Supplementary

MicroRNAs as Potential Mediators for Cigarette Smoking Induced Atherosclerosis

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Figure S1. Changes in (a) body, (b) lung, (c) liver, and (d) kidney weights of ApoE KO mice after 2-month exposure to CS at low or high dose. Data are mean±SEM of seven animals per group. *P<0.05 versus the control group.
Figure S2. Gene expression levels of NADPH oxidase subunits in ApoE KO mice exposed to CS. The mRNA levels of (a) p47phox and (b) p67phox in the aortic tissues were determined by quantitative RT-PCR analysis. Data are normalized by the abundance of β-actin mRNA. Quantitative data are expressed relative to the values for the control group. Data are mean±SEM of six or seven animals per group.

Figure S3. Expression levels of miRNAs in ApoE KO mice exposed to CS. The levels of (a) miR-126 in the aortic tissues were determined by quantitative RT-PCR analysis. Data are normalized by the abundance of snoRNA135. Quantitative data are expressed relative to the values for the control group. Data are mean±SEM of six or seven animals per group. The scatter plots showing the correlation between expression levels of miR-126 and (b) VCAM-1, (c) ICAM-1, (d) MCP1, and (e) creatinine adjusted level of 24-h urinary 8-iso-prostaglandin F2α. The coefficients and p-values were shown in the plots.
Figure S4. Expression levels of miRNAs in ApoE KO mice exposed to CS. The levels of (a) miR-21 in the aortic tissues were determined by quantitative RT-PCR analysis. Data are normalized by the abundance of snoRNA135. Quantitative data are expressed relative to the values for the control group. Data are mean±SEM of six or seven animals per group. The scatter plots showing the correlation between expression levels of miR-21 and (b) VCAM-1, (c) ICAM-1, (d) MCP1, and (e) creatinine adjusted level of 24-h urinary 8-iso-prostaglandin F2α. The coefficients and p-values were shown in the plots.