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Data article

Data in support of NFκB and JNK pathways involvement in TLR3-mediated HIV-1 transactivation, expression of IL-6 and transcription factors associated with HIV-1 replication

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ABSTRACT

In the present article, using human monocyte-derived macrophages and cell lines containing integrated copies of the HIV-1 promoter, we show the effects of TLR3 ligands on the pro-inflammatory cytokine IL-6. We further show the effects of TLR3 ligands on HIV-1 transactivation and transcription factors involved in HIV-1 replication. This article complements the data reported by the authors, "Toll-Like receptor-3 mediates HIV-1 transactivation via NFκB and JNK pathways, and histone acetylation, but prolonged activation suppresses Tat and HIV-1 replication" (Bhargavan et al., 2015) [1], and the interpretation of these data can be found in the research article published by the authors in Cellular Signaling in 2015 (Bhargavan et al., 2015) [1].

Specifications Table

Subject area	Biology
More specific subject area	Molecular biology, Virology
Type of data	Graph, Figure

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How data was acquired	Cell culture, Real-time PCR, Luciferase assay, CAT-ELISA
Data format	Analyzed
Experimental factors	Human monocyte-derived macrophages and cell lines containing integrated copies of the HIV-1 promoter; HIV-1 infection; treatment with TLR3 ligands, and pharmacological inhibitors
Experimental features	Real-time PCR, luciferase assay, and chloramphenicol acetyltransferase assay
Data source location	University of Nebraska Medical Center, Omaha, USA
Data accessibility	Data are with this article

Value of the data

- The approach used in this data article allows for the prediction of biological pathways involved in receptor–ligand interactions.
- Data on the mechanistic basis of TLR3-HIV interactions would be useful to others investigating viral transactivation and replication.
- Others interested in studying the functional effects of receptor–ligand interactions could learn from our approach.
- This approach would be of interest when determining the critical role of time on gene transcription and expression.

1. Data

To determine whether the TLR3 ligand polyinosinic-polycytidylic acid (PIC) can interact with HIV-1 to affect gene transcriptional regulation in human monocyte-derived macrophages (MDM), time-dependent and concentration-dependent real-time PCR analyses were performed on uninfected or HIV-1 infected human MDM treated with PIC. Genes analyzed included the pro-inflammatory cytokine IL-6 and transcription factors known to regulate the HIV-1 promoter activity (STAT-1, REL-B, JUN, CEBPA, and CEBPG). To investigate the role and involvement of these transcription factors in TRL3-mediated HIV-1 transactivation, pharmacological inhibitors targeting their signaling pathways were used, with and without TLR3 ligands, in luciferase and chloramphenicol acetyltransferase (CAT) ELISA assays on TZM-bl and U38 cells, both of which contained integrated copies of the HIV-1 promoter.

2. Experimental design, materials and methods

2.1. HIV-1 infection of MDM and real-time PCR

Human MDM were obtained from freshly elutriated human monocytes as previously described [1,2], cultured in 6-well plates (2 million cells per well), and treated with 10 or 25 μg/ml PIC for 2–120 h, with each experimental condition performed in triplicate. In separate experiments, MDM were infected with HIV-1_{ADA} at a multiplicity of infection of 0.01 as previously described [1–4], with or without PIC treatment (10 and 25 μg/ml) for 2–120 h, with each experimental condition performed in triplicate. Following treatment, cells were harvested, total RNA was extracted using the Trizol reagent, and real-time PCR performed as described in the main manuscript [1]. All PCR reagents, probes and primers were from Applied Biosystems and primers IDs were as follows: CEBPA (Hs00269972_s1), CEBPG (Hs01922818_s1), JUN (Hs99999141_s1), STAT1 (Hs00234829_m1), RELB (Hs00232399_m1), IL-6 (Hs00985639), and GAPDH

(Hs99999905_m1). For each gene and each sample, data was normalized to the sample's GAPDH to quantify the effects of PIC (10 and 25 μg/ml) on IL-6, STAT-1, REL-B, JUN, CEBPA, and CEBPG mRNA in uninfected (Fig. 1) and HIV-1-infected (Fig. 2) human MDM.

2.2. Luciferase and chloramphenicol acetyltransferase (CAT) assays

TZM-bl and U38 cells were treated for 48 h with PIC (25 or 50 μg/ml), with or without the inhibitor of NFκB transcriptional activation (481406, 20 nM), the JNK inhibitor (420119, 10 μM), the inhibitor of

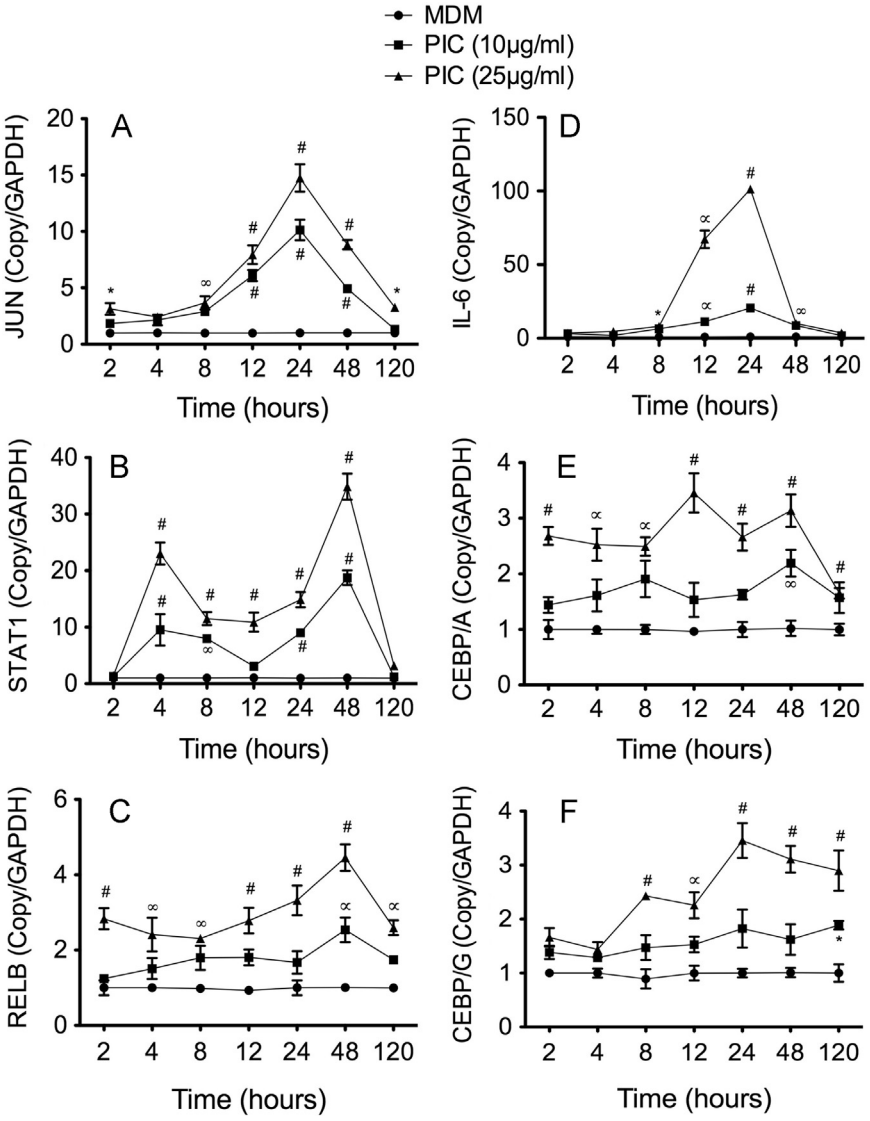

Fig. 1. Real-time PCR quantification of JUN (A), STAT1 (B), RELB (C), IL-6 (D), CEBPA (E), and CEBPG (F) mRNA in untreated and human macrophages treated with the TLR3 ligand PIC. For both MDM treated with 10 μg/ml and 25 μg/ml PIC, $^{*}P < 0.05$, $\infty P < 0.001$. $\propto P < 0.001$, $P < 0.0001$, compared to untreated MDM.

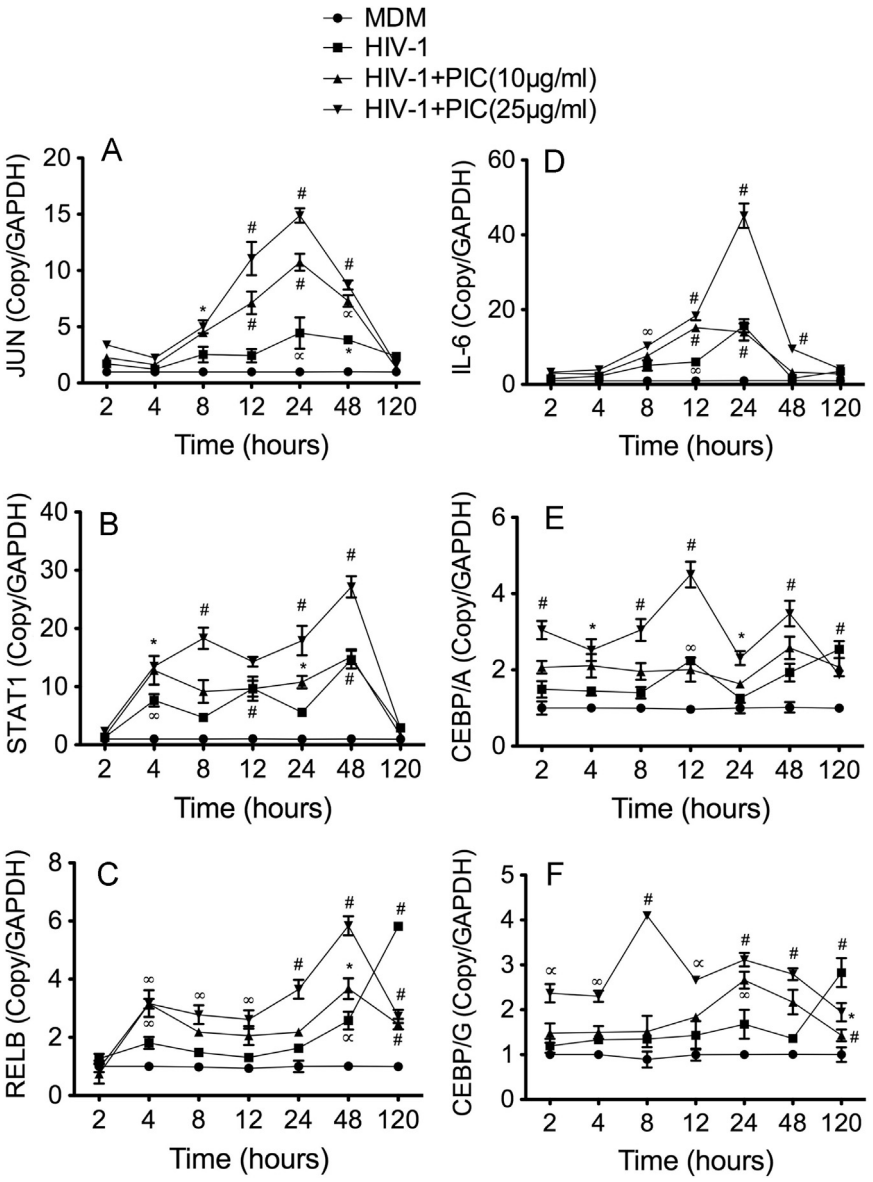

Fig. 2. Real-time PCR quantification of JUN (A), STAT1 (B), RELB (C), IL-6 (D), CEBPA (E), and CEBPG (F) mRNA in HIV-1-infected human macrophages, untreated or treated with the TLR3 ligand PIC. $^{*}P < 0.05$, $\infty P < 0.001$. $\propto P < 0.001$, $P < 0.0001$. *P*-values of infected MDM (HIV-1) are in comparison to non-infected controls (MDM), and *P*-values of infected MDM treated with PIC (HIV-1+PIC(10 μg/ml), and HIV-1+PIC(25 μg/ml)) are in comparison to infected MDM.

c-Jun/JNK complex (420130, 5 μM), and the MEKK7/MKK7 inhibitor (5ZO, 5 μM). Each experimental condition was performed in triplicate and following treatment, cells were harvested, washed with phosphate-buffered saline, and lysed as described [1]. Cell lysates were then used to quantify the luciferase activity (Fig. 3A) and the CAT activity (Fig. 3B) in each sample using the Luciferase Assay

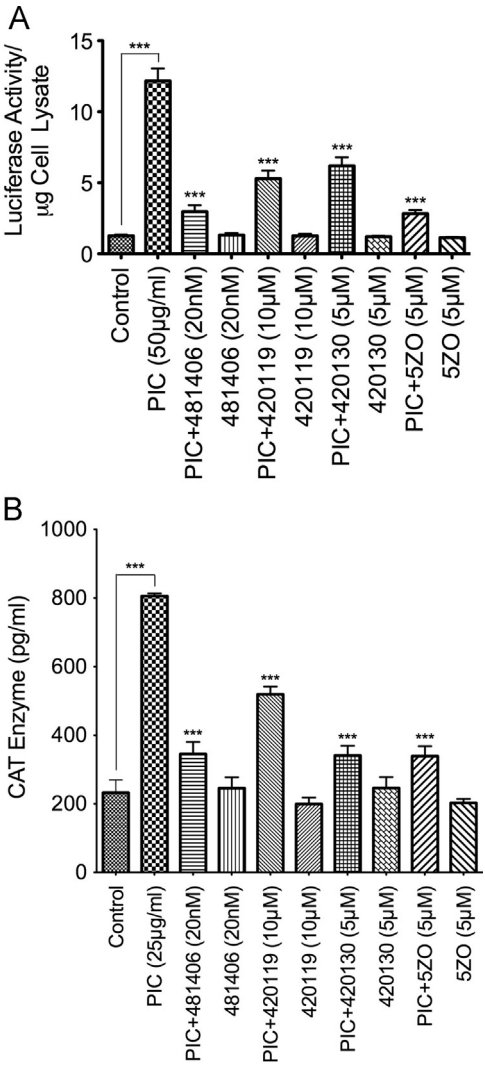

Fig. 3. Quantification of HIV-1 transactivation in TZM-bl (A) and U38 (B) cells treated with TLR3 ligands, with or without the inhibitor of NFκB transcriptional activation (481406), the JNK inhibitor (420119), the inhibitor of c-Jun/JNK complex (420130), and the MEKK7/MKK7 inhibitor (5ZO). ***$P < 0.001$; *P*-values for inhibitors-treated samples are in comparison to the HIV-1 promoter activity in PIC-treated cells.

System (Promega, Madison, WI) and the CAT ELISA kit (Roche Diagnostics Indianapolis, IN), as described in the main manuscript [1].

In separate experiments, cells were treated for 48 h with PIC (25 μg/ml), with or without the AP-1 inhibitor (SR11302, 2 μM and 10 μM), the JNK inhibitor V (420129, 10 μM and 20 μM), and the IRAK-1/4 inhibitor (5 μM and 10 μM). Each experimental condition was performed in triplicate and following treatment, the effects of PIC and inhibitors on HIV-1 transactivation was quantified in TZM-bl cells (Fig. 4A) or U38 cells (Fig. 4B). The main manuscript [1] includes the manufacturer's names, catalog numbers, and mechanisms of action of each inhibitor.

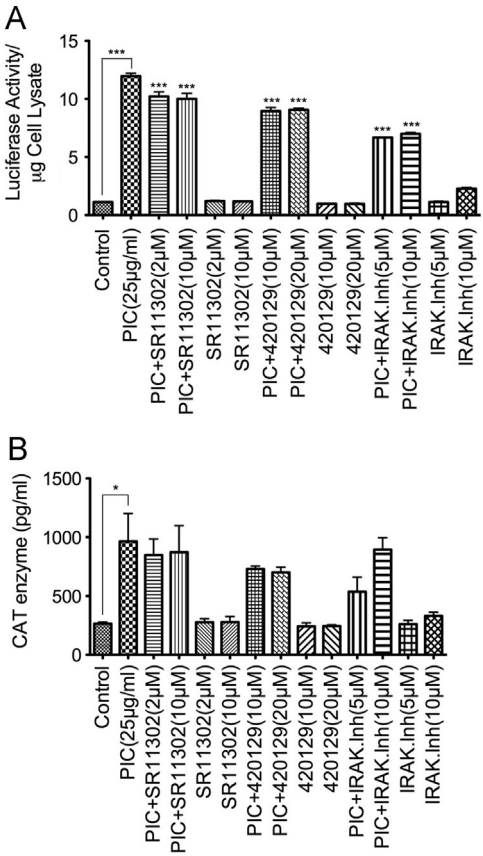

Fig. 4. Quantification of HIV-1 transactivation in TZM-bl (A) and U38 (B) cells treated with TLR3 ligands, with or without the inhibitor of AP-1 transcriptional activity (SR11302), the ATP-competitive inhibitor of JNK (420129), and the IRAK1/4 inhibitor ***$P < 0.001$; P-values for inhibitors-treated samples are in comparison to the HIV-1 promoter activity in PIC-treated cells.

Acknowledgments

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2015.12.022.

References

[1] Biju Bhargavan, Shawna M. Woollard, Georgette D. Kanmogne, Toll-Like receptor-3 mediates HIV-1 transactivation via NFκB and JNK pathways, and histone acetylation, but prolonged activation suppresses Tat and HIV-1 replication, Cell Signal. 28 (2015) 7–22.

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