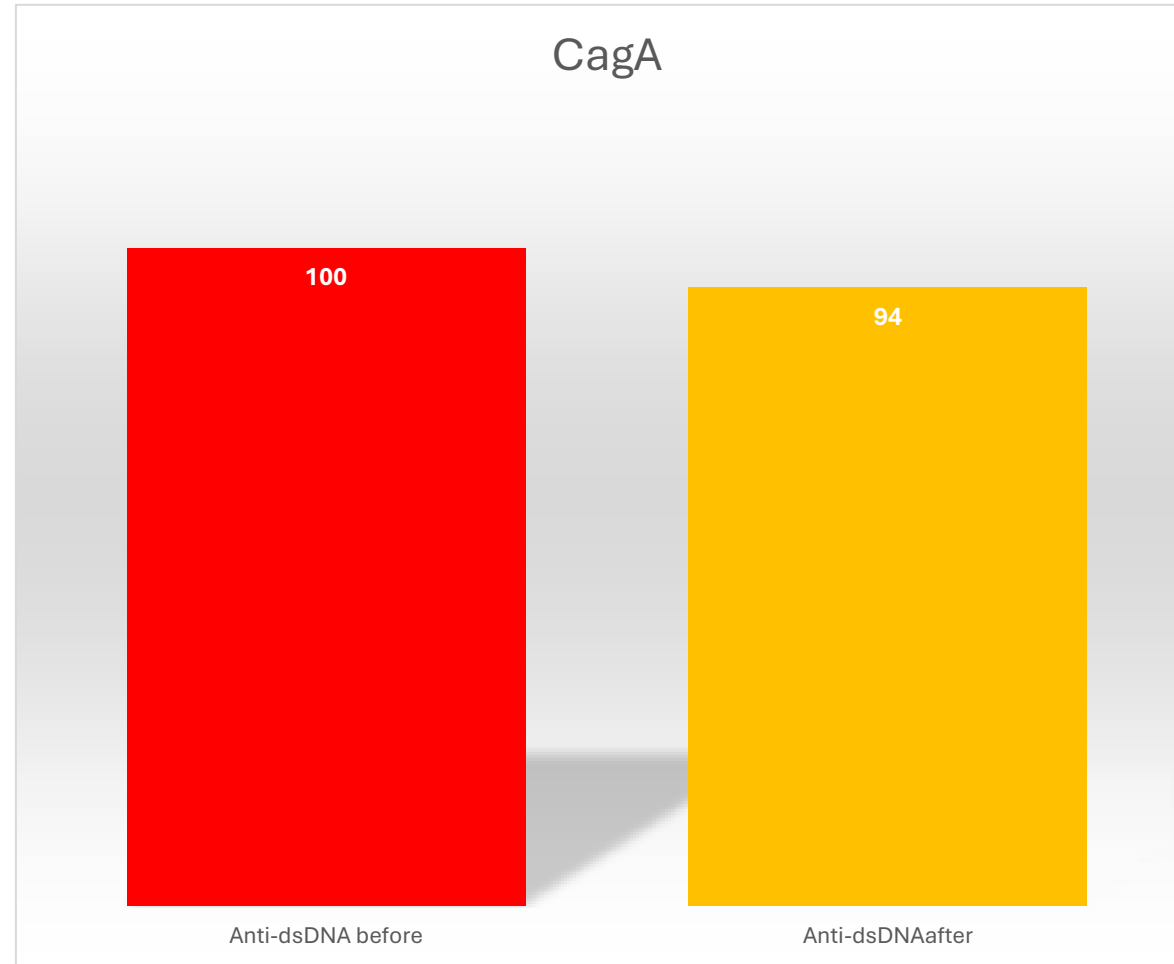
Anti-Scl70 ab reactivity before and after solid phase inhibition



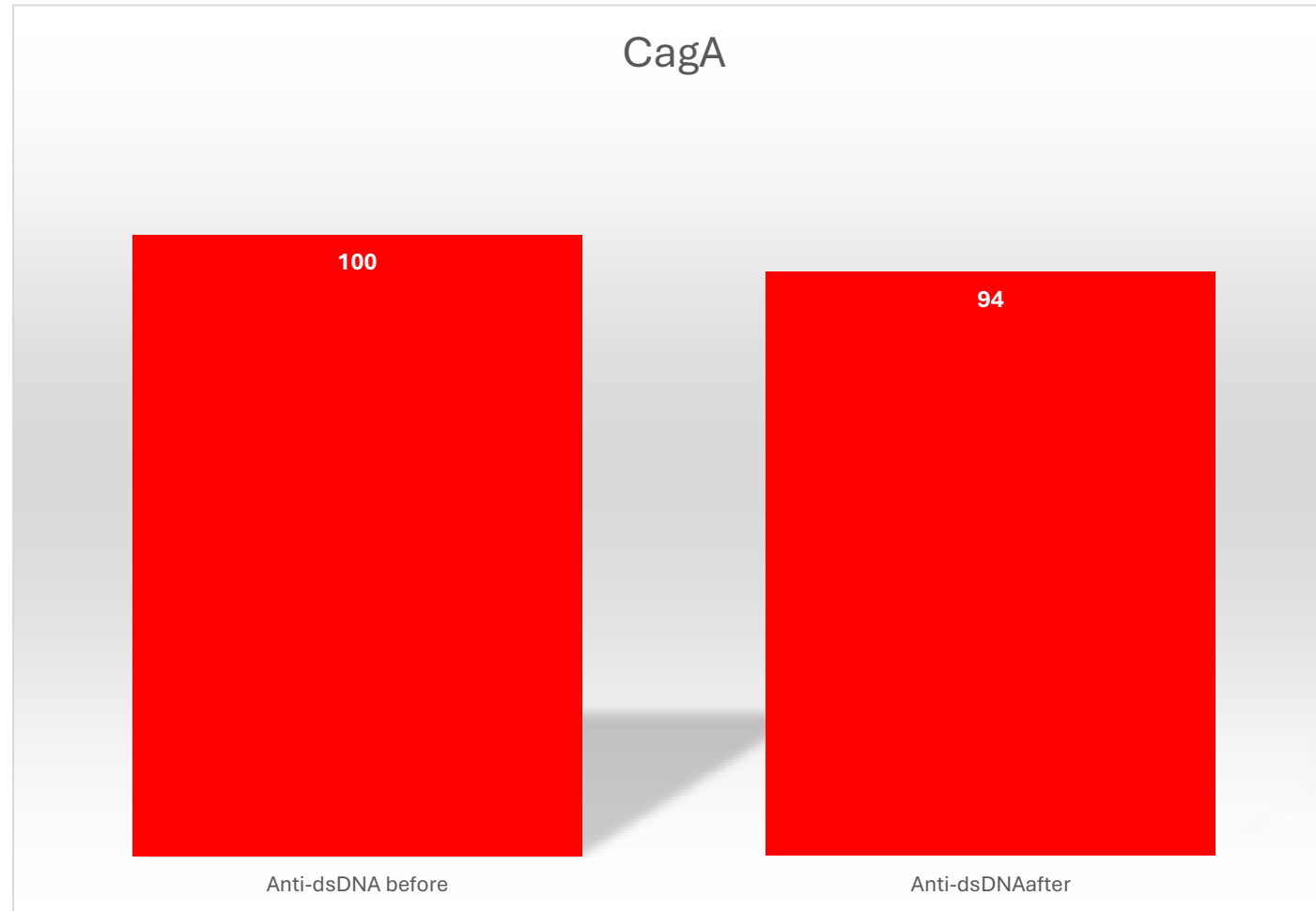
Anti-Scl70 ab reactivity % before and after solid phase inhibition



Anti-Centromere ab reactivity % before and after solid phase inhibition

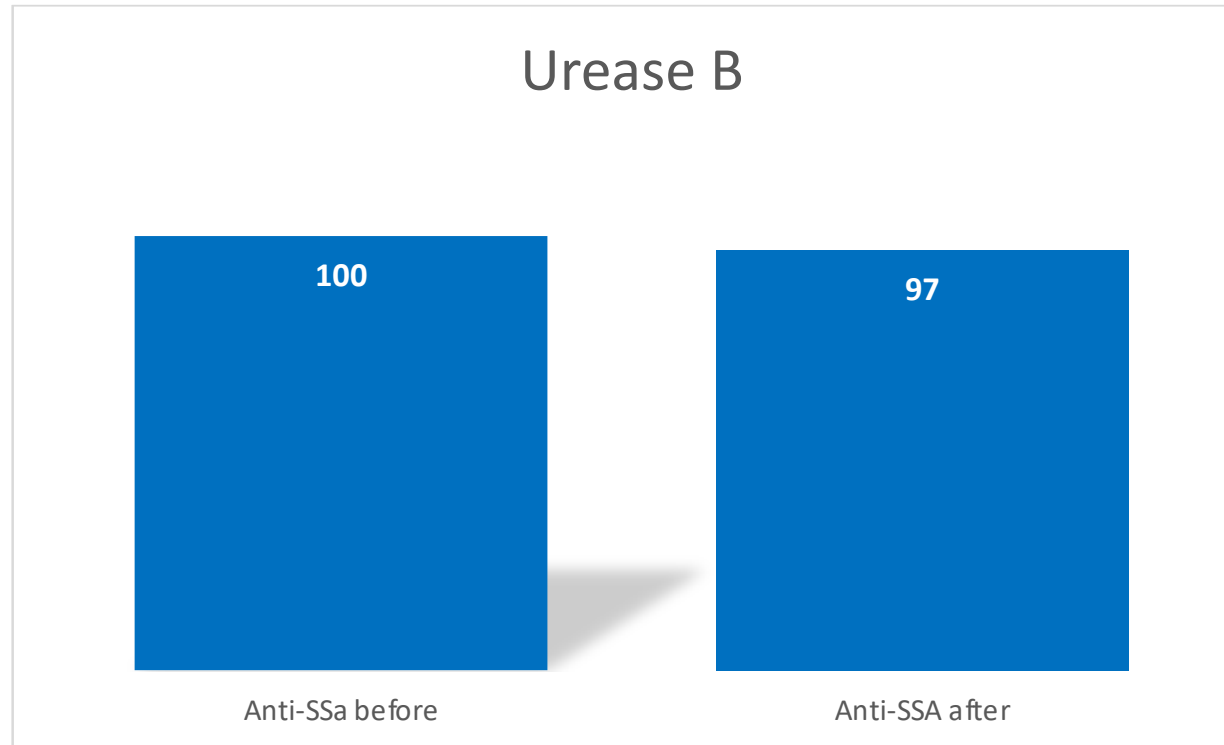


Anti-Centromere ab reactivity % before and after solid phase inhibition

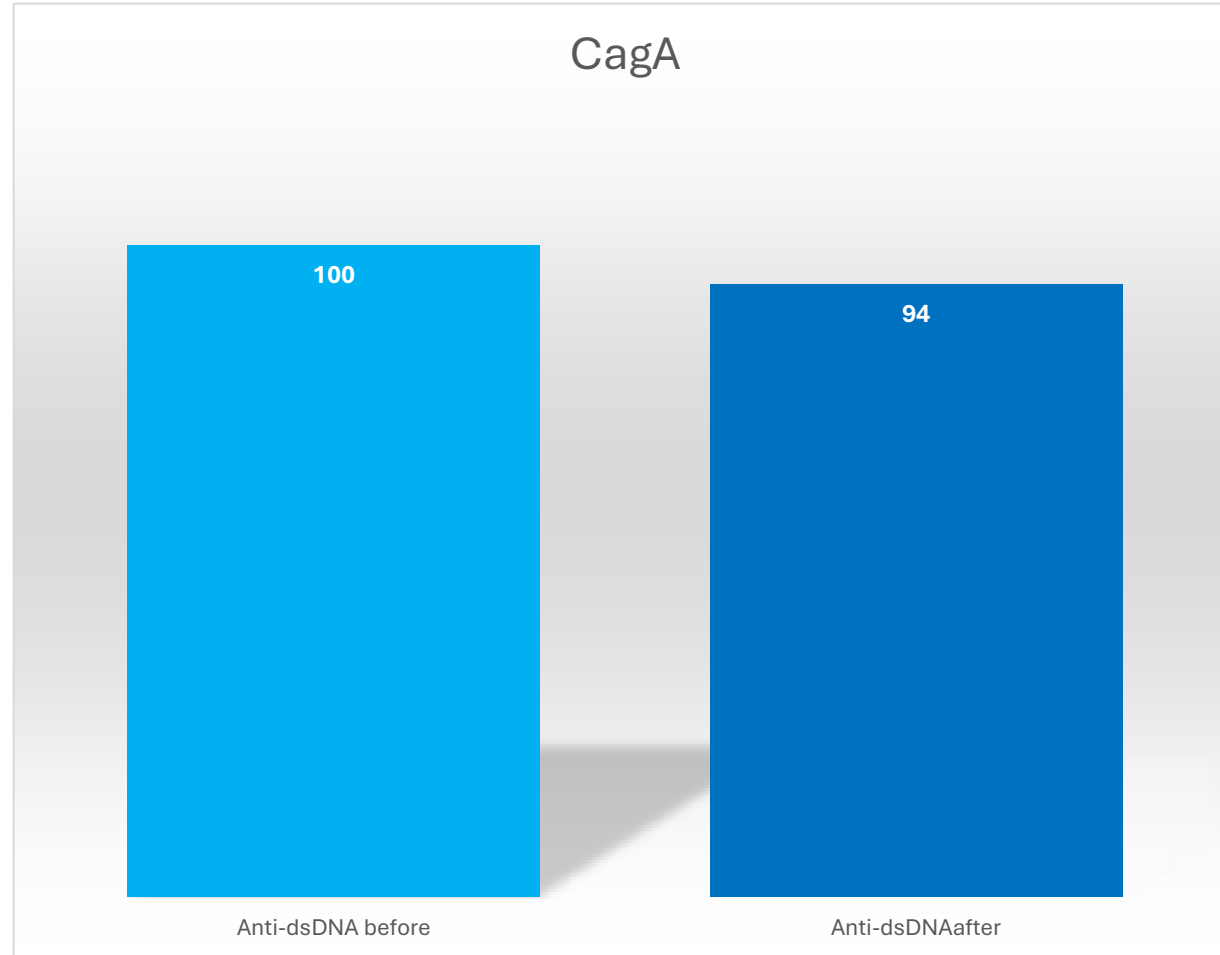


Hp as a trigger of SjS-ANA

Anti-SSA (Ro60) ab reactivity % before and after solid phase inhibition

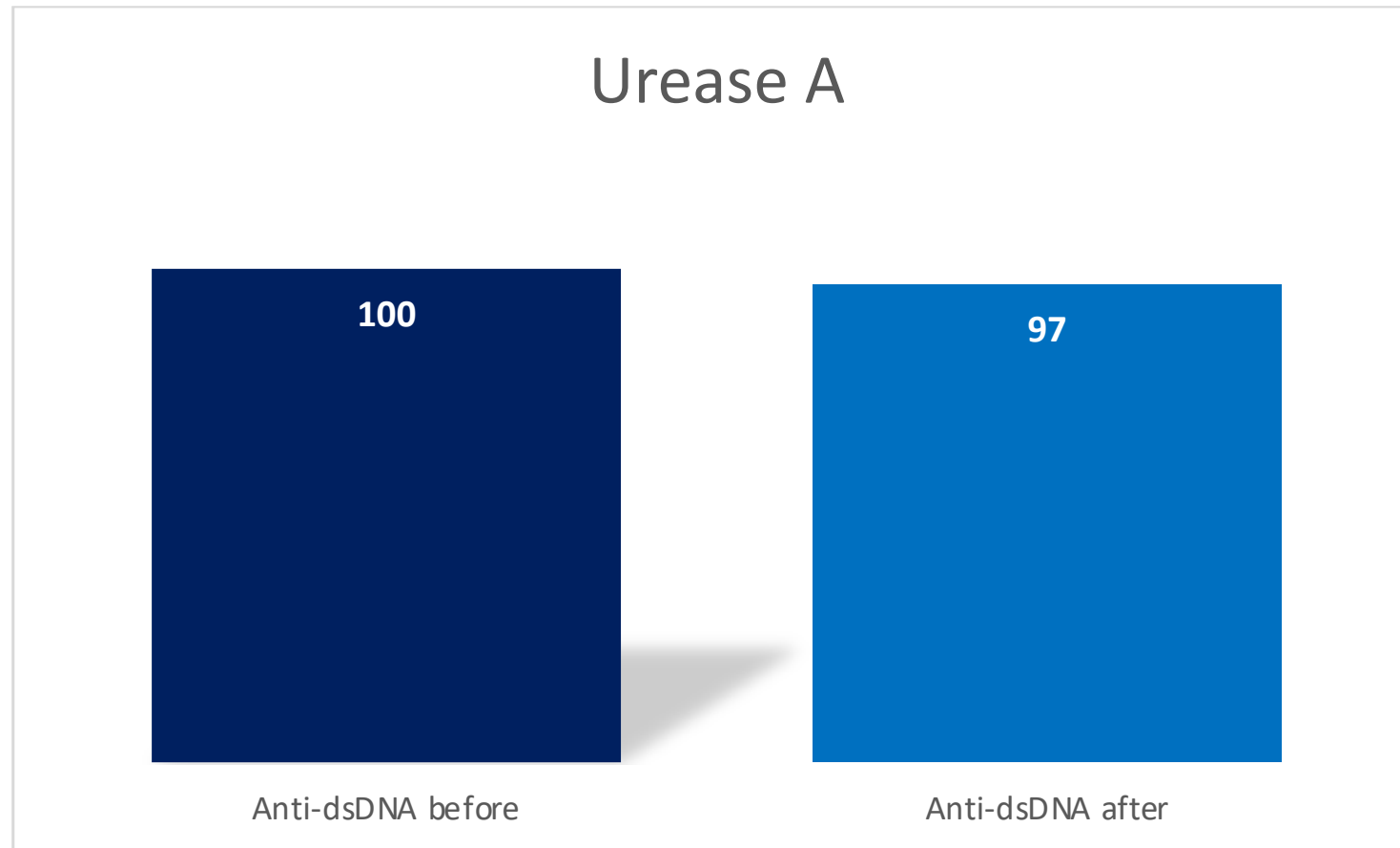


Anti-SSA (Ro52) ab reactivity % before and after solid phase inhibition

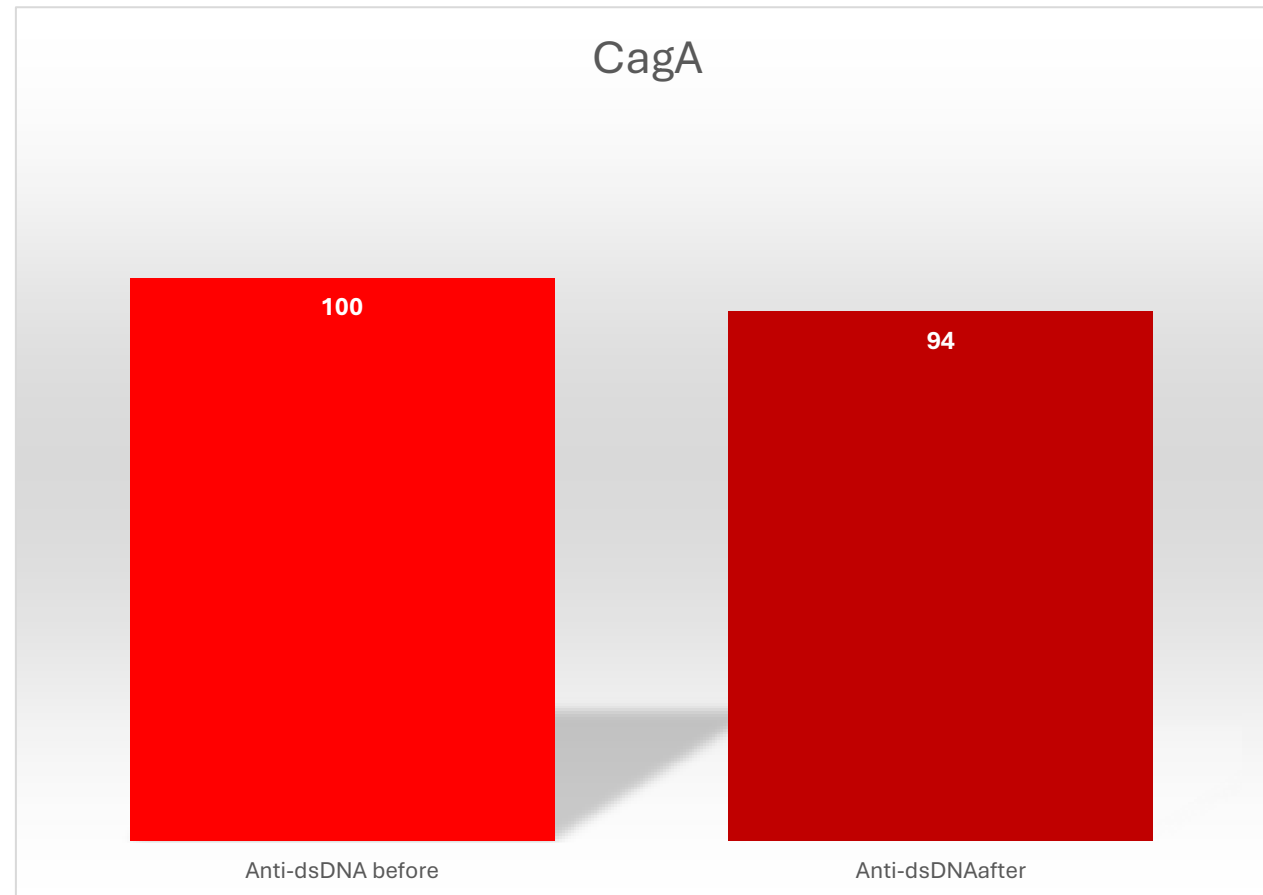


Hp as a trigger of SLE-ANA

Anti-dsDNA ab reactivity % before and after solid phase inhibition

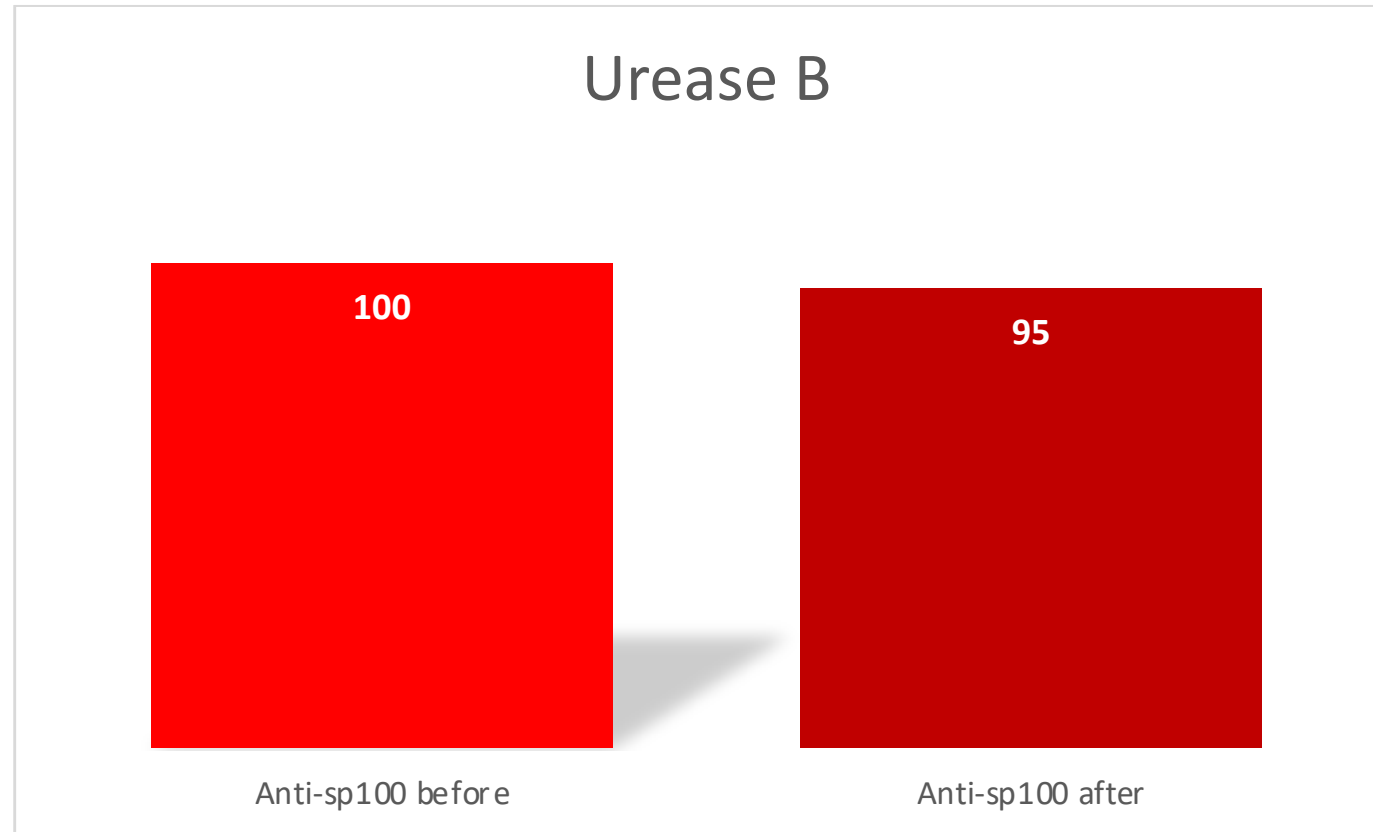


Anti-SSA (Ro52) ab reactivity % before and after solid phase inhibition

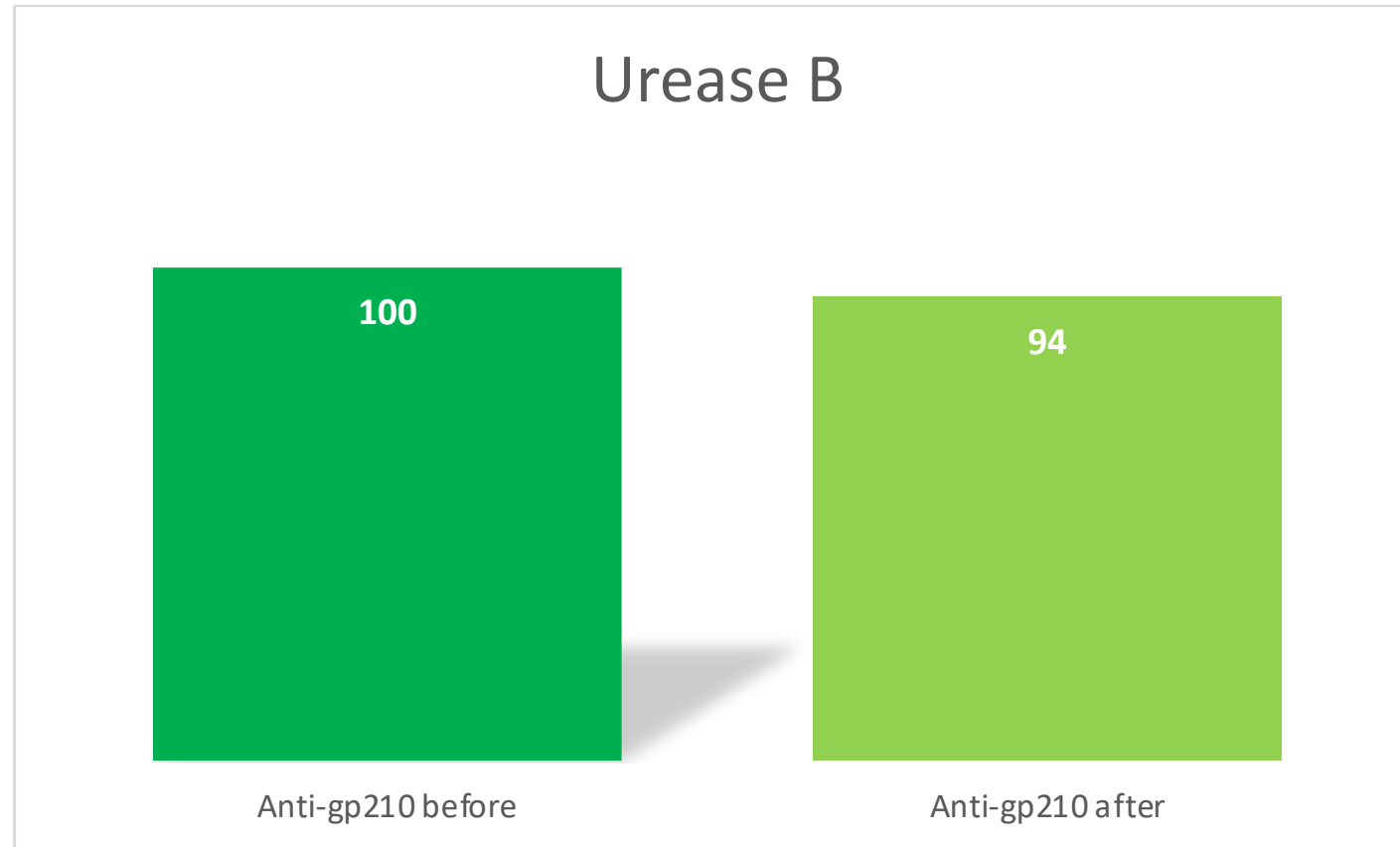


Hp as a trigger of PBC-ANA

Anti-Sp100 ab reactivity % before and after solid phase inhibition

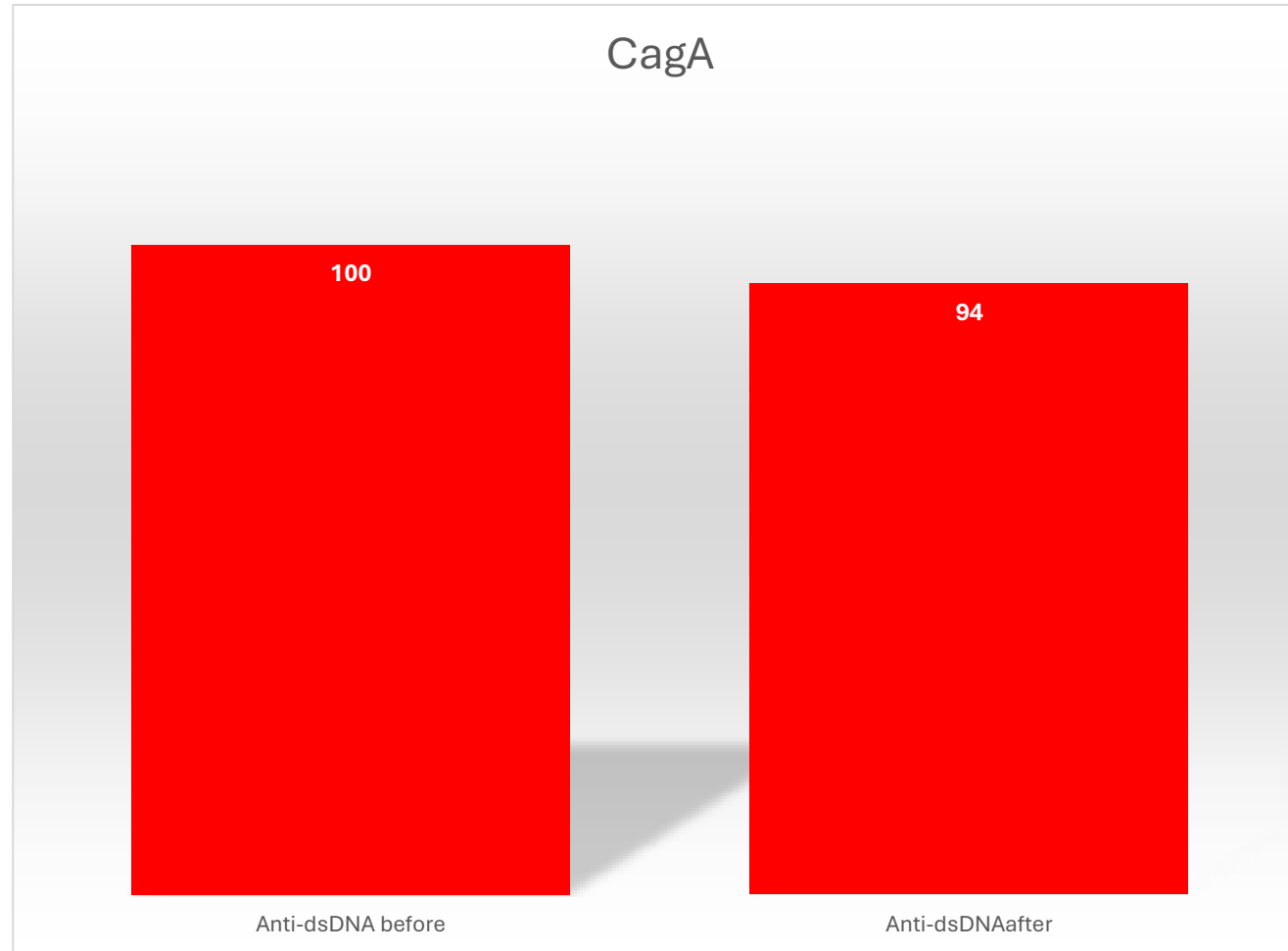


Anti-gp210 ab reactivity % before and after solid phase inhibition



Hp as a trigger of AIH-ANA

Anti-dsDNA ab reactivity % before and after solid phase inhibition in a patient with autoimmune hepatitis type 1



Conclusions

- Disease-specific ANAs are not triggered by anti-Hp specific antibodies
- Molecular mimicry between nuclear antigens and Hp antigens is unlikely in most if not all ANA associated autoimmune diseases