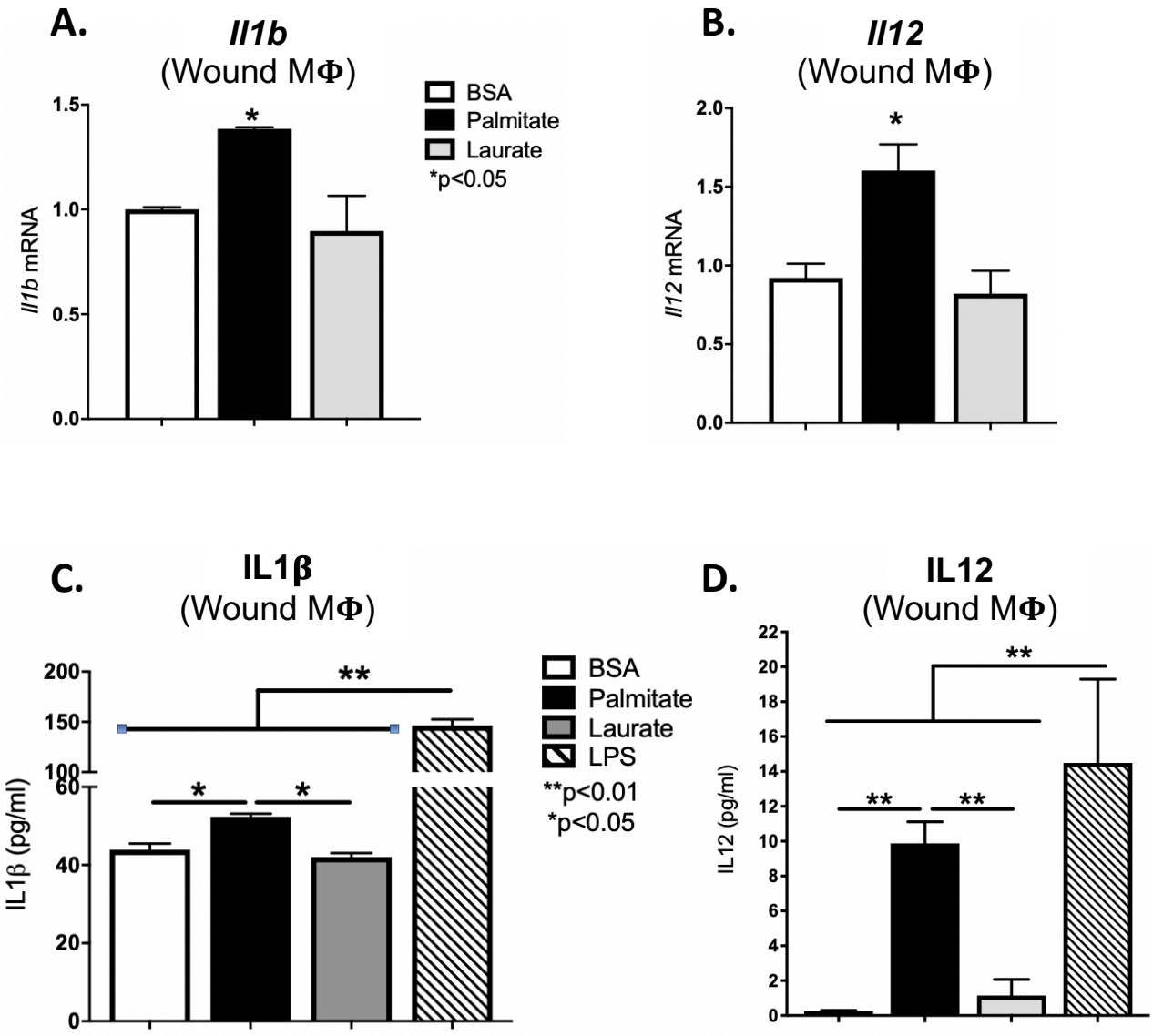
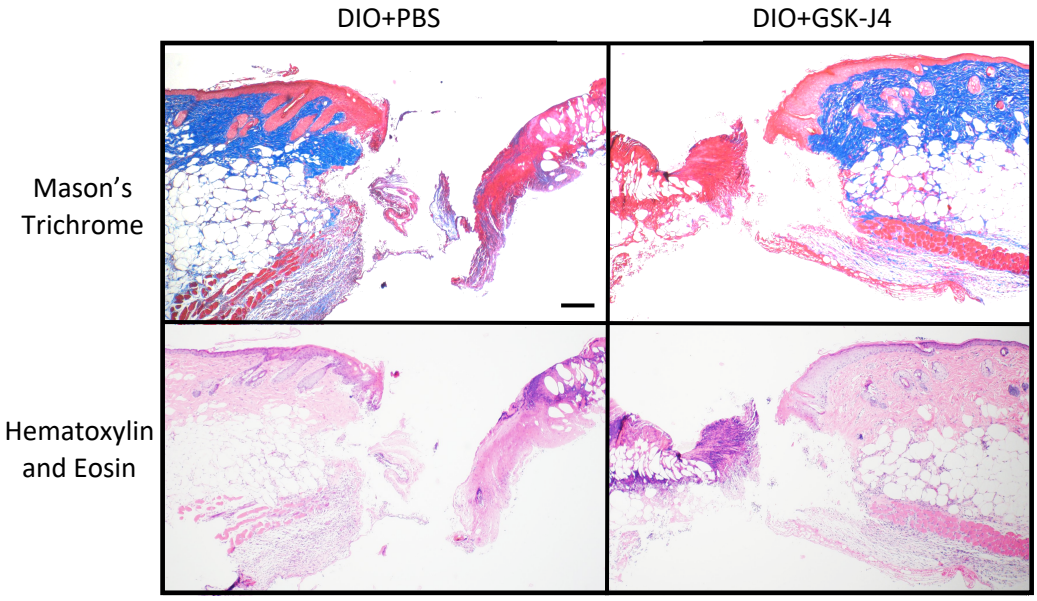
Supplemental Figure 1



Supplemental Figure 1. Palmitate induces proinflammatory phenotype in *in vivo* wound macrophages. **A, B.** Wound macrophages [CD11b$^+$(CD3$^-$, CD19$^-$, NK1.1$^-$, Ly6G$^-$)] were MACs isolated from control C57BL/6 on post-injury day 5 and stimulated with BSA, palmitate (200 μM), or Laurate (50 μM) for 24 hours. *Il1b* and *Il12* expression was quantified by qPCR and expressed as fold comparison to BSA treatment. (n =3 mice per group, data shown above is of a single representative experiment with experiment repeated two times with three technical replicates in each experiment). *p<0.05 by ANOVA followed by Newman–Keuls post hoc. **C, D.** Wound macrophages [CD11b$^+$(CD3$^-$, CD19$^-$, NK1.1$^-$, Ly6G$^-$)] were MACs isolated from control C57BL/6 on post-injury day 3 and stimulated with BSA, palmitate (200 μM), Laurate (50 μM), and LPS for 36 hours. IL1β and IL12 protein production was analyzed with ELISA (n =3 mice per group, data shown above is of a single representative experiment with experiment repeated two times with three technical replicates in each experiment). *p<0.05, **p<0.01 by ANOVA followed by Newman–Keuls post hoc. Data are presented as the mean±SEM.

Supplemental Figure 2



Supplemental Figure 2. Histological analysis of pharmacological inhibition of JMJD3 in diabetic wound healing. Wounds were harvested on day 3, paraffin embedded and sectioned. 5 μm sections were stained with hematoxylin and eosin and with Masson's Trichrome stain. Representative images are shown in 4X magnification. The black line represents 200 μm in length.

Supplemental Table I

Gene	Forward Primer	Reverse Primer
Il1b	5’ ACCTTTGTTCCGCACATC 3’	5’ GGGATTATTTCCCCCTGG 3’
Il12	5’ ACCCCGAAGTCATTTCCT 3’	5’ ACCCACTGTTCCTTCTGCT 3’