main clinical problem, i.e. distinguishing strains of the *Mycobacterium tuberculosis* complex from other atypical mycobacteria.

Such a protocol could be used in conjunction with one of the two techniques described by Pao et al. (8), one system as a control and the other as a confirmatory system. For example, on DNA extracted from seven pure cultures of mycobacterial strains *(M. xenopi, tuberculosis, bovis, gordonnae, terrae, kansasii, fortuitum)* and from 21 clinical isolates, we confirmed the positive results obtained with our method by using the protocol proposed by Pao et al. (8). Furthermore, the two PCR methods can be performed under the same cycle conditions.

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Rapid Spread of HIV Infections in Abidjan, Ivory Coast, 1987–1990

In 1986 relatively high HIV seroprevalence rates were observed among prostitutes, tuberculosis patients and pregnant women living in Abidjan, Ivory Coast (1). Soon thereafter, a high number of AIDS cases were reported (2).

In order to evaluate the spread of HIV-1 and HIV-2 infections in Abidjan, we continued the surveillance of these populations from 1987 to 1990. The populations tested were selected each year in the same hospital for the pregnant women and the tuberculosis patients, and in the same areas of Abidjan for the prostitutes. The number tested each year is shown in Table 1. The mean ages in the groups were similar. The serum samples were tested for antibodies to HIV-1 and HIV-2 by an ELISA (ELAVIA-1 and ELAVIA-2; Diagnostics Pasteur, France). All positive sera were retested by a corresponding Western blot (Dupont de Nemours, USA, and Diagnostics Pasteur). The criterion for positivity was the presence of antibodies to at least two envelope proteins (WHO criteria). Comparison of the groups was done by chi-square analysis.

Among prostitutes, overall HIV seroprevalence increased from 39.7 % ± 4.5 % to 69.4 % ± 10.9 % ($x^2 = 15.7$, $p < 0.001$) between 1987 and 1990. This overall increase was due to increases in HIV-1 (22.4 % ± 7.7 % to 41.7 % ± 11.6 %; $x^2 = 7.76$, $p < 0.01$) and dual HIV-1 + HIV-2 infection (6.9 %

Table 1: HIV seroprevalence rates in different populations in Abidjan, Ivory Coast.

Population	Year	No. tested	HIV-1		HIV-2		HIV-1 + HIV-2		Total	
			n	(%)	n	(%)	n	(%)	n	(%)
Prostitutes	1986[a]	101	20	(19.8)	11	(10.9)	7	(6.9)	38	(37.6)
	1987	116	26	(22.4)	12	(10.3)	8	(6.9)	46	(39.7)
	1989	120	40	(33.3)	10	(8.3)	8	(6.7)	58	(48.3)
	1990	72	30	(41.7)	5	(6.9)	15	(20.8)	50	(69.4)
Pregnant women	1986[a]	331	10	(3.0)	1	(0.3)	–	–	11	(3.3)
	1987	152	4	(2.6)	3	(2.0)	2	(1.3)	9	(5.9)
	1989	201	11	(5.5)	4	(2.0)	1	(0.5)	16	(8.0)
	1990	200	8	(4.0)	4	(2.0)	4	(2.0)	16	(8.0)
Tuberculosis patients	1986[a]	40	4	(10.0)	2	(5.0)	–	–	6	(15.0)
	1987	640	64	(10.0)	29	(4.5)	80	(12.5)	173	(27.0)
	1989	664	174	(26.2)	24	(3.6)	82	(12.3)	280	(42.2)
	1990	326	99	(30.4)	9	(2.8)	18	(5.5)	126	(38.7)

[a] Data from Reference 1.

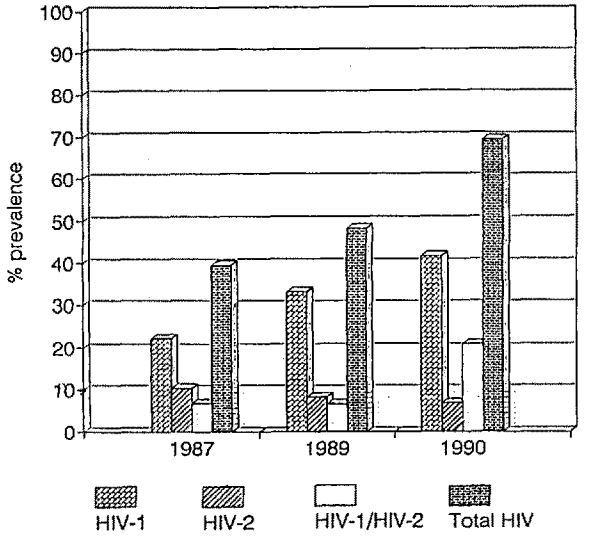

Figure 1: HIV seroprevalence rates among prostitutes in Abidjan, Ivory Coast, 1987–1990.

± 4.7 % to 20.8 % ± 9.5 %; $x^2 = 8$, $p < 0.01$), whereas HIV-2 infection remained stable (Figure 1). Among pregnant women, no significant increase of HIV infection was observed (5.9 % ± 3.8 % to 8.1 % ± 3.8 %; $x^2 = 0.55$, n.s.). Among tuberculosis patients, overall HIV seroprevalence increased from 27 % ± 3.5 % to 38.7 % ± 5.5 % ($x^2 = 13.5$, $p < 0.001$) and was due to an increase of HIV-1 infection (10 % ± 2.4 % to 30.4 % ± 5 %; $x^2 = 63.8$, $p < 0.001$).

These data illustrate the dramatic spread of HIV infection in Abidjan, especially among Abidjan prostitutes. Considering the low frequency of effective preventive measures taken, the women are at very high risk of acquiring and/or transmitting HIV. Targeted interventions aiming at increasing condom use are therefore of highest priority. AIDS is still the leading cause of death in Abidjan (3), and our data show that the impact of AIDS on the public health will be still greater in the coming years.

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In Vitro Activity of Antifungal Azoles against *Helicobacter pylori*

Helicobacter pylori is known to cause gastritis in previously healthy persons (1). Its role in duodenal ulcer disease has also been ascertained, since eradication of *Helicobacter pylori* and the concomitant gastritis has been followed by drastic decreases in ulcer relapses (2, 3). To date, the most successful results in the eradication of *Helicobacter pylori* from the gastric mucosa have been obtained with triple therapy consisting of amoxicillin or tetracycline, a nitroimidazole and a bismuth compound (2, 3). This current therapy, however, is far from optimal, and resistance to metronidazole has been related to treatment failures (4).

The mechanism by which nitroimidazoles exert their antibacterial action against *Helicobacter pylori* is not known. Recently, two other imidazole compounds, the benzimidazoles omeprazole and lansoprazole, have been shown to be active against this bacterium (5). Both of these drugs are H^+,K^+ ATPase inhibitors which might be useful in reducing gastric acid secretion. Whether the activity of these drugs against *Helicobacter pylori* is mediated by helicobacterial ATPase inhibition is not yet known. In this report we show that *Helicobacter pylori* is also susceptible to antifungal imidazoles and that the minimal inhibitory concentrations (MICs) of some of these agents are significantly low.

MICs were determined for clinical isolates and for two reference strains (NCTC 11637 and NCTC 11638) of *Helicobacter pylori* using an agar dilution method with Brucella agar plates supplemented with horse blood (7 %). Metronidazole was dissolved in sterile water, whereas omeprazole was dissolved in ethanol. Miconazole, ketoconazole, clotrimazole and fluconazole were dissolved in polyethylene glycol (PEG) 400 (Sigma, USA), and itraconazole was first dissolved in PEG 400 and ethanol (1:1) by gently heating and was then further diluted in PEG 400. Control plates with solvent only were included to rule out the possibility that the diluent itself inhibited the growth of the bacteria. The turbidities of suspensions of *Helicobacter pylori* strains grown in Mueller-Hinton broth in an atmosphere of 5 % O_2, 10 % CO_2 and 85 % N_2 at 37 °C for 24 h were adjusted to MacFarland standard 0.5 using Cobas Inocheck Instrument (Hoffman-La Roche, USA). A multipoint inoculator (Mast, UK) was used to apply 10^5 cfu per spot. The plates with neutral pH were incubated in a microaerobic atmosphere for 48 h. The MIC was defined as the lowest concentration of the drug that completely inhibited the visible growth.

For the two reference strains, NCTC 11637 and NCTC 11638, the MICs of the various agents tested were, respectively, as follows: metronidazole 0.5 and 2, omeprazole 16 and 8, itraconazole 2 and 2, miconazole 8 and 4, clotrimazole 8 and 4, ketoconazole 16 and 8, and fluconazole > 64 and > 64 mg/l.

Results for the clinical isolates tested are shown in Table 1. Most strikingly, itraconazole, miconazole, clotrimazole, and ketoconazole had relatively low MICs. In contrast, fluconazole had no activity. *Helicobacter pylori* strains exhibited very homogeneous susceptibility to the antifungal azoles. This was demonstrated by the identical MIC50 and MIC90 values of almost all of these drugs. On the other hand, susceptibility to metronidazole and omeprazole varied widely. Accordingly, no cross-resistance was found between metronidazole or omeprazole and the antifungal azoles.

In spite of relatively low MIC values, omeprazole has failed to eradicate *Helicobacter pylori* in vivo (6). The local direct effect of drugs on the bacteria in the gastric lumen certainly plays an important role in the eradication of *Helicobacter pylori,* and thus the in vitro results do not necessarily predict the outcome in vivo. With respect to the considerably low MICs of ketoconazole and clotrimazole, and especially those obtained with itraconazole and miconazole, the question arises whether these drugs could be potential alternatives to metronidazole in the eradication of *Helicobacter pylori.* Antifungal azoles have previously been reported to have antibacterial activity, mainly against some gram-positive bacteria and *Bacteroides* species (7). On the basis of the present