SUPPLEMENTARY INFORMATION –

S1. Demographic characteristics of groups.

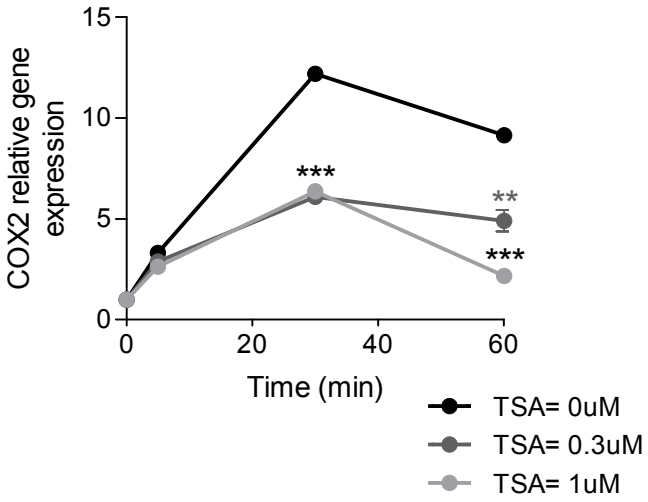
Group	Control	MT
n	21	19
Age	50.38±8.96	49.89±11.18
Race	95.23% Caucasian	84.21% Caucasian
Gender	12 F. 9 M	11 F. 8 M
BMI	23.55±3.76	24.16±3.51

Standard deviations for age and BMI variables are indicated.
BMI, body mass index; F, females; M, males.

S2. COX2 gene expression in MT2 cells is inhibited by HDACi TSA.

HDAC downregulation might lead to a decrease in HDAC activity, which we could not assess due to biological sample limitations. To analyze the potential mechanistic link between HDAC activity and the decrease in *COX2* expression in human blood cells, we explored the *in vitro* effect of the HDACi trichostatin A (TSA) on the MT2 human lymphocyte-derived cell line. HDAC inhibitors are extensively used to reduce HDAC activity in cellular and animal models and have been successfully used to treat several inflammatory diseases, such as inflammatory bowel disease and arthritis (Halili, 2009; Shakespear, 2011; Akimova, 2012).

We induced inflammation in MT2 cells with human recombinant TNF-α in the absence or simultaneous presence of two different concentrations of TSA. We observed that *COX2* gene expression was increased by TNF-α treatment but the peak of COX2 was significantly lower in the presence of TSA (Fig. 3 H). There was a significant interaction between the effects of TSA and time on *COX2* expression ($F(6, 17) = 51.112$, $p<0.001$; two-way ANOVA). Main effects of TSA were observed at 30 and 60 min after stimulation with TNF-α ($F(2, 3)= 182.918$, $p=0.001$; $F(2, 3) = 129.809$, $p=0.001$, respectively; one-way ANOVA). Tukey post-hoc comparisons indicated that TSA significantly lowered the expression of *COX2* at both 30 and 60 minutes after stimulation (TSA=0.3uM: $p=0.001$ at 30 min and $p=0.012$ at 60 min; TSA=1uM: $p=0.001$ both at 30 and 60 min). These data show that HDAC modulation of *COX2* levels is a mechanism that operates in human lymphocytes and indicate that the decrease in HDAC gene expression is likely to contribute to lower *COX2* gene and protein expression in the mindfulness meditation group.



COX2 gene expression in MT2 cells treated with human recombinant TNF-α (10ng/mL; 0-60 min) at different concentrations of TSA (0; 0.3μM; 1μM). TNF-α and TSA were added simultaneously to the cells. P-values from Tukey's Posthocs are indicated as *: $p< 0.05$, **: $p<0.01$; *** $p<0.001$. All error bars indicate SEM.

METHODS

COX2 gene expression was analyzed in MT2 cells (a T-cell line established from cord lymphocytes that had been cocultivated with leukemia cells; Miyoshi, 1981) treated with human recombinant TNF-α (Peprotech; 10ng/mL; 0, 5, 30 and 60 min). Each time was run in triplicate and the complete time-course was carried out at three different concentrations (0; 0,3uM; 1uM) of the HDACi TSA (Sigma-Aldrich) which was added to the cells at the same time as the TNF-α (time 0).

MT2 cells were cultured in RPMI 1640 with L-glutamine containing 10% heat-inactivated FBS and 1% penicillin/streptomycine. One million cells were seeded per well in 6-well plates. Two independent experiments were performed in which each condition was run by duplicates. TSA and time effects on COX2 expression was assessed by two-way ANOVA. One-way ANOVA and the corresponding Tukey post-hoc comparisons between the three different TSA concentrations were performed. COX2 relative gene expression was log-transformed in order to normalize the variable. P-values under 0.05 were considered statistically significant.

REFERENCES

-Miyoshi, I., Kubonishi, I., Yoshimoto, S., Akagi, T., Ohtsuki, Y., Shiraishi, Y., Nagata, K. and Hinuma, Y., 1981. Type C virus particles in a cord T-cell line derived by co-cultivating normal human cord leukocytes and human leukaemic T cells. Nature 294(5843), 770-771.

-Other references in the main manuscript.