Regional pattern of the molecular types of *Cryptococcus neoformans* and *Cryptococcus gattii* in Brazil

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The molecular types of 443 Brazilian isolates of Cryptococcus neoformans *and* Cryptococcus gattii *were analyzed to determine their geographic distribution within Brazil and their underlying host conditions. The following data, imported from previous epidemiological studies as well as two culture collections, were analyzed for: place of isolation, source (clinical or environmental), host risk factors, species, serotype, mating type, and molecular type. Molecular typing by PCR-fingerprinting using primers for the minisatellite-specific core sequence of the wild-type phage M13 or microsatellites [$(GACA)_4$, $(GTG)_5$], restriction fragment length polymorphism of* URA5 *gene analysis, and/or amplified fragment length polymorphism (AFLP) identified eight major genotypes: VNI/AFLP1, VNII/AFLP1A, VNIII/AFLP2, and VNIV/AFLP3 for* C. neoformans, *and VGI/AFLP4, VGII/AFLP6, VGIII/AFLP5, and VGIV/AFLP7 for* C. gattii. *The most common molecular type found in Brazil was VNI (64%), followed by VGII (21%), VNII (5%), VGIII (4%), VGI and VNIV (3% each), and VNIII (< 1%). Primary cryptococcosis caused by the molecular type VGII (serotype B, MATα) prevails in immunocompetent hosts in the North and Northeast regions, disclosing an endemic regional pattern for this specific molecular type in the Northern Brazil.*

Key words: *Cryptococcus neoformans* - *Cryptococcus gattii* - molecular types - epidemiology - Brazil

Cryptococcosis is a life-threatening, systemic mycosis affecting humans and animals. It is acquired by inhalation of viable propagules from the environment, and the most common clinical manifestation of cryptococcosis is meningoencephalitis (Lin & Heitman 2006). Cryptococcosis is caused by two different species of the genus *Cryptococcus*: *C. neoformans* (serotypes A, D, and hybrid AD) and *C. gattii* (serotypes B and C) (Kwon-Chung et al. 2002). While *C. neoformans* infections occur worldwide and are an important cause of morbidity and mortality in immunocompromised hosts (especially AIDS-patients), *C. gattii* usually infects normal hosts (Kwon-Chung & Bennett 1992, Lazéra et al. 2005) and is considered to be a tropical disease. The ongoing Vancouver Island outbreak in a temperate climate, however, suggests that *C. gattii* can adapt to new environments (Kidd et al. 2004).

In Brazil, cryptococcosis caused by *C. neoformans* occurs in all regions; however, *C. gattii* behaves as a primary pathogen infecting native immunocompetent hosts and mainly infects young people and children in the North and Northeast (NE) regions of Brazil. In this group of patients, the infection is characterized by a high lethality rate that ranges from 40.6% to 56% (Correa et al. 1999, Lazéra et al. 2005) and frequently causes incapacity (e.g., visual deficits or blindness) and hydrocephalus (Rozenbaum & Gonçalves 1994, Nishikawa et al. 2003). Brazilian epidemiological data suggest a geographic, macroregional, north-south trend in *C. gattii* infections in Brazil. The Northern macro region (NM), which is comprised of the states of Amazonas, Roraima, Pernambuco, Piauí, and Bahia, is endemic for *C. gattii*. The Southern macro region (SM), which are represented by the states of Mato Grosso do Sul, Minas Gerais, São Paulo (SP), Rio de Janeiro (RJ), Paraná, and Rio Grande do Sul (RS), show sporadic infections by *C. gattii*.

Serotyping was largely used for epidemiological studies of *C. gattii* and *C. neoformans*, but the lack of an available commercial serotyping kits and the search for a more reliable technique led to an increased use of molecular tools. Additionally, the analysis of genotypes within a species could answer several questions that may impact management, therapy, surveillance, and prophylactic actions. An attempt to standardize a technique for a global molecular epidemiological survey of the agents of cryptococcosis identified eight major molecular types via PCR fingerprinting with the minisatellite-specific core sequence of the wild-type phage M13 or microsatellites [$(GACA)_4$, $(GTG)_5$]. Thus, *C. neoformans* was grouped into the types VNI (serotype A), VNII (serotype A), VNIII (serotype AD), and VNIV (serotype D); *C. gattii* was grouped into the types VGI, VGII, VGIII, and VGIV (serotypes B and C) (Meyer et al. 1999). This

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grouping has been confirmed by others using different techniques, such as amplified fragment length polymorphism analysis (AFLP) (Boekhout et al. 2001), restriction fragment length polymorphism analysis (RFLP) of *URA5* and *PLB1* genes (Latouche et al. 2003, Meyer et al. 2003), and multilocus sequence typing (MLST) (Litvintseva et al. 2006). Recently, AFLP and MLST have revealed the existence of a new molecular type of *C. neoformans* in Botswana (genotype VNB) that seems to be geographically restricted to sub-Saharan Africa (Litvintseva et al. 2006).

Although several previous studies have analyzed the molecular types of Brazilian isolates according to the previously described typing system (VNI/AFLP1, VNII/AFLP1A, VNIII/AFLP2, VNIV/AFLP3, VGI/AFLP4, VGII/AFLP6, VGIII/AFLP5, and VGIV/AFLP7 types), they demonstrated only the presence of *C. neoformans* and *C. gattii* in certain cities or states (Casali et al. 2003, Igreja et al. 2004, Abegg et al. 2006, Matsumoto et al. 2007). Even when more than one region was analyzed, the number of isolates was limited (Trilles et al. 2003).

In the current study, the main objectives were to obtain an up-to-date picture of the molecular type distribution of *C. gattii* and *C. neoformans* in Brazil and correlate the genotypes to geographic regions and host conditions by reanalyzing all of data from previously published cryptococcal strains and combining them with data obtained from new strains using statistics and descriptive analysis.

MATERIALS AND METHODS

Data from 356 Brazilian isolates were imported from different sources: (i) previous epidemiological studies (Boekhout et al. 2001, Casali et al. 2003, Trilles et al. 2003, Meyer et al. 2003, Igreja et al. 2004, Katsu et al. 2004, Ribeiro et al. 2006), (ii) the database of the Cryptococcal Culture Collection at Laboratório de Micologia, Instituto de Pesquisa Clínica Evandro Chagas, RJ, and (iii) the database of the Australian Medical Fungal Collection, at the Molecular Mycology Research Laboratory at Westmead Hospital, Sydney, Australia.

The data collected for the current analysis included: place of isolation, source (clinical or environmental), host risk factors, species, serotype, mating type, and molecular type identified by PCR-fingerprinting, *URA5*-RFLP according to Meyer et al. (2003), or AFLP according to Boekhout et al. (2001). In addition to the imported data described above, we determined the mating type, serotype, molecular type, and polymorphisms of additional 87 *C. gattii* and *C. neoformans* isolates.

Serotyping - Serotyping was carried out using the slide agglutination test according to the manufacturer's instructions (Crypto Check Iatron RM 304-K kit; Iatron Laboratories, Tokyo, Japan).

Molecular typing - The 87 clinical and environmental Brazilian isolates were typed by *URA5*-RFLP and PCR-fingerprinting using the minisatellite-specific core sequence of the wild-type phage M13. The following standard strains representing each molecular type were included in the analysis: WM 148 (serotype A, VNI/AFLP1), WM 626 (serotype A, VNII/AFLP1A), WM 628 (serotype AD, VNIII/AFLP2), WM 629 (serotype D, VNIV/AFLP3), WM 179 (serotype B, VGI/AFLP4), WM 178 (serotype B, VGII/AFLP6), WM 175 (serotype B, VGIII/AFLP5), and WM 779 (serotype C, VGIV/AFLP7) (Meyer et al. 2003). The DNA was extracted as previously described (Meyer et al. 1999). The isolates were grown on Sabouraud's dextrose agar at 37°C for 48 h. The tube containing the yeast cell pellet was frozen in liquid nitrogen. The pellet was ground with a miniature pestle, and 500 µl of cell lysis solution (0.5% sodium dodecyl sulfate, 1.4% NaCl, 0.73% EDTA, and Tris-HCl 0.2M) was added to the frozen, ground cells. The tubes were incubated for 5 min at room temperature with constant shaking, and 500 µl phenol:chloroform:isoamyl alcohol (v:v:v 25:24:1) was added and mixed thoroughly for 2 min to obtain a homogenous suspension. The tubes were centrifuged for 20 min at 16,110 *g*. After centrifugation, the upper aqueous layer was transferred to a new tube, an equal volume of chloroform:isoamyl alcohol (v:v 24:1) was added, and the mixture was shaken and centrifuged. To precipitate the genomic DNA, an equal volume of isopropanol was added to the supernatant. The mixture was then gently shaken and incubated at -20°C for at least 1 h or overnight. The DNA pellet was washed with 70% ethanol and suspended in 200 µl sterile, deionized water at 4°C overnight.

PCR of the *URA5* gene was performed in a final volume of 50 µl. Each reaction contained 50 ng of DNA, 1 X PCR buffer [160 mM $(NH_4)_2SO_4$, 670 mM Tris-HCI (pH8.8 at 25°C), 0.1% Tween-20 - Bioline], 0.2 mM each of dATP, dCTP, dGTP, and dTTP (Roche Diagnostics GmbH), 3 mM magnesium chloride, 1.5 U BioTaq DNA polymerase (Bioline), and 50 ng of each primer URA5 (5' ATGTCCTCCCAAGCCCTCGACTCCG 3') and SJ01 (5' TTAAGACCTCTGAACACCGTACTC 3') (Meyer et al. 2003). PCR was performed for 35 cycles in a Perkin-Elmer thermal cycler (model 480) at 94°C with a 2 min initial denaturation, 45 s denaturation at 94°C, 1 min annealing at 61°C, 2 min extension at 72°C, and final extension cycle for 10 min at 72°C. A total of 30 µl of PCR products were double digested with *Sau*96I (10 U/µl) and *Hha*I (20 U/µl) for 3 h, and the fragments were separated by 3% agarose gel electrophoresis at 100 V. RFLP patterns were assigned visually by comparison with patterns obtained from standard strains (VNI-VNIV and VGI-VGIV).

PCR-fingerprinting reactions were carried out in a volume of 50 µl containing 25 ng genomic DNA, 10 mM Tris-HCl, pH 8.3, 50 mM KCl, 1.5 mM MgCl, 0.2 mM each of the dATP, dCTP, dGTP, and dTTP (Roche Diagnostics GmbH, Mannheim, Mannheim, Germany), 3 mM magnesium acetate, 30 ng primer (5' GAGGGTGGCGGTTCT 3'), and 2.5 U Amplitaq DNA polymerase (Applied Biosystems, Foster City, CA). PCR was performed for 35 cycles in a Perkin-Elmer thermal cycler (model 480) with a 20 s denaturation at 94°C, 1 min annealing at 50°C, 20 s extension at 72°C, and final extension cycle for 6 min at 72°C. Amplification products were concentrated to a volume of approximately 15 µl and separated by electrophoresis on 1.4% agarose gels stained with

ethidium bromide in 1 X Tris-borate-EDTA (TBE) buffer at 70 V for 9 h, and they were visualized under a UV light (Meyer et al. 2003). PCR-fingerprinting profiles were visually compared to the standard strains to determine the molecular types. The genetic relationships of the isolates were analyzed using the 1D gel analysis module (BioGalaxy, BioAware, Hannut, Belgium) in BioloMICS version 7.5.30 (BioAware). Similarity coefficients were calculated by using the Dice algorithm, and cluster analyses were performed by the Unweighted Pair Group Method with the Arithmetic mean (UPGMA).

AFLP analysis was performed according to the AFLP Microbial Fingerprinting Protocol of the manufacturer (Applied Biosystems), and we used *Mse*I and *Eco*RI for the DNA restriction digestion (Boekhout et al. 2001). The restriction ligation was performed using 1 unit *Mse*I, 5 units *Eco*RI, and 3 units T4 DNA ligase. The first PCR procedure was performed with two preselective primers (*Eco*RI core sequence and *Mse*I core sequence) and the AFLP Amplification Core Mix from the AFLP Microbial Fingerprinting Kit according to the manual. A second PCR procedure used more selective primers (*Eco*RI-AC FAM and *Mse*I-G).

Mating type - The mating type was determined by PCR. MAT alpha-specific mating type primers were MatalphaF (5' CTTCACTGCCATCTTCACCA 3') and MatalphaR (5' GACACAAAGGGTCATGCCA 3'), and the amplification reactions were carried out according to the procedure in Chaturvedi et al. (2000). MAT a-specific mating type primers were MFa2U (5' ACACCGCCTGTTACAATGGAC 3') and MFa2L (5' CAGCGTTTGAAGATGGACTTT 3') (Fraser et al. 2003), and the amplifications reactions were performed according to the procedure in Halliday and Carter (2003).

Statistical analysis - Statistical analysis of 443 *C. neoformans* and *C. gattii* isolates was performed using SPSS version 11.0 software, and a p-value < 0.05 was used to define significance. The most representative molecular types in terms of the numbers of isolates were included in the logistical analysis. Univariate analysis was performed using the Fisher's exact and chi-square tests. Odds ratio (OD) and 95% confidence intervals was calculated to assess the univariate risk of a particular molecular type or species occurring in a certain geographic area. The variables selected for the current study were source, host factors, and region (NM or SM). Host factors were analyzed according to the immunological status of the patient as either immunocompetent, HIV negative with no other conditions or immunocompromised, HIV positive or other conditions (e.g., corticotherapy and tumor). The complete data of the isolates are shown in the supplementary data. A multivariate logistic regression model was use to analyze 291 clinical samples and was calculated with SAS software version 8.0 using the Genmod procedure. The log-binomial model was used for estimating the adjusted relative risk of the most common molecular type of each species occurring in different host conditions as well as different Brazilian regions. The variables for which the Wald test showed a $p < 0.05$ were included in the final model. Two models were tested separately to examine the interactions between host factors and the macro region (i.e., VNI was compared to all other molecular types, and VGII was compared to all other molecular types).

RESULTS

A total of 443 *C. neoformans* (n = 320) and *C. gattii* (n = 123) isolates from all Brazilian regions, representing 11 Brazilian states, were analyzed (Fig. 1). Data from 356 isolates were imported from previous studies and that from 87 isolates were newly typed using the molecular tools described above. Out of the 320 *C. neoformans* isolates, 251 were clinical and 69 environmental isolates. Out of the 123 *C. gattii* isolates, 86 were clinical (84 human and 2 veterinary) and 37 were environmental isolates.

Information regarding geographic origin was not available for two isolates (0.5%). Mating type data was available for 262 (59.1%) isolates, and 249 (95%) of these were of mating type alpha. Ten isolates were of mating type a (5 VNIV environmental and 5 VGII clinical isolates, all from the NM). Host risk factor data were available for 293 of the 335 (87%) clinical isolates. The distribution of the molecular types of the *Cryptococcus* species complex in Brazil according to geographic region, mating type, source, and host factors is shown on Table I.

Overall, the most common molecular types were VNI (64%) and VGII (21%), followed by VNII (5%), VGIII (4%), VGI and VNIV (3% each), and VNIII (< 1%). The molecular type VGIV was not identified among the studied Brazilian isolates. Fig. 1 shows the distribution of the molecular types according to the Brazilian states included in the analysis. Fig. 2A demonstrates the RFLP profiles of the eight molecular types (VNI-VNIV, VGI-VGIV) resulting from the *URA5*-RFLP technique using the standards isolates, and Fig. 2B shows the PCR fingerprinting profiles from the eight standards isolates as well as a representative example of the Brazilian isolates. Univariate analysis of the dataset showed that the OD of

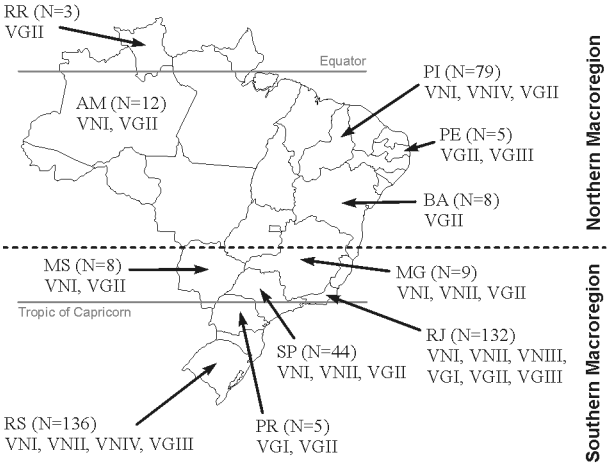

Fig. 1: map of Brazil showing the two macro regions (Northern and Southern macro regions) and the distribution of the molecular types and number of isolates according to the states involved in the study: AM: Amazonas; BA: Bahia; MG: Minas Gerais; MS: Mato Grosso do Sul; PE: Pernambuco; PI: Piauí; PR: Paraná; RJ: Rio de Janeiro; RR: Roraima; RS: Rio Grande do Sul; SP: São Paulo.

TABLE I

Distribution of the molecular types according the main characteristics of 443 Brazilian isolates included in this study

Characteristics	Molecular types – n (%)							
	VGI	VGII	VGIII	VNI	VNII	VNIII	VNIV	Total
Geographic region								
Northern Macro region	-	68 (63.6)	4 (3.7)	30 (28)	-	-	5 (4.7)	107
Southern Macro region	14 (4.2)	24 (7.2)	12 (3.6)	252 (75.4)	22 (6.6)	1 (0.3)	9 (2.7)	334
Mating Type								
Alpha	9 (3.6)	63 (25.3)	12 (4.8)	151 (60.6)	5 (2.0)	-	9 (3.6)	249
A	-	5 (50)	-	-	-	-	5 (50)	10
Alpha/a	-	-	-	3 (100)	-	-	-	3
Source								
Clinical	14 (4.2)	54 (16.1)	16 (4.8)	234 (69.9)	17 (5.1)	-	-	335
Environmental	1 (0.9)	36 (34)	-	49 (46.2)	5 (4.7)	1 (0.9)	14 (13.2)	106
Veterinary		2 (100)						2
Host Factors								
Immunocompetent	5 (11.9)	25 (59.5)	6 (14.3)	6 (14.3)	-	-	-	42
Immunocompromised	-	9 (3.6)	3 (1.2)	222 (88.4)	17 (5.8)	-	-	251

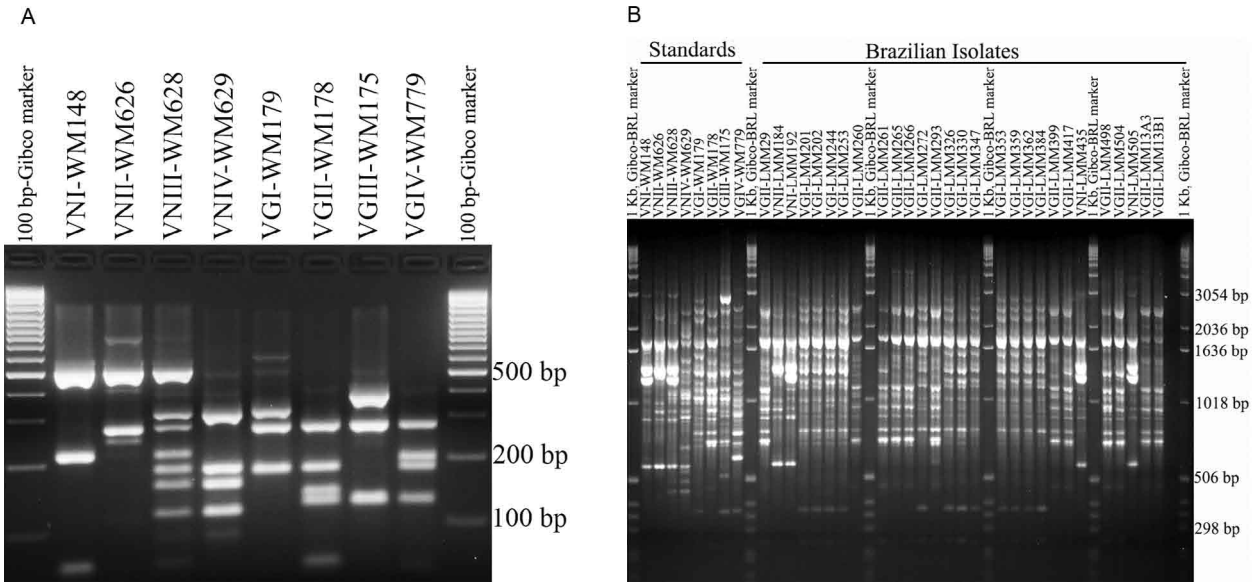

Fig. 2: A: restriction fragment length polymorphism (RFLP) profiles of *URA5* gene obtained by double digestion of the gene with *Sau*96I and *Hha*I of the standards isolates representing the eight molecular types VNI-VNIV and VGI-VGIV; B: banding patterns generated by PCR-fingerprints with the primer M13 obtained from a selection of Brazilian *C. neoformans* and *C. gattii* isolates, given as a representative example for the patterns obtained from clinical and environmental isolates. Standard patterns obtained from the reference strains of the eight major molecular types by PCR-fingerprinting with the microsatellite specific primer M13 (right-hand side of 2B).

an isolate being molecular type VNI or VGII (compared to all other molecular types) varied according to the region (NM or SM) when both clinical and environmental datasets were analyzed separately. Investigation of clinical (OD 9.6; $p < 0.001$) and environmental (OD 2.4; $p < 001$) samples suggests that the molecular type VGII is more likely to occur in the NM. On the contrary, VNI isolates are more likely to occur in the SM and show an OD of 1.5 for both clinical and environmental isolates ($p < 0.001$ and $p = 0.018$, respectively). *C. gattii* is 4.4 times more likely to occur in the NM, whereas *C. neoformans* is 1.6 times more likely to occur in the SM ($p < 0.001$).

The Wald test for the VNI log-binomial model revealed that host factors were significant ($p < 0.05$), whereas the macro region was not important for the occurrence of this molecular type in Brazil ($p > 0.05$). VNI was five times more likely than the other molecular types to infect immunocompromised hosts (Table II). When the major molecular type VGII was analyzed, the interaction analysis showed a significant correlation between host factors and macro region. This reflects a demographic pattern of distribution, because the majority of immunocompromised patients (mainly AIDS patients) live in big cities. The largest Brazilian cities

are located in the SM. This correlation may partially explain the predominance of VGII (*C. gattii*) in the NM. However, the Wald test for the VGII log-binomial model showed that the geographic region and host condition were significant predictors (p = 0.006) (Table III). Although it is slightly higher, the relative risk is quite similar to the adjusted relative risk. Immunocompetent hosts are ten times more likely to be infected by VGII than by all other molecular types. The molecular type VGII is 16 times more likely to occur in the NR (Table III). Nine out of 34 VGII (26%) clinical isolates were associated with HIV infections; two of these were from the NM and seven were from the SM.

Molecular polymorphisms identified via PCR-fingerprinting using the primer M13 grouped the 87 isolates into the major molecular types of VNI, VNII, VNIV, VGI, and VGII (Fig. 4). Overall, the similarity of the *C. neoformans* isolates was 87%, whereas that of the *C. gattii* isolates was 77%. According to the banding pattern, VNI isolates (n = 25) were divided into six subtypes: one major group comprised 50% of the VNI isolates, three groups contained three or four isolates, and two clinical isolates clustered individually. The VNII isolates (n = 3) showed individual patterns. The only three VNIV isolates analyzed by PCR-fingerprinting showed a unique, identical banding pattern. The VGI isolates (n = 13) were all from RJ (SN) and were divided into one major group (10 isolates), and three clinical isolates showed individual banding patterns. The highest degree of polymorphism was observed among the VGII isolates, which showed 14 individual patterns (1 major group with 8 isolates and a second group with 6 isolates). There was no association between these subtypes and their geographical distribution.

DISCUSSION

In this study, two major epidemiological trends were identified in Brazil: *C. gattii* predominantly occurred in the NM (OD 5.4; p < 0.001), and *C. neoformans* predominantly occurred in the SM (OD 2.6; p < 0.001). These results are in agreement with a phenotypic study of 467 Brazilian *C. neoformans* isolates by Nishikawa et al. (2003), which showed serotype A (*C. neoformans*) associated with the Southeastern region (which is in the SM), and serotype B (*C. gattii*) associated with the NE region (which is in the NM) of Brazil.

C. neoformans is the most common agent of cryptococcosis worldwide, and it mainly affects AIDS patients. In Brazil, over 70% of AIDS patients live in the South and Southeast regions (Brito et al. 2001). These regions contain the largest cities of the country, and deforestation and anthropic actions are more evident in them (Lazéra et al. 2005, Pedroso et al. 2007). On the contrary, *C. gattii* affects mainly immunocompetent hosts and has a natural habitat predominantly related to wood decay in tropical and subtropical countries. The predominance of *C. gattii* as the main agent of cryptococcosis in the NM of Brazil is reinforced by the similar results found during analysis of the environmental isolates (OD 2.3; p < 0.001).

With regard to the molecular types of the NM and SM of Brazil, no significant differences were observed regarding the occurrence of VNI. VGII clearly occurs endemically in the NM, where it is responsible for 89% of the *C. gattii* infections. In the SM, only 43% of the *C. gattii* infections are caused by VGII. Moreover, the majority of the VGII clinical isolates in RJ were obtained from patients coming from the NE region of Bra-

TABLE II

Logistic regression model of probability of VNI occurrence in clinical samples according to the host status (n = 291)

Host status	Relative Risk (95% CI)	p-value	Adjusted Relative Risk (95% CI)	p-value
Immunocompromised	2.18 (1.7-2.9)	< 0.001	6.19 (3.0-13.0)	< 0.001
Immunocompetent	1.00	-	1.00	-

CI: confidence interval.

TABLE III

Logistic regression model of probability of VGII occurrence in clinical samples with interactions between the predictors host status and macro region (n = 291)

Characteristics	Risk Relative (95% CI)	p-value	Adjusted Risk Relative (95% CI)	p-value
Host status				
Immunocompetent	11.20 (6.8-18.5)	< 0.001	11.71 (4.4-31.2)	< 0.001
Immunocompromised	1.00	-	1.00	-
Macro region				
Northern (NM)	16.0 (8.4-30.7)	< 0.001	17.57 (5.2-59.6)	< 0.001
Southern (SM)	1.00	-	1.00	-
Host status × Macro region				
Immunocompetent × NM		-	0.14 (0.03-0.57)	0.006
Immunocompetent × SM	-	-	1.00	
Immunocompromised × NM	-	-	1.00	
Immunocompromised × SM	-	-	1.00	

CI: confidence interval.

zil (Rozenbaum & Gonçalves 1994, MS Lazéra, unpublished data) who went to the large cities of the South in search of better living conditions. This explanation may also account for the VGII clinical isolates obtained in other cities (e.g. São Paulo, the most important center of internal migration in Brazil). Considering the large extent of inland migration, therefore, the molecular types of clinical origin of a certain region should be correlated with the molecular typing of the environmental isolates of that region. In the present study, 36 of the 37 *C. gattii* environmental isolates were isolated in the NM. All of these were VGII, which reinforces once again its predominance in that region. The only environmental *C. gattii* isolate obtained in the SM was molecular type VGI. However, the molecular type VGI was not detected among the clinical and environmental isolates from the NM, suggesting that this type may occur in higher latitudes of tropical or subtropical regions. Similar results were described by Escandón et. al. (2006) in Colombia, where only one of 425 isolates of the *Cryptococcus* species complex was identified as VGI; the majority (99.2%) of the *C. gattii* isolates were of the molecular type VGII. Recent molecular typing studies in Brazil have shown the presence of VNI and VNII in clinical samples and VNI, VNIV, and VGI in environmental samples in the SM (i.e. states of SP and RS) (Abegg et al. 2006, Ribeiro et al. 2006, Matsumoto et al. 2007).

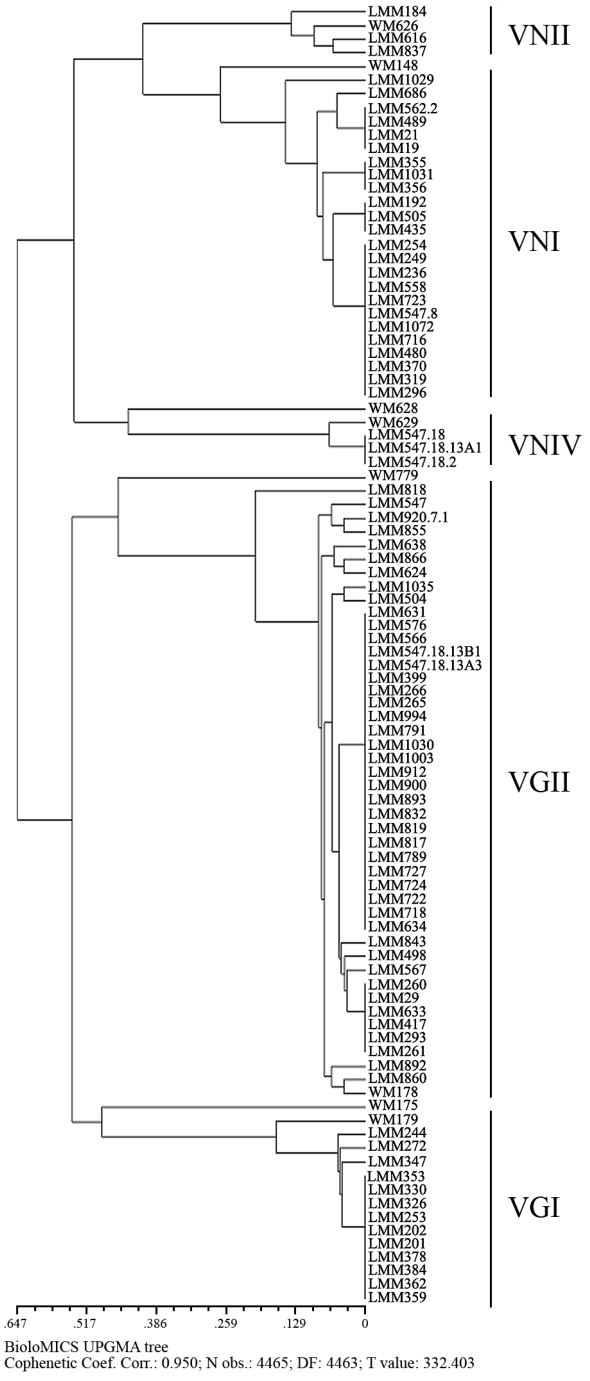

Fig. 3: phenogram of PCR-fingerprinting profiles obtained with the M13 primer from 87 Brazilian isolates. The isolates clustered into five major groups corresponding to the molecular types: VNI, VNII and VNIV (*C. neoformans*), and VGI and VGII (*C. gattii*). The Brazilian *C. neoformans* isolates generated 10 different bands pattern profiles, while the Brazilian *C. gattii* isolates formed 20 bands pattern profiles.

Studies of molecular polymorphisms in infective agents have led to a better understanding of those agents' epidemiology and may contribute to the evaluation of interventions and treatments. Martins et al. (2007) analyzed serial isolates from HIV patients using pulsed field gel electrophoresis and RAPD-PCR, and their findings suggested that patients may be infected by more than one isolate. Similarly, Almeida et al. (2007) used RAPD-PCR to analyze primarily Brazilian *C. neoformans* isolates. These authors observed a high correlation between a distinct genetic profile in serial samples and the tendency to become resistant to antifungal drugs. In the current study, the molecular polymorphism analysis showed a lower similarity rate (77%) among VGII than VNI isolates (87%). The VGII isolates may have a higher rate of mutation, which could produce a poorer response to antifungal therapy (Yee-Chun et al. 2000).

Mat alpha cells are more virulent than Mat a cells. As in the present study, these cells predominate in clinical and environmental samples worldwide. In the NM region, VGII Mat a was identified as agent of systemic cryptococcosis in five immunocompetent patients. These findings denote an uncommon picture of the pathogenesis of cryptococcosis and warrant further study to elucidate the impact of the mating locus in *C. gattii* infections. The finding of both mating types in Brazilian VGII isolates is of great interest in connection with the search for the possible origin of the recent outbreak of cryptococcosis on Vancouver Island British Columbia, Canada. In this case, all isolates of the causative agent were VGII Mat alpha strains (Kidd et al. 2004).

Because the VGII molecular type existed in Brazil long before the British Columbia outbreak and we have identified both mating types, Mat alpha and Mat a, geographically proximal to one another, we suggest the possibility of genetic recombination among those yeast populations in South America. The results of such recombination may spread in the environment as adapted polymorphic populations.

The first VGII strain (LMM 293) identified in Brazil was isolated in RJ in 1988 from a patient coming from the NM. Brazil is a large continental country with many different geographic and demographic patterns. In the northern region, the Amazon rainforest encompasses partially preserved wild areas surrounding urban cities or settlements. In the NE region, the central semi-arid area is covered by brushwood known as "caatinga". Despite these differences, our results showed the occurrence of *C. gattii* VGII in the environment of both regions. It behaved as a primary pathogen of human infection in native, normal hosts and primarily caused meningoencephalitis, attaining a prevalence of 20 to 30% in children and adolescents.

In accordance with the human infections by *C. gattii* VGII in the NM of Brazil, several genera of trees (e.g., *Cassia* sp., *Ficus* sp., *Guetarda* sp., *Erythrina* sp., and *Licania* sp.) can harbor this yeast in their hollows (Lazéra et al. 1998, 2000, Fortes et al. 2001). Clinical isolates are thus more likely to be found in deforestation areas, which are located mainly on the border of rainforests. A recent diagnosis (2006) of meningoencephalitis due to a VGII strain in a 5-year-old child (not included in the present analysis) who lived his entire life in RJ (SM) suggests that the VGII type may be spreading from the NM. It may be adapting to new areas in the SM due to anthropic activity and global climatic changes. It is also possible that this molecular type has long been present at a low density in the SM, thereby causing occasional human cases. Similarly, it has been suggested that *C. gattii* VGII has recently colonized the temperate region of Vancouver Island via unknown events and that forestry activities and the distribution of tree by-products may have facilitated the mobility of *C. gattii* through aerosolization and mechanical dispersal to non-endemic areas within the Pacific Northwest (Fraser et al. 2005, Kidd et al. 2007, Upton et al. 2007). This emphasizes the need for an active surveillance program of new human and animal infections by VGII strains in the SM.

It is very important to note that VGII is not a rare genotype of *C. gattii* in South America. In fact, it behaves as a primary fungal pathogen and causes endemic cryptococcosis in immunocompetent hosts in the northern macro region of Brazil, where it is particularly well-adapted to environmental biotypes associated with wood decay. Our findings suggest that this eco-epidemiological pattern of the VGII genotype in Brazil is not a recent event and has been recognized for at least the last 20 years.

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Supplementary data

List of the Brazilian isolates and their respective data included in the analysis

LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
LA 70	HEC 4744, LMM 210	A	VNI		alpha	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 71	HEC 4759, LMM 207	A	VNI		alpha	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 88	HEC 3385, LMM 275	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 89	HEC 3393, LMM 276	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 90	HEC 3451, LMM 277	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 64	HEC 4262, LMM 226	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 65	HEC 4407, LMM 221	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 66	HEC 4418, LMM 220	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 67	HEC 4436, LMM 219	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 68	HEC 4532, LMM 217	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 69	HEC 4576, LMM 216	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 72	HEC 4859, LMM 205	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 97	HEC 5988, LMM 656	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 98	HEC 6047, LMM 126	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 99	HEC 6196, LMM 117	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 91	HEC 7389, LMM 92	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 92	HEC 7576, LMM 663	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 94	HEC 7835, LMM 658	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 95	HEC 7836, LMM 657	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 96	HEC 8104, LMM 69	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 93	HEC 8462, LMM 59	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 100	HEC 10720, LMM 33	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 82	HEC 10724, LMM 32	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 83	HEC 10726, LMM 31	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 85	HEC 11116, LMM 20	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 86	HEC 11127, LMM 19	A	VNI	1	not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 87	HEC 11376, LMM 13	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 101	HEC 12284, LMM 228	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 75	HEC 13245, LMM 529	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 76	HEC 13247, LMM 617	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 77	HEC 13249, LMM 673	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 78	HEC 13262, LMM 619	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 79	HEC 13341	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 73	HEC 13670, LMM 550	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 74	HEC 13722, LMM 555	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
	HEC 13912	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 81	HEC 14047, LMM 674	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 108	HUCFF B-19/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 141	HUCFF B-30/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238

LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
LA 109	HUCFF B-49/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
	HUCFF B-229/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 143	HUCFF B-294/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 119	HUCFF B-444/94	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 105	HUCFF B-493/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 123	HUCFF B-545/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 131	HUCFF B-573/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 145	HUCFF B-605/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 133	HUCFF B-683/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 135	HUCFF B-960/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 139	HUCFF B-1295/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 118	HUCFF B-1616/93	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 110	HUCFF B-2128/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 137	HUCFF B-2202/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 106	HUCFF B-2218/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 107	HUCFF H-06/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 140	HUCFF H-10/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 113	HUCFF H-88/96 (15/2)	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 115	HUCFF H-88/96 (26/6)	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 142	HUCFF H-106/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 128	HUCFF H-120/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
	HUCFF H-137/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 112	HUCFF H-146/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 104	HUCFF H-154/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 130	HUCFF H-165/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
	HUCFF H-195/97	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 132	HUCFF H-197/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 134	HUCFF H-243/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 111	HUCFF H-293/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 136	HUCFF H-305/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 138	HUCFF H-377/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
	Hamdan F2'1	A	VNI	1	alpha	MG	environmental	N/A	*Microbiology* 2001; *147:* 891-907
	Hamdan I3'1	A	VNI	1	alpha	MG	environmental	N/A	*Microbiology* 2001; *147:* 891-907
LA 419		not typed	VNI		alpha	RJ	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 420		not typed	VNI		alpha	RJ	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 421		not typed	VNI		alpha	RJ	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 422		not typed	VNI		alpha	RJ	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 147	RJ 62, LMM 381	A	VNI	1	not typed	RJ	environmental	N/A	*Med Mycol* 2004; *42:* 229-238; *Microbiology* 2001; *147:* 891-907
LA 148	RJ 63, LMM 380	A	VNI	1	not typed	RJ	environmental	N/A	*Med Mycol* 2004; *42:* 229-238; *Microbiology* 2001; *147:* 891-907
LA 102	HUCFF B-64/97	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238

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LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
LA 103	HUCFF B-175/97	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 127	HUCFF B-308/96	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 117	HUCFF B-583/97	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 116	HUCFF B-1267/96	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 125	HUCFF B-1396/95	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 120	HUCFF B-1773/94	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 126	HUCFF H-91/96	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 121	HUCFF H-97/96	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 124	HUCFF H-318/95	A	VNII		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
	Hamdan C3-1	A	VNII	1A	alpha	RJ	environmental	N/A	*FEMS Yeast Res* 2004; *4:* 377-388; *Microbiology* 2001; *147:* 891-907
LA 146	RJ 24, LMM 385	A	VNII	1A	alpha	RJ	environmental	N/A	*Med Mycol* 2004; *42:* 229-238; *Microbiology* 2001; *147:* 891-907
LA 149	RJ 64, LMM 379	A	VNII		not typed	RJ	environmental	N/A	*Med Mycol* 2004; *42:* 229-238
LA 150	HEC 3724, LMM 456	not typed	VGI		not typed	RJ	clinical	unknown	*Med Mycol* 2004; *42:* 229-238
LA 84	HEC 11102, LMM 21	not typed	VGII	6	alpha	RJ	clinical	HIV+	*FEMS Yeast Res* 2004; *4:* 377-388; *Med Mycol* 2004; *42:* 229-238
	HEC 11241, LMM 16	not typed	VGII		not typed	SP	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
LA 55	LMM 417	B	VGII	6	a	PI	clinical	HIV-	*Emerg Infect Dis* 2003; *9:* 189-195
LA 57	LMM 506	B	VGII		not typed	PI	clinical	HIV-	*Emerg Infect Dis* 2003; *9:* 189-195
LA 59	LMM 513	A	VNI		not typed	PI	clinical	HIV-	*Emerg Infect Dis* 2003; *9:* 189-195
LA 61	LMM 557	not typed	VGII		not typed	PI	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 346	481(5)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 347	482(5)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 348	540(9)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 349	541(9)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 350	14(9)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 351	13(11)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 352	49(11)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 353	98(17)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 354	14(17)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 355	221(23)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 356	18(23)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 357	222(24)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 358	65(24)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 359	62(27)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 360	66(27)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 361	67(27)	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 363	1342	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 364	1755	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 365	1920	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 366	2177	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195

LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
LA 362	Bird1	B	VGII		not typed	SP	veterinary	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 326	LCS-534	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 328	GFR-1505	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 329	AFG-34	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 330	AFG-14	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 331	CMS-1226	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 332	AFG-25/00	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 333	PRL-1133	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 334	JCJ-1157	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 335	MAAC-1866	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 336	VPD-25	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 337	AFG-09/00	not typed	VGII		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 338	AFG-15/00	not typed	VGII		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 339	LCS-394	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 340	IFOC-1446	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 341	MO-438	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 342	KSO-948	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 343	CSC-1016	A	VNII		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 344	MDS-949	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 345	MEM-1064	A	VNI		not typed	SP	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 755	HC2	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 756	HC3	A	VNII		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 757	HC4	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 758	HC6	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 759	HC8	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 760	HC9	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 761	HC10	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 762	HC11	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 763	HC13	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 764	HC14	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 765	HC15	A	VNI		not typed	RS	clinical	HIV+	*Emerg Infect Dis* 2003; *9:* 189-195
LA 44	LMM 414	B	VGII		not typed	PI	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 45	LMM 415	B	VGII		not typed	PI	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 47	LMM 434	B	VGII		not typed	PI	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 52	LMM 561	A	VNI		not typed	PI	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
LA 54	LMM 564	A	VNI		not typed	PI	environmental	N/A	*Emerg Infect Dis* 2003; *9:* 189-195
	LMM 9	AD	VNII	1A	alpha	RJ	clinical	HIV+	*Med Mycol* 2003; *41:* 383-390
	LMM 23	B	VGII	6	alpha	PI	clinical	HIV-	*Med Mycol* 2003; *41:* 383-390
	LMM 29	B	VGII	6	alpha	PI	clinical	HIV-	*Med Mycol* 2003; *41:* 383-390
	LMM 34	A	VNII	1A	alpha	RJ	clinical	HIV+	*Med Mycol* 2003; *41:* 383-390
	LMM 379	A	VNII	1A	alpha	RJ	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 388	D	VNIII	3	not typed	RJ	environmental	N/A	*Med Mycol* 2003; *41:* 383-390

LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
	LMM 414	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 484.1	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 484.2	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 484.3	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 489	A	VNI	1	alpha	RJ	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 495	A	VNI	1	alpha	RJ	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 496	A	VNI	1	alpha	RJ	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 498	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 526	A	VNI	1	alpha	AM	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 527	A	VNI	1	alpha	AM	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 544.1	A	VNI	1	alpha	RJ	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 544.2	A	VNI	1	alpha	RJ	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.1	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.2	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.3	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.4	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.5	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.6	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.7	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.9	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.10	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.11	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.13	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.14	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.15	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.16	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.17	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.18	D	VNIV	2	a	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 547.19	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 561.5	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 561.6	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.1	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.2	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.3	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.4	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.5	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.6	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.7	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.9	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.10	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.11	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.12	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390

LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
	LMM 562.13	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.15	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 562.16	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.9	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.10	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.11	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.14	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.17	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.19	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.21	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.26	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 564.27	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 655	B	VGII	6	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 706	A	VNI	1	alpha	RS	clinical	unknown	*Med Mycol* 2003; *41:* 383-390
	LMM 715	A	VNI	1	alpha	RS	clinical	unknown	*Med Mycol* 2003; *41:* 383-390
	LMM 736	A	VNI	1	alpha	MS	clinical	HIV+	*Med Mycol* 2003; *41:* 383-390
	LMM 739	A	VNI	1	alpha	MS	clinical	HIV+	*Med Mycol* 2003; *41:* 383-390
	LMM 744	A	VNI	1	alpha	MS	clinical	HIV+	*Med Mycol* 2003; *41:* 383-390
	LMM 866	B	VGII	6	alpha	RR	clinical	HIV-	*Med Mycol* 2003; *41:* 383-390
	LMM 876.1	D	VNIV	2	a	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 876.2	D	VNIV	2	a	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 878.3	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 878.4	A	VNI	1	alpha	PI	environmental	N/A	*Med Mycol* 2003; *41:* 383-390
	LMM 916	C	VGIII	5	alpha	RJ	clinical	HIV-	*Med Mycol* 2003; *41:* 383-390
	LMM 421	A	VNI	1	not typed	RJ	environmental	N/A	*Microbiology* 2001; *147:* 891-907
	LMM 422	A	VNI	1	not typed	RJ	environmental	N/A	*Microbiology* 2001; *147:* 891-907
	Hamdan 214-L	A	VNII	1A	not typed	MG	clinical	HIV+	*Microbiology* 2001; *147:* 891-907
	Hamdan 299	A	VNII	1A	not typed	MG	clinical	HIV+	*Microbiology* 2001; *147:* 891-907
	Hamdan 822-B	A	VNI	1	not typed	MG	clinical	HIV+	*Microbiology* 2001; *147:* 891-907
	Hamdan C31	A	VNII	1A	not typed	MG	clinical	HIV+	*Microbiology* 2001; *147:* 891-907
	Hamdan MCP-2	A	VNII	1A	not typed	MG	environmental	N/A	*Microbiology* 2001; *147:* 891-907
	Hamdan WP	A	VNI	1	not typed	MG	clinical	HIV+	*Microbiology* 2001; *147:* 891-907
	RV63642	A	VNI	1	not typed		clinical	HIV+	*Microbiology* 2001; *147:* 891-907
	RV66095	B	VGI	4A	not typed		clinical	HIV-	*Microbiology* 2001; *147:* 891-907
	IFM 5880, UFPR 2262	B	VGI		not typed	PR	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 48219, A465	B	VGI		not typed	PR	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 5883, UFPR 2527	B	VGII		not typed	PE	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 48213, FT 84	B	VGII		not typed	PR	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 48216, FT 119	B	VGII		not typed	PR	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 48221, A 288	B	VGII		not typed	MG	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 48297, FT 130	B	VGII		not typed	PR	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	CN011	B	VGII		not typed	SP	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388

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LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
	CN015	B	VGII		not typed	SP	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	CN025	B	VGII		not typed	SP	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 5875, UFPR-D1	C	VGIII		not typed	PE	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 5882, UFPR 2526	C	VGIII		not typed	PE	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 5884, UFPR 2528-1	B	VGIII		not typed	PE	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
	IFM 5885, UFPR 2528-2	B	VGIII		not typed	PE	clinical	unknown	*FEMS Yeast Res* 2004; *4:* 377-388
LA 55	C1	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 56	C2	B	VGIII		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 57	C3	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 58	C4	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 59	C5	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 60	C6	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 61	C7	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 62	C8	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 63	C9	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C10	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C11	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C12	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C13	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C14	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C15	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C16	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C17	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C18	A	VNI		a/alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C19	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C20	B	VGIII		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C21	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C22	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C23	A	VNI		a/alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C24	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C26	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C27	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C28	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C29	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C30	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C31	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C32	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C33	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C34	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C35	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C36	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C37	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415

LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
	C38	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C39	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C40	B	VGIII		alpha	RS	clinical	unknown	*FEMS Yeast Res* 2003; *3:* 405-415
	C41	B	VGIII		alpha	RS	clinical	HIV-	*FEMS Yeast Res* 2003; *3:* 405-415
	C42	B	VGIII		alpha	RS	clinical	Tumor	*FEMS Yeast Res* 2003; *3:* 405-415
	C43	B	VGIII		alpha	RS	clinical	Polio, toxoplasmosis	*FEMS Yeast Res* 2003; *3:* 405-415
	C44	B	VGIII		alpha	RS	clinical	HIV-	*FEMS Yeast Res* 2003; *3:* 405-415
	C45	B	VGIII		alpha	RS	clinical	Corticosteroid	*FEMS Yeast Res* 2003; *3:* 405-415
	C46	B	VGIII		alpha	RS	clinical	HIV-	*FEMS Yeast Res* 2003; *3:* 405-415
	C47	B	VGIII		alpha	RS	clinical	unknown	*FEMS Yeast Res* 2003; *3:* 405-415
	C48	B	VGIII		alpha	RS	clinical	HIV-	*FEMS Yeast Res* 2003; *3:* 405-415
	C49	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C50	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C51	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C52	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C53	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C54	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C55	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C56	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C57	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C58	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C59	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C60	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C61	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C62	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C63	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C64	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C65	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C66	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C67	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C68	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C69	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C70	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C71	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C72	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C73	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C74	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C75	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C76	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C77	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C78	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C79	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415

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LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
	C80	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C81	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C82	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C83	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C84	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C85	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C86	A	VNI		alpha	RS	clinical	unknown	*FEMS Yeast Res* 2003; *3:* 405-415
	C87	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C88	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C89	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C90	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C91	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C92	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C93	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C94	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C95	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C96	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C97	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C98	A	VNI		alpha	RS	clinical	unknown	*FEMS Yeast Res* 2003; *3:* 405-415
	C99	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C100	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C101	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C102	A	VNI		alpha	RS	clinical	unknown	*FEMS Yeast Res* 2003; *3:* 405-415
	C103	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C104	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
	C105	A	VNI		alpha	RS	clinical	HIV+	*FEMS Yeast Res* 2003; *3:* 405-415
LA 44	E1	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 45	E2	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 46	E3	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 47	E4	A	VNI		a/alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 48	E5	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 49	E6	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 50	E7	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 51	E8	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 52	E9	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 53	E10	A	VNI		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 54	E11	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 337	E12	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 338	E13	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 339	E14	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 340	E15	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 341	E16	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415

LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
LA 362	E17	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 343	E18	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 344	E19	D	VNIV		alpha	RS	environmental	N/A	*FEMS Yeast Res* 2003; *3:* 405-415
LA 129	HUCFF B-352/96	A	VNI		not typed	RJ	clinical	HIV+	*Med Mycol* 2004; *42:* 229-238
	LMM 201	B	VGI		alpha	RJ	clinical	unknown	
	LMM 202	B	VGI			RJ	clinical	HIV-	
	LMM 236	AD	VNI	1	alpha	PI	clinical	HIV-	
	LMM 244	B	VGI		alpha	RJ	clinical	unknown	
	LMM 249		VNI			RJ	clinical	HIV+	
	LMM 254	A	VNI		alpha	RJ	clinical	HIV-	
	LMM 260	BC	VGII	6	alpha	RJ	clinical	unknown	
	LMM 261	B	VGII	6	a	RJ	clinical	HIV-	
	LMM 265	B	VGII		alpha	RJ	clinical	HIV+	
	LMM 266	B	VGII		alpha	RJ	clinical	HIV-	
	LMM 272	B	VGI		alpha	RJ	clinical	HIV-	
	LMM 293	B	VGII		alpha	RJ	clinical	HIV-	
	LMM 296		VNI		alpha	RJ	clinical	HIV+	
	LMM 319	A	VNI		alpha	RJ	clinical	HIV-	
	LMM 326	B	VGI		alpha	RJ	clinical	unknown	
	LMM 330	B	VGI		alpha	RJ	clinical	HIV-	
	LMM 347	B	VGI		alpha	RJ	clinical	unknown	
	LMM 353	B	VGI			RJ	clinical	HIV-	
	LMM 355	AD	VNI		alpha	RJ	clinical	HIV+	
	LMM 356	A	VNI		alpha	RJ	clinical	unknown	
	LMM 359	B	VGI		alpha	RJ	clinical	unknown	
	LMM 362	B	VGI		alpha	RJ	clinical	unknown	
	LMM 370	A	VNI		alpha	RJ	clinical	HIV+	
	LMM 384	B	VGI	4	alpha	RJ	environmental	N/A	
	LMM 399	B	VGII		alpha	RJ	clinical	unknown	
	LMM 435		VNI		alpha	RJ	clinical	HIV+	
	LMM 480		VNI		alpha	RJ	clinical	HIV+	
	LMM 498	B	VGII		alpha	PI	environmental	N/A	
	LMM 504		VGII	6	a	PI	clinical	unknown	
	LMM 547.8	A	VNI		alpha	PI	environmental	N/A	
	LMM 547.18.2	D	VNIV		a	PI	environmental	N/A	
	LMM 547.18.13A1	D	VNIV	2	a	PI	environmental	N/A	
	LMM 547.18.13A3	B	VGII	6	alpha	PI	environmental	N/A	
	LMM 547.18.13B1	B	VGII		alpha	PI	environmental	N/A	
	LMM 566		VGII	6	alpha	PI	clinical	HIV-	
	LMM 567	B	VGII	6	alpha	PI	clinical	HIV-	
	LMM 574		VGII	6	alpha	PI	clinical	unknown	
	LMM 576	B	VGII		alpha	PI	clinical	unknown	

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LA code	Original codes/others	Serotype	Molecular Type	AFLP Type[a]	Mating Type	State	Source	Host Factors	Reference
	LMM 624		VGII		alpha	AM	clinical	HIV-	
	LMM 631	B	VGII		alpha	BA	clinical	HIV-	
	LMM 633	B	VGII		a	BA	clinical	HIV-	
	LMM 634	B	VGII		alpha	BA	clinical	HIV-	
	LMM 638	B	VGII		alpha	BA	clinical	HIV+	
	LMM 716	A	VNI		alpha	AM	clinical	HIV+	
	LMM 718	B	VGII		alpha	AM	clinical	HIV-	
	LMM 722	B	VGII		alpha	AM	clinical	HIV-	
	LMM 723	A	VNI		alpha	AM	clinical	HIV-	
	LMM 724	B	VGII		alpha	AM	clinical	HIV-	
	LMM 727	B	VGII		alpha	PI	veterinary	N/A	
	LMM 789		VGII		alpha	RJ	clinical	HIV-	
	LMM 791	B	VGII		a	PI	clinical	HIV-	
	LMM 817	B	VGII		alpha	BA	clinical	unknown	
	LMM 818	B	VGII		alpha	BA	clinical	unknown	
	LMM 819	B	VGII		alpha	BA	clinical	unknown	
	LMM 832	B	VGII			MS	clinical	HIV-	
	LMM 843	B	VGII		alpha	BA	clinical	unknown	
	LMM 855	B	VGII		alpha	RR	clinical	HIV-	
	LMM 860	B	VGII	6	alpha	RR	clinical	HIV-	
	LMM 892	B	VGII	6	alpha	MS	clinical	HIV+	
	LMM 893	B	VGII	6		MS	clinical	HIV+	
	LMM 900	B	VGII			MS	clinical	HIV-	
	LMM 912	B	VGII		alpha	MS	clinical	HIV-	
	LMM 920.7.1	B	VGII	6		PI	clinical	HIV-	
	LMM 1003	BC	VGII	6		PI	clinical	unknown	
	LMM 1029	A	VNI			AM	clinical	HIV-	
	LMM 1030	B	VGII			AM	clinical	HIV-	
	LMM 1031	A	VNI			AM	clinical	HIV+	
	LMM 1035	B	VGII			AM	clinical	HIV-	
	LMM 1072	A	VNI			RJ	clinical	HIV+	

a: the isolates typed by AFLP (Amplified Fragment Length Polymorphism) technique show the respective AFLP type; N/A: not applicable; AM: Amazonas; BA: Bahia; MG: Minas Gerais; MS: Mato Grosso do Sul; PE: Pernambuco; PR: Paraná; PI: Piauí; RJ: Rio de Janeiro; RR: Roraima; RS: Rio Grande do Sul; SP: São Paulo.