Supplemental Figure 1: Pilot kinetic studies conducted in fasting white adipose tissue (WAT) of 4 subjects to optimize experimental conditions to be used for LPS and ATP as priming and activation positive controls of WAT NLRP3 inflammasome, respectively. Shown here is WAT IL-1β-secretion following incubation with (A) LPS concentration-curve for 6 hours followed by 5 mmol/L ATP for 20 min, N=1; (B) 1 µg/mL LPS time-curve followed by 5 mmol/L ATP for 20 min, N=1; (C) 0.3 µg/ml LPS for 6 hours followed by ATP concentration-curve for 20 minutes, N=3; and (D) 0.3 µg/ml LPS for 4 hours followed by 3 mmol/L ATP time-curve, N=2. Minimal LPS and ATP concentrations and incubation period that induced maximal WAT IL-1β-secretion were then used as positive controls for all experiments described in manuscript and were: 0.3 µg/ml LPS for 4 hours followed by 3 mmol/L ATP for 3 hours (highlighted in red rectangular below). N.B. LPS was used for 4 hours for practical reasons to account for the 8 hours needed to complete experiments in postprandial WAT (not included in this manuscript).
Supplemental Figure 2: Sex differences in fasting WAT IL-1β-secretion induced by 5% FBS medium/medium (baseline or negative control), LDL/LDL, medium/ATP, LDL/ATP, LPS/medium, LPS/LDL, or LPS/ATP (positive control) for the priming/activation periods of the NLRP3 inflammasome. Data was analyzed by mixed-model analysis with treatment and sex interaction as in methods and presented as average +/- SEM. N=23 women and N=11 men except with LDL/LDL, where N=21 women and N=11 men for missing data.
**Supplemental Figure 3:** Cytotoxic assay measuring LDH release into WAT medium induced by 5% FBS medium/medium (baseline or negative control), LDL/LDL, medium/ATP, LDL/ATP, LPS/medium, LPS/LDL, or LPS/ATP (positive control) for the priming/activation periods of the NLRP3 inflammasome. Also shown are LDH concentrations measured in WAT-free medium alone or supplemented with 1.2 g/L apoB incubated for 3 hours compared to the LDH positive control provided by the commercial kit (Invitrogen). Notably, LDL presence alone (without WAT) represent a modest false positive signal for LDH measurement above baseline (medium).
Supplemental Figure 4: Heat map representing Pearson correlation of subject weight, BMI, waist circumference, hip circumference, waist/hip ratio, android fat, gynoid fat, total body fat, android/gynoid fat ratio with fasting \( \log_{10} \) WAT IL-1\( \beta \)-secretion at baseline (medium/medium) and following incubation with LDL/LDL, medium/ATP, LDL/ATP, LPS/medium, LPS/LDL, or LPS/ATP for the priming/activation periods, and with \( \log_{10} \) WAT NLRP3, \( \log_{10} \) WAT IL1B mRNA and WAT pro-IL-1\( \beta \) protein in all subjects. N=40 for WAT mRNA and pro-IL-1\( \beta \) data and N=34 for all WAT IL-1\( \beta \)-secretion conditions except with LDL/LDL when N=31 for missing data. Grey cells represent insignificant and red cells represent significant positive correlations.

![Heat map image](image-url)
Supplemental Figure 5: Differences between subjects with low-apoB versus high-apoB in % fasting plasma phospholipid fatty acids. N=19 for low-apoB and N=20 for high-apoB for all fatty acids except for % C17:0 where N=5 for low and N=2 for high-apoB, % C16:1n-7 where N=16 for low-apoB, and % C18:3n-6 where N=18 for low and N=19 for high-apoB as the sample for one subject with low-apoB could not be measured and the named fatty acids were below detection limit of the GC-MS in some subjects.
Supplemental Figure 6: Differences between subjects with low-apoB versus high-apoB in fasting baseline WAT mRNA expression of ADIPOQ, PPARγ, HMGCR, SREBP1C, SREBP2, LDLR, and CD36 normalized for HPRT (A), and of MCP1, ADGRE1, IL10, NLRP3, CASP1, and IL1B normalized for HPRT (B) analyzed by mixed-model analysis with group x gene interaction and presented as boxes with whiskers representing 10th – 90th percentile and line at the average. N=20 for low and N=20 for high plasma apoB.
Supplemental Figure 7: Pearson correlation of fasting plasma apoB with 2nd phase GIIS\textsubscript{IVGTT} (A), total GIIS\textsubscript{IVGTT} (B), 2nd phase C-peptide secretion\textsubscript{IVGTT} (C), total C-peptide secretion\textsubscript{IVGTT} (D), insulin sensitivity as M/I\textsubscript{clamp} (E), fasting plasma IL-1Ra (F), AUC\textsubscript{6hr} plasma TG (G) and AUC\textsubscript{6hr} plasma apoB48 (H) in women (N=27, closed circles, dotted regression line) and men (N=13, open circles, dashed regression line) except for panels A-D where N=11 men and panel E where N=12 men for missing data. Solid regression line represents pooled data for both sexes.

- **A**: 2nd phase GIIS\textsubscript{IVGTT} (μU/mL) vs. Plasma apoB (g/L) with r = 0.33 and p = 0.038.
- **B**: Total GIIS\textsubscript{IVGTT} (μU/mL) with r = 0.80 in men and p = 0.003.
- **C**: 2nd phase C-peptide secretion\textsubscript{IVGTT} (ng/mL) with r = 0.36 and P = 0.025.
- **D**: Total C-peptide secretion\textsubscript{IVGTT} (ng/mL) with r = 0.35 and p = 0.031.
- **E**: Insulin sensitivity M/I\textsubscript{clamp} (mg/kg/min)/(μU/mL) with r = -0.50 and p = 0.001.
- **F**: Fasting plasma IL-1Ra (log_{10} (pg/mL)) with r = 0.34 and p = 0.032.
- **G**: AUC\textsubscript{6hr} plasma TG (log_{10} (mmol/L)) with r = 0.61 and p < 0.001.
- **H**: AUC\textsubscript{6hr} plasma apoB48 (log_{10} (mmol/L)) with r = 0.35 and p = 0.029.
Supplemental Figure 8: Pearson correlation of fasting WAT IL-1β-secretion induced by LPS/LDL with 2\textsuperscript{nd} phase GIIS\textsubscript{IVGTT} (A), total GIIS\textsubscript{IVGTT} (B), insulin sensitivity as M/I\textsubscript{clamp} (C), AUC\textsubscript{6hrs} plasma apoB48 (D), AUC\textsubscript{6hrs} plasma TG (E), fasting baseline WAT mRNA expression of ADIPOQ (F) and CD36 (G) normalized for HPRT and fasting plasma apoB (H), and estimated LDL size as LDL-C/apoB ratio (I) in subjects with low-apoB (N=15, open circles) and high-apoB (N=19, closed circles). Solid regression line represents correlation in all subjects.
Supplemental Figure 9: Pearson correlation of fasting baseline WAT mRNA expression of *NLRP3* normalized for *HPRT* with 1st phase C-peptide secretion$_{IVGTT}$ (A), 2nd phase C-peptide secretion$_{IVGTT}$ (B), total C-peptide secretion$_{IVGTT}$ (C), insulin sensitivity as M/I$_{clamp}$ (D), total disposition index (total C-peptide$_{IVGTT}$ x M/I$_{clamp}$) (E), AUC$_{6hrs}$ plasma TG (F), fasting plasma IL-1Ra (G), TG (H), apoB/PCSK9 ratio (I), LDL-C/apoB ratio (J), and HDL-C (K), fasting baseline WAT mRNA expression of *PPARG* (L), *ADIPOQ* (M), *CD36* (N), *SREBP2* (O), *MCP1* (P), *ADGRE1* (Q), and *IL1B* (R) normalized to *HPRT*, and % fasting plasma phospholipid palmitate (S), arachidonate (T) and stearate (U) in subjects with low-apoB (N=20, open circles, dotted regression line) and high-apoB (N=20, closed circles, dashed regression line). Solid regression line represents pooled data for all subjects.