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Supplemental Material

Evaluation of Early Biomarkers of Atherosclerosis Associated with Polychlorinated Biphenyl Exposure: An *in Vitro* and *in Vivo* Study

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Figure S1. Functional analyses of genes targeted by differentially expressed miRNAs. High-throughput sequencing analysis of differentially expressed miRNAs after 5 μ M PCB29-pQ treatment of HUVECs for 24 h ($n = 3$). Target gene of differentially expressed genes were screened and functional analyses performed. Gene ontology has three ontologies: molecular function, cellular component, and biological process. The exact data are presented in Excel Table S3.

Figure S2. KEGG pathway analyses of genes targeted by differentially expressed miRNAs. High-throughput sequencing analysis of differentially expressed miRNAs in HUVECs with 5 μ M PCB29-pQ treatment for 24 h ($n = 3$). KEGG pathway enrichment in miRNA, the rich factor represents the degree of enrichment. The node size shows the number of selected genes, and the color scale represents $-\log(P \text{ value})$. The exact data are presented in Excel Table S4.

Figure S3. The expression of *HDAC7-AS1*, *MIR-7-5p* and *Ago2* in HUVECs exposed to PCB29-pQ. RT-qPCR was performed to validate (a) lncRNAs and (b) miRNAs identified as differentially expressed by RNA sequencing assay. β -*Actin* was used as housekeeping genes for lncRNAs quantification and *U6* was used as housekeeping genes for miRNAs quantification, respectively. RP5-1057120.4 (termed as *HDAC7-AS1*) and *MIR-7-5p* were selected as target lncRNA and miRNA for further investigation (indicated with red boxes). Data are presented as mean \pm SD ($n = 3$). (c) Anti-Ago2 RNA binding protein immunoprecipitation assay was used to pull down endogenous RNAs associated with Ago2; IgG served as the control. Ago2 in proteins from Ago2-RIP assay was measured by western blotting. Data are presented as mean \pm SD ($n = 3$). P values were determined by two-way ANOVA, followed by Tukey's *post hoc* test. Data are graphed relative respective vehicle controls. The exact mean and SD value are presented in Table S20.

Figure S4. The expression of *HDAC7* level in HUVECs transfected with *HDAC7-AS1* siRNA or pEZ-M61-*HDAC7-AS1*. (a) Predicted relationship between ENST00000080059 (*HDAC7*) and ENST00000599515 (*HDAC7-AS1*) via RNAplex (<http://www.tbi.univie.ac.at/RNA/RNAplex.1.html>) (b) After transfection with NC siRNA or *HDAC7-AS1* siRNA (25, 50, or 100 nM) for 48 h, the silencing efficiency of *HDAC7-AS1* siRNA in HUVECs was determined by RT-qPCR. Data are presented as mean \pm SD (n = 3). (c) After transfection with 2.5 μ g pEZ-M61-NC or pEZ-M61-*HDAC7-AS1* (2.5, 5, or 7.5 μ g) for 6 h, HUVECs were treated with 5 μ M PCB29-pQ. *HDAC7-AS1* level was determined by RT-qPCR. Data are presented as mean \pm SD (n = 3). *HDAC7* expression of PCB29-pQ-exposed cells or control after (d) transfection with 25 nM NC siRNA or 25 nM *HDAC7-AS1* siRNA for 24 h or (e) transfection with 2.5 μ g pEZ-M61-NC or 2.5 μ g pEZ-M61-*HDAC7-AS1* for 6 h. Data are presented as mean \pm SD (n = 3). The housekeeping gene *β -actin* was used for quantification, and the primer information is shown Table S3. P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. For b and c, expression is shown relative to NC siRNA control and pEZ-M61-NC controls, respectively. The exact mean and SD values are presented in Table S21.

Figure S5. The localization of LncRNA *HDAC7-AS1* in HUVECs. HUVECs were treated with 5 μ M PCB29-pQ for 24 h. (n = 3). Fluorescent *in situ* hybridization (FISH) assay was used to investigate the localization of *HDAC7-AS1* in the cells. *HDAC7-AS1* was stained red using an RNA probe, and the nucleus was stained blue with DAPI. Scale bar = 10 μ m.

Figure S6. Volcano plots analyzing differential expression with mRNA sequencing (mRNA-Seq) in the control group and PCB29-pQ group. The abscissa represents the logarithmic values of two different groups, and the ordinate represents mRNAs differences (fold change ≥ 2 and P value < 0.05) between two groups. Red dots indicated PCB29-pQ group is higher relative to control group). Green dots indicated lower expression in PCB29-pQ group than control group (judgment standard is P value < 0.05 , and the difference multiple is more than 2). Black dots indicated no difference between two groups.

Figure S7. Target genes TGF- $\beta 2$ and PPME1 protein levels in HUVECs exposed to PCB29-pQ with *MIR-7-5p* inhibitor or TGF- $\beta 2$ /PPME1 siRNA. After co-transfection with 100 nM NC inhibitor or *MIR-7-5p* inhibitor and NC siRNA or TGF- $\beta 2$ /PPME1 siRNA (25 nM) for 24 h, HUVECs were treated with 5 μ M PCB29-pQ for 24 h. (n = 3). Protein levels of (a) PPME1 and (b) TGF- $\beta 2$ were detected by western blotting (upper panel). The relative protein expression levels (lower panel) were quantified by ImageJ software. Data are presented as mean \pm SD (n = 3). β -Actin was used as an internal loading control. Data are graphed relative to the expression in cells exposed to the NC inhibitor and NC siRNA together. The exact mean and SD values are presented in Table S22.

Figure S8. Luciferase analysis of the activity of *MIR-7-5p* bind to *TGF-β2*, *PPME1*, and *HDAC7-ASI* in HUVECs transfected with NC mimic or *MIR-7-5p* mimic. (a) HUVECs were transfected with NC mimic or *MIR-7-5p* mimic for 48 h, together with NC-3'UTR, *TGF-β2* (WT)-3'UTR, or *TGF-β2* (MUT)-3'UTR. (b) HUVECs were transfected with 50 nM NC mimic or 50 nM *MIR-7-5p* mimic for 48 h, together with NC-3'UTR, *PPME1* (WT)-3'UTR, or *PPME1* (MUT)-3'UTR. (c) HUVECs were transfected with 50 nM NC mimic or 50 nM *MIR-7-5p* mimic for 48 h, together with Luc-NC, Luc-*HDAC7-ASI*-WT, or Luc-*HDAC7-ASI*-MUT. For all panels, Gaussia luciferase activity was analyzed, corresponding to *TGF-β2*, *PPME1*, and *HDAC7-ASI* transcription. Data are presented as mean ± SD (n = 3). P values were determined by two-way ANOVA, followed by Tukey's *post hoc* test. Data are graphed relative to the NC-mimic treated, NC-3'UTR controls. The exact mean and SD values are presented in Table S23.

Figure S9. The tube forming ability in HUVECs exposed to PCB29-pQ and transfected with a *MIR-7-5p* inhibitor. (Left panel) After transfection with 100 nM NC inhibitor or 100 nM *MIR-7-5p* inhibitor for 48 h, followed by exposure to 5 μM PCB29-pQ for 24 h, cells were plated on Matrigel to conduct tube formation assay. Scale bar = 200 μm. (Right panel) Quantification of tube formation through measurement of branch point number with ImageJ software. Data were presented as mean ± SD (n = 3). P values were determined by two-way ANOVA, followed by Tukey's *post hoc* test. The exact mean and SD values are presented in Table S24.

Figure S10. Apoptosis and proliferation rates in HUVECs exposed to PCB29-pQ and transfected with a *MIR-7-5p* mimic or pEZ-M61-*HDAC7-ASI*. (a) Cell apoptosis was assessed by Annexin V-FITC/PI double staining with a flow cytometer. (n = 3). (b) Cell proliferation was measured by BrdU/PI double staining with a flow cytometer. (n = 3).

Figure S11. *HDAC7-ASI*, *MIR-7-5p*, *TGF-β2*, *PPME1* mRNA levels, *TGF-β2* and *PPME1* protein levels, and apoptosis or proliferation rates in HUVECs exposed to PCB29-pQ and transfected with a *MIR-7-5p* inhibitor or *HDAC7-ASI* siRNA. After co-transfection with 100 nM NC inhibitor or 100 nM *MIR-7-5p* inhibitor and NC siRNA or *HDAC7-ASI* siRNA (25 nM) for 24 h, HUVECs were treated with 5 μM PCB29-pQ for 24 h. RNAs expression of (a) *MIR-7-5p*, (b) *HDAC7-ASI*, (c) *TGF-β2* and (d) *PPME1* were detected by RT-qPCR. *β-actin* was used as a housekeeping gene, except U6 was used for *MIR-7-5p* quantification. The primer information is shown in Table S3-4. (e) Protein levels of *TGF-β2* and *PPME1* were detected by western blotting. *β-Actin* was used as an internal loading control. (f) *TGF-β2*, (g) *PPME1* protein expression levels were quantified by ImageJ software. Data were presented as mean ± Standard Deviation (SD) (n = 3). (h) Cell viability was measured by CCK-8 kit. Data were presented as mean ± SD (n = 3). (i) Cell apoptosis was performed by Annexin V-FITC/PI assay with a flow cytometer. (j) Cell proliferation was performed by BrdU/PI staining. P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. Data are graphed relative to the cells exposed to NC inhibitor, NC siRNA, and vehicle control. The exact mean and SD values are presented in Table S25.

Figure S12. HE staining of aortic root cross-sections and TC and TG levels in *ApoE*^{-/-} mice were intravenous (*i.v.*) injected with AAV-*HDAC7-AS1*. Male *ApoE*^{-/-} mice were *i.v.* injected with AAV-*HDAC7-AS1* via tail vein (4×10^{10} particles/mouse) to create an *HDAC7-AS1* overexpressed mice model. Male *ApoE*^{-/-} mice that received the AAV vector were used as AAV control mice. Control and *HDAC7-AS1* overexpressed *ApoE*^{-/-} mice were fed a western high-fat diet for 12 weeks and tap water ad libitum. Mice were injected with 5 mg/kg body weight of PCB29-pQ or equal volumes of corn oil by *i.p.* injection once a week for 12 continuous weeks with the first two injections during the 1st week (3 days apart). (a) (Left panel) HE of the aortic root (Right panel) plaque area in the aortic root was quantified by ImageJ software. Data are presented as mean \pm Standard Deviation (SD). Scale bar = 200 μ m. (b) TC level and (c) LDL-C level. Data are presented as mean \pm SD (n = 5). P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. The exact mean and SD values are presented in Table S26.

Figure S13. CAV1 phosphorylation and TGF- β , PPME1, and inflammatory factor levels in HUVECs exposed PCB29-pQ with CAV1 siRNA. (a) HUVECs were treated with 5 μ M PCB29-pQ for 1, 3, and 6 h. (upper panel) Protein levels of p-CAV1 and CAV1 were detected by western blotting. β -Actin was used as an internal loading control. The relative protein expression levels (lower panel) were quantified by ImageJ software. Data are presented as mean \pm SD (n = 3). After transfection with 25 nM NC siRNA or *CAVI* siRNA for 48 h, HUVECs were treated with 5 μ M PCB29-pQ for 24 h. The siRNA information is shown in Table S6. (b) Protein levels of p-CAV1, CAV1, IL-1 β , IL-6, and TNF α were detected by western blotting. β -Actin was used as an internal loading control. (c-h) p-CAV1, CAV1, IL-1 β , IL-6, and TNF α were quantified by ImageJ software. Data are presented as mean \pm SD (n = 3). P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. Data are graphed relative to the NC siRNA group. The exact mean and SD values are presented in Table S27.

Figure S14. p-CAV1 level in *ApoE*^{-/-} mice that were *i.v.* injected with AAV-*HDAC7-AS1*. Male *ApoE*^{-/-} mice were *i.v.* injected with AAV-*HDAC7-AS1* via tail vein (4×10^{10} particles/mouse) to create an *HDAC7-AS1* overexpressed mice model. Male *ApoE*^{-/-} mice that received the AAV vector were considered as AAV control mice. Control and *HDAC7-AS1* overexpressed *ApoE*^{-/-} mice were fed a Western high-fat diet for 12 weeks and tap water ad libitum. Mice were injected with 5 mg/kg body weight of PCB29-pQ or equal volumes of corn oil by *i.p.* injection once a week for 12 continuous weeks with the first two injections during the 1st week (3 days apart). (Left panel) The presence of p-CAV1 in aortic root cross-sections was detected by double immunostaining with the use of antibodies against p-CAV1 (red), endothelial cell marker CD31 (green), and nucleus marker DAPI (blue), respectively. Scale bar = 100 μ m. White arrows represent the colocalization of p-CAV1 (red) and CD31 (green). (Right panel) Colocalization of p-CAV1 and CD31 was analyzed by Pearson's correlation coefficient. Data were presented as mean \pm SD (n = 3). The exact mean and SD values are presented in Table S28.

Figure S15. Gene type identification of *ApoE* and *CAVI* knockout mice. DNA from the tail of offspring mice was extracted and analyzed by PCR and agarose gel electrophoresis. (a) Homozygous of *ApoE*: 245 bp; Heterozygous of *ApoE*: 245 bp & 155 bp; WT of *ApoE*: 155 bp. (b) Homozygous of *CAVI*: 410 bp; Heterozygous of *CAVI*: 690 bp & 410 bp; WT of *CAVI*: 690 bp. The PCR primer sequences for *ApoE* and *CAVI* mice genotyping are shown in Table S2.

Figure S16. Immunohistochemistry staining of CD68 and TC and LDL-C levels in *ApoE*^{-/-} and *ApoE*^{-/-}/*CAVI*^{-/-} mice. *ApoE*^{-/-} mice were crossed with *CAVI*^{-/-} mice to generate *ApoE*^{-/-}/*CAVI*^{-/-} mice. *ApoE*^{-/-} or *ApoE*^{-/-}/*CAVI*^{-/-} mice were fed a Western high-fat diet for 12 weeks and tap water ad libitum. Mice were treated with 5 mg/kg body weight of PCB29-pQ or equal volumes of corn oil by *i.p.* injection. (a) (Left panel) Immunohistochemistry staining of CD68 detected macrophage infiltration located at the aortic wall. Scale bar = 200 μm. (Right panel) Quantification proportion of positive CD68 expressions area to total aortic section using was performed by ImageJ software. Data are presented as mean ± SD (n = 3). Serum (b) TC level and (c) LDL-C levels. Data are presented as mean ±SD (n = 5). P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. The exact mean and SD values are presented in Table S29.

Figure S17. Adhesion molecules, pro-inflammatory cytokines and p65 protein expression levels in HUVEC exposed to PCB29-pQ. HUVECs were treated with 5 μM PCB29-pQ for 1, 3, or 6 h. (a) IL-1β, IL-6, TNF-α, ICAM-1, and VCAM-1 expressions in cell lysates were analyzed by western blotting. (b-f) IL-1β, IL-6, TNF-α, ICAM-1, and VCAM-1 expressions were quantified by ImageJ software. Data are presented as mean ± Standard Deviation (SD) (n = 3). (g) Calcein-AM-loaded THP-1 cells (10⁶ cells/ml) were added to HUVECs and then incubated for 3 h. The unbound cells were washed off, and attached fluorescent monocytes were visualized using an optical microscope. (Left panel) The fluorescence intensity of Calcein-AM was quantified using ImageJ software. Data are presented as mean ± SD (n = 3). P value was determined by unpaired Student's t-test. (h) HUVECs were treated with 5 μM PCB29-pQ for 1, 3, or 6 h. IκBα and p-p65 levels were analyzed by western blotting analysis. β-Actin was used as an internal loading control. (i-j) IκBα and p-p65 levels were quantified by ImageJ software. Data are presented as mean ± SD (n = 3). (k) HUVECs were pretreated with 5 μM p65 inhibitor PDTC for 1 h, followed with 5 μM PCB29-pQ exposure for 6 h. IL-1β, IL-6, and TNF-α in cell lysates were analyzed by western blotting. β-Actin was used as an internal loading control. (l-n) IL-1β, IL-6, and TNF-α protein expression levels were quantified by ImageJ software. Data were presented as mean ± SD (n = 3). P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. The exact mean and SD values are presented in Table S30.

Figure S18. Adhesion molecules, pro-inflammatory cytokines and p65 protein expression levels in HUVEC exposed to PCB29-pQ with *CAVI* siRNA. HUVECs were transfected with NC siRNA or 25 nM *CAVI* siRNA for 48 h and then treated with 5 μM PCB29-pQ for 6 h. (a) IL-1β, IL-6, TNF-α, ICAM-1 and VCAM-1 expressions were analyzed by western blotting. β-Actin was used as an internal loading control. (b-f) IL-1β, IL-6, TNF-α, ICAM-1 and VCAM-1 expression levels were quantified by ImageJ software. Data were presented as mean ± Standard Deviation (SD) (n = 3). (g) IκBα, p-p65, and p65 expressions were analyzed by western blotting. β-Actin was used as an internal loading control. (h-i) IκBα, p-p65, and p65 expression levels were quantified by ImageJ software. Data were presented as mean ± SD (n = 3). (j) (Left panel) Immunofluorescence analysis of p65. Green staining represents the location of p65. Nuclei shown in blue were stained with DAPI. Scale bar = 10 μm. (Right panel) Co-localization of p-CAVI and DAPI was analyzed by Pearson's correlation coefficient. Data were presented as mean ± SD (n = 3). P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. The exact mean and SD values are presented in Table S31.

Figure S19. Inflammatory factors and ROS levels in HUVEC exposed to PCB29-pQ.

HUVECs were pretreated with 40 μ M VC, 20 μ M VE or 5 mM NAC for 1 h, followed with 5 μ M PCB29-pQ exposure for 6 h. (n = 3). (a) p-p65, p65, IL-1 β , IL-6, TNF α , ICAM-1 and VCAM-1 in cell lysates were analyzed by western blotting. (b-g) p-p65, p65, IL-1 β , IL-6, TNF α , ICAM-1 and VCAM-1 expression levels were quantified by ImageJ software. Data were presented as mean \pm Standard Deviation (SD) (n = 3). (h) ROS levels were detected by DCFH-DA (10 μ M) probe. Data were presented as mean \pm SD (n = 3). P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. In all graphs, data is normalized to the control cell groups. The exact mean and SD values are presented in Table S32.

Figure S20. p-CAV1 and CAV1 levels in HUVEC exposed to PCB29-pQ and antioxidants.

HUVECs were pretreated with 40 μ M VC, 20 μ M VE or 5 mM NAC for 1 h, followed by 5 μ M PCB29-pQ exposure for 6 h. HUVECs were pretreated with (a) 40 μ M VC, 20 μ M VE or 5 mM NAC, (b) 200 U/ml PEG-SOD, 500 U/ml PEG-CAT and 5 mM GSH-MEE for 1 h, followed with 5 μ M PCB29-pQ exposure for 6 h. (Left panel) p-CAV1 and CAV1 in cell lysates were analyzed by western blotting. β -Actin was used as an internal loading control. (Right panel) p-CAV1 and CAV1 expression levels were quantified by ImageJ software. Data were presented as mean \pm Standard Deviation (SD) (n = 3). P values were determined by one-way ANOVA, followed by Tukey's *post hoc* test. All data is graphed relative to the control cell groups. The exact mean and SD values are presented in Table S33.

Figure S21. Heatmap showing correlation scores between factors. Plasma RNA was extracted by TRNzol universal reagent. CHD group (n = 77) contains patients with > 50% coronary artery stenosis, and the control group (n = 50) contains subjects with < 50% coronary artery stenosis. RT-qPCR analysis of *HDAC7-AS1*, *MIR-7-5p*, *TGF- β 2*, *PPME1*, *IL-1 β* , *IL6* and *TNF α* expressions. Spearman's rank correlation coefficient was used to assess the correlation between the two indicated factors. P value of < 0.05 was considered significant. The size and color of circle represents the correlation between two factors. X's mean no significance. Summary data can be found in Table S11.

Table S1. Sources of antibodies using in this study.

Table S2. PCR primer sequences for *ApoE* and *CAVI* mice genotype.

Table S3. PCR program for *ApoE* and *CAVI* mice genotype.

Table S4. RT-qPCR primer sequences for lncRNAs and mRNAs.

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Table S20. The expression of select lncRNAs in HUVECs exposed to PCB29-pQ.

Table S21. The expression of *HDAC7* level in HUVECs transfected with *HDAC7-AS1* siRNA or pEZ-M61-*HDAC7-AS1*.

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Table S23. Luciferase analysis of the activity of *MIR-7-5p* bind to *TGF- β 2*, *PPME1*, and *HDAC7-AS1* in HUVECs transfected with NC mimic or *MIR-7-5p* mimic.

Table S24. The tube forming ability in HUVECs exposed to PCB29-pQ and transfected with a *MIR-7-5p* inhibitor.

Table S25. *HDAC7-AS1*, *MIR-7-5p*, *TGF-β2*, *PPME1* levels and apoptosis or proliferation rates in HUVECs exposed to PCB29-pQ and transfected with a *MIR-7-5p* inhibitor or *HDAC7-AS1* siRNA.

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Table S29. Immunohistochemistry staining of CD68, total cholesterol (TC), and low-density lipoprotein level (LDL) in *ApoE*^{-/-} and *ApoE*^{-/-}/*CAVI*^{-/-} mice.

Table S30. Adhesion molecules, pro-inflammatory cytokines and *p65* protein expression level in HUVEC exposed to PCB29-pQ.

Table S31. Adhesion molecules, pro-inflammatory cytokines and *p65* protein expression levels in HUVEC exposed to PCB29-pQ with *CAVI* siRNA.

Table S32. Protein expression of inflammatory factors and ROS levels in HUVEC exposed to PCB29-pQ.

Table S33. p-*CAV1* protein expression relative to *CAV1* total protein in HUVEC exposed to PCB29-pQ and antioxidants.

Additional File- Excel Document