Supplementary figure legends

Figure S1. Western Immunoblots demonstrating PAR-2 expression in coeliac ganglia neurons. Immunoblots for PAR-2 (52 KD). Lung was used as positive control and liver used as a negative control. Tissue of origin is showed below each lane.

Figure S2. Effect of thrombin on I_{Ca} in coeliac ganglia neurons. (A) Left, a representative trace of thrombin (30 nM) effect on I_{Ca}. (B) time course of thrombin (30 nM) effect on I_{Ca}. (C) Summary of I_{Ca} inhibition by thrombin.

Figure S3. Effect of BT and LR-NH₂ on repetitive ction action potentials (APs) in coeliac ganglia neurons. (A) Representative traces of repetitive AP firing before (left panel) and during application of 30 nM BT (Right panel) in current-clamp mode. Action potentials were elicited by positive current (100–200 pA) injection for 300 ms via the patch pipette. (B) Representative traces of repetitive AP firing before (left panel) and during presence of 100 μ M LR-NH₂ (right panel) in current-clamp mode.

Figure S4. Effect of thrombin on repetitive firing of a firing of action potentials (APs) in CG neurons. (A) representative traces of thrombin (30 nM) effect on repetitive APs evoked by current injection in current-clamp mode. Action potentials were elicited by positive current (100–200 pA) injection for 300 ms via the patch pipette.

Figure S5. Contribution of N-type (ω -CgTx-sensitive) and L-type (nifendipine-sensitive) currents to the total I_{Ca} . (A) a representative trace of the effects induced by application of nifedpine (1 μ M) (left) and ω -CgTx (1 μ M) (right) on I_{Ca} . (B) Time course of the effects induced by application of nifedpine (1 μ M) and ω -CgTx (1 μ M) on I_{Ca} . Trace 1, 2 and 3, 4 in (A) panel represent the traces recorded at the corresponding time indicated in right panel respectively. (C) Summary of I_{Ca} inhibition by nifedpine and ω -CgTx.

Figure S6. Effect of nifedpine and ω -CgTx on I_{Ca} and PAR-2 agonist-induced I_{Ca} inhibition. (A) Left, a representative trace of the effects induced by consecutive application of nifedpine (1 μ M) and trypsin (30 nM) on I_{Ca}. Right, time course of effects induced by consecutive application of nifedpine (1 μ M) and trypsin (30 nM) on I_{Ca}. Trace 1, 2 and 3 in left panel represent the traces recorded at the corresponding time indicated in right panel respectively. (B) Left, a representative trace of the effects induced by consecutive application of nifedpine (1 μM), ω-CgTx (1 μM) and trypsin (30 nM) on I_{Ca}. Right, time course of effects induced by consecutive application of nifedpine $(1 \ \mu M)$, ω -CgTx $(1 \ \mu M)$ and trypsin $(30 \ nM)$ on I_{Ca}. Trace 1, 2 and 3, 4 in left panel represent the traces recorded at the corresponding time indicated in right panel respectively. (C) Summary of I_{Ca} inhibition by trypsin in the absence and presence of nifedpine and ω -CgTx. (D) Left, a representative trace of the effects induced by consecutive application of nifedpine (1 μ M) and SL-NH₂ (100 μ M) on I_{Ca}. *Right*, time course of effects induced by consecutive application of nifedpine (1 μ M) and SL-NH₂ (100 μ M) on I_{Ca}. Trace 1, 2 and 3 in left panel represent the traces recorded at the corresponding time indicated in right panel respectively. ((E) Left, a representative trace of the effects induced by consecutive application of nifedpine (1 μ M), ω -CgTx (1 μ M) and SL-NH₂ (100 μ M) on I_{Ca}. *Right*, time course of effects induced by consecutive application of nifedpine (1)

 μ M), ω -CgTx (1 μ M) and SL-NH₂ (100 μ M) on I_{Ca}. Trace 1, 2 and 3, 4 in left panel represent the traces recorded at the corresponding time indicated in right panel respectively. (F) Summary of I_{Ca} inhibition by SL-NH₂ in the absence and presence of nifedpine and ω -CgTx.





Liver Lung CG







sup2





В



sup3

Control





sup4





sup.5





D





Supplementary Fig. S1







В



Supplementary Fig. S3

400

Control





Supplementary Fig. S4







В

2

C

Control

O nifedipine



С



Supplementary Fig. S5









