

SUPPLEMENTARY INFORMATION

**TNF- α -induced Inflammation Stimulates Apolipoprotein-A4 via
Activation of TNFR2 and NF- κ B Signaling in Kidney Tubular
Cells**

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Supplementary Figure

Figure S1

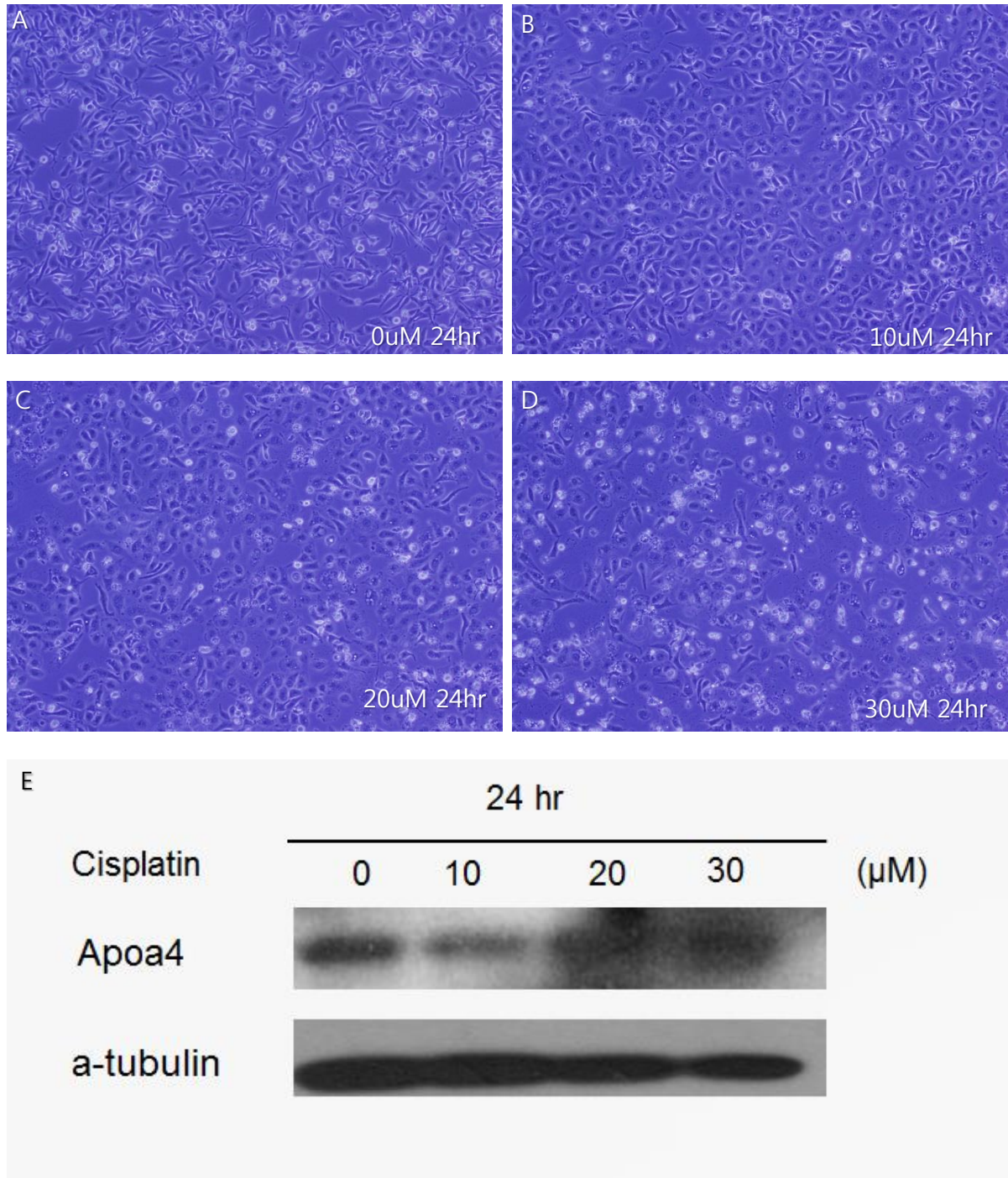


Figure S1. Cisplatin induced acute kidney injury model.

We treated HK-2 cells with the nephrotoxic cancer drug cisplatin. HK2 were treated with various concentrations of cisplatin, as indicated, for 24 hours. Microscopic findings showed decreased cell viability with increasing cisplatin concentration(A, B, C, D). There was no significant difference in expression of apo-A4 in western blot (E).

Figure S2

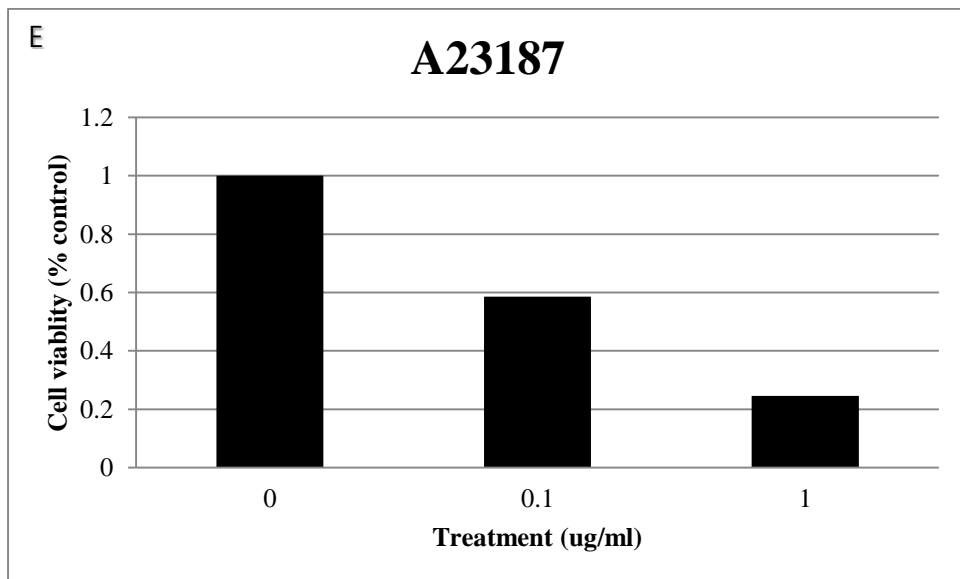
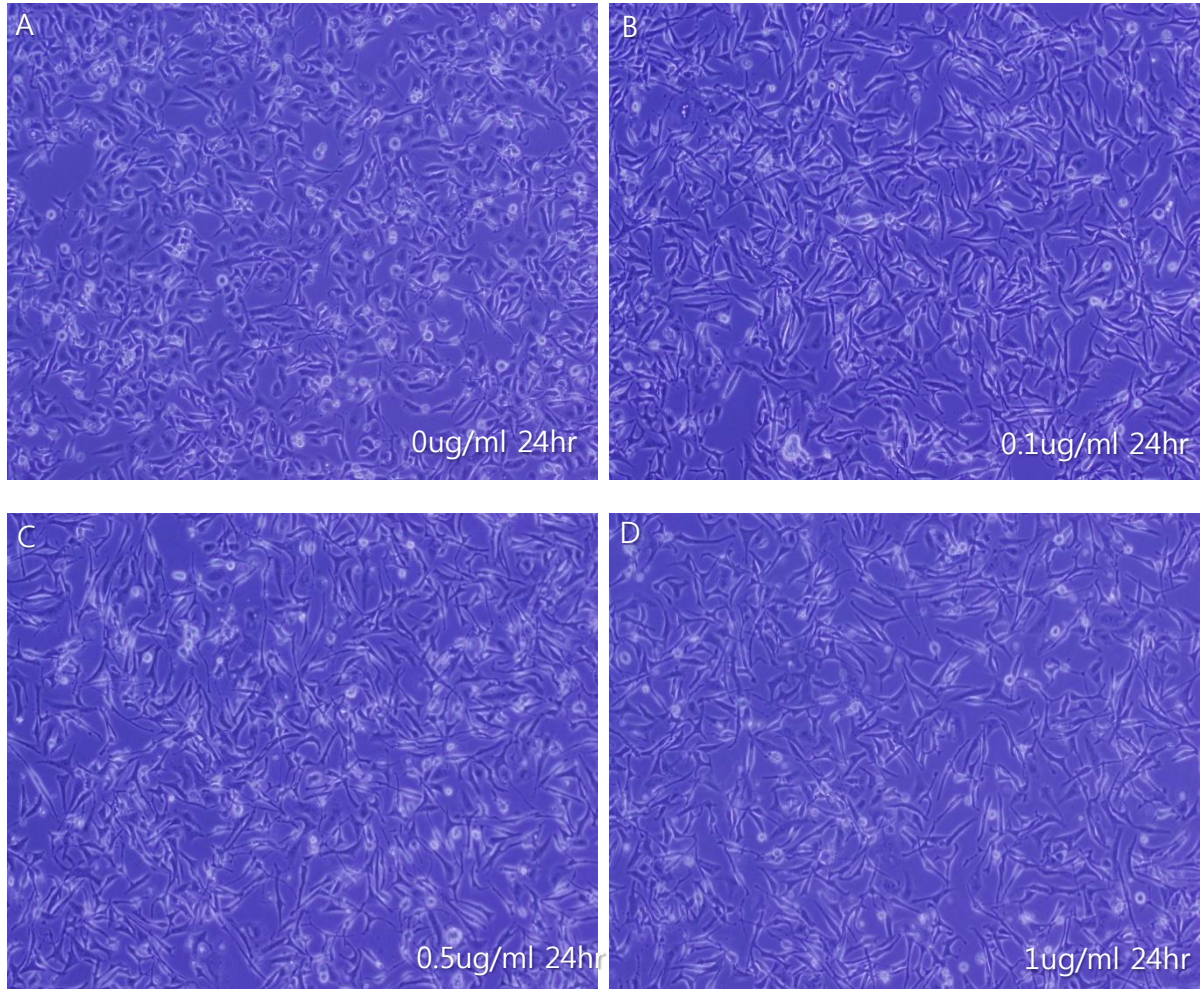


Figure S2. Calcium ionophore A23187 induced acute kidney injury model.

We treated HK-2 cells with the Calcium ionophore A23187. HK2 were treated with various concentrations of A23187 for 24 hours Microscopic findings showed decreased cell viability with increasing cisplatin concentration(A, B, C, D). Cell viability was decreased in diagram(E).

Figure S3

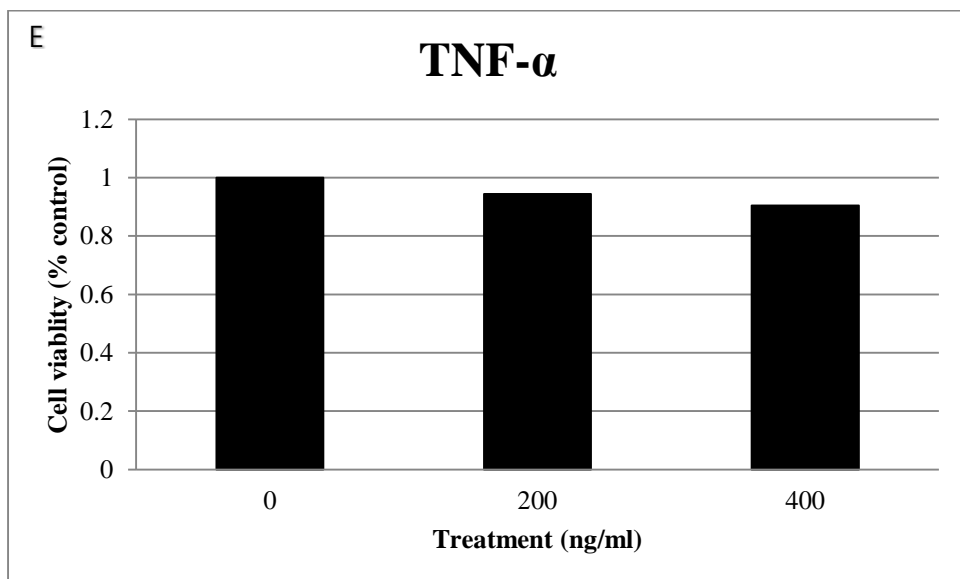
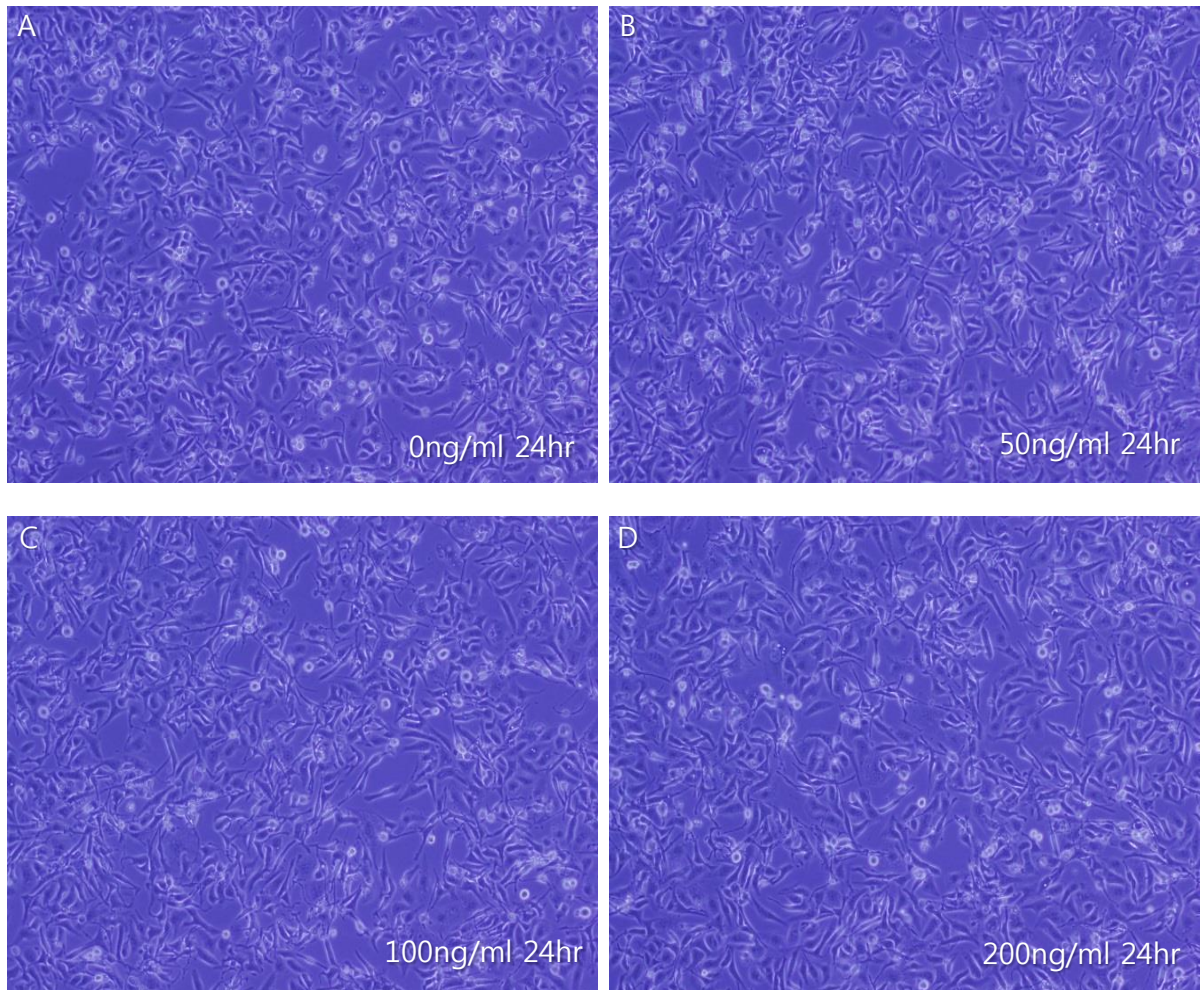
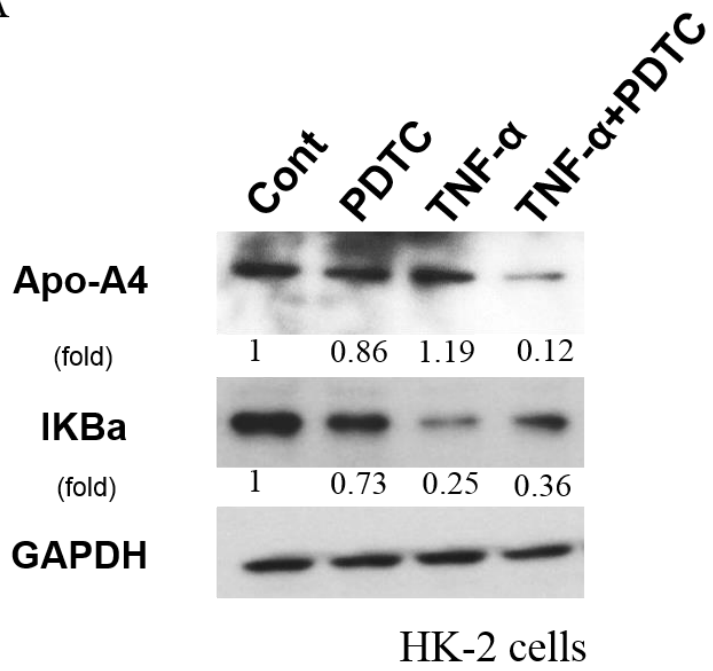


Figure S3. TNF- α induced acute kidney injury model.

We treated HK-2 cells with the TNF- α . Microscopic findings showed cell viability reduction was less than cisplatin and A23187(A, B, C, D). Cell viability was less decreased in diagram(E).

Figure S4

A



B

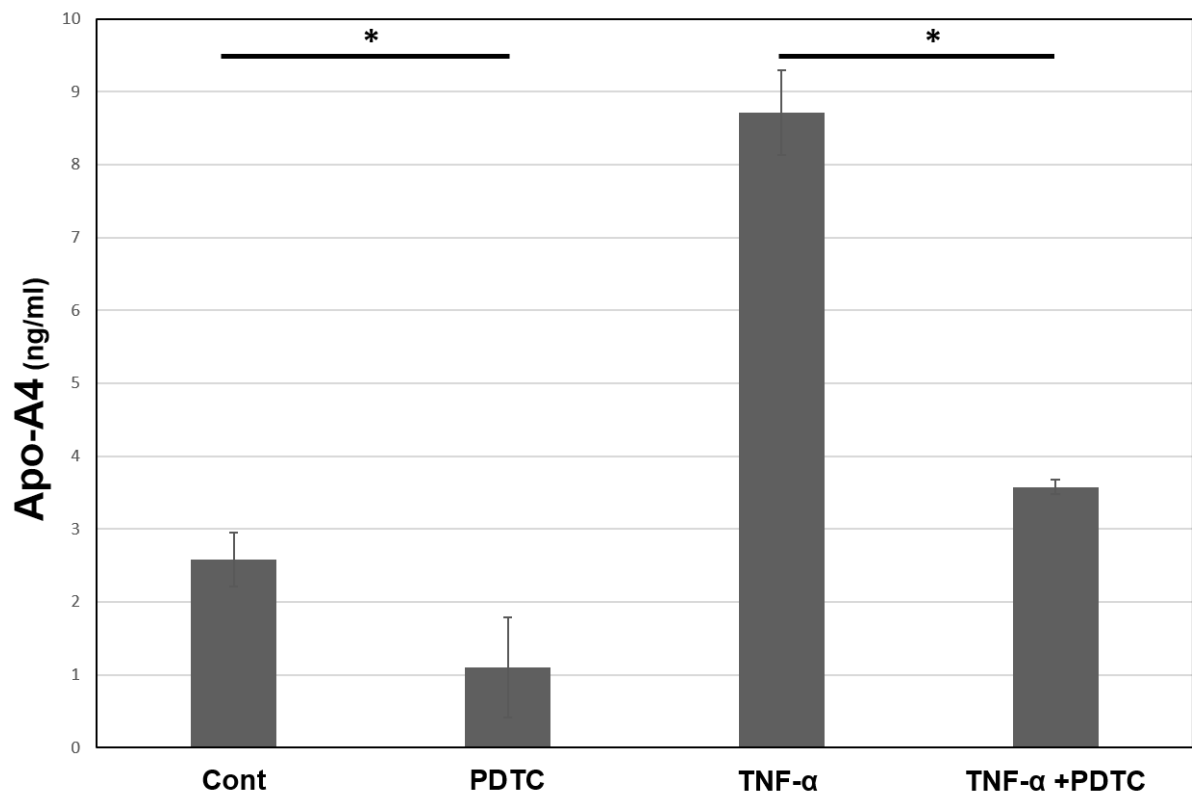


Figure S4. NF- κ B regulates apo-A4 expression

We treated HK-2 cells with the TNF- α and Pyrrolidine dithio-carbamate ammonium (PDTC). PDTC attenuated apo-A4 expression in western blot (Figure S4A). The expression of apo-A4 was reduced by PDTC(NF- κ B inhibition) treated group (Figure S4B). In addition, apo-A4 was increased in the group treated with TNF- α , but apo-A4 expression was decreased when PDTC was administered, statistically (* p <0.05).