*Supplementary Materials*

# Dysregulation of Gap Junction Function and Cytokine Production in Response to Non-Genotoxic Polycyclic Aromatic Hydrocarbons in an In Vitro Lung Cell Model

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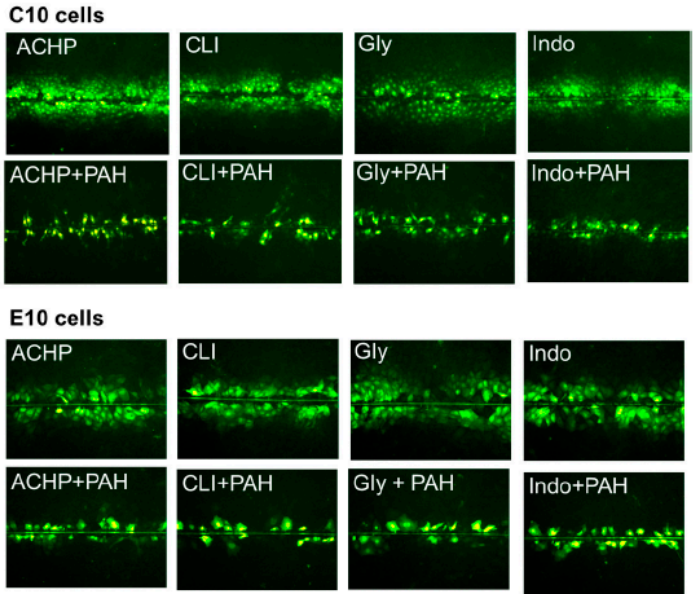


**Figure S1.** Representative images for the other inhibitor combinations with 4 h PAH treatment in C10 and E10 cells. C10 and E10 cells were treated with the binary LMW PAH mixture (40 mM at 4 h; 1:1 ratio of 1-methylanthracene and fluoranthene) for 4 h following 1 h pre-incubation with these inhibitors (ACHP, 1 mM; CLI-095, 3 mM; glyburide, 50 mM; indomethacin, 1 mM). Representative images of C10 and E10 cells following the SL/DT assay used to quantify the gap junction activity in these cells in response to the LMW PAHs and ACHP, CLI-095, or indomethacin combinations. Experiments were repeated 3 times; $n$ = 3 per treatment per experiment. Gly, glyburide; Indo, indomethacin. The control and PAH alone treatments are found in Figure 3.

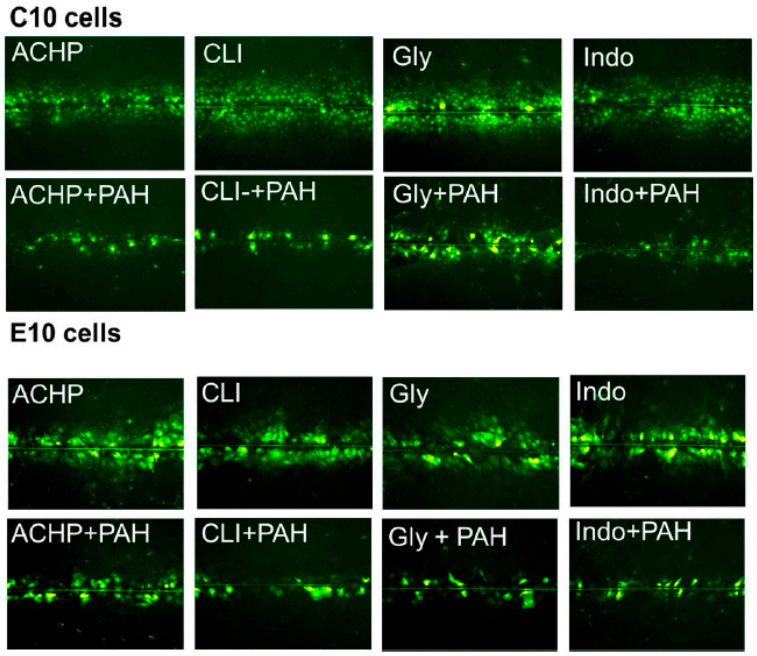


**Figure S2.** Representative images for the other inhibitor combinations with 24 h PAH treatment in C10 and E10 cells. C10 and E10 cells were treated with the binary LMW PAH mixture (15 mM at 24 h; 1:1 ratio of 1-methylanthraceneand fluoranthene) following a 1 h pre-incubation with these inhibitors (ACHP, 1 mM; CLI-095, 3 mM; glyburide, 50 mM; indomethacin, 1 mM). Representative images of C10 and E10 cells following the SL/DT assay used to quantify the gap junction activity in these cells in response to the LMW PAHs and all inhibitor combinations, except parthenolide. Experiments were repeated 3 times; $n$ = 3 per treatment per experiment. Gly, glyburide; Indo, indomethacin. The control and PAH alone treatments are found in Figure 4.

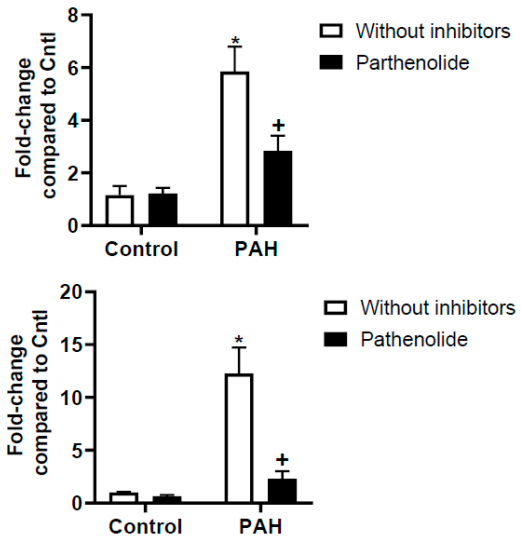


**Figure S3.** *Tnf* transcript expressionin C10 and E10 cells is inhibited by parthenolide in the presence of PAH mixture treatment. C10 and E10 cells were treated for 4 h with binary LMW PAH mixture (40 mM; 1:1 ratio of 1-methylanthracene and fluoranthene) following a 1 h pre-incubation with parthenolide (10 mM). qRTPCR was then performed for TNF, normalized to 18S followed by comparison to control treated cells (DMSO). Experiments were repeated twice with $n$ = 3 per experiment. * $p < 0.05$ for treatment compared to control; $^{+}$ $p < 0.05$ for PTL + PAH compared to PAH treatment.

**Table S1.** Primer sequences for qRTPCR analysis *.

| Name | Primer Sequence | | Genbank Accession/NCBI |
|---|---|---|---|
| Il1β | Forward (5′→3′) | CAA CCA ACA AGT GAT ATT CTC CAT G | M15131.1 |
| | Reverse (5′→3′) | GAT CCA CAC TCT CCA GCT GCA | |
| NALP3 | Forward (5′→3′) | CTC TAT CAA GGA CAG GAA CG | Nm_145827.3 |
| | Reverse (5′→3′) | TAG GAT GGT TTT CCC GAT GC | |
| TLR4 | Forward (5′→3′) | ATT GAA GAC AAG GCA TGG CAT | AF095353 |
| | Reverse (5′→3′) | GTG AGC CAC ATT GAG TTT CTT | |

* Kc, Cox-2, Il6, Mcp-1, 18S primers are published in Osgood et al., 2017 [37]; Tnf primers are published in Osgood et al, 2013 [36].