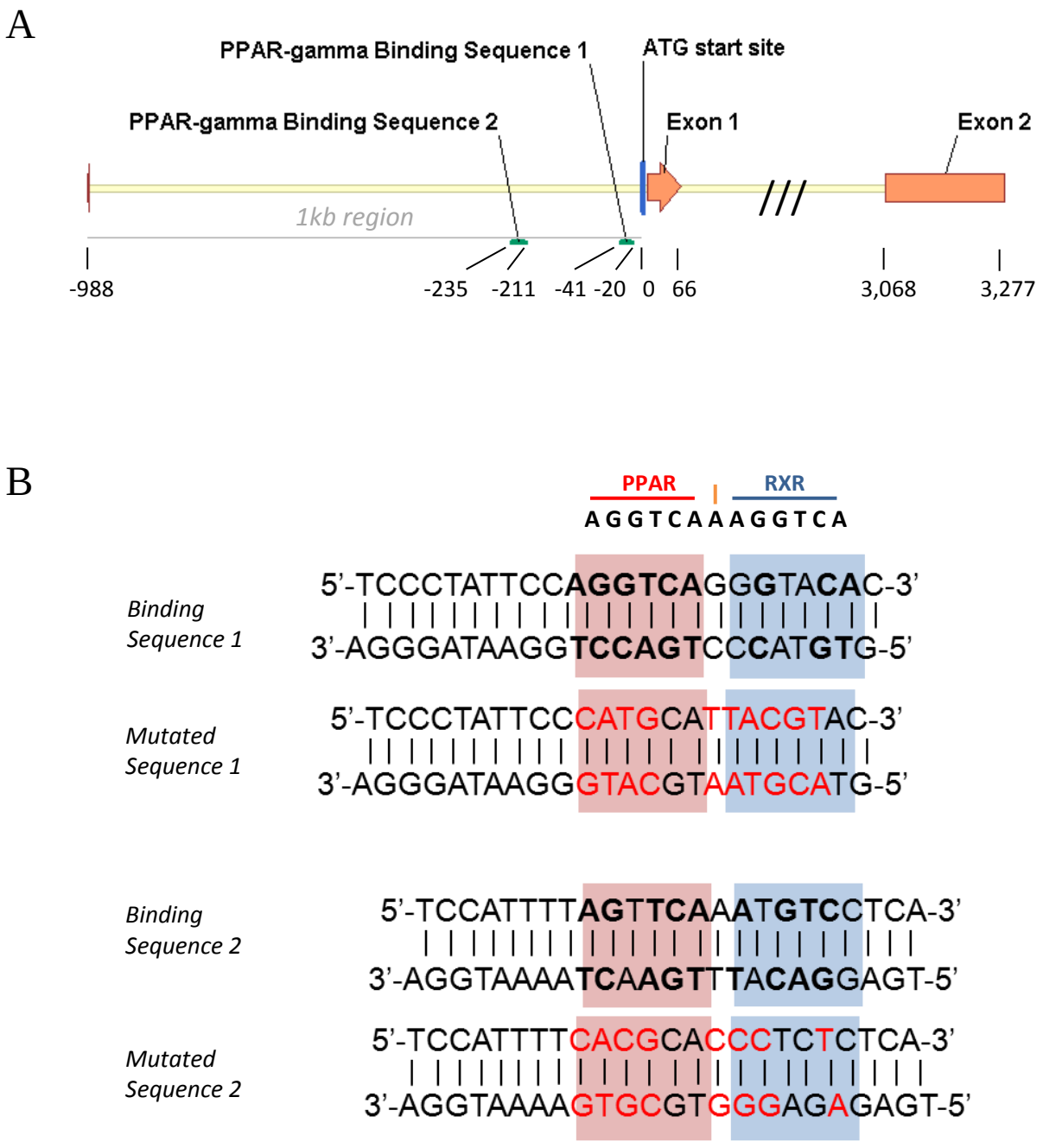
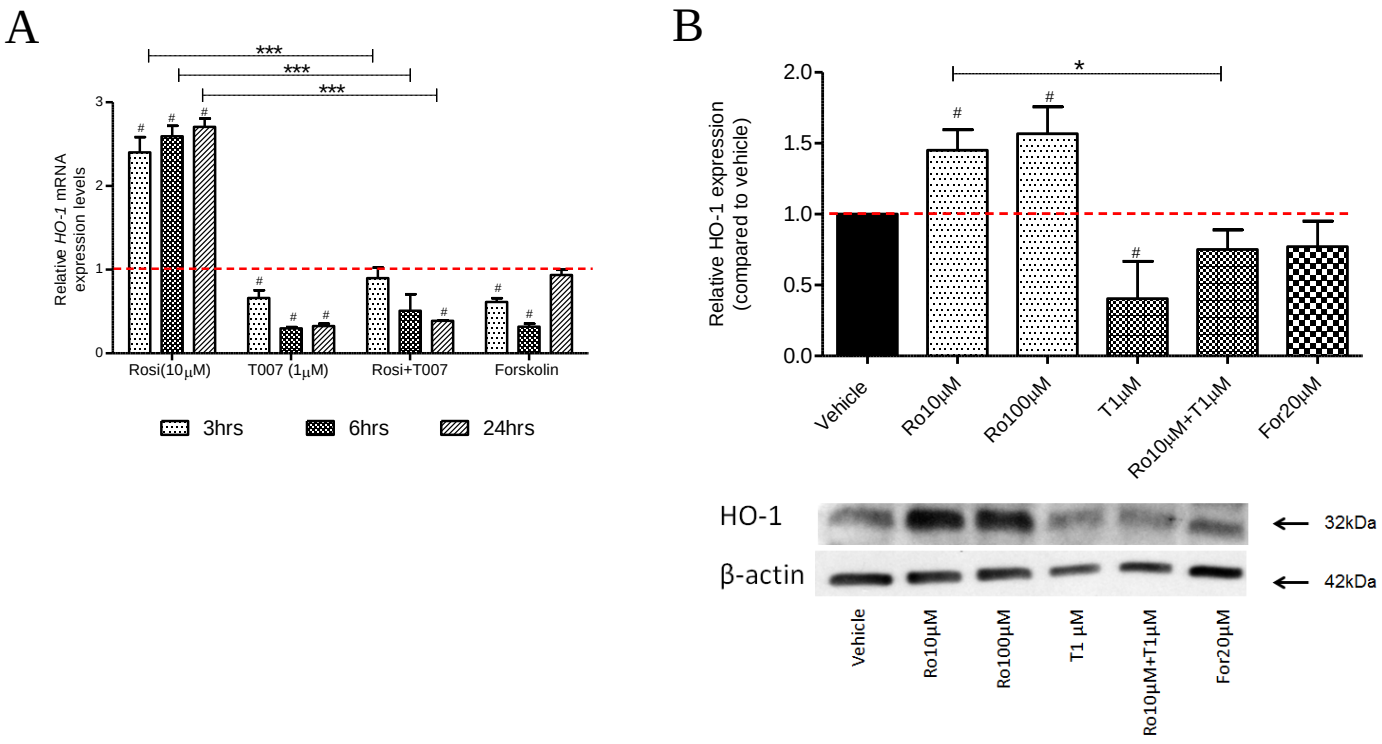
Supplementary Table 1. Antibodies and dilutions.

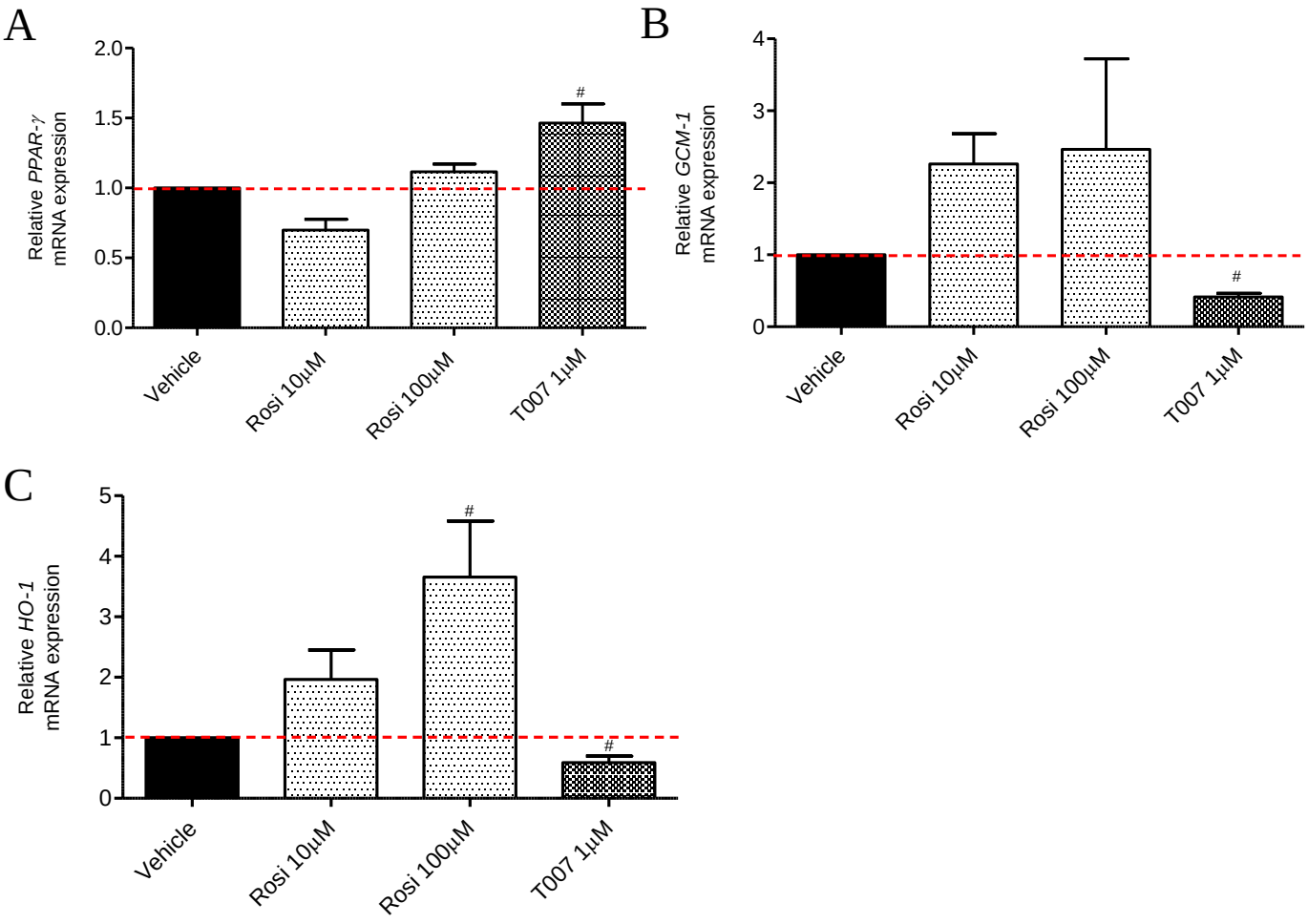
Antigen	Origin	Size (kDa)	Dilution	Supplier
HO-1	Rabbit	32	1:500 (WB)	Enzo Life Sciences Burlington, ON, Canada
PPAR-γ	Rabbit	54-57	1:100 (WB) 1:100 (F-IHC)	SantaCruz Biotechnology Dallas, TX, USA
E-Cadherin	Mouse	110	1:1000 (WB) 1:100 (F-IHC)	Abcam Toronto, ON, Canada
Lamin B	Mouse	67	1:500 (WB)	SantaCruz Biotechnology Dallas, TX, USA
α-Tubulin	Mouse	50	1:2000 (WB)	Sigma Oakville, ON, Canada
β-actin	Mouse	42	1:4000 (WB)	Abcam Toronto, ON, Canada



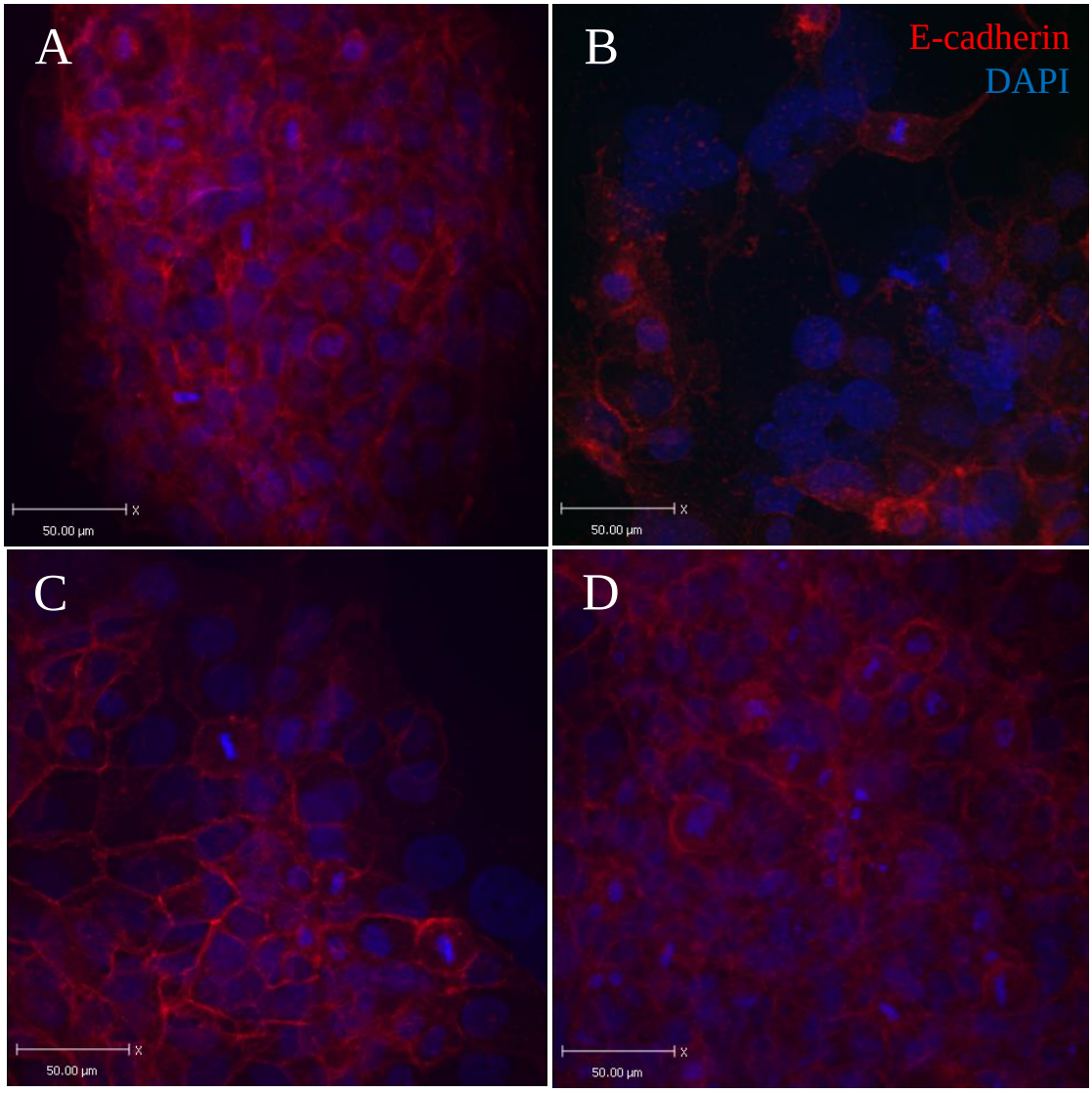
Supplementary Figure 1. PPAR-γ response element (PPRE) identification and binding and mutated sequence design. (A) Gene map of the 1kb upstream region of the human *GCM-1*. The 1kb region upstream of the human *GCM-1* gene was analyzed for PPAR-γ binding sites. Two putative PPREs were found within 250 base pairs from the transcription start site (sequence 1 and sequence 2). The gene map shows only exons 1 and 2 of *GCM-1* gene (not to scale). **(B)** Duplex oligonucleotide sequences for binding and mutated sequence 1 (top) and binding and mutated sequence 2 (bottom). Nucleotides conserved between the consensus PPRE (shown above for comparison) and our identified binding sites are bolded. Mutated nucleotides are shown in red. PPAR-γ binding site is highlighted in pink, RXR-α binding site shown in blue.



Supplementary Figure 2. Induction of PPAR-γ activity positively correlates with HO-1 mRNA and protein expression in the BeWo cell line. (A) Compiled *HO-1* mRNA expression levels overtime in response to rosiglitazone, T0070907, their combination and forskolin treatment. **(B)** Cellular HO-1 protein expression at 24 hours correlates with mRNA expression profile (representative blot shown in lower panel). All treatments are compared to their respective vehicle controls (set as 1, red dashed line). Values are represented as mean ± SEM; $^{\#}p<0.05$ *vs* vehicle control; *p<0.05; ***p<0.001 (n=3-4). For, forskolin; Ro/rosi, rosiglitazone; T/T007, T0070907.



Supplementary Figure 3. Response of isolated human primary cytotrophoblast cells to PPAR-γ activity-modulating drugs. mRNA expression profiles of *PPAR-γ* **(A)**, *GCM-1* **(B)** and *HO-1* **(C)** in primary human cytotrophoblast cells are analogous to those observed in BeWo cells (t=24 hours). All treatments are compared to their respective vehicle controls (set as 1, red dashed line). Values are represented as mean ± SEM; $^{\#}p<0.05$ *vs* vehicle control (n=3-4). Rosi, rosiglitazone; T007, T0070907.



Supplementary Figure 4. Effect of PPAR-γ modulation on BeWo cell fusion. Cell fusion was assessed at 48 hours post-treatment. Cells were treated with forskolin (**B**) as a positive cell-fusion control, rosiglitazone (**C**), and T0070907 (**D**) and compared to vehicle control (**A**). E-cadherin (red) was used as a cell surface marker; DAPI (blue) was used as the nuclear stain (200X magnification).