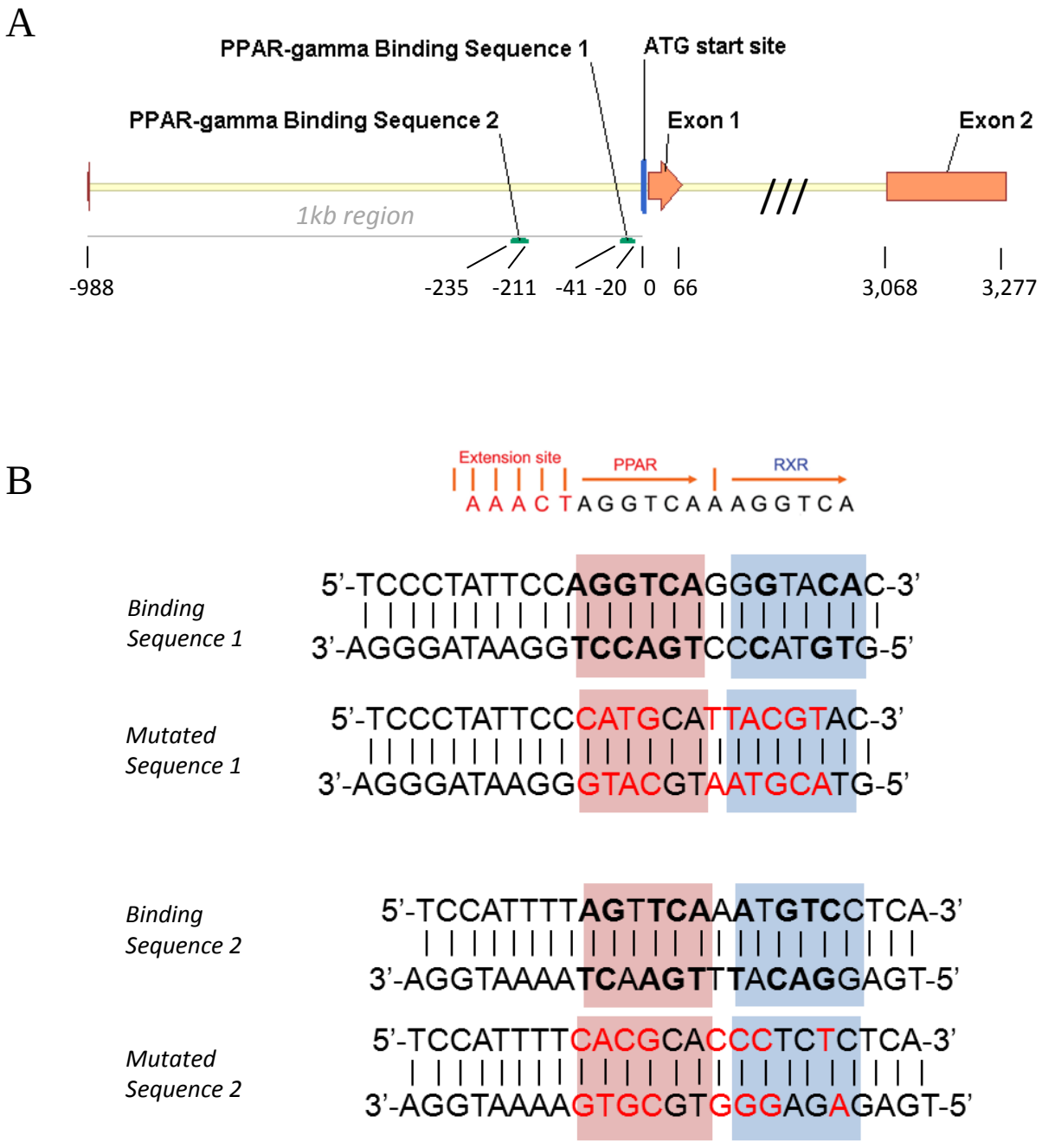
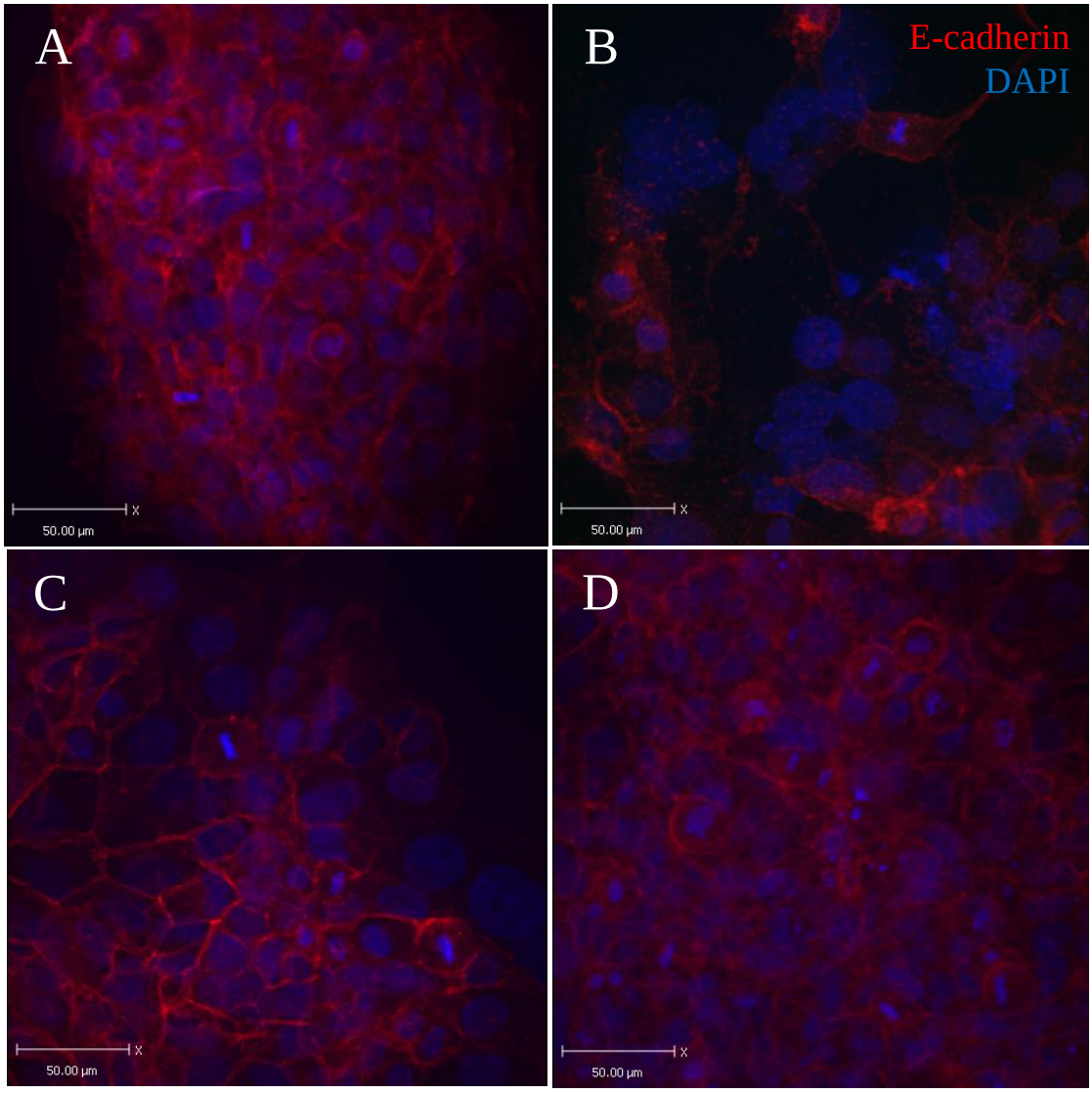
Supplementary Table 1. Antibodies and dilutions.

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



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. 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).