

Supplementary Materials: PEDF Inhibits the Activation of NLRP3 Inflammasome in Hypoxia Cardiomyocytes through PEDF Receptor/Phospholipase A2

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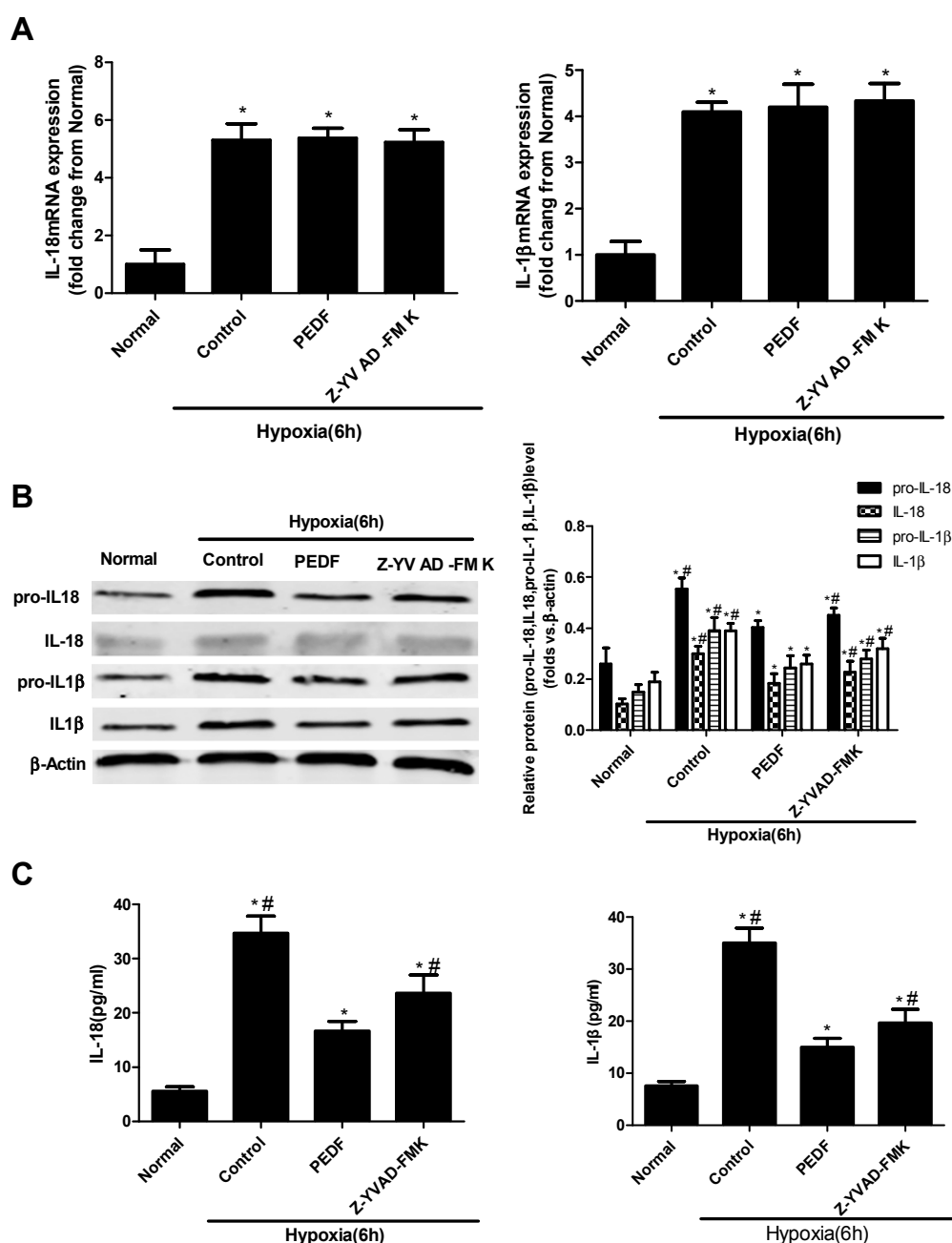


Figure S1. Effects of PEDF on the mRNA expression of IL-1β/18 and the protein levels of pro-IL18/1β and IL-1β/18. (A) IL-1β/18 mRNA expression was examined by real-time PCR. The results were expressed as the relative expression to β-actin and plotted as the ratio of the control group ($n = 4$; $* p < 0.05$ vs. the control group); (B) Representative Western blot analyses of pro-IL18/1β and IL-1β/18 expression ($n = 4$; $* p < 0.05$ vs. the control group; $\# p < 0.05$ vs. the PEDF group); (C) ELISA analyzed the concentrations of IL-1β/18 in the culture medium ($n = 4$; $* p < 0.05$ vs. the control group; $\# p < 0.05$ vs. the PEDF group). Data are expressed as the mean \pm SD.

PEDF inhibited the hypoxia-induced increase of pro-IL18/1 β and IL-1 β /18 protein levels and secreted levels. Real-time RT-PCR revealed that the expression of IL-1 β /18 mRNA was significantly increased in cardiomyocytes undergoing hypoxia. However, compared with the control group, treatment with PEDF and Z-YVAD-FMK had no effect on the mRNA expression of IL-1 β /18 (Figure S1A). Western blot analysis showed that protein levels of pro-IL18/1 β and IL-1 β /18 were significantly increased in cardiomyocytes. However, pro-IL18/1 β and IL-1 β /18 protein levels were significantly lower in the PEDF and Z-YVAD-FMK group compared with the control group (Figure S1B). Similar to the Western blot result, the secreted levels of IL-1 β and IL-18 in the cultured supernatant of neonatal cardiomyocytes were significantly lower in the PEDF and Z-YVAD-FMK group compared with the control group (Figure S1C).