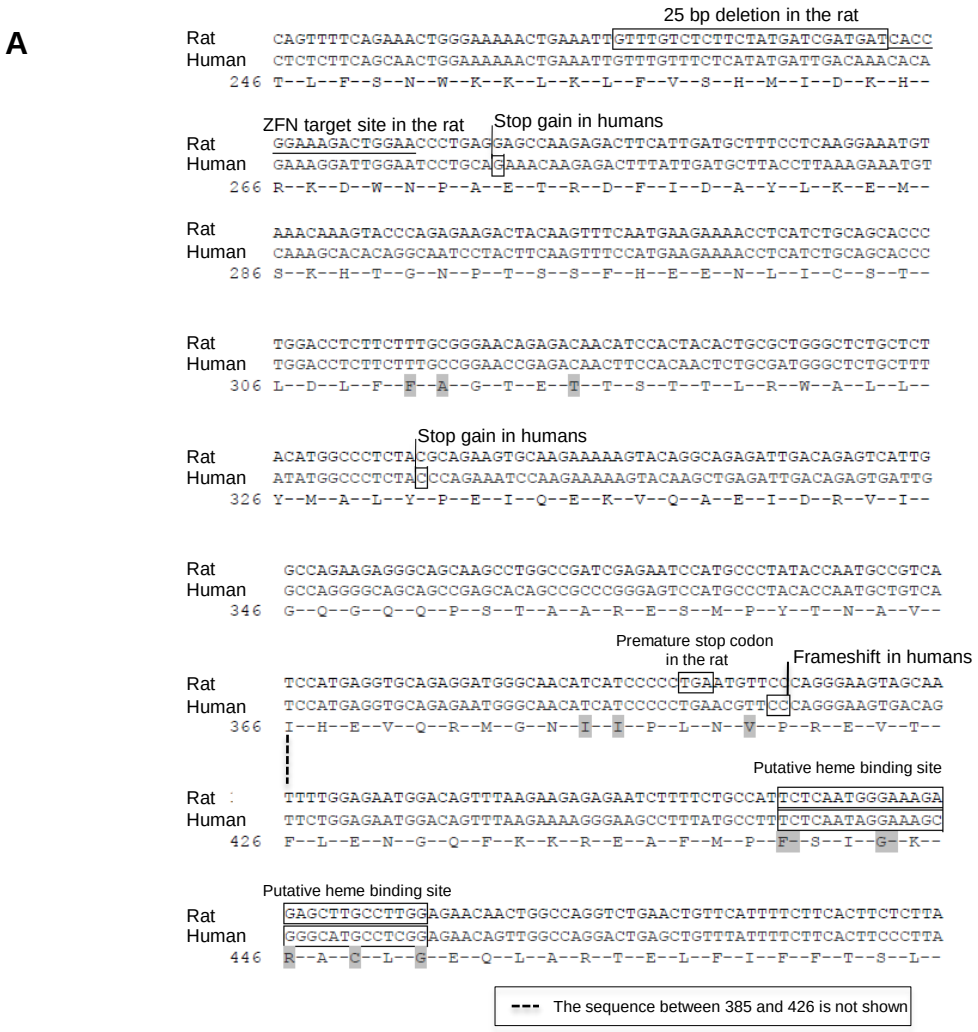
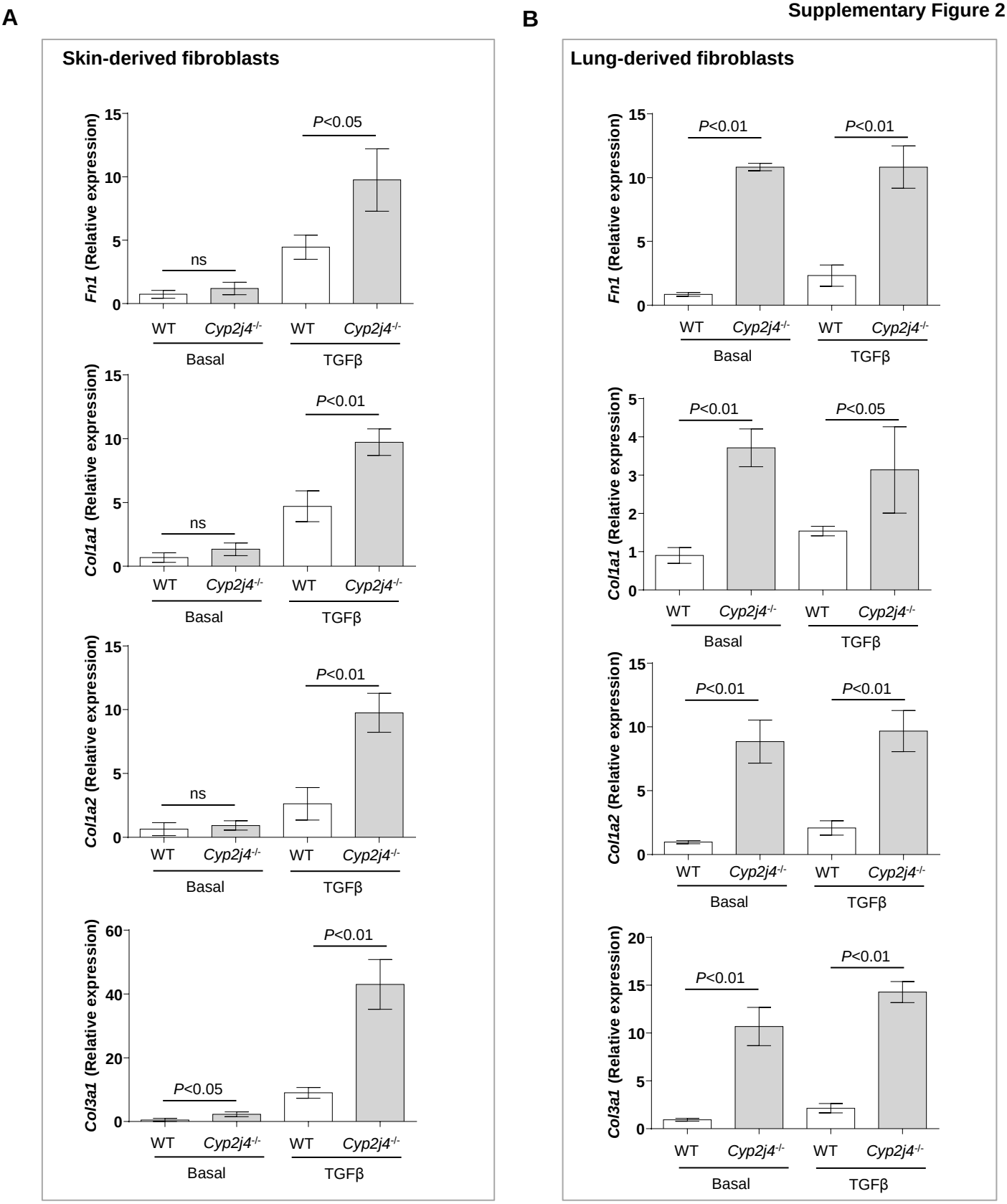
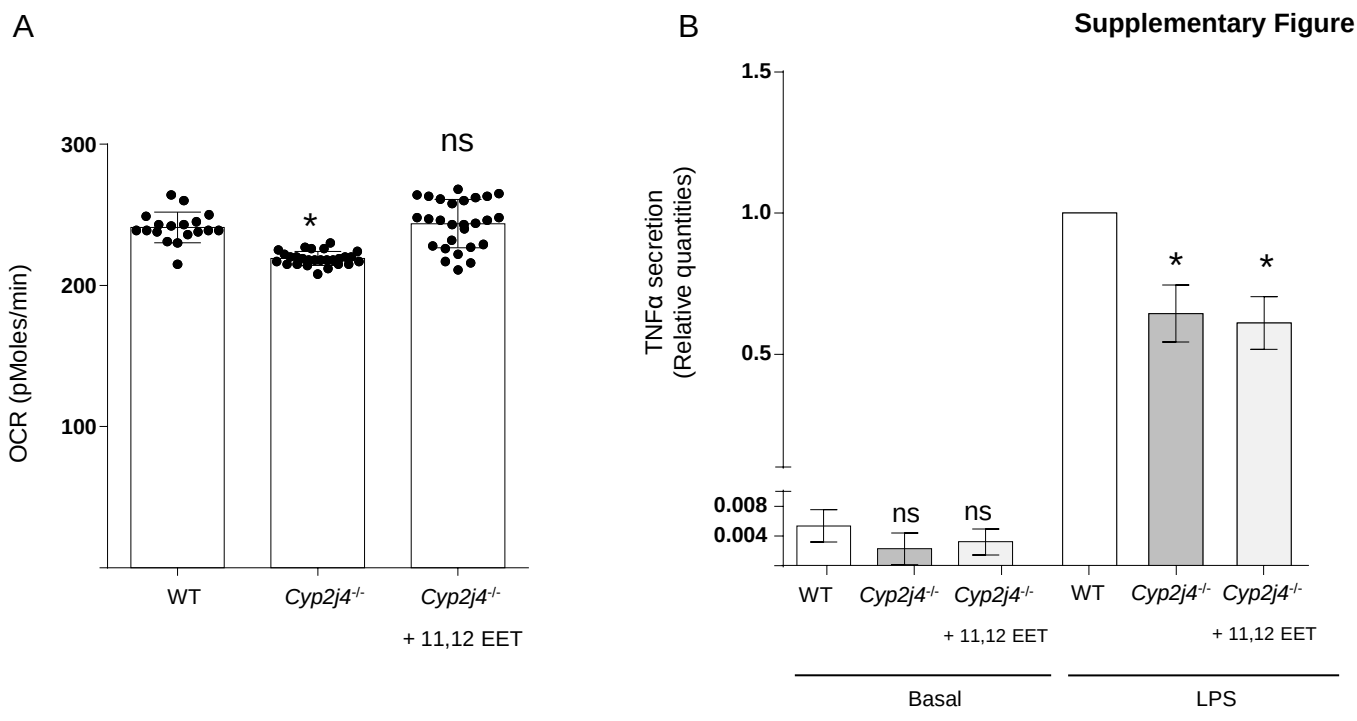


Supplementary Figure 1. **A**. The comparative illustration of rat *Cyp2j4* and its human orthologue *CYP2J2* cDNA sequence and its corresponding peptide sequence in humans. The 25 bp deletion in the rat causes a premature stop codon upstream the putative heme binding site (residues 441, 444, 446, 448, 450 highlighted in grey). The frameshift deletion affects also the active site cavity identified in human CYP2J2 with residues I127, F310, A311, T315, I375, I376 and V380 (highlighted in grey) that are in close proximity to heme, providing a restricted access of substrates through a narrow hydrophobic channel (Lafite et *al*. Biochemistry 2007 11;46(36)). Note that human *CYP2J2* presents stop/gain mutations (identified by 1000 Genomes), one of them being 143 bp upstream the premature rat stop codon. **B**. The amino acid sequence of wild-type (WT) and *Cyp2j4*$^{-/-}$ rat from 241-420 illustrating the frameshift caused at position 255. The frameshift sequence (shown in green colour) causes a stop codon at position 372.



Supplementary Figure 2. Expression levels of *Fn1*, *Col1a1*, *Col1a2*, *Col3a1* in primary fibroblasts derived from WT and *Cyp2j4*$^{-/-}$ skin (**A**) or lung (**B**) measured by qRT-PCR. Fibroblasts were cultured in either basal conditions or with addition of TGFβ (10ng/ml for 48 hours). Note the marked expression differences between basal lung-derived WT vs *Cyp2j4*$^{-/-}$ fibroblasts for all the tested transcripts. n=3 rats/strain were used in four replicates and the results are representative of two independent experiments.



Supplementary Figure 3. **A**. Oxygen consumption rate (OCR) in WT and *Cyp2j4*$^{-/-}$ BMDMs measured by extracellular flux analyser in WT and *Cyp2j4*$^{-/-}$ BMDMs in basal conditions (assay medium) and in conditions were 11,12 EETs (1µM) were added onto *Cyp2j4*$^{-/-}$ BMDMs. OCR measurements were taken every 10min following addition of either basal or +11,12 EETs media for a total duration of 3h (See experimental procedures for details). The experiment was repeated three times with at least 4 technical replicates each time. *, P<0.001 when compared with WT; ns, non-significant when compared with WT. B. TNFα levels were quantified by sandwich ELISA in WT and *Cyp2j4*$^{-/-}$ BMDMs in basal (untreated) and lipopolysaccharide (LPS, 100ng/ml, 12h) treated samples. *, *P*<0.05 compared to WT (LPS) by nonparametric Wilcoxon signed-rank test; ns, non-significant when compared to WT (basal). The experiment was repeated four times using 4 biological replicates in duplicate.