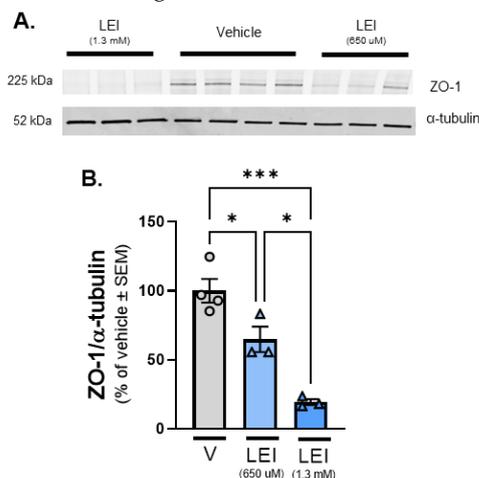


Suppl. Fig 1. PKC inhibitor, G06983 did not influence the effect of LEI-106 on VE-cadherin. bEnd.3 cells were treated with protein kinase c inhibitor, G06983 (100 nM) 30 min before the application of LEI-106 (650 uM or 1.03 mM), then subjected to Western to detect VE-cadherin. (A) Representative image showing the expression of VE-cadherin along with α -tubulin as loading control. (B) The pretreatment of PKC inhibitor did not influence the effect of LEI-106 on VE-cadherin main band (LEI-106-650 uM vs. LEI-106-650 uM+ G06983: $p=0.9990$, LEI-106-1.3 mM vs. LEI-106-1.3 mM+ G06983: $p=0.9320$, as assessed by one-way ANOVA with Tukey post-test, $F(5,18)=4.986$). All data presented as % of vehicle-treated \pm SEM ($n=4$ /condition). ns=non-significant. (C) The PKC inhibitor did not have a significant impact on the fragmentation of VE-cadherin caused by LEI-106 in bEnd.3 cells (LEI-106-650 uM vs. LEI-106-650 uM+ G06983: $p=0.9999$, LEI-106-1.3 mM vs. LEI-106-1.3 mM+ G06983: $p=0.9548$, as assessed by one-way ANOVA with Tukey post-test, $F(5,18)=21.88$). All data presented as % of vehicle-treated \pm SEM ($n=4$ /condition). ns=non-significant.



Suppl. Fig 2. DAGL α inhibitor, LEI-106 reduced the detection of ZO-1 in bEnd.3 cells. bEnd.3 cells were treated with LEI-106 (650 uM or 1.03 mM) or vehicle for 15 min, then subjected to Western to detect ZO-1. (A) Representative image showing the expression of ZO-1 along with α -tubulin as loading control. (B) The blockade of DAGL α significantly decreased the detection of ZO-1, compared to vehicle control (vehicle vs. LEI-106-650 uM: $p=0.034$, vehicle vs. LEI-106-1.3 mM: $p=0.0004$, LEI-106-650 uM vs. LEI-106-1.3 mM: $p=0.0142$ as assessed by one-way ANOVA with Tukey post-test, $F(2,7)=27.41$). All data presented as % of vehicle-treated \pm SEM ($n=3-4$ /condition). * $p<0.05$, *** $p<0.001$.