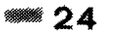
ELISA RESULTS* ON PAIRED SERA 2 WEEKS APART FROM RECENTLY HIV-1 INFECTED SUBJECTS A AND B

ELISA	A		B	
	June 17, 1988	June 30	June 2, 1989	June 16
Diagnostics Pasteur 'ELAVIA' (HIV-1, proteins from virus culture)	1·5	2·6	3·3	5·9
Abbott recombinant (HIV-1, recombinant proteins)	1·8	3·4	3·0	4·2
Behring (HIV-1 + HIV-2, synthetic peptides)	5·0	>8·0	4·6	7·6

*As sample/cut-off ratios.

Such patterns may represent HIV-2 infection or reactivity non-specific to HIV infection or early seroconversion to HIV-1. Specific ELISA and WB assays will confirm a possible HIV-2 infection and the two other possibilities can usually be differentiated on the first sample by the use of different ELISAs (two or three if necessary) and a WB lot sensitive to anti-gp160.

Because not all ELISAs have the same capacity to detect p24 reactive only samples—assays using culture viral proteins recognise these samples better than assays based on recombinant or synthetic proteins—and because not all ELISAs have the same sensitivity to detect early seroconversions, a discrepancy between ELISA results will indicate probable non-specific reaction while positive results in all ELISAs would favour a specific reaction. WB analysis will complete this first approach to differentiating between false and true reactivities, provided lots sensitive for anti-gp160 are used. Under these conditions we diagnose non-specific reactivity to HIV-1 when at least one ELISA is clearly negative and when anti-gp160 is absent; we diagnose early seroconversion when all the ELISAs are reactive and when a gp160 band is present together with p24.



HIV-1 WBs on three samples from same subject.

Left = Aug 5; middle = Sept 2; right = Oct 7, 1987.

If there is still doubt a second sample must be taken but this can be done after as little as 2 weeks because titres of antibodies to HIV increase very quickly after infection (the table and figure illustrate this point) while p24 reactive only sera give the same results for many years.[4]

A WB pattern need not remain indeterminate for 2 weeks because p24 reactive only sera from people who are not infected by HIV-1 can be rapidly differentiated from sera from people recently infected. A blood donor infected by HIV must be notified as quickly as possible to avoid spread of the infection; waiting 6 months, as proposed by Genesca et al and Dock et al,[5] is much too long.

Institut National de Transfusion Sanguine,
6 rue Alexandre-Cabanel,
75015 Paris, France

ANNE-MARIE COUROUCÉ

1. Van der Poel CL, Reesink HW, Tersmette Th, Lelie PN, Huisman H, Miedema F. Blood donations reactive for HIV in western blot, but non-infective in culture and recipients of blood. *Lancet* 1986; ii: 752–53.
2. Courouce AM, Muller JY, Richard D. False-positive Western blot reactions to human immunodeficiency virus in blood donors. *Lancet* 1986; ii: 921–22.
3. Van der Poel CL, Lelie PN, Reesink HW, et al. Blood donors with indeterminate anti-p24gag reactivity in HIV-1 Western blot: absence of infectivity to transfused patients and in virus culture. *Vox Sang* 1989; **56:** 162–67.
4. Lefrère JJ, Courouce AM, Lucotte G, et al. Follow-up of subjects with isolated and pesistent anti-core (anti-p24 or anti-p17) antibodies to HIV. *AIDS* 1988; **2:** 287–90.
5. Dock NL, Lamberson HV, O'Brien TA, et al. Evaluation of atypical human immunodeficiency virus immunoblot reactivity in blood donors. *Transfusion* 1988; **28:** 412–18.

FATAL TOXIC EPIDERMAL NECROLYSIS AFTER GRISEOFULVIN

SIR,—Griseofulvin is used as a fungistatic drug in dermatophytosis. Cutaneous side-effects include macular and/or papular rashes, urticaria, and photosensitisation.[1,2] We report a case of fatal toxic epidermal necrolysis following oral griseofulvin.

A nineteen-year-old woman was admitted to our hospital with a diagnosis of toxic epidermal necrolysis. She had previously been healthy. Seven days before admission, she had been treated with griseofulvin, two 500 mg tablets daily, because of two lesions on the knee and thigh, consistent with dermatophytosis. One day later she received one application of topical econazole. Three days before admission (when she was still on griseofulvin), metronidazole, four 250 mg tablets daily, was given because of leucorrhoea. The next day, she had macular and generalised rash with fever (39°C) and itching, which led to withdrawal of all drugs. Bullae were noted in the mouth and Nikolsky's sign was seen in the erythematous areas. She was therefore referred to our burns centre. On admission, the clincial diagnosis of toxic epidermal necrolysis was histologically confirmed. Unfortunately, evolution was rapid, with complete skin necrolysis three days later. *Staphylococcus aureus* and *Escherichia coli* septicaemia occurred, and despite antibiotic therapy her condition deteriorated with the onset of metabolic acidosis, septic shock, adult respiratory distress syndrome, and acute renal failure. She died with multiple organ failure two weeks after admission.

Toxic epidermal necrolysis is rare, has a high mortality, and is most frequently attributable to an adverse drug reaction. Sulphonamides, anticonvulsants, and non-steroidal anti-inflammatory drugs are usually inplicated in this condition.[2]

According to standardised criteria for drug culpability,[2] the points implicating griseofulvin in our patient are: (1) the absence of any other illness before admission; (2) the time interval between drug administration and the onset of bullous eruption being six days, when it is only one day for metronidazole; and (3) the absence of antibodies to other possible causes such as mycoplasma, cytomegalovirus, hepatitis virus, and human immunodeficiency virus. Moreover, staphylococcal scaled skin syndrome was excluded by clinical and histological findings. To our knowledge, this is only the second report of toxic epidermal necrolysis following griseofulvin administration.[2]

G. MION
R. VERDON
Y. LE GULLUCHE
H. CARSIN
A. GARCIA
J. GUILBAUD

Centre for Treatment of Burns,
Hôpital d'Instruction des Armées Percy,
92141 Clamart, France

1 Guillaume JC, Roujeau JC, Revuz J, et al. The culprit drugs in 87 cases of toxic epidermal necrolysis (Lyell's syndrome). *Arch Dermatol* 1987; **123:** 1166–70.
2. Taylor B. Duffill M. Toxic epidermal necrolysis from griseofulvin. *J Am Acad Dermatol* 1988; **19:** 565–67.