Entire JCV DNAs were cloned into pUC19 at the unique *Bam*HI site as described previously (Yogo et al., 1991). The resultant complete JCV DNA clones were prepared using a QIAGEN Plasmid Mini kit (QIA GEN GmbH, Hilden, Germany). Purified plasmids were used for a cycle sequencing reaction set up using the DYEnamic ET Terminator Cycle Sequencing Kit (Amersham Biosciences, Buckinghamshire, England). The primers used were a set reported previously (Agostini et al., 1997), excluding JIG-9, JIG-10, JIG-53 and SEC-11, and four additional primers: W-11 [5'- AGTTTTTGAGGGAACAGAGGAG -3', corresponding to nucleotide (nt) 283 to 310 in the JCV (Mad-1) genome (Frisque et al., 1994), W-1a (5'-CTTCGCCAGCTGTCACGTAAGGCTTCTG-3', corresponding to nt 283 to 310) and the M13 universal forward and reversal primers. DNA sequencing was performed using an automated sequencer (ABI PRISM 373S DNA sequencer, Applied Biosystems, Foster City, USA).

Phylogenetic analysis

The noncoding control region of the JCV genome was excluded from the phylogenetic analysis, as this region is hypervariable especially in JCV isolates derived from the brain and the cerebrospinal fluid of PML patients [one isolate, SA21_01, was recovered from the cerebrospinal fluid of a PML patient (Venter et al., 2004)] (Yogo and Sugimoto, 2001). DNA sequences were aligned using CLUSTAL W (Thompson et al., 1994) with a gap opening penalty of 15.00 and gap extension penalty of 6.66. To evaluate the phylogenetic relationships among DNA sequences, we employed the neighbor-joining (NJ) method (Saitou and Nei, 1987) using the CLUSTAL W program. Divergences were estimated with Kimura's two-parameter method (Kimura, 1980). To assess the confidence of branching patterns of the

NJ tree, bootstrap probabilities (BPs) were estimated with 1,000 bootstrap replicates (Felsenstein, 1985) using CLUSTAL W. A phylogenetic tree was visualized using the TREEVIEW program (Page, 1996).

Results

We sequenced 15 complete JCV (SC) DNA clones, including nine established previously (Guo et al., 1996) and six established in this study (the origins of these clones are shown in Table 1 and Figure 1). We constructed an NJ phylogenetic tree from these sequences plus 50 complete SC sequences reported previously (Table 1). The latter included seven isolates (710A to 716A) described by Cui et al. (2004), one (732A) described by Yanagihara et al. (2003) and one (SA21_01) described by Venter et al. (2004), all of which were left unclassified into subgroups. Thus, in this study, we subclassified 11 SC isolates in China, three in Vietnam, two in Malaysia, two in Myanmar, one in Indonesia, two in Mauritius, one in Zambia, one in South Africa, and one in Hawaii, USA.

According to the resultant tree (Figure 2), the SC isolates worldwide were classified into several clusters, previously designated SC-a to -f and SC-x (Saruwatari et al., 2002; Takasaka et al., 2004), with BPs ranging from 46% to 100%. Although the BP for SC-f was not high (63%), we found that all SC-f isolates examined, but none belonging to the other subgroups, carried the unique pentanucleotide deletion in the transcriptional control region (Saruwatari et al., 2002) (Table 1). Thus, it is evident that SC-f constitutes a distinct clade.

The number of isolates belonging to each SC subgroup is shown for each country in Table 2, and the findings are summarized as follows: SC-a to -e contained mainly isolates localized

to an area that included Myanmar, Thailand and southwestern China; SC-f contained those spread in all of China, southeastern Asia and southern Africa; and SC-x contained those localized to the Philippine and a single Hawaiian isolate.

Discussion

In this study, we classified SC isolates worldwide into seven subgroups (SC-a to SC-f and SC-x) using a phylogenetic analysis based on complete coding DNA sequences. As only a lower BP was obtained for the clustering of SC-f isolates in the phylogenetic tree, we confirmed this classification by detecting the unique pentanucleotide deletion in the regulatory region. This deletion has been detected in JCV isolates classified as SC-f by phylogenetic analysis, but has never been detected in those classified in the other SC subgroups (Saruwatari et al., 2002; see Table 1). It should be noted, however, that as minor isolates classified as 7B1 [or CY according to Yogo et al. (Yogo et al., 2004)] also carried this deletion (Cui et al., 2004), the presence of the pentanucleotide deletion is not sufficient to identify an isolate as belonging to SC-f.

The geographic distributions of various subgroups of SC clarified in this and previous studies (Saruwatari et al., 2002; Cui et al., 2004; Takasaka et al., 2004) are summarized as follows. Only SC-f spread throughout southeastern Asia and southern China, with some SC-f in other Asian and southern-African countries. In contrast, most of the other subgroups (i.e. SC-a to -e) remained in an area of mainland Asia, encompassing Myanmar, Thailand and southwestern China. In addition subgroup SC-x mainly occurs in the Philippines, with a single isolate belonging to this subgroup detected in Hawaii. A single isolate belonging to

SC-b was detected in Mongolia.

As there exists a close correlation between JCV genotypes and human populations (Yogo et al., 2004), based on the findings outlined in the above paragraph, we infer ancient dispersals of southeastern Asians accompanied by SC subgroups. Ancestral southeastern Asians, probably living somewhere within an area (enclosed by a circle in Fig. 1) including Myanmar Thailand and southeastern China, would have carried the proto-SC. [These ancestral southeastern Asians are assumed to have been derived from a population that migrated out of Africa (Sugimoto et al., 2002).] The ancestral Asians would have diverged into several populations each carrying a distinct subgroup of SC. Most populations, carrying SC-a to SC-e, expanded mainly within the enclosed area (Fig. 1), but a population carrying SC-f would have migrated out of this area and dispersed throughout southeastern Asia and all of China, even with migration far to southern Africa (see Fig. 1). Furthermore, a population carrying SC-x would have migrated to the Philippines and the Pacific islands, and a population carrying SC-b would have migrated to Mongolia.

Acknowledgements

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Table 1. Origins of JCV isolates whose complete DNA sequences were used in the phylogenetic analysis (Figure 1)

Geographic origin (site no. [a])	Ethnic origin	Subgroup[b]	Isolates	5-bp deletion[c]	Accession no.[d]	Ref.[e]
Manzhouli, China (1)	Mongolian	SC-f	714A	+	AF300964	A
Chifeng, China (2)	Mongolian	SC-f	711A	+	AF300961	A
Chifeng, China (2)	Han	SC-f	713A	+	AF300963	A
Shenyang, China (3)	Han	SC-f	715A	+	AF300965	A
Kunming, China (4)	Dai	SC-f	710A	+	AF300960	A
Kunming, China (4)	Dai	SC-f	712A	+	AF300962	A
Kunming, China (4)	Dai	SC-a	716A	-	AF300966	A
Taipei, China (5)	Chinese	SC-f	C1	+	AB198940	–
Taipei, China (5)	Chinese	SC-f	C3	+	AB077873	B
Wuhan, China (6)	Chinese	SC-f	CW-2	+	AB048579	C
Wuhan, China (6)	Chinese	SC-f	CW-7	+	AB198942	–
Wuhan, China (6)	Chinese	SC-f	CW-10	+	AB077872	B
Wuhan, China (6)	Chinese	SC-f	CW-11	+	AB198943	–
Urumqi, China (7)	Chinese	SC-f	UR-8	+	AB198941	–
Ulaanbaatar, Mongolia (8)	Mongolian	SC-b	MO-11	-	AB048582	C
Hanoi, Vietnam (9)	Vietnamese	SC-f	Han-1	+	AB198944	–
Hanoi, Vietnam (9)	Vietnamese	SC-f	Han-3	+	AB198945	–
Hanoi, Vietnam (9)	Vietnamese	SC-f	Han-4	+	AB198946	–
Chiang Mai, Thailand (10)	Thai	SC-a	TL-2	-	AB077855	B
Chiang Mai, Thailand (10)	Thai	SC-a	TL-5	-	AB077856	B
Chiang Mai, Thailand (10)	Thai	SC-c	TL-7	-	AB077858	B
Yangon, Myanmar (11)	Burmese	SC-f	MN-3	+	AB077879	B
Yangon, Myanmar (11)	Burmese	SC-d	MN-6	-	AB077866	B
Yangon, Myanmar (11)	Burmese	SC-d	MN-7	-	AB077867	B
Yangon, Myanmar (11)	Burmese	SC-e	MN-11	-	AB077871	B
Chaungtha Beach, Myanmar (12)	Burmese	SC-e	CH-2	-	AB077869	B
Chaungtha Beach, Myanmar (12)	Burmese	SC-f	CH-7	+	AB077875	B
Chaungtha Beach, Myanmar (12)	Burmese	SC-f	CH-17	+	AB077870	B

Myitkyina, Myanmar (13)	Burmese	SC-c	MT-1	-	AB077859	B
Myitkyina, Myanmar (13)	Burmese	SC-c	MT-2	-	AB077860	B
Myitkyina, Myanmar (13)	Burmese	SC-f	MT-10	+	AB077874	B
Myitkyina, Myanmar (13)	Burmese	SC-f	MT-14	+	AB077861	B
Myitkyina, Myanmar (13)	Burmese	SC-f	MT-15	+	AB077857	B
Myitkyina, Myanmar (13)	Burmese	SC-e	MT-22	-	AB077868	B
Tiddim, Myanmar (14)	Burmese	SC-d	TD-4	-	AB077862	B
Tiddim, Myanmar (14)	Burmese	SC-d	TD-6	-	AB077863	B
Tiddim, Myanmar (14)	Burmese	SC-d	TD-15	-	AB077864	B
Tiddim, Myanmar (14)	Burmese	SC-d	TD-19	-	AB077865	B
Peinnebeen, Myanmar (15)	Burmese	SC-f	PB-3	+	AB077876	B
Peinnebeen, Myanmar (15)	Burmese	SC-f	PB-4	+	AB077877	B
Peinnebeen, Myanmar (15)	Burmese	SC-f	PB-5	+	AB077878	B
Rakhine, Myanmar (16)	Burmese	SC*	RH-2	-	AB198947	–
Rakhine, Myanmar (16)	Burmese	SC-f	RH-5	+	AB198948	–
Masai, Malaysia (17)	Malaysian	SC-f	ML-2	+	AB198950	–
Masai, Malaysia (17)	Malaysian	SC-f	ML-4	+	AB198951	–
Masai, Malaysia (17)	Malaysian	SC-f	ML-6	+	AB048581	C
Jakarta, Indonesia (18)	Indonesian	SC-f	ID-1	+	AB048580	C
Jakarta, Indonesia (18)	Indonesian	SC-f	ID-2	+	AB198949	–
Luzon, Philippines (19)	Filipino	SC-f	Luz-1	+	AB113125	D
Luzon, Philippines (19)	Filipino	SC-f	Luz-2	+	AB113132	D
Luzon, Philippines (19)	Filipino	SC-f	Luz-3	+	AB113134	D
Luzon, Philippines (19)	Filipino	SC-x	Luz-18	-	AB113130	D
Luzon, Philippines (19)	Filipino	SC-x	Luz-19	-	AB113131	D
Luzon, Philippines (19)	Filipino	SC-x	Luz-20	-	AB113133	D
Cebu, Philippines (20)	Filipino	SC-f	Ceb-1	+	AB113118	D
Cebu, Philippines (20)	Filipino	SC-f	Ceb-2	+	AB113122	D
Cebu, Philippines (20)	Filipino	SC-f	Ceb-4	+	AB113123	D
Cebu, Philippines (20)	Filipino	SC-x	Ceb-14	-	AB113119	D
Cebu, Philippines (20)	Filipino	SC-x	Ceb-15	-	AB113120	D
Cebu, Philippines (20)	Filipino	SC-x	Ceb-16	-	AB113121	D

Kwazulu-Natal, South Africa (21)		SC-f	SA21_01	+	AY536239	E
Lusaka, Zambia (22)	Zambian	SC-f	ZA-3	+	AB198952	–
Port Louis, Mauritius (23)	Mauritian	SC-f	MU-4	+	AB198953	–
Port Louis, Mauritius (23)	Mauritian	SC-f	MU-7	+	AB198954	–
Hawaii, USA (24)	Hawaiian	SC-x	732A	-	AF396427	F

[a]Indicated in Figure 1.

[b]Determined based on the phylogenetic analysis (Figure 2).

[c]Presence (+) or absence (-) of the pentanucleotide deletion spanning nucleotides nt 218 to 222 [these nucleotide numbers were those of the archetype (Yogo et al., 1990)] in the transcriptional control region.

[d]GenBank/EMBL/DDBJ accession numbers.

[e]References: A, Cui et al., 2004; B, Saruwatari et al., 2002; C, Sugimoto et al., 2002; D, Takasaka et al., 2004; E, Venter et al., 2004; F, Yanagihara et al., 2003; –, this study.

Table 2. Distribution of various SC subgroups in the world

Geographic origin	Total no. of isolates analyzed	No. of isolates classified as:							
		SC-x	SC-a	SC-b	SC-c	SC-d	SC-e	SC-f	Others
China	14	0	1	0	0	0	0	13	0
	(11)		(1)					(10)	
Vietnam	3	0	0	0	0	0	0	3	0
	(3)							(3)	
Thailand	3	0	2	0	1	0	0	0	0
Myanmar	22	0	0	1	3	6	4	7	1
	(2)							(1)	(1)[b]
Malaysia	3	0	0	0	0	0	0	3	0
	(2)							(2)	
Indonesia	2	0	0	0	0	0	0	2	0
	(1)							(1)	
Philippines	12	6	0	0	0	0	0	6	0
Mongolia	1	0	0	1	0	0	0	0	0
Mauritius	2	0	0	0	0	0	0	2	0
	(2)							(2)	
Zambia	1	0	0	0	0	0	0	1	0
	(1)							(1)	
South Africa	1	0	0	0	0	0	0	1	0
	(1)							(1)	
Hawaii, USA	1	1	0	0	0	0	0	0	0
	(1)	(1)							
Total	65	7	3	2	4	6	4	38	1
	(24)	(1)						(21)	(1)

[a]Numbers within parentheses indicate those of isolates subgrouped in the present study.

[b]One isolate (RH-2) was grouped as others, as this isolate clustered with no other isolate in the present analysis (Figure 1).

Figure Legends

Figure 1. Locations of the sites of sample collection and proposed migrations carrying SC-f. Dots indicate the sites where samples (usually urine) were collected, and numbers beside the dots indicate the site numbers (see Table 1). One site (Hawaii; site no, 24) is not shown. We assumed that populations carrying SC-a to -e expanded within the enclosed area, while those with SC-f migrated out of this area and dispersed in various directions shown by arrows (see text).

Figure 2. NJ phylogenetic tree relating 65 complete JCV (SC) DNA sequences. A phylogenetic tree was constructed from complete sequences, excluding regulatory sequences, using the NJ method. The phylogenetic tree was visualized using the TREEVIEW program. The tree was rooted using isolate CY as the outgroup, as this genotype is a distinct genotype closely related to SC (Sugimoto et al., 2002). The symbols for sequences are shown in Table 1. The numbers at nodes in the tree indicate BPs (percent) obtained by 1,000 replicates (only those for major clusters are shown). SC subgroups (SC-a to SC-f and SC-x) are indicated. Isolates whose sequences were determined in this study are shown in white on a black background, while those whose sequences were determined previously but left unclassified into subgroups are shown in white on a grey background.

Figure 1

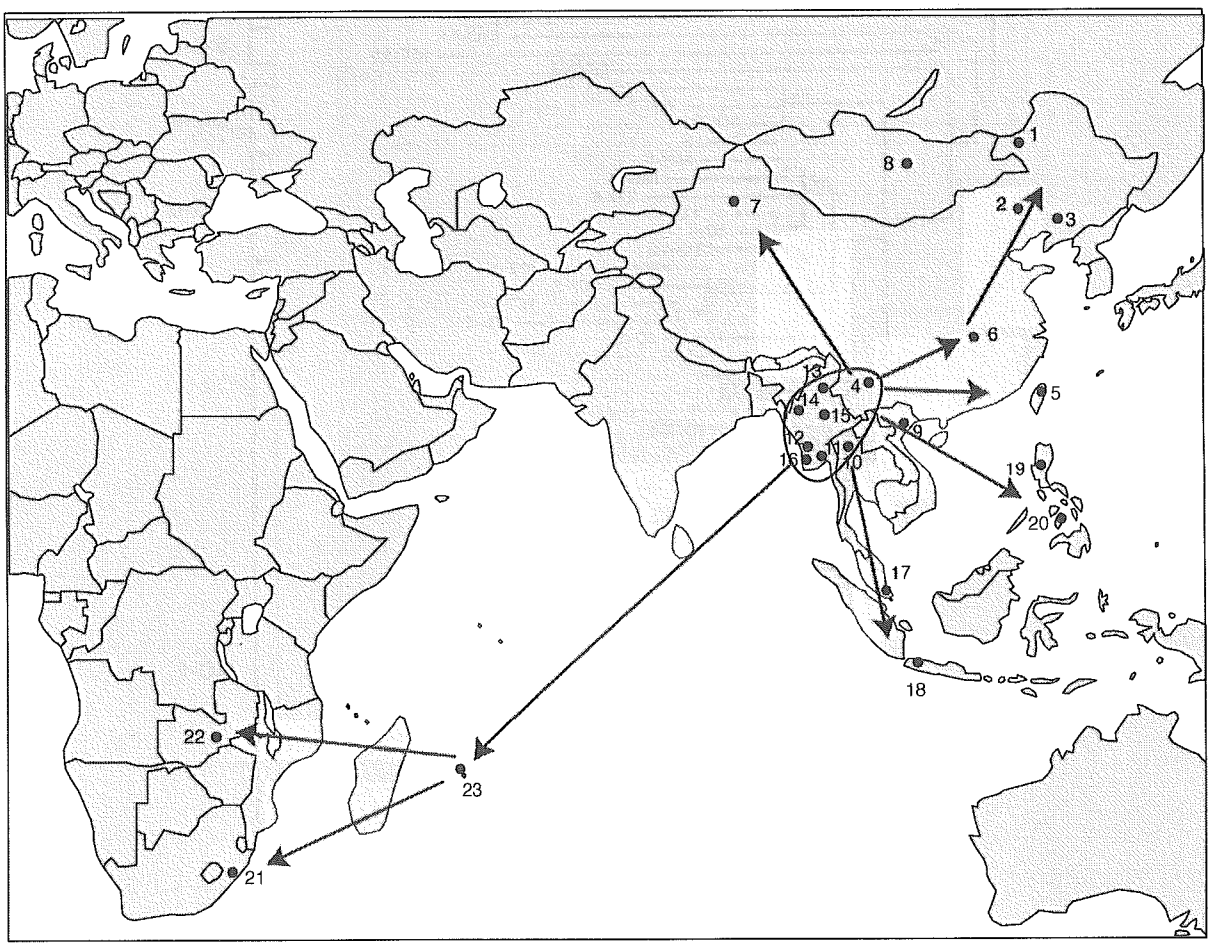
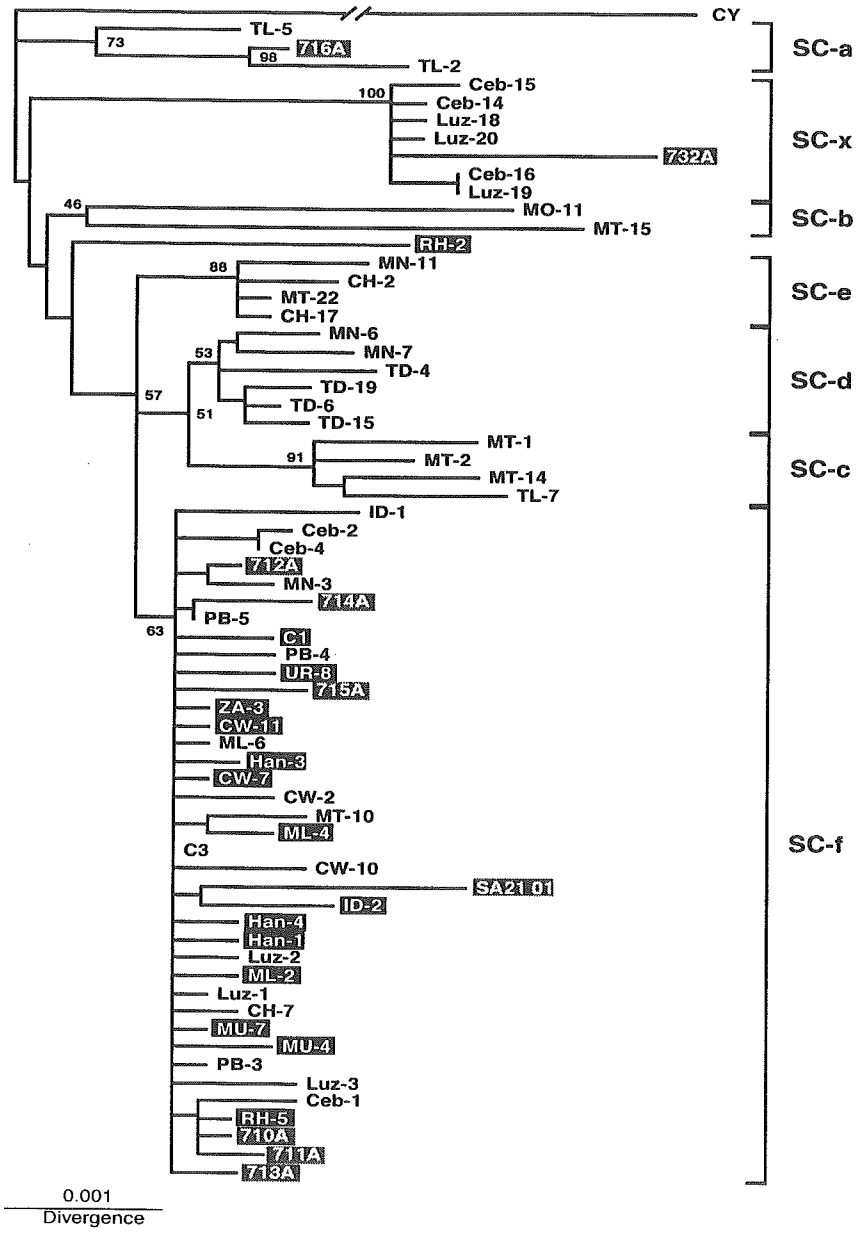


Figure 2


CY
TL-5
73
716A
98
TL-2
100
Ceb-15
Ceb-14
Luz-18
Luz-20
732A
Ceb-16
Luz-19
46
MO-11
MT-15
RH-2
88
MN-11
CH-2
MT-22
CH-17
MN-6
53
MN-7
TD-4
57
TD-19
51
TD-6
TD-15
MT-1
91
MT-2
MT-14
TL-7
ID-1
Ceb-2
Ceb-4
712A
MN-3
714A
PB-5
63
C1
PB-4
UR-8
715A
ZA-3
CW-11
ML-6
Han-3
CW-7
CW-2
MT-10
ML-4
C3
CW-10
SA21 01
ID-2
Han-4
Han-1
Luz-2
ML-2
Luz-1
CH-7
MU-7
MU-4
PB-3
Luz-3
Ceb-1
RH-5
710A
711A
713A
SC-a
SC-x
SC-b
SC-e
SC-d
SC-c
SC-f
0.001
Divergence

平成１７年度論文（HIV 感染症関連のみ）

1）Mitsuru Konishi,, Ken Takahashi, Eiichiro Yoshimoto, Kenji Uno, Kei Kasahara, Keiichi Mikasa：Association between osteopenia/osteoporosis and the serum RANKL in HIV-infected patients. AIDS 19：1240－1241, 2005.

2）Shimura M, Tokunaga K, Konishi M, Sato Y, Kobayashi C, Sata T, Ishizaka Y：Premature sister chromatid separation in HIV-1-infected peripheral blood lymphocytes. AIDS 19：1434－1438, 2005.

3）古西　満：HIV/AIDS 患者・家族のための緩和医療―抗 HIV 治療確立前後の比較―. 死の臨床 28：27－29, 2005.

4）Mitsuru Konishi, Eiichiro Yoshimoto, Kenji Uno, Kei Kasahara, Keiichi Mikasa：A case of pulmonary *Mycobacterium avium* infection with growing nodules after commencing highly active antiretroviral therapy. Respir Med Extra 1：93－95, 2005.

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Association between osteopenia/osteoporosis and the serum RANKL in HIV-infected patients

The association between osteopenia/osteoporosis and HIV infection, identified unexpectedly in many patients who received dual-energy X-ray absorptiometry to assess lipodystrophy, has become the focus of attention in the past few years [1]. Many reports have indicated that HIV-related osteopenia/osteoporosis was associated with antiretroviral therapy. However, the underlying mechanism triggering bone mineral loss in HIV-infected patients has yet to be defined. Recently, the tumor necrosis factor family molecule RANKL (receptor activator of nuclear factor κB ligand) and its receptor RANK have been recognized as important regulators of bone remodeling, and are considered essential for the development and activation of osteoclasts [2].

The aim of this study was to define the role of RANKL in the development of HIV-related osteopenia/osteoporosis. We measured bone mineral density (BMD), the concentration of urinary deoxypyridinoline, which was a marker of bone resorption, and the serum RANKL in 39 HIV-seropositive men (mean age 41.4 years). Eight patients did not receive any antiretroviral therapy and 15 were on antiretroviral therapy including protease inhibitors. BMD was measured at the L2–4 vertebrae level using a dual-energy X-ray absorptiometer (DPX-L; Lunar Radiation Corp., USA). A fasting blood sample and a second morning voided urine sample were obtained and stored at −80° until analysed. The concentration of urinary deoxypyridinoline was measured using a sandwich enzyme-linked immunosorbent assay kit (Quidel Co., USA). The sample results from a single urine collection were normalized for urine dilution by urine creatinine analysis. The serum level of RANKL was measured using a sandwich enzyme-linked immunosorbent assay kit (Pepro Tech Inc., UK), the performance of which was characterized within 32–2000 pg/ml.

The mean BMD of the lumbar spine was 1.079 g/cm^2 (range 0.779–1.229 g/cm^2). Osteoporosis was detected in one patient (2.6%) and osteopenia in nine patients (23.1%) according to the World Health Organization (WHO) criteria of osteopenia/osteoporosis. The mean urinary deoxypyridinoline level was 6.25 nmol/mmol creatinine (range 3.4–12.1 nmol/mmol creatinine). This bone turnover marker showed a bone resorption pattern in 20 patients (51.3%). The mean serum RANKL concentration was 199.7 pg/ml (range 32.6–781.0 pg/ml). The mean serum RANKL levels were 241.0 pg/ml (SD 235.5) in patients on highly active antiretroviral therapy (HAART), 106.7 pg/ml (SD 102.3) in patients not on HAART. The serum RANKL levels were thus significantly higher in HAART-treated patients than in non-HAART-treated or untreated patients ($P < 0.05$). The serum RANKL levels were inversely proportional to the BMD of the lumbar spine ($r = 0.57$, $P < 0.01$). Furthermore, the serum RANKL levels positively correlated with the urinary deoxypyridinoline concentration (Figure 1).

There have been increasing numbers of reports on the high incidence of osteopenia/osteoporosis in HIV-infected patients receiving antiretroviral treatment. The epidemiology of these complications, however, remains undefined. Our results demonstrate that RANKL may correlate with the development of osteopenia/osteoporosis in HIV-infected patients. The binding of RANKL to RANK on the pre-osteoclasts initiates the differentiation and proliferation of these cells, and promotes osteoclast fusion and activation [2]. Therefore, the activation of the RANKL–RANK pathway is recognized

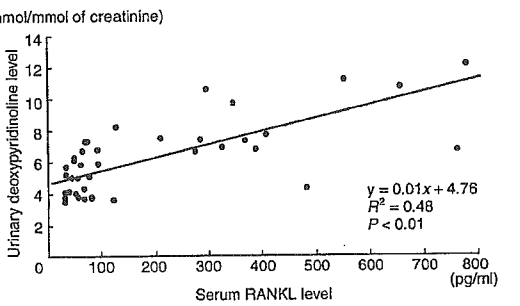

Fig. 1. Correlation between the serum RANKL level and the urinary deoxypyridinoline level. The serum RANKL levels positively associated with the urinary deoxypyridinoline concentration.

as one of the mechanisms of development of osteopenia/osteoporosis. It has been reported that the production of RANKL by the peripheral blood mononuclear cells is significantly greater in patients with the postmenopausal osteoporosis [3]. A phase II trial recently indicated that the human monoclonal antibody to RANKL significantly increases BMD in postmenopausal women with low BMD within one month. RANKL is expressed not only on osteoblast/stromal cells but also on activated T cells. Fakruddin and Laurence [4] found that soluble HIV-1 envelope gp120 could promote osteoclastogenesis and bone resorption via the induction of RANKL production by T cells *in vitro*, and that certain protease inhibitors might remove the normal physiological block to RANKL overactivity. Furthermore, they reported that the HIV-1 accessory protein Vpr mediated the upregulation of RANKL [5].

In conclusion, we suggest that RANKL may act as an important mechanism of development of osteopenia/osteoporosis in HIV-infected patients, and that HIV itself or some antiretroviral drugs may produce RANKL by activated T cells.

Mitsuru Konishi, Ken Takahashi, Eiichiro Yoshimoto, Kenji Uno, Kei Kasahara and Keiichi Mikasa, *Center for Infectious Diseases, Nara Medical University, Nara, Japan.*

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Response to 'Limited benefit of antiretroviral resistance testing in treatment-experienced patients: a meta-analysis'

We read with interest about the meta-analysis performed by Panidou *et al.* [1] in a previous issue of *AIDS*, which appeared to demonstrate the limited utility of antiretroviral resistance testing and expert advice. We believe that this meta-analysis has several limitations, and that the conclusions reached by the authors cannot be generalized to all clinical situations. First, the analysis was not controlled for the number of active drugs available to the patients. More recent studies have documented this variable as a genotypic or phenotypic sensitivity score, which is unmeasured in the standard of care arms. In addition, studies included in the meta-analysis had a high proportion of patients who had failed several regimens and had very few active agents available to them, especially in the era studied 1997–1998 [2–4].

We have experience reviewing over 1000 genotypes for a large urban public HIV clinic in the United States. In our clinic, approximately one patient in four tested by genotypic resistance testing (GRT) has complete resistance to two or more classes of medications. The lack of useful agents would dampen the ability of any resistance testing method to affect virological outcome as we recently demonstrated [5]. Our experience suggests that patients who have failed one or two regimens benefit more from GRT than patients with a greater number of previous regimens, which logically correlates with a decreasing number of available antiretroviral agents. Similarly, there appears to be a range of the number of previous regimen failures in which expert advice contributes more and more as medication choices diminish, and then decreases again as the number of fully active choices dwindles towards zero. In our study, we demonstrated a highly statistically significant benefit for GRT plus expert advice versus GRT alone [6]. The study was not reviewed for meta-analysis, as it was a retrospective study. However, the study represents real world outcomes in our clinic. We agree with Panidou *et al.* [1] that phenotypic antiretroviral resistance testing (PART) is not very useful, and suspect that this is due to the tendency of PART and virtual PART to overestimate the activity of some nucleosides, as demonstrated by Haubrich *et al.* [7].

In conclusion, our experience suggests that GRT and expert advice are most useful for patients with limited antiretroviral exposure and fewer resistance mutations in choosing antiretroviral regimens for patients failing virologically on highly active antiretroviral therapy. Further studies are needed better to define the utility of GRT in patients who are highly drug experienced.

Sheila M. Badri[a,b,c], Oluwatoyin M. Adeyemi[a,b,c], Blake E. Max[c,d] and David E. Barker[a,b,c], *[a]Division of Infectious Diseases, John H. Stroger, Jr. Hospital of Cook County, Chicago, IL, USA; [b]Rush Medical College, Chicago, IL, USA; [c]CORE Center, Cook County Bureau of Health, Chicago, IL, USA; and*

compared with four in the experienced group. Four of these five patients had HCV co-infection. Two events arose after one month of treatment and the other three after a year, confirming the multifaceted mechanisms causing this toxicity. In all these cases the treatment had to be stopped, and the patients regressed.

To the best of our knowledge, this study comprises the biggest series to date of patients treated with lopinavir/ritonavir and followed prospectively outside clinical trials. In addition, this HIV-positive population had a high prevalence of co-infection with hepatitis viruses.

The frequency of hepatotoxicity was actually low, unlike in other studies. This might partly be the result of methodological differences, reflecting how the data were collected. Retrospective studies can suffer major selection bias. Gonzalez-Requena *et al.* [11] also reported a low incidence of adverse events, but their case series was small and was followed up for not more than one year.

In conclusion, the present study found that lopinavir/ritonavir caused only limited hepatic toxicity in this population of HIV-positive patients with a high prevalence of co-infection with hepatitis B virus or HCV.

The CISAI Study Group

Coordination: T. Quirino, P. Bonfanti, G.M. Vigevani, F. Parazzini, E. Ricci

Recruitment sites and investigators: R. Cinelli, U. Tirelli (Aviano); G. Cocca, G. Rizzardini (Busto Arsizio); C. Grosso, A. Stagno (Cesena); L. Pusterla, D.Santoro (Como); C. Magnani, P. Viganò (Cuggiono); S. Carradori, F. Ghinelli (Ferrara); F. Vichi, F. Mazzotta (Firenze, S. Maria Annunziata); C. Martinelli, F. Leoncini (Firenze, Careggi); G. Penco, G. Cassola (Genova); S. Miccolis, A. Scalzini (Mantova); S. Landonio, M. (I Divisione, Ospedale Sacco, Milano); L. Valsecchi, L.Cordier, A. Cargnel (II Divisione, Ospedale Sacco, Milano); T. Bini, S. Melzi, M. Moroni (Clinica Malattie Infettive, Ospedale Sacco, Milano); E. Rosella, G. Fioni (Milano); M. Gargiulo, A. Chirianni (Napoli); M. Franzetti, P. Cadrobbi (Padova); C. Sfara, G. Stagni (Perugia); G. Parruti, G. Marani Toro (Pescara); B. Adriani, A. Paladini (Prato); G. Madeddu, M.S. Mura (Sassari); G. Liuzzi, A. Antinori (Roma); G. Orofino, P. Caramello (Torino); G. Cristina, F. Carcò (Vercelli); D. Migliorini, O. Armignacco (Viterbo).

[a]*I Divisione di Malattie Infettive, Ospedale Luigi Sacco, Milan, Italy;* [b]*Divisione di Malattie Infettive, Ospedale Galliera, Genoa, Italy;* [c]*Divisione A di Malattie Infettive, Ospedale Amedeo di Savoia, Turin, Italy;* [d]*Clinica di Malattie Infettive, Ospedale Luigi Sacco, Milan, Italy;* [e]*Clinica di Malattie Infettive, Perugia, Italy;* [f]*Divisione di Malattie Infettive, Cremona, Italy;* [g]*Divisione di Malattie Infettive, Vercelli, Italy; and* [h]*Divisione di Malattie Infettive, Busto Arsizio, Italy.*

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Premature sister chromatid separation in HIV-1-infected peripheral blood lymphocytes

Mari Shimura[a], Kenzo Tokunaga[b], Mitsuru Konishi[c], Yuko Sato[d], Chizuko Kobayashi[e], Tetsutaro Sata[b] and Yukihito Ishizaka[a]

To investigate the mechanism of aneuploidy that is frequently observed in AIDS, we examined premature sister chromatid separation (PCS), a sign of genomic instability, in peripheral blood cells of HIV-1-infected individuals. PCS was found in all