Table 1 Comparison of V3 sequences from distinct HIV-1 biotypes

HIV-1 isolates	V3 sequences	% homology to cons. B seq.	Positive charges	Cell tropism
	*			
B Cons.	CTRPNNNTRKS..IHI..GPGRAFYTTGEIIGDIRQAHC	100	3	
BaL	----------..---..----------	100	3	M
Ada	----------..---..----------	100	3	M
JRFL	----------..---..----------	100	3	M
JRCSF	----S-----..---..----------	97	3	M
SF162	----------..-T-..-------A--D----------	91	3	M
HXB2	----------R..-R-QR------V-I-K.--NM-----	74	9	T
LAI	----------IR-QR..------V-I-K---NM-----	77	8	T
NL43	----------..-R-QR------V-I-K---NM-----	80	8	T
MN	-----Y-K--R..---..---------KN---T------	83	8	T
SF2	----------..-Y-..------H---R------K---	89	6	T

A period (.) represents a deletion; *Location where most M-tropic viruses have acidic amino acid residues whereas T-tropic strains often have basic residues[3]. Sequences were aligned according to Myers, G. *et al.*[7].

Table 2 Comparison of net charges in extracelluar domains of CXCR4 and CCR5

Extracellular domains	Net charges in the domains CXCR4	CCR5
First	−6 (1–39)	−1 (1–30)
Second	+1 (100–110)	0 (90–102)
Third	−3 (176–200)	+2 (167–198)
Fourth	−2 (262–285)	0 (261–277)
Total	−10	+1

The numbers in parentheses are the first and last residues of the named domains (from Swiss-Prot: P30991 and P51681).

more complementary negatively charged residues than those of CCR5. Comparing the sequences of these two proteins, it is apparent that the net negative charges in the extracellular domains of CXCR4 are indeed much higher than those of CCR5 (Table 2). This may explain why T-tropic viruses preferentially bind to CXCR4. It seems that in the early stages of HIV-1 infection, most of the primary isolates have M-tropic V3 sequences which preferably bind to CCR5 on M/M. In contrast, in the later stages, due to the increasing pressure of antibody response against the principal neutralizing domains (PND) in V3 loops, the sequences of V3 loops in gp120 of these viruses have to mutate into T-tropic structures positively charged residues. The outcome of this viral mutation is twofold: First, T-tropic viruses can preferably bind to CXCR4 on T lymphocytes, induce syncytium and become more virulent than M-tropic isolates. Second, T-tropic strains may escape attack by antibodies directed against the M-tropic V3 sequences.

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Transmission of *L. infantum* by blood donors

To the editor — The possible transmission of *Leishmania* via blood transfusion was raised by the World Health Organization in 1990. We screen blood from donors to the Monaco Blood Bank who live in areas endemic for *L. infantum*, checking for potential asymptomatic *Leishmania* carriers and searching for parasitemia. To date, sera of 463 donors have been analyzed by western blotting, and 61 of them exhibited 14- and/or 18-kDa bands previously shown to correlate with a positive leishmanin skin test[1]. To date, *L. infantum* promastigotes have been detected by culture in blood samples from nine donors. In some cases detection of parasites was only possible by maintaining cultures for more than three months. The necessity of such long culture periods (observed also in Balb/c infection, unpublished data) might be due to a low concentration of the autocrine growth regulating factor secreted by promastigotes[2], when the initial parasite load in the culture is low, resulting in slow proliferation. It is interesting that the blood of six (out of nine) culture-positive donors was drawn in April, before the annual emergence of a new generation of *Leishmania*-transmitting sandflies, indicating that parasitemia may occur long after infection. We also confirmed that the blood of culture-positive donors can transmit *Leishmania* to Syrian hamsters (injected with high loads of PBMCs purified from the buffy coats).

Although Monaco is an endemic area, it is difficult to prove that leishmaniasis results from a transfusion rather than from the bite of a sandfly.

Our preliminary data show that *L. infantum*, the agent of visceral leishmaniasis and common around the Mediterranean and in Brazil, circulates in the blood of asymptomatic subjects and raise important epidemiological and public health questions.

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