Macrophages and neutrophils constitute one of the main effectors of nonspecific immunity, phagocytosing conventional and opportunistic microorganisms and presenting their antigens to T lymphocytes. This consequently induces specific immune functions (13, 91). Moreover, activated macrophages, but also phagocytosing neutrophils, produce some cytokines and other humoral mediators (e.g., defensins) that intervene in defensive and/or inflammatory processes (2, 19, 63). Reactive oxygen and nitrogen species are produced by these cells in response to cytokines released by immunocompetent cells or induced by other signaling processes (23). Not only microorganisms but also viruses induce the activation of macrophages (54) and neutrophils (40). However, various viruses, analogously to some microorganisms, especially intracellular ones, can affect the regular functions of these cells. This phenomenon predisposes to different coinfections or superinfections and increases their severity (5). In addition, acquired disorders of phagocytic activity of macrophages and neutrophils of noninfective conditions, such as traumatic, dysmetabolic, and autoimmune disorders, can predispose an organism to microbial and viral infections (29).

Finally, smoking reduces the oxidative burst of monocytes and neutrophils, and increases neutrophil chemotaxis but reduces that of monocytes. Abstinence for almost 20 days repairs oxidative burst activity (93). However, monocytes/macrophages may represent an important reservoir of human immunodeficiency virus (HIV) infection (28), and other viruses, e.g., measles (85) and hepatitis C viruses (84), may propagate the infection to other target cells. Neutrophils can bind HIV-1 and transfer the infection to lymphocytic cells (35); this occurs with other viruses, for instance, in the case of cytomegalovirus infection (39).

MECHANISMS OF MONOCYTE/MACROPHAGE IMPAIRMENT INDUCED BY HIV INFECTION AND PATHOLOGICAL CONSEQUENCES

Lafrin et al. (57) demonstrated that HIV-1 Tat protein promotes the chemotaxis of monocytes, their adhesion to the endothelium, and their recruitment into extravascular tissues and consequently phagocytic function. This chemotactic activity modulation seems to be mediated by the Tat cysteine-rich domain (1). However, gp120 exposure also induces the macrophages to amoeboid and motile behavior (94), but down-regulation of interleukin-12 (IL-12) production induced by gp120 on peripheral blood mononuclear cells interferes with Th1 differentiation and contributes to a Th2 switch (76); this glycoprotein impairs the phagosome-lysosome fusion (66).

Moreover, Nef protein can inhibit not only the macrophagic phagocytosis of infective agents (15, 82) but also that of apoptotic bodies of immune cells and in particular neutrophils, which are more easily affected by apoptotic phenomena in patients with HIV infection (101). This phenomenon may contribute to the persistence of an inflammatory state in HIV patients, especially during opportunistic infections that are often favored by defective phagocyte activity (101). The phago-
TABLE 1. Immunologic disorders induced by HIV on monocytes and macrophages in vitro and ex vivo

| Disorder | 
|---|---|
| Enhancement of chemotaxis by Tat and inhibition of chemotaxis by Nef protein | 
| Increased production of proinflammatory cytokines, including tumor necrosis factor alpha, IL-1, and IL-6 | 
| Inhibition of phagocytosis: inhibition of apoptotic bodies induced by Nef protein | 
| Inhibition of phagosome-lysosome fusion | 
| Inhibition of antimicrobial oxygen-dependent burst and acidification, induced in part by gp120 | 
| Decrease of surface expression of CD4, CD49e, and CD62L | 
| Decrease of Fcγ receptor expression | 
| Decrease of production of macrophage-CSF | 
| Decreased activity of APC function | 

cytosis of apoptotic cells, although reduced in HIV-positive patients, may, however, increase HIV-1 replication in macrophages (59), thus creating a dangerous synergism with the previously reported inhibitory phenomena.

Alterations of mononuclear phagocyte function seem to be implicated in the neuropathogenic manifestations accompanying HIV-1 infection. In fact, the neuroinflammatory phenomena that particularly characterize the HIV-1 dementia complex may occur both because of the anomalous activation of brain phagocytes with production of proinflammatory cytokines and/or in consequence of the dysfunction of phagocytosis and intracellular killing of opportunistic neurotropic agents (50, 73). Pu et al. (80) demonstrated that HIV-1 Tat protein induces the activation of microglial and perivascular cells to proinflammatory protein production, leading to monocyte infiltration into the brain. Moreover, pathological manifestations occurring in HIV-positive patients as respiratory, cardiovascular, and gastroenteric mechanisms can have etiopathogenetic mechanisms similar to those described for the central nervous system (17, 50, 81, 92, 96).

As previously reported, the central role of monocyte/macrophage cells in HIV-1 infection is their expression of CD4 receptors and chemokine receptors, which allow the viral interaction. In particular, these cells are infected prevalently by macrophage-tropic strains of HIV-1; resistant to the cytopathic effect of the virus, they act as important viral reservoirs and may disseminate the infection to different tissues (50).

HIV infection affects all the immune functions of monocytes/macrophages, affecting chemotaxis (enhancement by Tat and inhibition by Nef protein), phagocytosis, intracellular killing, APC function, and cytokine production (underproduction and overproduction, according to the cytokine type) (Table 1). This impairment allows not only the establishment of various opportunistic infections, but also the reactivation of others (50). However, in early stages of HIV-1 infection, monocyctic and polymorphonucleated cells phagocytose, and reactive ox-ygen product release can be increased, as demonstrated by Bandres et al. (4). This may be due to a nonspecific stimulation of phagocytic cells in early-phase retrovirus infection. Moreover, Pfitz et al. (77) demonstrated that although the phagocytosis and the phagolysosomal dysfunction of monocytes were found at late stages of HIV infection, these phenomena are more evident when CD4⁺ T lymphocytes significantly decrease.

As previously recalled, in vitro studies demonstrated that, other than treatment with recombinant Nef (82), challenge with recombinant gp120 impaired macrophage activity and particularly phagolysosomal fusion at doses ranging from 1 to 1,000 ng/ml of viral protein (77). These data were confirmed by Moorjani et al. (66), who also demonstrated a defect of phagosome-lysosome fusion in blood monocyte-derived macrophages infected by HIV-1 or treated with recombinant gp120 only. These investigators suggested that this phenomenon initially depends on the interaction between the viral glycoprotein and CD4 macrophage receptor. Pietrella et al. (75) demonstrated that gp120 reduces the antifungal capacity of peripheral blood monocytes, affecting the antimicrobial oxygen-dependent burst and the phagolysosomal acidification. Durrbaum-Landmann et al. (26) studied the effects of recombinant gp120 on monocyte cultures and demonstrated a significant reduction in these cells’ ability to stimulate autologous lymphocytes in response to anti-CD3, accompanied by a significant decrease in CD4 and Fc receptor expression.

Kedzierska et al. (51) demonstrated that HIV-1 infection of human monocyte-derived macrophages markedly affects FcγR-mediated phagocytosis. This occurs perhaps because the infection inhibits the tyrosine phosphorylation of cellular proteins that is necessary for the initial receptor activation, a phenomenon particularly dependent on Nef protein activity. In addition, interaction with cellular kinases, downmodulation of CD4 expression, and inhibition of macrophage colony-stimulating factor (CSF) exerted by Nef protein on monocytes/macrophages were also described (12, 97).

Finally, Trail et al. (103) demonstrated that in HIV-1-infected patients at stage A, the percentage of monocytes expressing CD49d, HLA-DP, HLA-DQ, and CD11a/CD18 was increased. Instead, at stages B and C, when phagocytic activity and trans-endothelial migration ability were markedly depressed, surface expression of CD49e and CD62L decreased (Table 1). This happens in concomitance with the reduced percentage of monocytes expressing CD18, CD11a, CD29, CD49e, CD54, CD58, CD31, and HLA-I. This phenomenon, which particularly concerns receptors of activation and cellular adhesion, seems to depend on the presence of high levels of circulating immune complexes.

MECHANISMS OF NEUTROPHIL IMPAIRMENT INDUCED BY HIV INFECTION AND PATHOLOGICAL CONSEQUENCES

As previously recalled, neutrophil function can be impaired by HIV infection (4). HIV-1 produces neutrophilic damage in a direct fashion by interacting with envelope constituents in cell membranes (67). Moreover, leukopenia and neutropenia are often present in HIV-positive patients, especially when patients have been diagnosed with AIDS. Consequently, mi-
Microbial infections are a common problem of in late stages of HIV infection (11). In particular, Pitrak (78) demonstrated that this retroviral infection can produce defects in chemotaxis, phagocytosis, and microbial killing, especially when CD4⁺ T-cell levels were <200 cells/μl (Table 1). Increases in neutrophil apoptosis via Fas may worsen the biological functioning of these cells (87); this in part explains the neutropenia induced by HIV infection (11, 56). Perskvist et al. (73) noted that HIV-1 infection accelerated the apoptosis of neutrophils, especially in the presence of apoptotic infective stimuli, contributing to proinflammatory cytokine release. However, Pugliese et al. (83) previously demonstrated that Nef protein may increase the effect of apoptotic stimuli in HIV target cells.

Treatment with recombinant granulocyte-CSF and granulocyte-macrophage CSF seems to improve neutrophil defects (18, 56). On this topic, Coffey et al. (18) demonstrated that treatment with granulocyte-CSF of HIV-infected patients increases the anticytotoxic cell activity of neutrophils. Vecchiarelli et al. (104) observed a cytokine dysregulation of neutrophils in HIV-infected patients. Inflammatory stimuli of concomitant infections increase the binding ability of HIV-1 to neutrophils, thus facilitating the propagation of infection to other susceptible cells (36). Analogously to monocytes, decreased phagocytosis and respiratory burst of granulocytes during HIV-1 infection correlates with CD4⁺ T-cell concentrations (10, 25). Prakash et al. (79) demonstrated an enhancing effect of Tat protein on ethanol-induced impairment of neutrophil function with transgenic mice. Meddows-Taylor et al. (64) suggested that defects in polymorphonuclear leukocytes (PMNLs) in phagocytosis and oxidative burst, detected particularly in HIV-1 patients with pulmonary tuberculosis, depend in part on the reduced expression of IL-8 surface receptors. In fact, both A and B IL-8 receptors considerably decrease in these cells, causing a reduced response in such cytokines implicated in neutrophil PMNL degranulation. However, in vitro treatment with IL-2 or IL-8 does not improve the phagocytic activity of these cells (47). In conclusion, we recall that neutrophil function is regulated by some cytokines (7, 27, 31) and that the cytokine network is generally altered in HIV-infected patients (95).

**INTRACELLULAR MICROORGANISM PHAGOCYTOSIS AND HIV INFECTION**

Several opportunistic and nonopportunistic intracellular microorganisms, such as *Salmonellae*, *Mycobacteria*, *Toxoplasma gondii*, *Pneumocystis carinii*, *Cryptococcus neoformans*, and *Histoplasma capsulatum* (some of these are facultative intracellular parasites), often produce serious manifestations in last stages of HIV infection (Fig. 1). More rarely, this occurs with *Rhodococcus equi* (responsible for pyogranulomatous bronchopneumonia) and *Bartonella henselae* (responsible for bacillary panchymal angiomatosis) but also with many other infective agents (22). This happens in good part because neutrophils and monocytes/macrophages are seriously compromised in their functions (22).
SALMONELLOSIS, OFTEN PRODUCED BY MINOR SALMONELLA STRAINS, REPRESENTS A SERIOUS PROBLEM FOR AIDS PATIENTS. IN FACT, IN THESE PATIENTS NOT ONLY MAJOR BUT ALSO MINOR SALMONELLA STRAINS ACT AS INTRACELLULAR MICROORGANISMS, BECAUSE OF PHAGOCYTIC CELL DEFECTS (46).

Kedzierska et al. (49) demonstrated that the phagocytosis of intracellular opportunistic agents Mycobacterium avium complex and Toxoplasma gondii is impaired in human monocytes infected with wild-type strains of HIV-1, but not with nef gene-defective HIV-1 strains, confirming the role of this gene product in macrophagic cell inhibitory activity. Perskvist et al. (73) demonstrated a lower half-life in neutrophils in patients coinfected with HIV-Mycobacterium tuberculosis, and Bonecini-Almeida Mda et al. (10) found that AIDS patients suffering from pulmonary tuberculosis have a reduced level of phagocytic activity in alveolar macrophages and neutrophils, especially when advanced impairment of CD4+ T cells was present; in addition, they demonstrated that this phenomenon is not only innate in the phagocytes of HIV-1-positive subjects but was also indirect, i.e., it is mediated by cytokine dysregulation. Denis and Ghadirian (24), comparing the bronchialveolar lavage-derived macrophages of HIV-1 infected subjects with those of healthy individuals, showed significant alterations in some cytokine release in the case of HIV-positive patients. PMNL function is also significantly compromised during the last stages of HIV infection, particularly in the pathological association between HIV-1 and tuberculosis (91).

In regard to Pneumocystis carinii (responsible for pneumonitis in seriously immunocompromised patients), HIV-1-infected subjects show a significant reduction in macrophagic mannose receptors (especially in patients with CD4+ counts of <200 cells/mm³) with a consequent decrease in lung anti-infection protection. In fact, such receptors mediate macrophage binding and subsequent phagocytosis of this opportunistic microorganism (55). Moreover, neutrophils of HIV-positive subjects are impaired in their ability to counterattack this microorganism (58).

In addition, coinfection worsens neutrophilic impairment (36). Toxoplasma gondii is often responsible for disseminated reactivations in AIDS patients, especially in neurotoxoplasmosis (70). Peripheral blood monocyte-derived macrophages of healthy individuals infected with a monocytotropic strain of HIV-1 reduce their phagocytic activity against this protozoan; intracellular replication of the microorganism is enhanced by HIV infection (6). This observation postulates a suggestive mechanism of T. gondii reactivation in AIDS patients. In addition, the severity of leishmaniosis is increased by HIV infection, because the protozoan replicates easily within the macrophages of HIV-infected subjects (102).

Of particular interest is the observation of the impairment of anticytotoxic activity of peritoneal and blood-derived macrophages after in vitro infection with HIV-1, as demonstrated by Cameron et al. (14). The investigators think that this depression, induced by HIV-1, depends not only on viral tropism and replication but also on cell tissue origin. In fact, this phenomenon, restricted to phagocytic activity alone, cannot be detected in alveolar macrophages (14).

To explain the influence of HIV infection on alveolar macrophages that constitute a first barrier against Cryptococcus neoformans inhalation in immunocompetent subjects, Jeong et al. (43) performed an enlightening in vitro experimental study. These investigators demonstrated that HIV-1 infection of these cells significantly reduces or suppresses fungicidal activity against this microorganism without, however, affecting the binding or internalization of C. neoformans, only inhibiting the intracellular antimicrobial phenomena.

Monocyte-derived macrophages from HIV-infected subjects also present impaired phagocytic functions against Histoplasma capsulatum yeast and are more permissive for their intracellular replication (16). In vitro tests confirm these data and demonstrate that only infective virions can affect both phagocytic activity and intracellular killing of macrophages; instead, treatment with gp120 alone may reduce only the phagocytosis of the yeast (16).

EXTRACELLULAR MICROORGANISM PHAGOCYTOSIS AND HIV INFECTION

There is a good deal of data on phagocytosis and the impairment of intracellular killing of extracellular microorganisms in HIV-infected patients, especially in regard to yeasts. In particular, Candida albicans opsonophagocytosis in neutrophils and intracellular killing were progressively reduced in HIV-positive subjects, in parallel with the increasing severity of viral infection (Fig. 1) (98). With this yeast, Tascini et al. (99) demonstrated that the inhibition of fungicidal activity of polymorphonuclear leukocytes in HIV-infected patients was due in part to IL-4 and IL-10 overproduction. In fact, these cytokines are able to impair neutrophil activity at high doses. This observation is in accord with that of Yoo et al. (106), who demonstrated alteration of cytokine equilibrium and impairment of monocyte-macrophage function in HIV infection.

Crowe et al. (21) demonstrated that HIV-1 infection of monocytic-derived macrophages decreased the percentage of Candida albicans-phagocytosing cells from 83% to 53%, and the mean number of yeasts per cell from 6.1 to 2.5. These findings and the data previously reported are in accord with the high incidence of serious candidiasis in AIDS patients. In addition, we demonstrated a significant infection exerted by Nef protein on Candida albicans macrophagic phagocytosis and killing, depending on oxidative processes (82). Conversely, Tat protein is able to bind Candida albicans through its RGD motif (Arg-Gly-Asp) and to induce hyphae production, a phenomenon that seems to increase the in vitro phagocytosis of the yeast (38).

In the early stages of HIV-1 infection, monocytic and PMNL phagocytosis and reactive oxygen product release can be increased, as demonstrated by Bandres et al. (4) with Staphylococcus aureus or Escherichia coli as a target. This may be in consequence of a nonspecific stimulation of phagocytic cells in the early phases of retrovirus infection. In the late stages of HIV infection, with low levels of CD4 T cells and with extracellular microorganisms present, the defensive mechanisms of monocytic/macrophagic cells and granulocytes are depressed (25). In fact, a reduced level of phagocytosis with E. coli and S. aureus was found by Schuermann et al. (88) in neutrophils of HIV-infected patients with low levels of CD4+ T cells (Fig. 1).

Brettle (11) recalled that the high frequency of bacterial sepsis in HIV-positive patients depends in part on neutrophil function impairment (chemotaxis, bacterial killing, phagocyto-
sis, and superoxide production). Payeras et al. (71) underlined the role of opsonophagocytosis defects in HIV-positive patients in facilitating encapsulation of bacteria responsible for recurrent infections of the respiratory tract.

To complete the question, we note the effects of HIV infection on microglial cells, which are very important as nonspecific immunologic defenses for the nervous system (residential macrophage functions) but which can also present a pathological activation, with release of proinflammatory cytokines in AIDS patients (68). Microglial cells and brain macrophages are susceptible to HIV-1 infection; reducing their protective activities may promote the diffusion of HIV to the central nervous system (32).

Finally, dendritic cells, a particular set of monocyte-derived cells, can also be affected by HIV infection. They may absorb, internalize, and transfer the virus to lymphocytes; in parallel, they are also damaged in their defensive functions (105).

**EFFECT OF ANTIRETROVIRAL THERAPY ON PHAGOCYTIC CELL FUNCTION**

Mastroianni et al. (62) found that highly active antiretroviral therapy (HAART) produces a significant improvement in chemotaxis and microbicidal activity of monocyte and neutrophil cells, contributing to cell-mediated immune responses against opportunistic agents (for instance, APC function). Consequently, a correct antiretroviral treatment, possibly associated with a suitable immunotherapy and/or growth factor treatment, may improve the monocyte/macrophage and neutrophil functions, together with some correlated activities of the immune system, as underlined by various authors (52, 53, 74) This contributes to the reduction in incidence of concomitant infections, especially those produced by opportunistic microorganisms such as the Mycobacterium avium complex, Pneumocystis carinii, Toxoplasma gondii, and Candida albicans. Antiretroviral therapy may regulate cytokine production, in particular, the release of IL-12 (which modulates cell-mediated immune responses) and IL-10 (anti-inflammatory cytokine) by monocytes/macrophages (9). Of particular interest is the demonstration that indinavir (a protease inhibitor) can inhibit the production of urease and protease, virulence factors of Cryptococcus neoformans but also involved in capsule formation, which contributes to reducing the risk of this serious opportunistic infection in AIDS patients (65). Blasi et al. (8) found that the same antiretroviral drug increases Cryptococcus neoformans phagocytosis and killing produced by microglial cells. Nathoo et al. (69) demonstrated that the treatment with protease inhibitors increases nonopsonic macrophagic phagocytosis of Plasmodium falciparum-infected erythrocytes in HIV-1 patients with malaria.

Magnani et al. (61) suggested the use of a potent antiviral drug, such as the antileukemic fludarabine (9-fluoroadenine 5'-monophosphate) loaded to red blood cells for the eradication of HIV-1 in macrophage cells. This experimental therapy affects only infected macrophages, reduces the virus release from these cells, and improves their biological function.

Finally, Feldman (30) demonstrated that HAART reduces the serious risk of pneumonia by intracellular or extracellular microorganisms, not only by increasing CD4 T-cell levels but also in part by improving the activity of phagocytic cells.

**CONCLUSIONS**

Monocytes/macrophages and neutrophils play an important role in nonspecific immunity against opportunistic pathogens by acting as first-line defense against extracellular pathogens, especially against intracellular pathogens, which are able to survive and replicate into phagocytic cells (44). This is true in several immunodeficiency states, particularly during HIV-1 infection.

Several mechanisms are used by phagocytic cells (monocytes/macrophages and neutrophils) to inhibit bacterial replication, including APC activity, cytokine production, and more specifically phagocytosis and killing. All these mechanisms are altered in phagocytic cells from HIV-1-infected patients, particularly during the late stages of the disease, and parallel a persistent decrease in the number of CD4 T cells (86). HIV is able to decrease activity of monocytes/macrophages and neutrophils indirectly through its viral proteins, such as gp120, p24, and Nef protein (49, 77, 82). On the other hand, HIV-1 Tat protein promotes the chemotactic activity of monocytes in vitro (1, 57).

Finally, HAART is partially able to restore phagocytic activity in HIV-1-infected patients, and thus preventing or reducing opportunistic infections related to these immunologic disorders.

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