Corrections

Functional Interactions between Synthetic Alkyl Phospholipids and the ABC Transporters P-Glycoprotein, Ste-6, MRP, and Pgh-1, by Stephan Ruetz, Martine Brault, William Dalton, and Philippe Gros*, Volume 36, Number 26, July 1, 1997, pages 8180−8188.

We have been unable to reproduce complementation of the yeast *Saccharomyces cerevisiae ste6* mutant by either the human *MRP* gene or the *pfmdr1* gene of *Plasmodium falciparum*. Additional studies suggest that the original *MRP* and *pfmdr1*-associated mating activity were caused by contaminating *STE6* sequences that were detected by Southern blotting in frozen vials of *MRP* and *pfmdr1* transformants from that period. This invalidates the use of mating complementation to monitor *MRP* and *pfmdr1* function, including monitoring possible interactions of these two proteins with synthetic alkylphospholipids (ALP). However, we were able to verify mating complementation for both wild-type *STE6* and *Mdr3* and could also verify that ALP can block mating complementation by *Mdr3* and *STE6*. Low levels of cellular resistance to ALP in the *Mdr3* and *MRP* transformants were detected in some experiments only. These levels of resistance were low, difficult to reproduce systematically, and varied considerably in different populations of transformants and in individual clones from the same transformation group. We were unable to reproducibly detect ALP resistance in *pfmdr1*, *Mdr2*, and *STE6* transformants. The reason for the discrepancy in drug resistance phenotype of current vs former transformants could not be determined with certainty but may be due either to unique populations of transformants available at the time or to a unique set of experimental conditions that we could not recreate. Thus, we are retracting the above article (with the exception of the effects of ALP on *Mdr3* and *STE6*). Please contact P. Gros (gros@med.mcgill.ca), should you need additional information or wish to discuss further any aspect of this work.

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