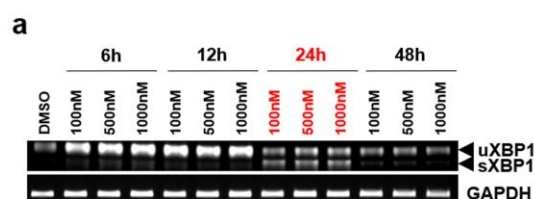


Supplementary Fig. 1

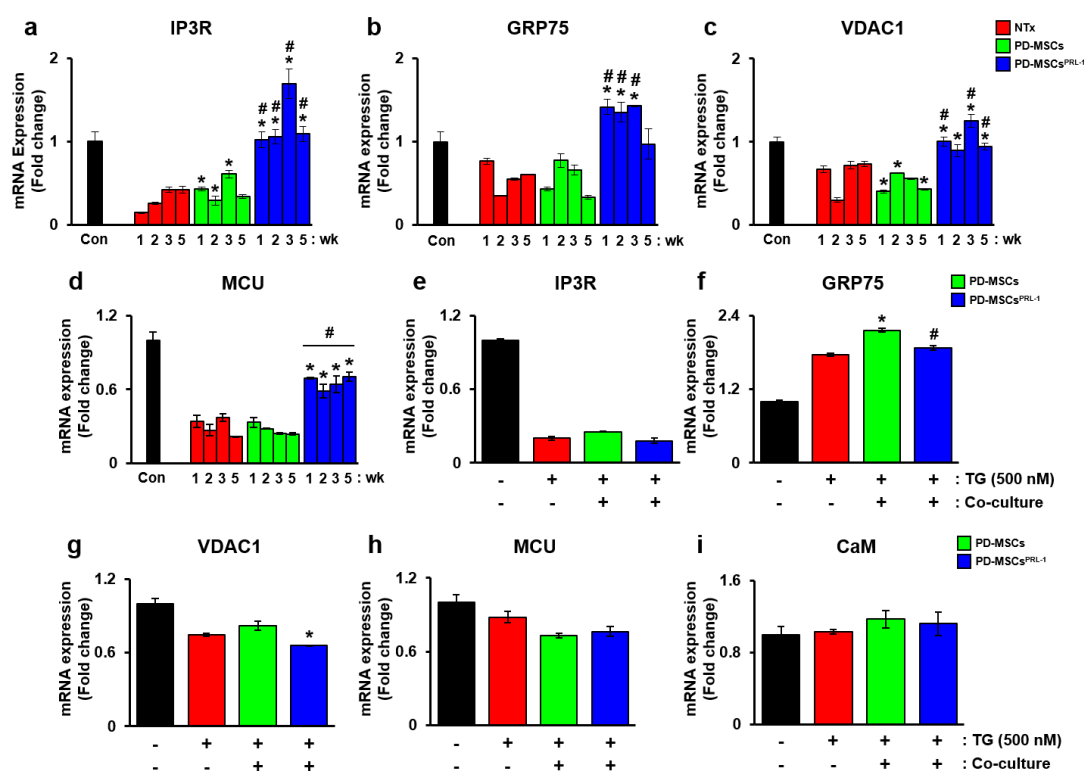
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Supplementary Fig. 1 Optimal concentration of thapsigargin (TG) in WB-F344. **a** TG was treated with each concentration (100, 500, and 1000 nM) for 6, 12, 24, and 48 h in WB-F344. XBP1 splice form was transformed upon TG 24 h treatment. GAPDH was used as loading control. GAPDH, glyceraldehyde-3-phosphate dehydrogenase; TG, thapsigargin

Supplementary Fig. 2

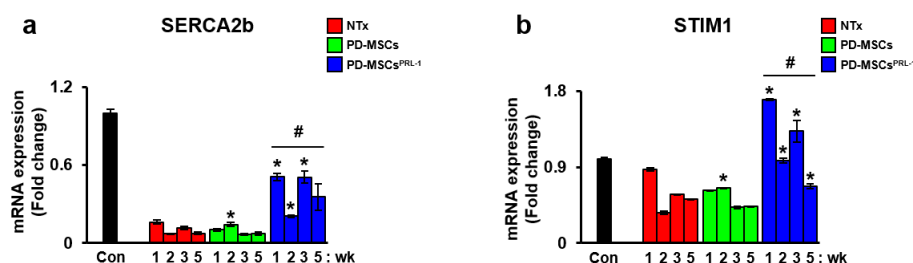
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Supplementary Fig. 2 PD-MSCs^{PRL-1} regulated calcium channels in a rat BDL and hepatocyte treated with TG. **a-d** mRNA levels of calcium channels (e.g., IP3R, GRP75, VDAC1, and MCU) in a rat model with BDL. **e-i** mRNA levels of calcium channels (e.g., IP3R, GRP75, VDAC1, MCU, and CaM) exposed to TG (500 nM) for 24 h in WB-F344 by qRT-PCR. Data from each group are shown as the means $\pm$ SD and were assessed using Student's t-test. * $p < 0.05$ vs. NTx, # $p < 0.05$ vs. PD-MSCs. BDL, bile duct ligation; CaM, calmodulin; GRP75, glucose regulated protein 75; IP3R, inositol trisphosphate receptor; MCU, mitochondria calcium uniporter; NTx, nontransplantation; PRL-1, phosphatase of regenerating liver-1; TG, thapsigargin; VDAC1, voltage dependent anion channel 1

Supplementary Fig. 3

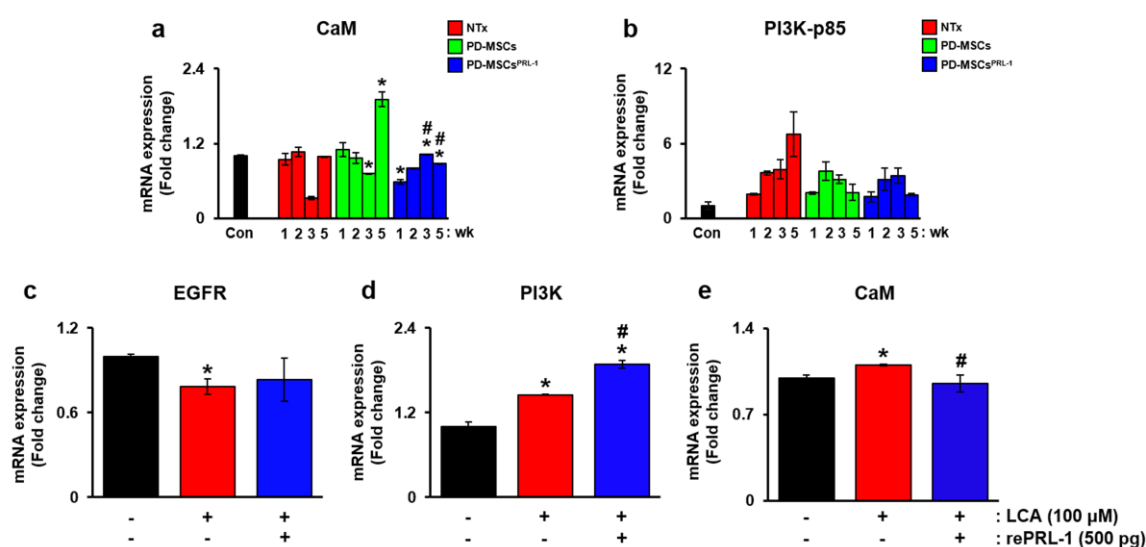
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Supplementary Fig. 3 Increased expression of SERCA2b and STIM1 in PD-MSCs^{PRL-1} groups in a rat model with BDL. **a, b** mRNA levels of ER-specific Ca²⁺ channel factor SERCA2b and Ca²⁺ sensor factor STIM1 in a rat model with BDL. Data from each group are shown as the means $\pm$ SD and were assessed using Student's t-test. * $p < 0.05$ vs. NTx, # $p < 0.05$ vs. PD-MSCs. BDL, bile duct ligation; NTx, nontransplantation; SERCA2b, sarco/endoplasmic reticulum Ca²⁺ ATPase; PRL-1, phosphatase of regenerating liver-1; STIM1, stromal interaction molecule 1

Supplementary Fig. 4

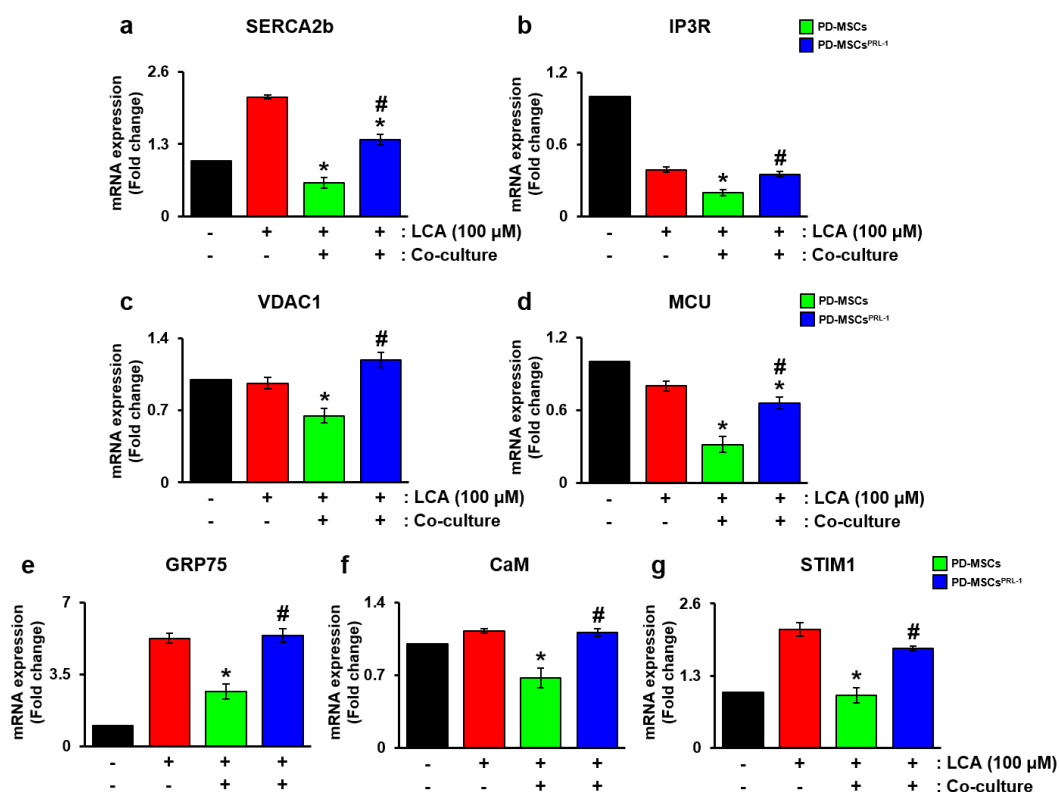
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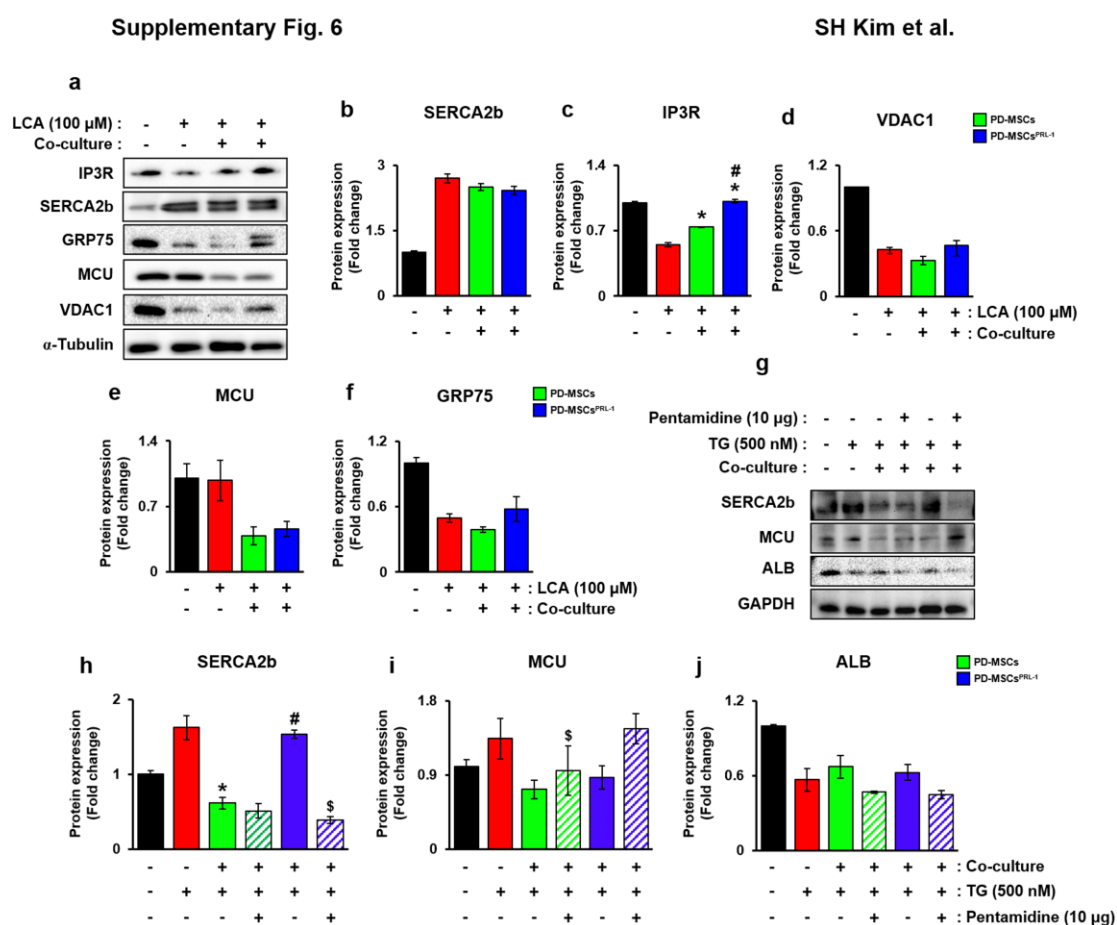
Supplementary Fig. 4 PRL-1 regulates EGFR-PI3K-CaM calcium signaling in a BDL-injured rat liver and rat hepatocyte treated LCA. **a** mRNA levels of CaM and **b** PI3K-p85 in a rat model with BDL. **c-e** mRNA levels of intracellular calcium signaling (e.g., EGFR, PI3K, and CaM) induced LCA (100 μM) and treated recombinant PRL-1 (rePRL-1; 500 pg) in WB-F344. Data from each group are shown as the means ± SD and were assessed using Student's t-test. *p < 0.05 vs. NTx, #p < 0.05 vs. PD-MSCs. BDL, bile duct ligation; CaM, calmodulin; EGFR, epidermal growth factor receptor; NTx, nontransplantation; PI3K, phosphatidylinositol-3-kinase

Supplementary Fig. 5

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Supplementary Fig. 5 PD-MSCs^{PRL-1} modulated calcium channels in a rat hepatocyte treated with LCA by qRT-PCR. **a-g** mRNA levels related to intracellular calcium transport channels factors (e.g., SERCA2b, IP3R, VDAC1, MCU, GRP75, CaM, and STIM1) induced LCA (100 μM) and co-cultivation with naïve PD-MSCs or PD-MSCs^{PRL-1} in WB-F344. Data from each group are shown as the means ± SD and were assessed using Student's t-test. *p < 0.05 vs. LCA (100 μM), #p < 0.05 vs. PD-MSCs. CaM, calmodulin; GRP75, glucose regulated protein 75; IP3R, inositol trisphosphate receptor; LCA, lithocholic acid; MCU, mitochondria calcium uniporter; PRL-1, phosphatase of regenerating liver-1; SERCA2b, sarco/endoplasmic reticulum Ca²⁺ ATPase; STIM1, stromal interaction molecule 1; VDAC1, voltage dependent anion channel 1

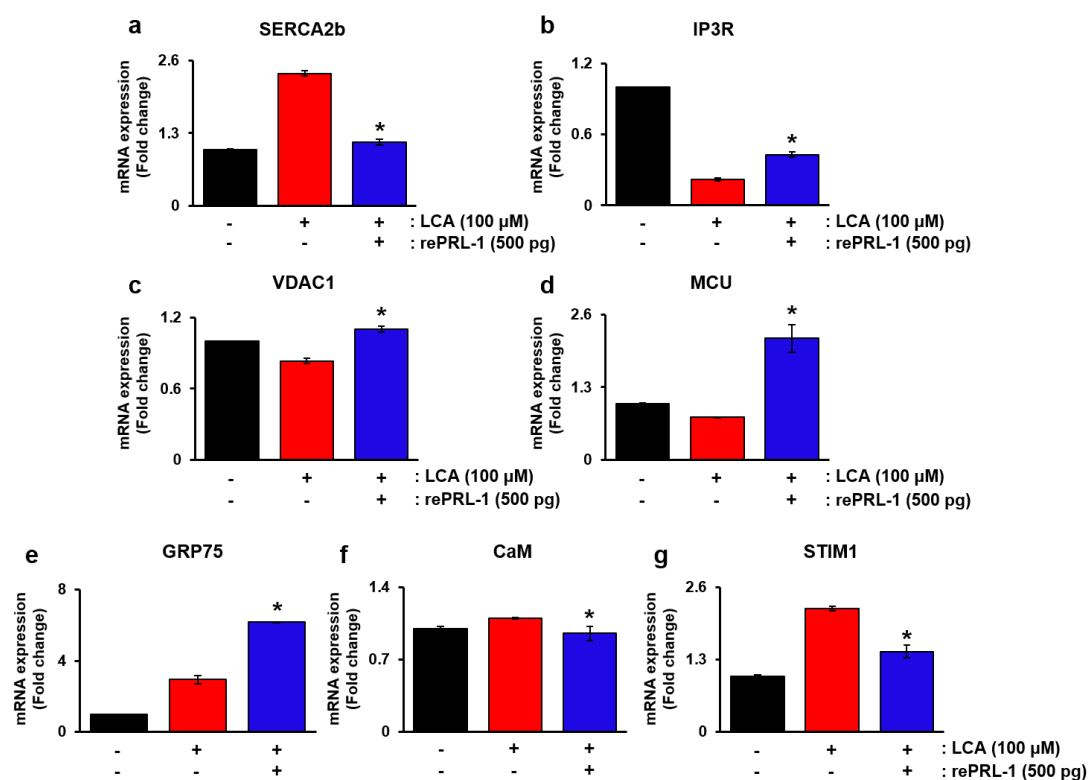


Supplementary Fig. 6 PD-MSCs^{PRL-1} regulated calcium channels in a rat hepatocyte treated with LCA and TG by western blotting. **a** Protein expression and **b-f** their intensities of intracellular calcium transport channels factors (e.g., SERCA2b, IP3R, VDAC1, MCU, and GRP75) induced LCA (100 μ M) and co-cultivation with naïve PD-MSCs or PD-MSCs^{PRL-1} in WB-F344. **g** Protein expression and **h-j** their intensities of intracellular calcium transport channels factors (e.g., SERCA2b, and MCU) and liver regeneration factor induced LCA (100 μ M) and co-cultivation with naïve PD-MSCs or PD-MSCs^{PRL-1} and Pentamidine (10 μ g) treated in rat primary hepatocytes. GAPDH and α -tubulin was used as loading control. Data from each group are shown as the means $\pm$ SD and assessed using Student's t-test. * $p < 0.05$ vs. LCA (100 μ M), # $p < 0.05$ vs. PD-MSCs, \$ $p < 0.05$ vs. PD-MSCs or PD-MSCs^{PRL-1}. ALB, Albumin; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; GRP75, glucose regulated protein 75; IP3R, inositol trisphosphate receptor; LCA, lithocholic acid; MCU, mitochondria calcium uniporter; PRL-1, phosphatase of regenerating liver-1; SERCA2b,

sarco/endoplasmic reticulum Ca^{2+} ATPase; TG, Thapsigargin; VDAC1, voltage dependent anion channel 1

Supplementary Fig. 7

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Supplementary Fig. 7 Recombinant PRL-1 increased calcium channels in a rat hepatocyte treated with LCA. **a-g** mRNA levels of intracellular calcium transport channels factors (e.g., SERCA2b, IP3R, VDAC1, MCU, GRP75, CaM, and STIM1) induced LCA (100 μM) and treated recombinant PRL-1 (rePRL-1; 500 pg) in WB-F344. Data from each group are shown as the means ± SD and assessed using Student's t-test. *p < 0.05 vs. LCA (100 μM). CaM, calmodulin; GRP75, glucose regulated protein 75; IP3R, inositol trisphosphate receptor; LCA, lithocholic acid; MCU, mitochondria calcium uniporter; PRL-1, phosphatase of regenerating liver-1; SERCA2b, sarco/endoplasmic reticulum Ca²⁺ ATPase; VDAC1, voltage dependent anion channel 1