Table 1. HLA-B*5701 Results According to Race.

Race	Patients Positive for HLA-B*5701 (N=7)	All Patients (N=617)
Black (African origin)	1	535
White	5	54
Other	1	28

TO THE EDITOR: In PREDICT-1, Mallal and colleagues found that prospective HLA-B*5701 screening reduced the incidence of hypersensitivity reactions to abacavir in a predominantly white population. In our 2006 study involving Martinican patients with HIV infection or AIDS, most of whom were of sub-Saharan African origin, abacavir had to be discontinued in 10 of 414 patients because of probable hypersensitivity reactions (2.4%; 95% confidence interval, 1.2 to 4.5). None of the black patients in this study carried the HLA-B*5701 allele. We subsequently screened 617 consecutive patients, seen at our hospital, for HLA-B*5701 (Table 1). In this Martinican cohort of mainly black African ancestry, abacavir exposure was frequent, hypersensitivity reactions were rare, and the prevalence of HLA-B*5701 was low. In the Caribbean region, which is significantly affected by the HIV pandemic, black populations of African ancestry do not appear to be at high risk for hypersensitivity reactions, and it therefore seems inappropriate to restrict the prescription of abacavir in this population when HLA-B*5701 genotyping is not possible.

Sylvie Abel, M.D.
Laure Paturel, M.D.
University Hospital of Fort-de-France
97200 Fort-de-France, France
sylvie.abel@chu-fortdefrance.fr
André Cabié, M.D.
Clinical Research Center of Antilles–Guyane
97200 Fort-de-France, France

THE AUTHORS REPLY: The PREDICT-1 study was specifically designed to study the effectiveness of screening for the HLA-B*5701 allele to prevent hypersensitivity to abacavir. The negative predictive value of HLA-B*5701 for patch-test–positive abacavir hypersensitivity was found to be 100%; we therefore disagree with Vandekerckhove et al. that our study will only modestly alter clinical practice. Randomized, placebo-controlled, double-blind clinical studies have shown a 2 to 7% rate of hypersensitivity among patients who were not receiving abacavir.[1] Therefore, the 3.4% rate among patients with a clinical diagnosis of hypersensitivity but a negative patch test was expected. Indeed, for the purposes of our calculation of statistical power, we anticipated a rate of clinical overdiagnosis of 3.6% among patients in the screened group. In addition, nonblinded studies have shown that clinical overdiagnosis decreases dramatically over time, further providing support for the clinical impact of HLA-B*5701 screening and therefore lessening the need for a rechallenge study.[1] Finally, it would not have been safe, practical, or methodologically sound to incorporate a double-blind, placebo-controlled rechallenge component into our study.

We commend Abel et al. for the approach that

they took to establish the very low frequency of HLA-B*5701 carriage in a population of Martinican blacks. However, the clinical usefulness of screening is predicated on the reduction in both immunologically confirmed and false positive clinical diagnoses, and Abel et al. do not state whether the discontinuation rate of 2.4% observed in the prescreening phase decreased toward zero after screening was introduced; this decrease has been observed in unblinded screening programs reported on elsewhere.[1] The approach that Abel and colleagues propose may be reasonable to use in small, static, and select populations in which the epidemiology of HLA-B*5701 carriage has been defined and access to screening may be unavailable; however, we caution against the generalization of this approach to other nonwhite populations with a low prevalence of HLA-B*5701. Given that HLA-B*5701 has been shown to have 100% sensitivity for immunologically confirmed hypersensitivity to abacavir in both black and white patients in the United States,[2] selective screening on the basis of the perceived race or ethnic background of the patient is unlikely to be practical, ethical, or safe in most settings because of increasing rates of racial or ethnic admixture and global migration and because of the unreliability of the assignment of race or ethnic group by the patient or his or her clinician.[3]

Elizabeth Phillips, M.D.
David Nolan, M.B., B.S.
Simon Mallal, M.B., B.S.
Murdoch University
Murdoch, WA 6150, Australia
s.mallal@murdoch.edu.au

1. Phillips EJ, Mallal SA. Pharmacogenetics and the potential for the individualization of antiretroviral therapy. Curr Opin Infect Dis 2008;21:16-24.
2. Saag M, Balu R, Phillips E, et al. High sensitivity of human leukocyte antigen-B*5701 as a marker for immunologically confirmed abacavir hypersensitivity in white and black patients. Clin Infect Dis 2008;46:1111-8.
3. Phillips EJ. Genetic screening to prevent abacavir hypersensitivity reaction: are we there yet? Clin Infect Dis 2006;43:103-5.