

Figure S1. Isotipic control of S100A8/A9-P-induced miR-146a and miR-155-5p expression through TLR4 signaling (Fig 3 of the manuscript). dHL-60 cells were incubated for 30 min with isotopic control (IgG), then stimulated with 10 μ g/mL S100A8/A9-P for 18h. 1 μ g/mL isotopic control was added at 6h and 12h of stimulation. Expression of **(a)** miR-146a-5p and **(b)** miR-155-5p were quantified by RT-qPCR. Data normalization was performed using three reference genes (RNUA1, RNUA5, SCARNA17) and expressed as fold induction compared to the stimulated control not treated with TLR4 neutralizing antibody. Results are presented as mean ± SEM of three independent experime







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Figure S2. Effect of miR-146a-5p and miR-155-5p mimics on cytokine secretion induced by LPS in dHL-60 cells. Transduced dHL-60 cells with miR-146a-5p and miR-155-5p were stimulated with 100 ng/mL LPS for 6h. Secretion of TNF α , IL-6, CCL2, CCL3, CXCL8 and IL-12b was quantified by cytometric bead array and secretion of CCL4 by ELISA. Cell viability for each condition was measured by LDH quantification. The secretion results are presented as mean ± SEM of six independent experiments. *p < 0.05; **p < 0.01; ***p < 0.001; ****p < 0.0001.















Figure S3. Effect of transduced miR-146a-5p and miR-155-5p on putative TLR4-dependent mRNA targets. Transduced dHL-60 cells were stimulated for 6 h with 100 ng/mL of LPS. The expression of potential mRNA targets TLR4, MyD88, IRAK1, IRAK2, IRAK4, TAB2 and NF-kB1 was assessed by real-time PCR. Data normalization was performed using Actin- β , B2M, and Gus β and expressed as fold induction compared to the stimulated negative control. Results are presented as mean ± SEM of six independent experiments. *p < 0.05; **p < 0.01; ***p < 0.001; ****p < 0.001.



Figure S4. Overlay between miRNA expression and cytokine mRNA expression upon S100A8/A9-P stimulation in dHL-60 cells. dHL-60 cells were stimulated for 3, 6, 12, 18 and 24h with 10 µg/mL S100A8/A9-P. The expression of miR-146a-5p and miR-155-5p as well as CCL2, CCL4 and CXCL8 was assessed by qPCR. Data normalization was performed using three reference genes (RNUA1, RNUA5, SCARNA17 for miRNAS and Actin- β , B2M and Gus β for cytokine expression) and expressed as fold induction compared to the respective non-stimulated control. The results of miRNA and cytokine expression have been superposed for CCL2 (A), CCL4 (B) and CXCL8 (C). Results are presented as mean \pm SEM of 3 independent experiments. For more convenience, all statistical analysis were summarized in the table D). * = p < 0.05; ** = p < 0.01; *** = p < 0.001; **** = p < 0.0001.