

## ***Supplemental Information***

### **Inhibition of Vascular Inflammation by Apolipoprotein A-IV**

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**Supplementary Table 1: Real-time PCR Primers**

<b>Gene</b>	<b>Direction</b>	<b>Primer sequence</b>
<b>Human ICAM-1</b>	Sense	5' CCA TCT ACA GCT TTC CGG CGC 3'
	Antisense	5' CTC TGG GGT GGC CTT CAG CA 3'
<b>Human VCAM-1</b>	Sense	5' ATG TAG TGT CAT GGG CTG TG 3'
	Antisense	5' GGA ATG AGT AGA GCT CCA CC 3'
<b>Human MCP-1</b>	Sense	5' TCC ACA ACC CAA GAA CAC A 3'
	Antisense	5' TCC ACA ACC CAA GAA CAC A 3'
<b>Human DHCR24</b>	Sense	5' CTC CTG CCG CTC TCG CTT ATC 3'
	Antisense	5' GTC TTG CTA CCC TGC TCC TTC C 3'
<b>Human HRPT-1</b>	Sense	5' TGA CAC TGG CAA AAC AAT GCA 3'
	Antisense	5' GGT CCT TTT CAC CAG CAA GCT 3'
<b>Human B2M</b>	Sense	5' CAT CCA GCG TAC TCC AAA GA 3'
	Antisense	5' GAC AAG TCT GAA TGC TCC AC 3'

**Supplementary Table 2: A single apoA-IV infusion does not affect plasma lipid, apoA-I or apoA-IV levels in NZW rabbits**

<b>Plasma Lipids</b>	<b>Treatment</b>	<b>Baseline (T=0 h)</b>	<b>Collar insertion (24 h)</b>	<b>Euthanasia (48 h)</b>
<b>Apolipoprotein (<math>\mu\text{mol/L}</math>)</b>	<b>Saline</b>	417.5 $\pm$ 35	412.2 $\pm$ 28	488.4 $\pm$ 35
	<b>ApoA-I</b>	415.6 $\pm$ 52	444.6 $\pm$ 61	448.5 $\pm$ 80
	<b>ApoA-IV</b>	359.6 $\pm$ 15	372.2 $\pm$ 26	456.9 $\pm$ 31
<b>Phospholipid (<math>\mu\text{mol/L}</math>)</b>	<b>Saline</b>	842.3 $\pm$ 37	856.7 $\pm$ 126	789.0 $\pm$ 36
	<b>ApoA-I</b>	904.0 $\pm$ 80	853.2 $\pm$ 87	887.8 $\pm$ 76
	<b>ApoA-IV</b>	843.0 $\pm$ 70	819.6 $\pm$ 87	887.4 $\pm$ 73
<b>Unesterified Cholesterol (<math>\mu\text{mol/L}</math>)</b>	<b>Saline</b>	144.7 $\pm$ 17	123.4 $\pm$ 10	135.5 $\pm$ 11
	<b>ApoA-I</b>	145.2 $\pm$ 8	155.2 $\pm$ 25	144.2 $\pm$ 6
	<b>ApoA-IV</b>	146.7 $\pm$ 11	147.4 $\pm$ 24	162.7 $\pm$ 14
<b>Total Cholesterol (<math>\mu\text{mol/L}</math>)</b>	<b>Saline</b>	769.1 $\pm$ 66	635.8 $\pm$ 50	704.5 $\pm$ 50
	<b>ApoA-I</b>	699.1 $\pm$ 74	727.3 $\pm$ 99	635.3 $\pm$ 83
	<b>ApoA-IV</b>	685.5 $\pm$ 75	685.5 $\pm$ 75	755.1 $\pm$ 68

NZW rabbits (n=6/group) were randomised to receive iv saline, lipid-free apoA-I (8 mg/kg), or lipid-free apoA-IV (1 mg/kg) at 24 h prior to placement of a non-occlusive, peri-arterial collar around the left common carotid artery. The animals were euthanised 24 h after collar placement and plasma lipid, apoA-I and apoA-IV levels were quantified as described in Materials and Methods.

**Supplementary Table 3: A single apoA-IV infusion does not affect HDL composition or size in NZW rabbits**

<b>Treatment</b>	<b>PL/UC/CE/apoA-I) (<i>mol/mol</i>)</b>	<b>Particle Diameter (<i>nm</i>)</b>
<b>Saline</b>	17.6±4.0/1.5±0.8/13.6±0.3/1.0	8.9
<b>ApoA-I</b>	21.8±3.0/1.6±0.5/11.0±2.0/1.0	9.0
<b>ApoA-IV</b>	19.5±6.0/1.8±0.2/10.6.0/1.0	9.2

NZW rabbits (n=6/group) were randomised to receive iv saline, lipid-free apoA-I (8 mg/kg iv), or lipid-free apoA-IV (1 mg/kg iv) at 24 h prior to placement of a non-occlusive, peri-arterial collar around the left common carotid artery. The animals were euthanised 24 h after collar placement, the HDLs were isolated by ultracentrifugation and their composition and size were quantified as described in Materials and Methods.