Involvement of Dihydropyridine-sensitive Calcium Channels in Human Dendritic Cell Function

COMPETITION BY HIV-1 TAT

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The entry of extracellular calcium in leukocytes mediates several cellular processes; however, unlike in excitable tissues, the underlying molecular mechanisms are poorly defined. In this paper we provide phenotypical and biochemical evidence that peripheral blood-derived human dendritic cells express dihydropyridine-sensitive calcium channels. Exposure to the dihydropyridine drug nifedipine, which binds L-type calcium channels blocking calcium influx, prevents two dendritic cell functions that are dependent on extracellular calcium entry: apoptotic body engulfment and interleukin-12 production induced by cross-linking of the surface lectin NKRP1A. It is known that exogenous human immunodeficiency virus, type 1 Tat affects several Ca\(^{2+}\)-dependent immune cell responses. Here we demonstrate that Tat inhibits apoptotic body engulfment and interleukin-12 production by blocking extracellular calcium influx. This inhibition is prevented by the calcium channel agonist dihydropyridine derivative Bay K 8644, suggesting the involvement of l-type calcium channels. This hypothesis is further supported by the observation that Tat and dihydropyridine drugs compete for binding to dendritic cells. Taken together, these findings indicate that exogenous Tat exerts its inhibitory effects on dendritic cells by blocking dihydropyridine-sensitive l-type calcium channels.

Calcium-linked cellular functions in excitable tissues are mediated by voltage-dependent calcium channels, which participate in the regulation of action potential generation, muscle contraction, and secretion of hormones or neurotransmitters (1). Although in neurons multiple types of calcium channels, which can be distinguished by their pharmacological properties, are expressed, in skeletal and cardiac muscles the principal calcium channels are l-type (1, 2). These channels are composed of three transmembrane subunits (\(\alpha\)1C, \(\gamma\), and the \(\alpha\)2\(\delta\) complex) and one cytoplasmic chain (the \(\beta\)1 chain). A spectrum of compounds, the dihydropyridine (DHP)\(^1\) derivatives, which specifically bind with high affinity to the \(\alpha\)1C chain of \(\lambda\)-type channels (3, 4), regulating their functional state from blocking to opening, allows both the identification and the functional analysis of this class of molecules (1–4).

Cytosolic calcium rise is an important signal also in nonexcitable cells, including immune cells, regulating fundamental processes such as activation, growth, and differentiation (5–7). Increase in free intracellular calcium concentration ([Ca\(^{2+}\)]\(_i\)) may result from calcium mobilization from either intracellular stores or extracellular medium or both (5). Unlike the mechanisms mediating mobilization from intracellular stores, the molecular structures mediating extracellular calcium influxes are still poorly characterized. Recently, the presence of functional calcium channels displaying DHP sensitivity has been observed in B lymphocytes (8), raising the possibility that similar structures are present also in nonexcitable cells. We have previously shown that some functions of dendritic cells (DC) are mediated by [Ca\(^{2+}\)]\(_i\), increase. DC are professional antigen presenting cells able to endocytose and process soluble or particulated antigens (9, 10) and prime naive T lymphocytes (9). Activated DC produce IL-12, a cytokine that amplifies the immune response promoting the differentiation of the T helper 1 lymphocyte subset, which in turn substanst the natural killer (NK) cell activity (11, 12). We reported that activation of DC by cross-linking of the surface lectin NKRP1A with consequent IL-12 production is accompanied by extracellular calcium influx (13); similarly, apoptotic body engulfment induces and is dependent on calcium entry in phagocytosing DC (10). Interestingly, the HIV-1 transactivating factor Tat, which can be released by infected cells and play a number of extracellular roles (14), affects several calcium-mediated events in immune cells (15–18), including the phagocytosis of apoptotic cells by DC (19). We thus investigated the presence of calcium channels on DC and the possible interference by exogenous Tat. Our data indicate that functional DHP-sensitive l-type calcium channels are expressed by DC and regulate both apoptotic body engulfment and NKRP1A-mediated IL-12 production. Interestingly, l-type calcium channels appear to be the molecular target of HIV-1 Tat on DC; indeed, binding of DHP derivatives to these channels is cross-inhibited by Tat. Moreover, the inhibitory effect of HIV-1 Tat on DC function is antagonized by the DHP agonist Bay K 8644 (2).

EXPERIMENTAL PROCEDURES

REAGENTS—The acetoxymethyl ester of FURA 2 (FURA 2-AM) and all the reagents for electrophoresis were from Sigma. Prestained molecular weight markers were from Bio-Rad.

\(^{1}\) The abbreviations used are: DHP, dihydropyridines; CCD, charged coupled device; DC, dendritic cells; GAM, goat anti-mouse; Fn-III, fibronectin type III repeat; mAb, monoclonal antibody; MFI, mean fluorescence intensity; NFP, nifedipine; NK, natural killer; IL, interleukin; HIV, human immunodeficiency virus; FURA 2-AM, acetoxymethyl ester of FURA 2.
Ionomycin, nifedipine (NFP), and (-) Bay K 8644 (the net functional effect of the racemic mixture is that of the negative enantiomer, which is a L-type Ca\(^{2+}\) channel agonist) were from Calbiochem-Intalco S.p.A. (Milan, Italy). Fluorescein DM-BODIPY\(^\circledR\) DHP was from Molecular Probes Europe (Leiden, the Netherlands). Chemically synthesized and biotinylated protein was produced by Technogenetics (Piano almonte, Cesena, Italy). Synthetic Tat preparations were purified by reverse phase high pressure liquid chromatography yielding a purity of 96%. The biological activities of synthetic Tat were superimposable to those of natural Tat in different assays (20, 21). Recombinant fibronectin type III repeat (Fn-III, from amino acids 1086–1172) was a kind gift of L. Zardi (National Institute for Cancer Research, Genos, Italy). RPMI 1640 medium, lipase, 1-glutamine, and penicillin-streptomycin were from Biochrom (Berlin, Germany). Recombinant granulocyte-macrophage colony-stimulating factor was from Schering-Plow (Milan, Italy).

Engulfment of Apoptotic Bodies by Dendritic Cells—Peripheral blood monocytes were isolated from healthy donors as described (10, 22) and cultured in RPMI 1640 medium supplemented with 2 mM L-glutamine, 100 mM sodium penicillin, 100 µg/ml streptomycin, 10% heat-inactivated fetal calf serum, and 20 ng/ml granulocyte-macrophage colony-stimulating factor. Cells were isolated on Ficoll-Hypaque and cultured in 96-well plates, in triplicate, at 3×10^6 cells/well, for 3 days in the presence of NFP or Tat. 

Cytokine Production—DC (2×10^6/sample), untreated or Tat-pretreated cells, were co-incubated with adherent DC (2:1 ratio) at 37 °C in the absence or presence of NFP (10 or 1 µM). One representative experiment out of six is shown. Supernatants were collected after 3 h, and secreted IL-12 (p40 and p70) purchased from Endogen (Woburn, MA) were measured using the enzyme-linked immunosorbent assay kit for human IL-12 (p40 and p70) purchased from Endogen (Woburn, MA).

Western Blot—After separation by SDS-polyacrylamide gel electrophoresis (12%) under reducing conditions, lysates from DC (50 µg) or A431 cells (positive control) were electrotransferred onto nitrocellulose filters, hybridized with anti-calcium channel β1 subunit mAb followed by horseradish peroxidase-GAM Ig, and developed with chemiluminescence.

RESULTS

The DHP Drug NFP Prevents Calcium Mobilization and Engagement of Apoptotic Bodies by Dendritic Cells—We have previously shown that apoptotic body engulfment by DC elicits Ca\(^{2+}\)\(^{2+}\), rises, mainly due to the entry of extracellular calcium, that are essential for phagocytosis (10, 19). The possibility of an involvement of calcium channels in this process has been in-

![FIG. 1.](http://www.jbc.org/Downloaded from http://www.jbc.org/2018-07-24-18.2.8)

**FIG. 1.** DHP-sensitive calcium channels participate in apoptotic body engulfment by DC. A, DC cultured on round coverslips and loaded with 1 µM FURA 2-AM were challenged at 37 °C with apoptotic bodies (AB) at a ratio of 1:2. [Ca\(^{2+}\)]\(_i\) was monitored with an inverted epi-fluorescence microscope connected to an intensified CCD camera. Results are expressed as [Ca\(^{2+}\)]\(_i\) nm. Calcium fluxes were measured during interaction between DC and apoptotic bodies as such or after pretreatment with NFP (10 or 1 µM). One representative experiment out of six is shown. B, apoptotic bodies labeled with 31Cr were co-incubated with adherent DC (2:1 ratio) at 37 °C in the absence or presence of NFP (10 or 1 µM). After 45 min, noningested apoptotic bodies were removed by four gentle washings and DC-associated radioactivity measured in a γ-counter (Beckman Instruments Inc., Irvine, CA). C, apoptotic bodies were loaded with Bay K 8644 10 µM or with different concentrations of Tat (from 100 to 1 nM) or with Tat 100 nM plus Bay K 8644 10 µM, pretreated for 20 min with different concentrations of Tat (from 100 to 1 nM) or with Tat 100 nM plus Bay K 8644 10 µM or with Bay K 8644 10 µM alone were challenged by cross-linking of the NRK1R1 molecule with F(ab\(^\prime\))\(_2\) of a specific monoclonal antibody followed by GAM F(ab\(^\prime\))\(_2\), as described (13). Cross-linking of CD31 or exposure of DC to GAM F(ab\(^\prime\))\(_2\), alone did not stimulate IL-12 production (13). Supernatants were collected after 3 h, and secreted IL-12 was measured using the enzyme-linked immunosorbent assay kit for human IL-12 (p40 and p70) purchased from Endogen (Woburn, MA).

Western Blot—After separation by SDS-polyacrylamide gel electrophoresis (12%) under reducing conditions, lysates from DC (50 µg of protein/sample; protein dosage performed with the Detergent-Compatible Bio-Rad kit based on the colorimetric Lowry method, Bio-Rad) or A431 cells (positive control, Transduction Laboratories, Lexington, KY) were electrotransfered onto nitrocellulose filters (Hybond ECL, Amersham Italia S.r.l., Milan, Italy) as described (13). Filters were blocked overnight with 10% nonfat dry milk in phosphate-buffered saline and then incubated for 1 h with the anti-calcium channel β1 subunit mAb (clone 44, Transduction Laboratories), at 1:2,000 dilution. The immunoreactive bands were revealed by lumino reaction (ECL, Amersham).
vestigated by using the inhibitory DHP derivative NFP, which specifically binds to the α1C subunit of l-type calcium channels (1–4). As shown in Fig. 1A, NFP inhibits in a dose-dependent manner the calcium mobilization that follows the interaction between apoptotic bodies and DC. Likewise, engulfment is prevented by DC exposure to this drug (Fig. 1B). These results suggest that DC express functional l-type calcium channels. This was confirmed by the finding of a specific 58-kDa band detectable by Western blot analysis of DC lysates with a monoclonal antibody recognizing the β1 calcium channel subunit (Fig. 1C). Unlike in excitable tissues, these channels are voltage-independent, because exposure of DC to 50 mM KCl failed to induce a calcium influx (not shown).

HIV-1 Tat Prevents the Opening of DHP-sensitive Calcium Channels and Competes with DHP Derivatives for Binding to DC—Exogenous HIV-1 Tat is able to inhibit both apoptotic body engulfment by DC and the [Ca\(^{2+}\)]\(_i\) rise induced by apoptotic body-DC interaction (19). We thus investigated whether the inhibitory effect of Tat is due to the block of l-type calcium channels on DC. DC were loaded with apoptotic bodies, and the engulfment was measured under different conditions. Fig. 2A shows that the inhibition of engulfment observed in the presence of HIV-1 Tat is prevented by DC pretreatment with Bay K 8644, a calcium channel agonist that induces Ca\(^{2+}\) entry by opening l-type channels (2, 8). In turn, once DC have been pretreated with Tat, Bay K 8644 is unable to revert the inhibition (Fig. 2A). The calcium influx elicited in DC by exposure to Bay K 8644 is blocked by pretreatment of DC with HIV-1 Tat in a dose-dependent manner (Fig. 2B); in contrast, Tat does not affect the calcium channel-independent calcium rise that follows DC exposure to the ionophore ionomycin (Fig. 2C). These data suggest that HIV-1 Tat competes with Bay K 8644 for binding to DHP-sensitive calcium channels. To further confirm this hypothesis, we performed fluorescence-activated cell sorter analysis of DC stained with DM-BODIPY\(^{®}\) DHP, a fluorescent DHP derivative that binds to the α1C chain of l-type calcium channels (4), in the presence or absence of Tat or Bay K 8644. As shown in Fig. 3A, DM-BODIPY\(^{®}\) DHP stains DC, indicating the presence of l-type calcium channel α1C subunits on these cells. Tat antagonizes the DM-BODIPY\(^{®}\) DHP binding with the same dose response as the α1C ligand Bay K 8644. In turn, the binding of biotinylated Tat to DC is cross-inhibited by pretreatment of the cells with Bay K 8644 (Fig. 3B). In both cases, the unrelated peptide Fn-III, displaying the same length as HIV-1 Tat, has no effects (Fig. 3, A and B).

Both HIV-1 Tat and the DHP Drug NFP Inhibit IL-12 Secretion by DC—An important function of DC is to support T helper cell differentiation by IL-12 production (11, 12). Secretion of IL-12 is induced by various stimuli (11, 12); one of them is cross-linking of the surface lectin NKRP1A (13). Because this triggering also results in Ca\(^{2+}\) entry (13), we investigated whether l-type calcium channels play a role in IL-12 release. NKRP1A molecules were cross-linked by the specific monoclonal antibody, and the secretion of IL-12 by DC in the presence or absence of NFP or HIV-1 Tat was measured. Fig. 4A shows that the engagement of NKRP1A results in release of IL-12, which is prevented by exposure of DC to NFP. HIV-1 Tat proves to exert the same inhibition of NFP on NKRP1A-induced IL-12 production (Fig. 4A). This inhibition was dose-dependent, being detectable up to 10 nM Tat (Fig. 4A). Again, the calcium channel agonist Bay K 8644 is able to reverse the inhibitory effect of HIV-1 Tat (Fig. 4A). In keeping with these results, the calcium rise elicited by NKRP1A cross-linking in DC is inhibited by either the DHP drug NFP (Fig. 4B) or HIV-1 Tat (Fig. 4C), further supporting the hypothesis that both compounds exert their effects by acting on l-type calcium channels.

DISCUSSION

In the present paper we show that molecular structures displaying pharmacological properties of l-type calcium channels (1, 2) mediate extracellular calcium influx in human DC. The presence of functional l-type Ca\(^{2+}\) channels in DC is supported by three lines of evidence: (i) Ca\(^{2+}\)-dependent DC functions, such as apoptotic body engulfment and IL-12 production, are inhibited by l-type Ca\(^{2+}\) channel blockers; (ii) fluorescent DHP drugs, specific for the α1C chain of l-type channels (3, 4), bind to DC surface; and (iii) the calcium channel β1 chain is detectable in DC lysates. In contrast, N-type calcium channels (1, 25) are not expressed, because fluorescent ω-conotoxin failed to stain DC (not shown). Although l-type calcium channels in excitable tissues are voltage-gated (1), DHP-sensitive Ca\(^{2+}\) channels in DC are voltage-independent, suggesting that they lack a membrane voltage sensor. This finding supports the recently reported data on the existence of voltage-independent
DHP-sensitive channels on B lymphocytes (8), raising the possibility of a common mechanism responsible for Ca\(^{2+}\) entry in immune cells.

An important novel finding of this paper is that HIV-1 Tat blocks two calcium-mediated DC functions by acting on DHP-sensitive Ca\(^{2+}\) channels. Indeed, Tat-mediated inhibition of both apoptotic body engulfment and NKRP1A-induced IL-12 production is reverted by the l-type Ca\(^{2+}\)-channel agonist Bay K 8644. In turn, Tat blocks the Ca\(^{2+}\) influx that follows triggering of DC with Bay K 8644. The finding that DHP derivatives and Tat compete for binding to DC strongly supports the hypothesis that l-type calcium channels are the molecular targets of the inhibitory effects of Tat on DC. Several Tat binding molecules have been described on different cell types; Tat binds with high affinity vascular endothelial growth factor receptor (21) and CD26 (26), with low affinity heparan sulfates (27), and with high affinity vascular endothelial growth factor receptor molecules have been described on different cell types; Tat binds gets of the inhibitory effects of Tat on DC. Several Tat binding sensitive Ca\(^{2+}\) blocks two calcium-mediated DC functions by acting on DHP-immune cells.

production is reverted by the L-type Ca\(^{2+}\) both apoptotic body engulfment and NKRP1A-induced IL-12 hypothesizing that L-type calcium channels are the molecular targets and Tat compete for binding to DC strongly supports the role in the progression of the disease (33, 34), which is associated with decreased NK cell function, loss of T helper cell subset 1 cells, and a corresponding increase in T helper cell subset 2 cells (33, 35). In light of these considerations, our data provide evidence for a molecular mechanism possibly underlying T helper cell subsets 1 and 2 embalance during HIV-1 infection, based on the Tat-mediated impairment of IL-12 production. The physiological relevance of our findings relies on the observation that nanomolar concentrations of HIV-1 Tat are detectable in the sera of AIDS patients (36). It is conceivable that the local amount of Tat in the mucosal and lymphoid tissues site of infection is higher due to the concentrating effect of extracellular matrix components, such as heparan sulfates, which bind to Tat (27). This may explain the finding that in vitro, in the absence of matrix components, Tat is usually active at concentrations higher than those found in AIDS patient sera (15, 17, 29, 31, 32). Finally, the finding that Tat can act through blocking calcium channels may provide a universal key for understanding the molecular basis of exogenous Tat-mediated immunosuppressive effects during HIV-1 infection.

![Fig. 3. Bay K 8644 and HIV-1 Tat compete for binding to DC. A, DC untreated or pretreated for 20 min with Tat or Bay K 8644 or Fn-III peptide at different concentrations, as indicated, were stained with 3 nM DM-BODIPY® DHP and analyzed by FACSort (Becton Dickinson). Results are expressed as MFI (arbitrary units). B, DC untreated or pretreated for 20 min with Tat or Bay K 8644 or Fn-III peptide at different concentrations, as indicated, were stained with biotinylated Tat (Tat-biot) followed by phycoerythrin-streptavidin (PE-Av-biot) and analyzed by FACSort. Results are expressed as MFI (arbitrary units).](http://www.jbc.org/)

![Fig. 4. NKRP1A-induced Ca\(^{2+}\) influx and IL-12 secretion are inhibited by NFP and HIV-1 Tat. A, DC (10\(^5\)/sample) were challenged by cross-linking of the NKRP1A molecule after the following treatments: no treatment (Nil, hatched column), NFP 10 \(\mu M\) (dotted column), 10–100 \(\mu M\) Tat (closed columns), Bay K 8644 10 \(\mu M\) followed by Tat 100 \(\mu M\) (dark gray column), or Bay K 8644 10 \(\mu M\) alone (light gray column). IL-12 was measured in the supernatants by enzyme-linked immunosorbent assay after 3 h. The open column (Nil) represents supernatants from noncross-linked DC. B and C, DC cultured on round coverslips and loaded with 1 \(\mu M\) FURA 2-AM, untreated or pretreated with NFP (B, 1 or 10 \(\mu M\)) or Tat (C, 10–100 \(\mu M\)) were challenged by cross-linking of the NKRP1A molecule. [Ca\(^{2+}\)]\(_i\) was monitored with an inverted epifluorescence microscope connected to an intensified CCD camera. Results are expressed as [Ca\(^{2+}\)]\(_i\), nm. One representative experiment out of six is shown.](http://www.jbc.org/)
Functional L-type Calcium Channels in Dendritic Cells

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