ERRATA

- In the Number 1 (February), Volume 11, 2007, paper entitled “Bilateral Adrenal Nodules Due to Histoplasmosis in an Elderly” by Flávio Carvalho, et al., page 160, the **Title** should be read: Bilateral Adrenal Nodules Due to Histoplasmosis in an Elderly.

- In the Number 3 (June), Volume 11, 2007, paper entitled “Heterogeneous Resistance to Vancomycin and Teicoplanin Among *Staphylococcus* spp. Isolated from Bacteremia ” by Ana Paula Ferreira Nunes, et al., page 347, the **Table 2** should be read:

Table 2. Characteristics of 13 clinical strains of CNS that grew on vancomycin agar screen plate

Patient Number	Strain Number	**Staphylococcal species**	Resistance to oxacillin (gene *mecA*)	**Vancomycin** MIC (automated system)	Vancomycin and teicoplanin MICs [a] (agar dilution method)		Growth on vancomycin agar with:		MIC from subpopulation gr on vancomycin	
					VC	TC	4 µg/mL	6 µg/mL	VC	TC
4	4a	S. hominis subsp novobiosepticus	+	4	2	4	+	-	2	4
	4b	*S. hominis subsp novobiosepticus*	+	4	2	4	+	-	2	4
19	19a	*S. haemolyticus*	+	≤ 2	2	32	+	-	2	16
31	31a	*S. haemolyticus*	+	≤ 2	< 1	8	+	-	2	16
53	53a	*S. haemolyticus*	+	≤ 2	< 1	4	+	-	2	32
	53b	*S. haemolyticus*	+	≤ 2	< 1	2	+	+	4	8
56	56a	*S. hominis subsp novobiosepticus*	+	≤ 2	2	4	+	-	2	8
115	115a	*S. hominis subsp hominis*	+	≤ 2	2	4	+	+	12	8
	115b	*S. hominis subsp hominis*	+	≤ 2	2	4	+	-	12	8
120	120a	*S. hominis subsp novobiosepticus*	+	≤ 2	< 1	< 1	+*	-	nd	nd
	120b	*S. hominis subsp novobios epticus*	+	≤ 2	2	8	+	+	4	4
127	127a	*S. haemolyticus*	+	4	2	8	+	-	2	32
168	168a	*S. epidermidis*	+	≤ 2	2	8	+	+	4	8

- In the Number 4 (August), Volume 11, 2007, paper entitled “Genotype Testing and Antiretroviral Resistance Profiles from HIV-1 Patients Experiencing Therapeutic Failure in Northeast Brazil ” by Melissa Soares Medeiros, et al., page 391-392, the item **Results** shoul be read in the following sequence:

Characteristics of the study population are found in Table 1. Patients initiated antiretroviral therapy between July/81 and September/02. With regards to the prior utilization of antiretrovirals, we found the percentage of patients which had used each drug to be the following: Zidovudine 82.2%, Lamivudine 79.2%, Stavudine 64.4%, Didanosine 77.2%, Zalcitabinae 10.9%, Efavirenz 27.7%, Nevirapine 23.8%, Nelfinavir 43.6%, Indinavir 27.7%, Saquinavir 9.9%, Ritonavir 26.7% (used as an active drug), Lopinavir 4% and Amprenavir 1% Amprenavir 1 (1%). Atazanavir was not utilized. Double therapy was utilized in 65 regimens

before triple therapy was initiated, but we were not able to evaluate the impact of double therapy on resistance because these regimens were changed for HAART before Genotype testing. These patients were failing three or more regimens at the time Genotype testing was performed [25]. From 101 isolates, 7.9% did not present with resistance mutations, 9.9% had mutations associated with one class of drugs (7.9% NRTI, 1% NNRTI and 1% PI), 73.3% to two classes (38.6% NRTI and PI, and 34.7% NNRTI and NRTI), and 8.9% to all classes.

In the protease gene, 97 (96%) had sequences with a resistant mutation in at least one of the 21 positions associated with resistance. L63P was the mutation most frequently encountered (73.3%) when considering all resistance positions in the protease gene. Excluding polymorphic positions, the main mutation was L90M (24.8%). Among the 101 sequences, 91 (90.1%) had one or more mutations in the 18 positions conferring resistance to NRTIs. M184V was the most common mutation found (60.4%) [23]. T215Y was the second most common, occurring in 42.6% sequences (presentation T215F/Y in 3% of cases), followed by M41L in 40.6% [26,27].

For NNRTIs, 44 (43.5%) of the sequences contained one or more mutations in the 12 positions associated with resistance. K103N was the most frequent mutation found (26.7%).

The antiretroviral susceptibility profile using the Stanford Database can be seen in Table 2. Of the samples evaluated, 49 (48.5%) demonstrated resistance to PIs, with 31 different patterns found. The six main mutation patterns, excluding polymorphic positions, corresponded to 49% of the sequences found. Analysis of these patterns by the Stanford Database was also performed (Table 3). In the NRTI class, we found 49 mutation patterns and the six standard corresponded to 38.5% of mutation sequences (Table 4). These patterns contained an average of 4 resistance mutations to NRTIs (minimum 1 and maximum 5).

K65R was found in 5.9% (6) patients, occurring alone in 3 patients, and in combination with M184V, or with M184V and K219E, or with M41L in one patient.

In the NNRTI class, 17 mutational patterns were detected, and the two main patterns corresponded to 40.9% of all sequences (Table 5).

Nelfinavir was utilized as the only protease inhibitor in 26 patients (25.7%), whose Genotype test analysis showed the presence of 9 patterns with D30N (30N+77I+88D = 3, 30N+36I+88D = 2, and 30N+36I+46L, 30N+77I, 30N+36I, 30N+36I+46I+77I+88D = 1), corresponding to 34.6%, and 6 patterns with L90M (36I+90M = 4, 90M = 1, 77I+90M = 1) that corresponded to 23%. In addition to these, four were isolated with 77I, three with 36I, one with 36I+88D and three patterns isolated did not contain PI mutations.

For patients treated with Indinavir (N=6), the main mutation found was 63P (66.6%), followed by 36I (50%), 82A (50%) and L90M (16.6%). In the NNRTI class, when the initial regimen utilized EFV (N=23), the following were found as the main mutations: 103N (60.8%), 190A (21.7%), 108I (13%) and 181C (8.7%). When NVP was utilized (N=21), we found 181C (52.4%), 103N (38.1%), 190A (38.1%) and 108I (4.7%) were the main mutations. There was not a significant difference in the predominance of the 103N mutation with prior use of Efavirenz or Nevirapine (p=0.13), nor for 190A (p=0.23) or 108I (p=0.33). However, there was a significant difference for the mutation 181C (p=0.0015) with prior use of NVP.

When the main individual mutation profiles were analyzed in first failure, second failure, and multi-failure (three or more regimens failure) subgroups, the results shown in Table 6 were obtained.