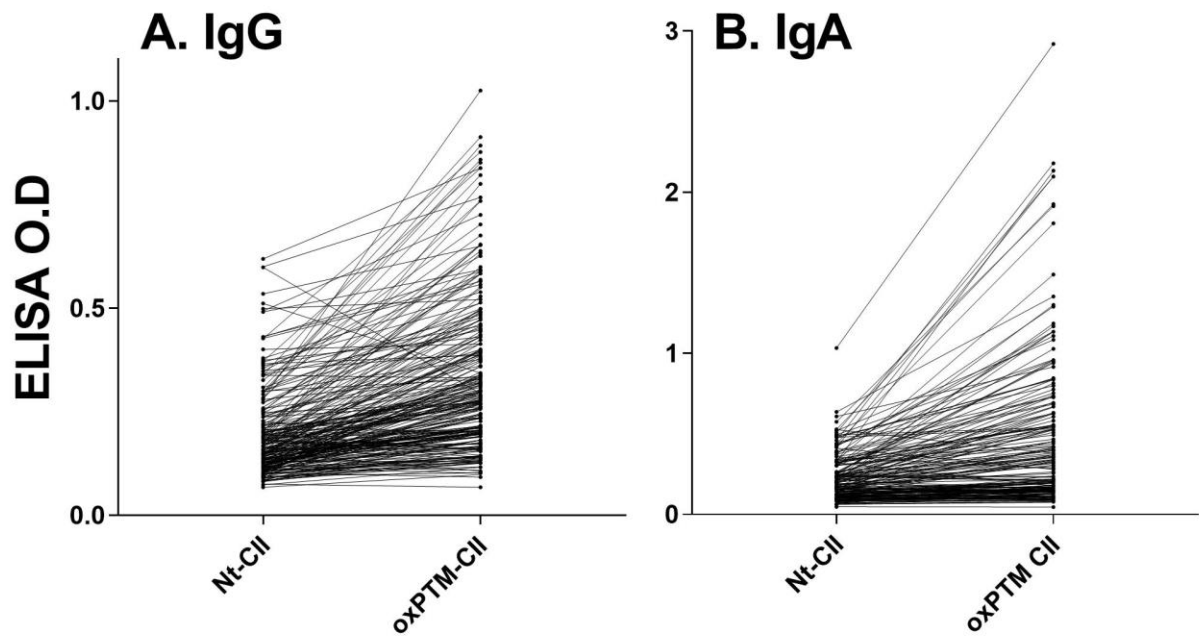


Supplementary files

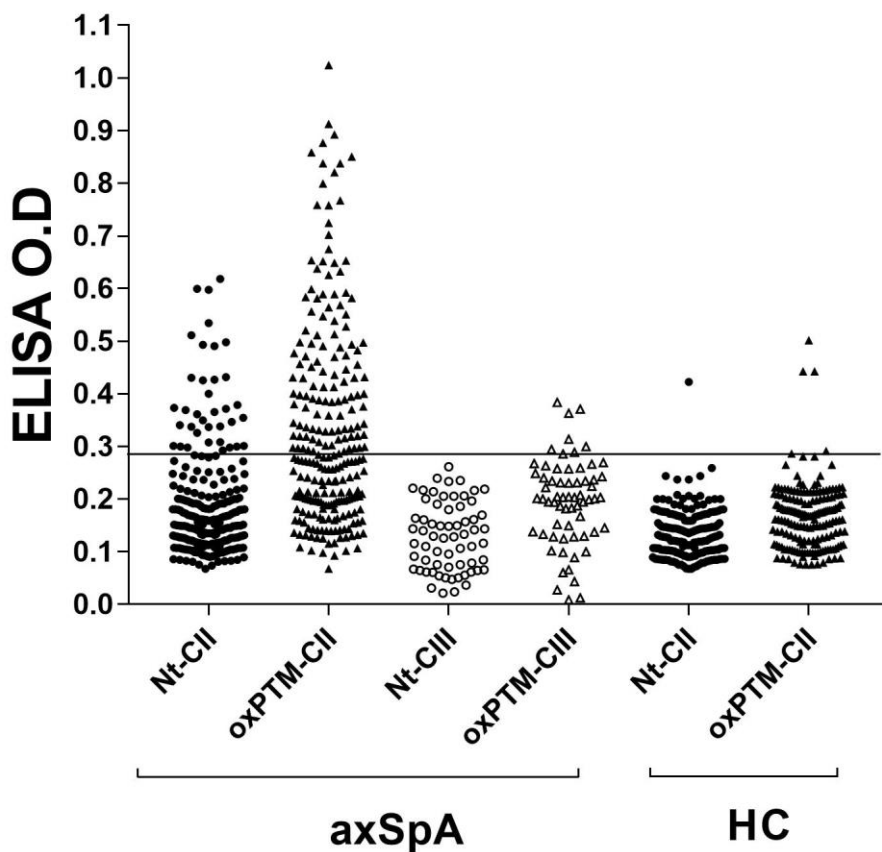
		Sensitivity	Specificity	LK	AUC	SE	95% CI
axSpA	IgG	<u>64.04</u>	<u>80.58</u>	3.298	0.8181	0.0201	0.7786 to 0.8576
	IgA	<u>80.58</u>	<u>85.71</u>	5.641	0.8325	0.0344	0.765 to 0.9
RA	IgG	<u>94.94</u>	<u>91.67</u>	11.39	0.9612	0.0183	0.9254 to 0.9971
	IgA	<u>32.14</u>	<u>89.29</u>	3	0.5561	0.0547	0.4488 to 0.6635
PSA	IgG	<u>42.13</u>	<u>86.67</u>	3.16	0.7269	0.0336	0.661 to 0.7928
	IgA	<u>87.18</u>	<u>92.86</u>	12.21	0.9441	0.0286	0.888 to 1
PS	IgG	27.53	88.57	2.409	0.6291	0.0530	0.5252 to 0.7331
	IgA	61.54	78.57	2.872	0.6429	0.0869	0.4725 to 0.8132
UA	IgG	34.27	90	3.427	0.7321	0.0419	0.6499 to 0.8143
	IgA	47.73	85.71	3.341	0.5901	0.0686	0.4556 to 0.7246
FM	IgG	69.66	73.68	2.647	0.7931	0.0426	0.7095 to 0.8765
	IgA	57.89	85.71	4.053	0.6109	0.1028	0.4094 to 0.8124

Supplementary table S1. Sensitivity and specificity for IgG and IgA anti-oxPTM-CII.

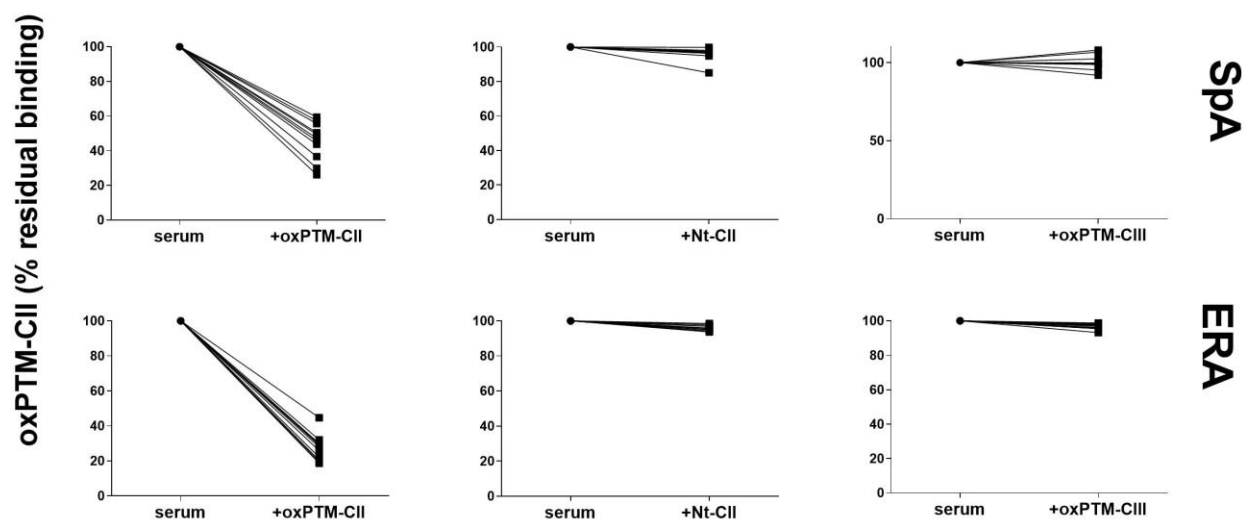
AUC is indicated for each group. LK is likelihood ratio and SE is standard error. 95% confidential interval (95% CI) is also indicated.



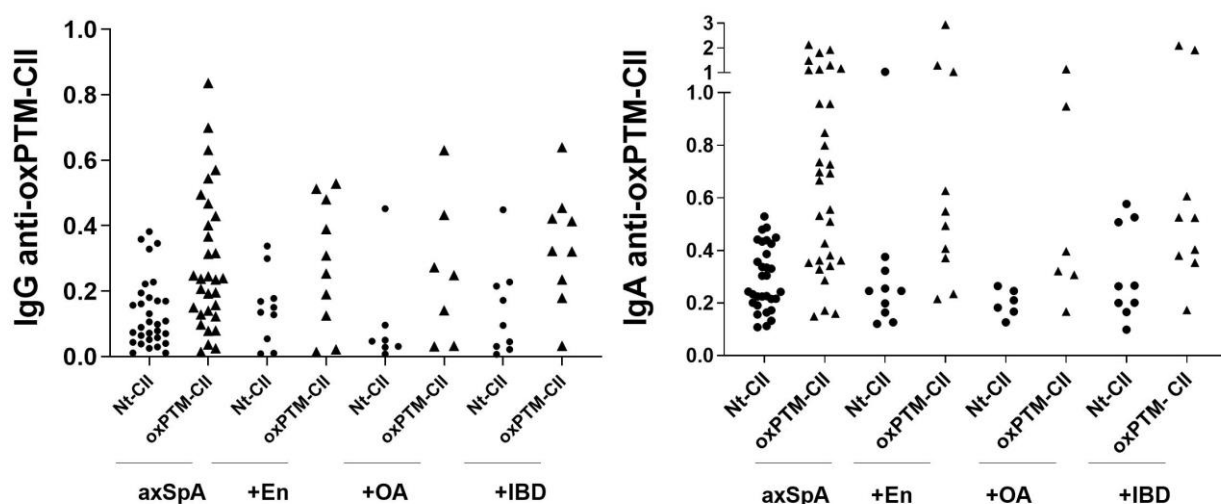
Supplementary figure S1: Comparative binding of IgG (A) and IgA (B) anti-native CII (Nt-CII) versus anti-oxPTM-CII. axSpA samples that responded to both Nt-CII and oxPTM-CII had stronger reactivity toward oxPTM-CII ($p < 0.0001$).



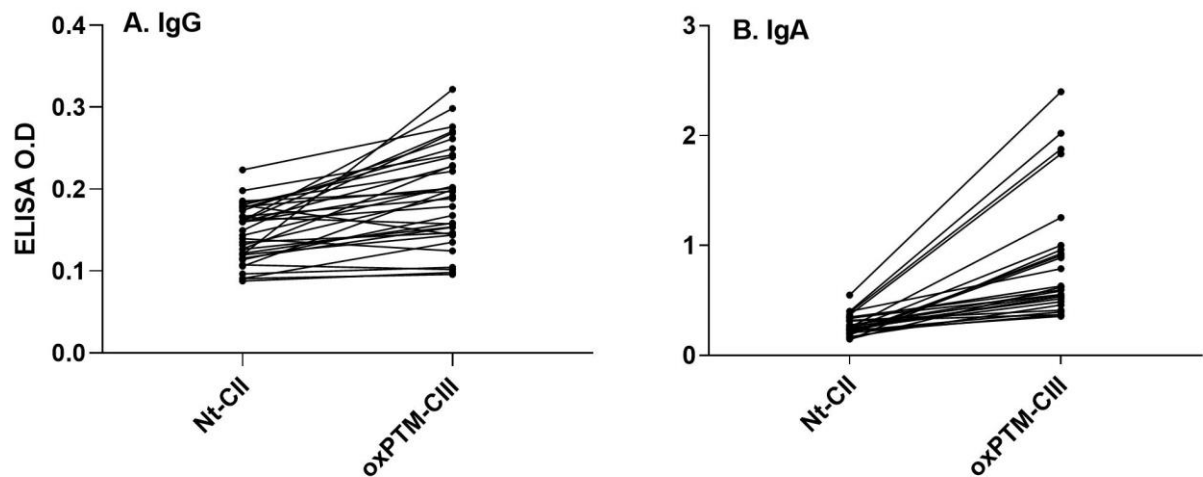
Supplementary figure S2. Binding to ox-PTM-CII appeared specific, as no reactivity to native and oxPTM collagen type III was detected. Nt-CII is native CII, oxPTM-CII is oxidised post-translationally modified CII. Nt-CIII is native CIII, oxPTM-CIII is oxidised post-translationally modified CIII. Dotted line is the ELISA O.D. cut-off that was determined arbitrarily as the 97th percentile of the oxPTM-CII antibodies levels detected in healthy individuals (O.D. = 0.2845).



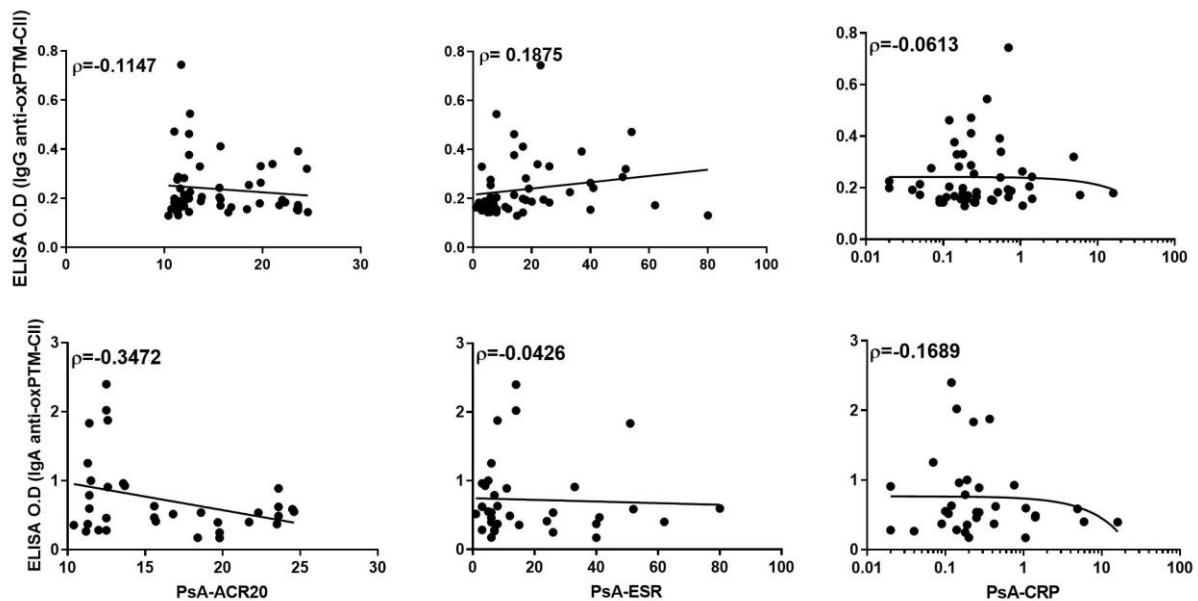
Supplementary figure S3. Only oxPTM-CII but not oxPTM-CIII was competing with the binding to oxPTM-CII in both axSpA and RA samples. Ten serum samples from axSpA patients were incubated with 50µg/ml native (+Nt-CII), oxPTM-CII (+oxPTM-CII) or oxPTM-CIII (+oxPTM-CIII) for 2 hours before binding to target oxPTM-CII in the ELISA plate. Pre-incubation with oxPTM-CII resulted in a significant reduction in the signal by both axSpA and RA sample ($p < 0.0001$). When the competitor was native CIII or oxPTM-CIII, the binding to ox-PTM-CII was reduced by only approximately 5%.



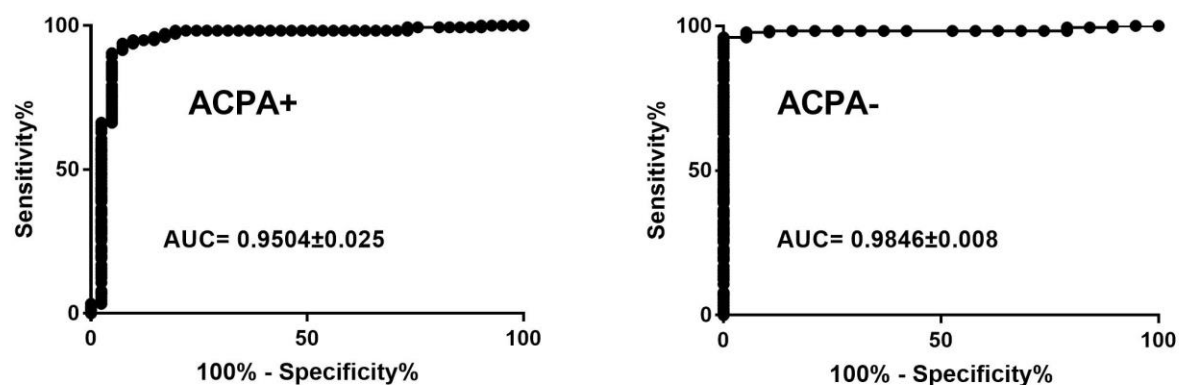
Supplementary figure S4. Binding to oxPTM-CII in samples from axSpA clinical subgroups. axSpA groups revealed no significant difference in anti-oxPTM-CII IgG or IgA reactivity in patients with 'pure' axSpA versus clinical subgroups e.g. axSpA-associated with oligoarthritis (axSpA-OA), inflammatory bowel disease (axSpA-IBD) or patients with enthesitis (axSpA-En), ($p > 0.05$).



Supplementary figure S5: Comparative binding of IgG (A) and IgA (B) anti-native CII (Nt-CII) versus anti-oxPTM-CII in PsA. PsA samples that responded to both Nt-CII and oxPTM-CII had stronger reactivity toward oxPTM-CII.



Supplementary figure S6. Anti-oxPTM-CII reactivity in PsA is not associated with markers of inflammation. There was no association between IgA and IgG anti oxPTM-CII reactivity with American College of Rheumatology (ACR20), erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP).



Supplementary figure S7. ROC analysis for sensitivity and specificity of IgG anti-oxPTM-CII in rheumatoid arthritis samples comparing ACPA positive to ACPA negative samples. High specificity and sensitivity observed for both ACPA positive and ACPA negative samples. AUC values are indicated in the figure.