

SUPPLEMENTARY INFO FILE

Relapsing-remitting multiple sclerosis patients display an altered lipoprotein profile with dysfunctional HDL

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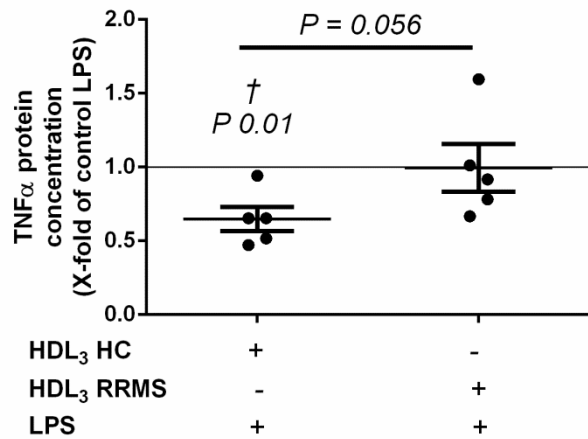
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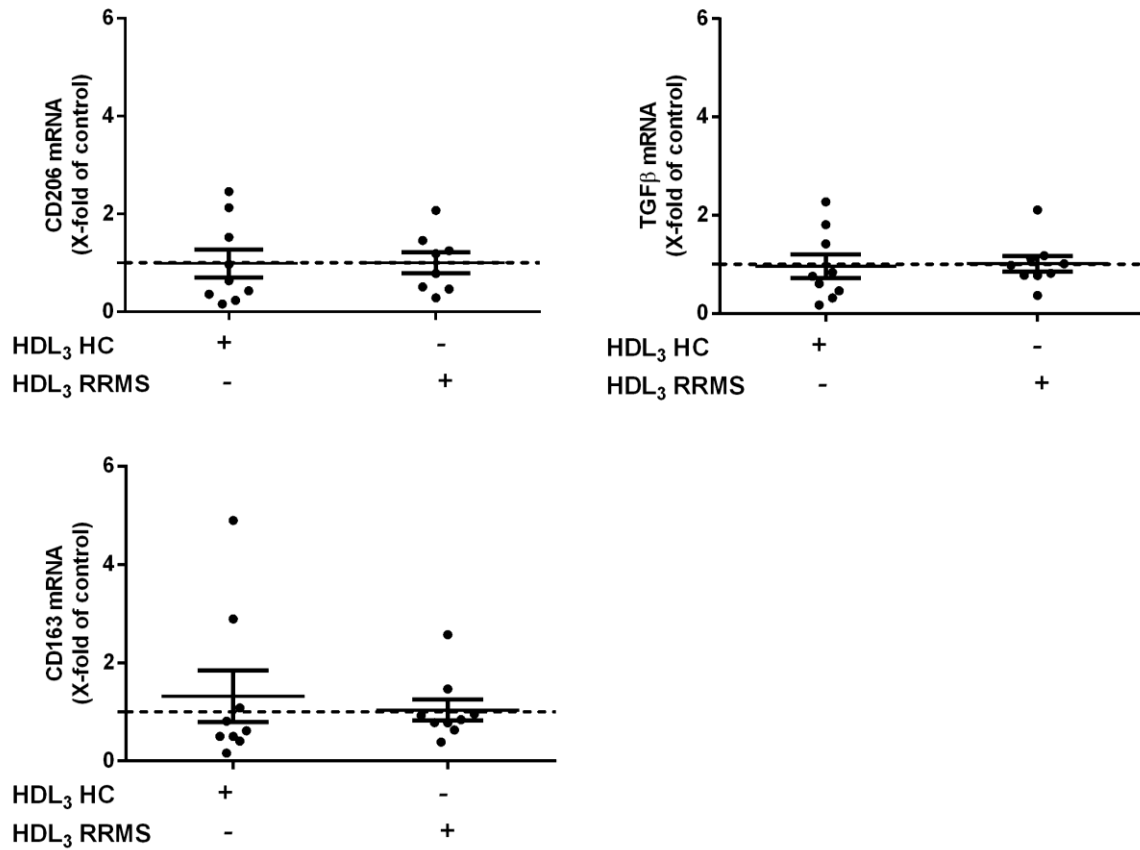
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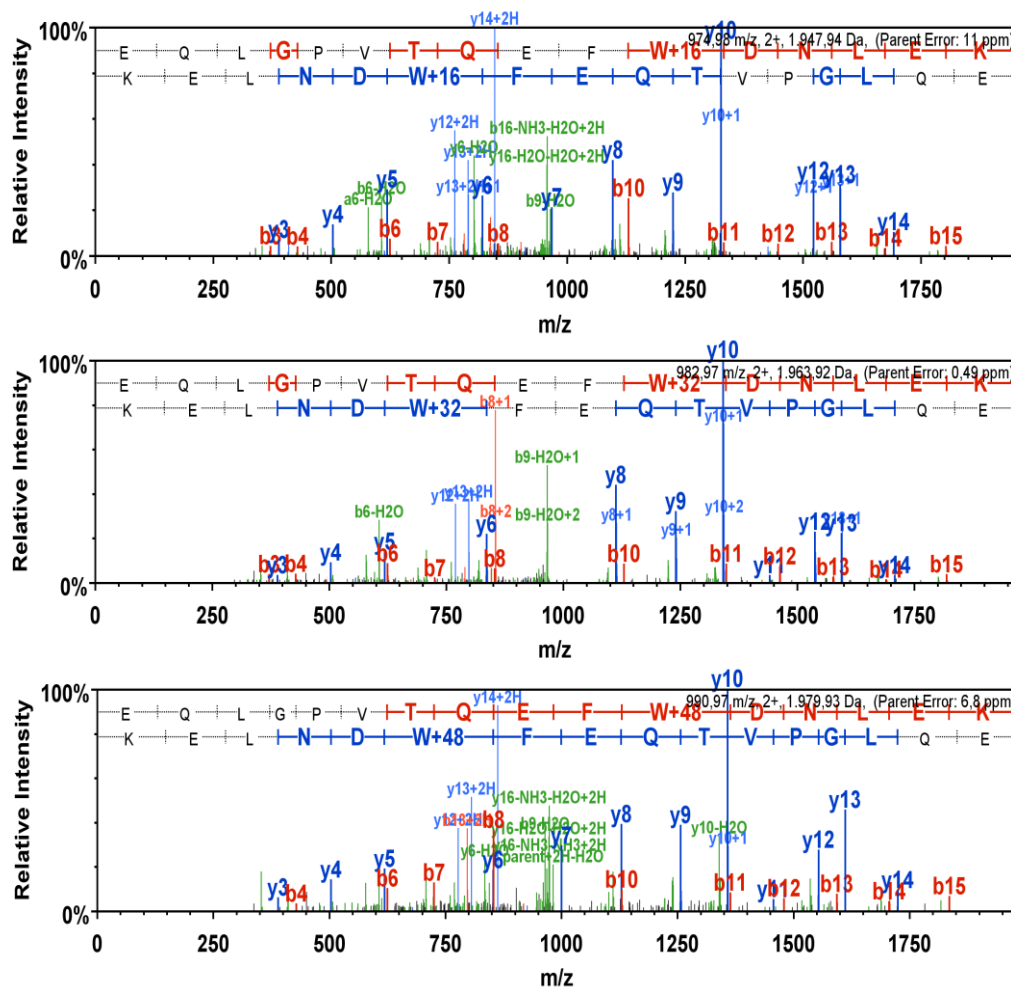


Supplemental Fig. 1. HDL₃ of low-BMI RRMS patients does not suppress inflammation-induced protein expression of TNF α Monocytes of HC were pre-incubated with pooled HDL₃ (60 mg/dl) isolated from low-BMI control subjects (n=8) or low-BMI RRMS patients (n=9) for four days followed by an overnight LPS (100 ng/ml) stimulus. Protein expression levels of TNF α present in the medium of monocytes were measured with ELISA. Results are expressed as fold change of control LPS conditions without HDL₃. HC = healthy controls; RRMS = relapsing-remitting multiple sclerosis; LPS = lipopolysaccharide. [†] versus control LPS (=1) ([†]P<0.05).

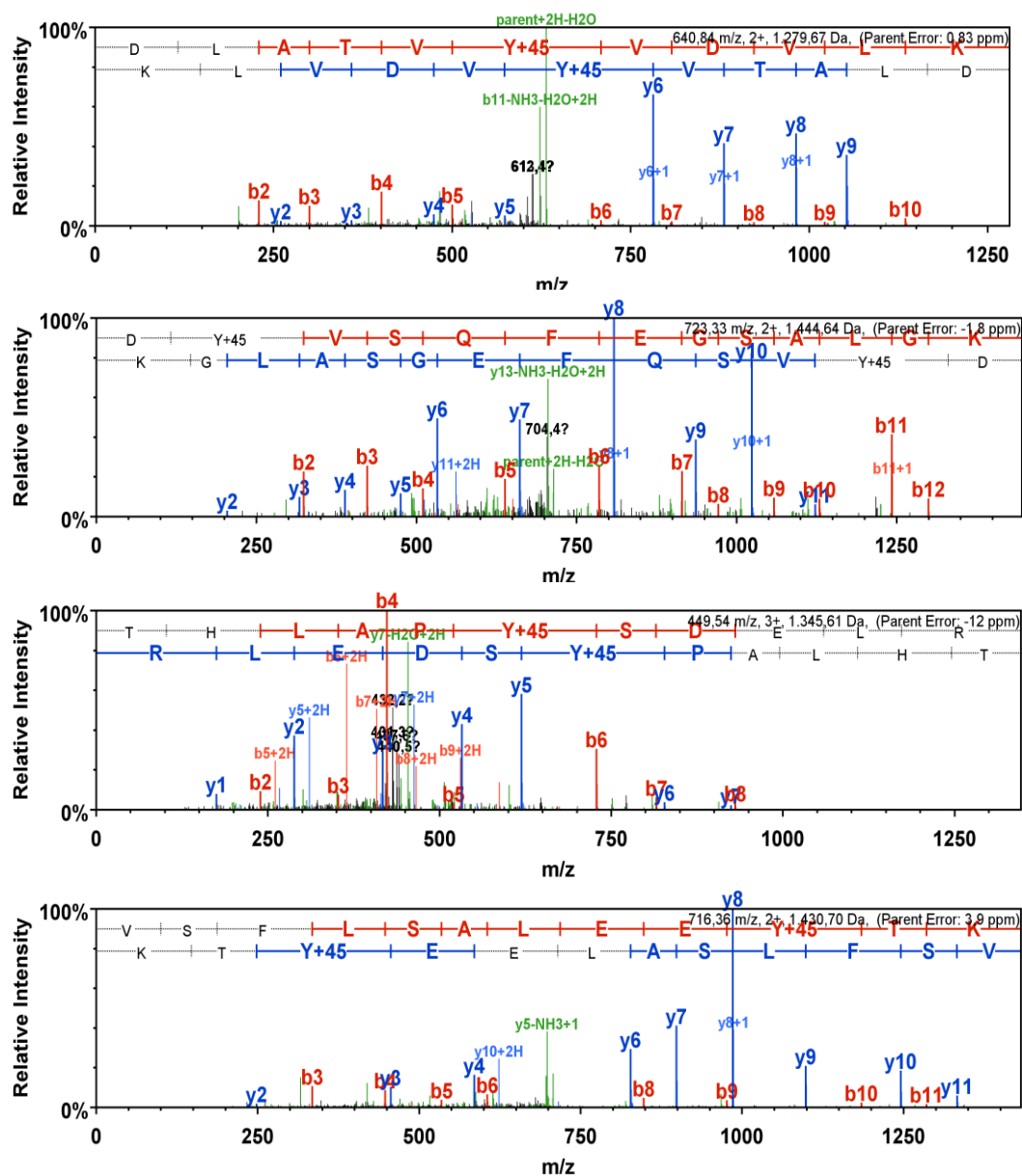


Supplemental Fig. 2. HDL₃ of low-BMI HC and RRMS patients does not influence gene expression of the anti-inflammatory markers CD163, TGFβ, and CD206 on monocytes

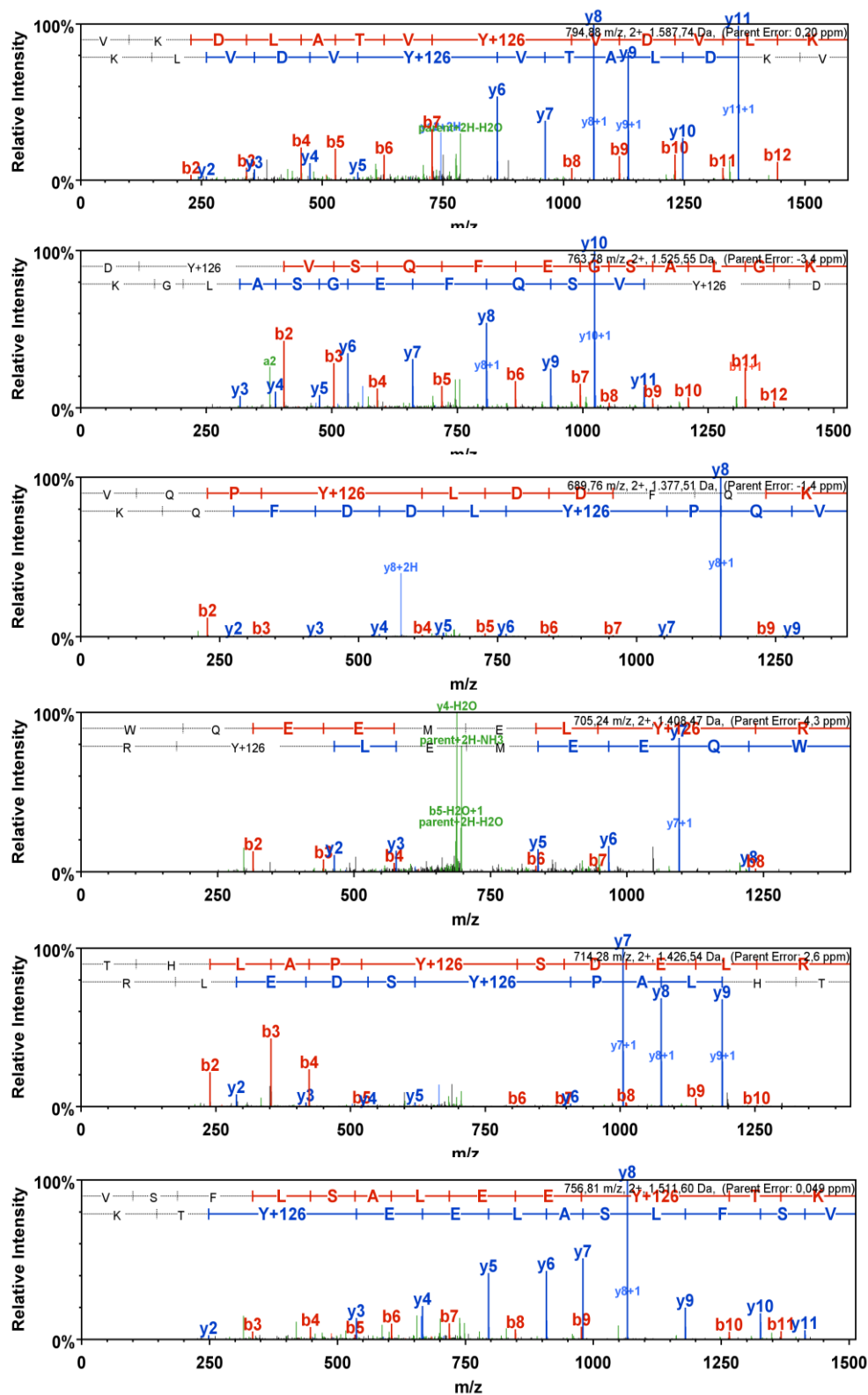
Monocytes of HC were pre-incubated with pooled HDL₃ (60 mg/dl) isolated from low-BMI control subjects (n=8) or low-BMI RRMS patients (n=9) for four days. mRNA expression levels of CD163, TGFβ, and CD206 were measured with qPCR. Results are expressed as fold change of control conditions without HDL₃. HC = healthy controls; RRMS = relapsing-remitting multiple sclerosis.



Supplemental Fig. 3. Mono-, di- and tri-oxidations of Trp-72 in ApoA-I of RRMS patients
CID spectra of mono-, di- and tri-oxidations of Trp-72 in ApoA-I of RRMS patients are shown respectively from top to bottom. Spectra were acquired during the analysis of in-gel tryptic digests of the ApoA-I band from sHDL isolated with sequential flotation ultracentrifugation. Modifications were detected in an LC-MS/MS experiment as described under “Methods”.



Supplemental Fig. 4. Nitrations of Tyr-18, 29, 166, and 236 in ApoA-I of RRMS Patients
CID spectra of Tyr-18, 29, 166, and 236 nitrosation sites in ApoA-I of RRMS patients are shown respectively from top to bottom.



Supplemental Fig. 5. 126 amu modification of Tyr-18, 29, 100, 115, 166, and 236 in ApoA-I of RRMS Patients CID spectra of 126 amu modifications of Tyr-18, 29, 100, 115, 166, and 236 in ApoA-I of RRMS patients are shown respectively from top to bottom.

Supplemental Table 1. MS patient therapies

	RRMS (n)	Progressive MS (n)
No treatment	5	9
Interferon β (Rebif®)	10	1
Glatiramer acetate (Copaxone®)	2	2
Cyclofosfamide (Endoxan®)	3	10
Fingolimod (Gilenya®)	9	1
Natalizumab (Tysabri®)	5	1
Teriflunomide (Aubagio®)	1	
Methotrexate (Ledertrexate®)		1
Dimethyl Fumarate (BG-12®)	1	

Supplemental Table 2. Primer sequences for qPCR

	Forward	Reverse
Human TNF α	AGCCCATGTTGTAGCAAACC	TGAGGTACAGGCCCTCTGAT
Human CD40	TGCGACCCCAACCTAGGGCTT	AAAGCCGGGCGAGCATGAGC
Human IL1 β	GATGAAGTGCTCCTTCCAGG	GCATCTTCCTCAGCTTGTCC
Human IFN γ	GGGGCCAACCTAGGCAGCCAAC	AAGCACTGGCTCAGATTGCAGGC
Human CD206	TCTCCTACTGGACACCAGGC	ATTTCTGTGATTGCGCATCC
Human CD163	TGGAGTGACCTGCTCAGATG	CATCACACACTGTTCCCCAC
Human TGF β	GTGGAAACCCACAACGAAAT	CACGTGCTGCTCCACTTTTA
Human ABCA1	AACGAGACTAACGAGGCAATC	ACACAATACCAGCCCAGAAC
Human ABCG1	CCAGAAGTCGGAGGCCATC	AAGTCCAGGTACAGCTTGGCA