

Supplementary Materials

Figure S1

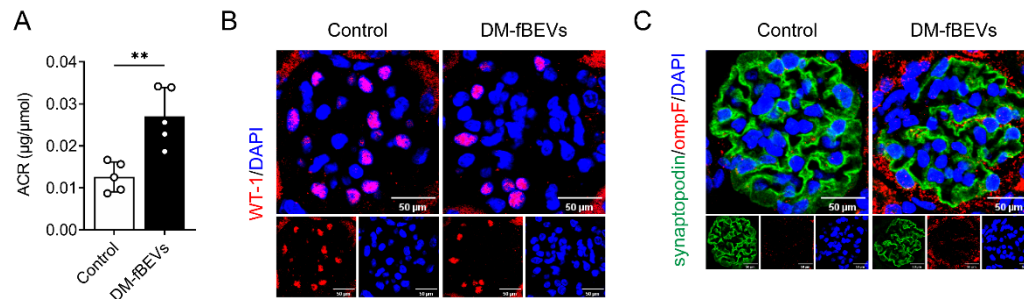


Figure S1. Orally administered DM-fBEVs translocate to glomerulus and induce podocyte damage. (A) Urinary albumin/ creatinine ratio (ACR) was measured in mice ($n=5$). $**P < 0.01$. (B) WT-1⁺ (red) podocytes were stained by immunofluorescence staining. Nuclei were stained with DAPI (blue) (scale bar, 50 μm , original magnification $\times 400$). (C) The presence of ompF⁺ OMVs (red dots) in mouse podocytes marked with synaptopodin (green) was determined by immunofluorescent staining. Nuclei were stained with DAPI (blue) (scale bar, 50 μm , original magnification $\times 400$).

Figure S2

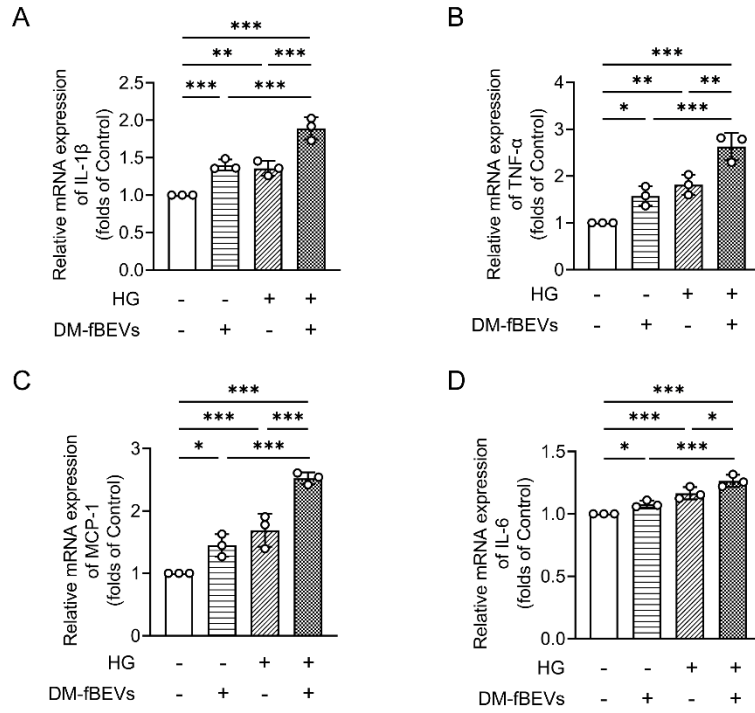


Figure S2. HK-2 cells were treated with high glucose (HG) (30 mmol/L), DM-fBEVs (5 μ g/mL) and HG (30 mmol/L) together with DM-fBEVs (5 μ g/mL) for 24 h. mRNA levels (n=3) of *IL-1 β* , *TNF- α* , *MCP-1* and *IL-6* were measured by qRT-PCR analysis.

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

Figure S3

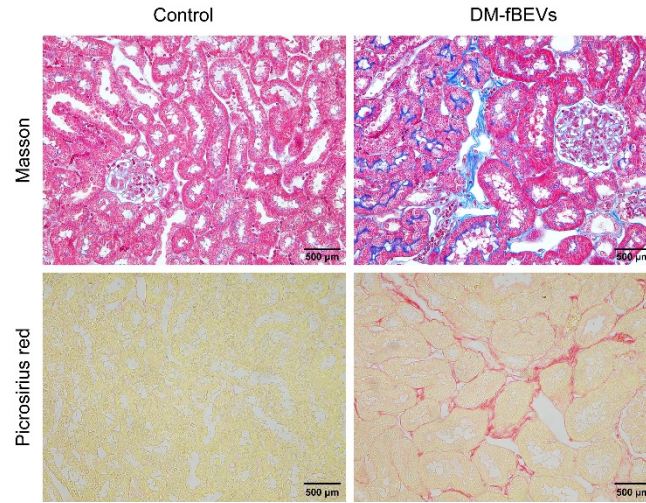


Figure S3. Tubulointerstitial pathological changes were detected by Masson staining and Picrosirius red staining. (Scale bar, 500 μm , original magnification $\times 400$).

Figure S4

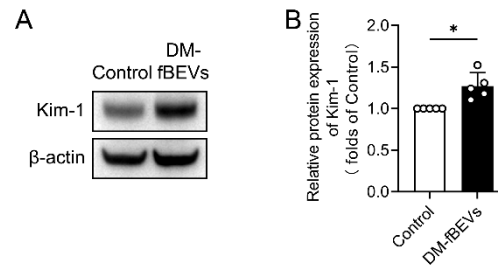


Figure S4. Protein expression levels of Kim-1 in the renal cortex of Control and DM-fBEVs gavage mice were detected by Western blot analysis (n=5). * $P < 0.05$.

Table S1. Primers for qRT-PCR

Gene	Forwards (5'-3')	Reverse (5'-3')
<i>Homo CASP4</i>	TCGGAAGGTACAGCAATCAT	TGCCAGGAAAGAGGTAGAAA
<i>Homo CASP1</i>	ACAGACAAGGGTGCTGAACAA	TCGGAATAACGGAGTCAATCA
<i>Homo IL-1β</i>	ATGATGGCTTATTACAGTGGCAA	GTCGGAGATTCGTAGCTGGA
<i>Homo TNF-α</i>	GGAAAGGACACCATGAGC	CCACGATCAGGAAGGAGA
<i>Homo MCP-1</i>	TGAAGCTCGCACTCTCG	GTGACTGGGGCATTGATT
<i>Homo IL-6</i>	ACTCACCTCTTCAGAACGAATTG	CCATCTTTGGAAGGTTTCAGGTTG
<i>Homo GAPDH</i>	GGTGTGAACCATGAGAAGTATGA	GAGTCCTTCCACGATACCAAAG