Supplementary Figure 1: Effects of choline and KYNA on LPS-induced PGE2 levels. Shown are the effects of LPS (A), choline (B), nicotinic ACh receptor antagonists (C), KYNA (D), choline + KYNA (E) on PGE2 levels. \*p<0.05, \*\*p<0.01, \*\*\*p<0.001 vs. control; †††p<0.001 vs. LPS group; ‡‡‡p*<*0.001 vs. LPS + choline, one-way ANOVA with post-hoc Tukey-Kramer multiple comparison test or Student’s t-test, n=4. MEC, mecamylamine; MLA, methyllycaconitine; KYNA, kynurenic acid.

Supplementary Figure 2: Effects of choline and KYNA on intracellular calcium levels. Shown are the effects of LPS (A), choline (B), nicotinic receptor antagonists (C), KYNA (D), choline + KYNA (E) on intracellular calcium levels, [Ca2+]i. \*p<0.05,\*\*p<0.01 vs. control; †p<0.05, ††p<0.01 vs. LPS group, one-way ANOVA with post-hoc Tukey-Kramer multiple comparison test or Student’s t-test, n=5. MEC, mecamylamine; MLA, methyllycaconitine; KYNA, kynurenic acid.

Supplementary Figure 3: Effects of choline and KYNA on LPS-induced TNFα levels. Shown are the effects of LPS, choline, selective or non-selective nAChR antagonists (MLA and MEC), KYNA, choline + KYNA on TNFα protein levels (by ELISA). \*\*\*p<0.001 vs. control; ††p<0.01, †††p<0.001 vs. LPS group; ‡p<0.05, ‡‡p<0.01, ‡‡‡p<0.001 vs. LPS + choline. One-way ANOVA with post-hoc Tukey-Kramer multiple comparison test or Student’s t-test, n=5. MEC, mecamylamine; MLA, methyllycaconitine; KYNA, kynurenic acid.

Supplementary Figure 4: Effects of choline and KYNA on LPS-induced NF-κB p65 protein expressions. Shown are the effects of LPS, choline, KYNA, choline + KYNA on NF-κB protein expression. \*p<0.05; \*\*p<0.01; one-way ANOVA with post-hoc Tukey-Kramer multiple comparison test or Student’s t-test, n=3. CH, choline; KYNA, kynurenic acid.

Supplementary Figure 5: Kynurenic acid (KYNA) potentiates the anti-inflammatory effects of choline (CH) as well as showing dual anti-inflammatory role both by decreasing calcium (Ca2+) influx and NF-κB levels through alpha 7 nicotinic acetylcholine receptors (α7nAChR) and suppressing the lipopolysaccharide (LPS)-induced prostaglandin (PG) E2 synthesis (graphical abstract).