Dear Sirs,

The term “aspirin resistance” (1) has been used to describe a number of different phenomena, including inability of aspirin to: (i) protect against cardiovascular events despite its regular intake; (ii) to affect various laboratory tests, reflecting platelet activity. Indeed, there is a good evidence to indicate that there are subpopulations which do not respond to antithrombotic actions of aspirin in various laboratory tests. This question was recently addressed in Thrombosis and Haemostasis by P. Henry and his colleagues (2) and discussed in an accompanying editorial by M. Lordkipanidze (3). The possible explanations, given by these authors, include: lack of compliance, deficient aspirin absorption, enhanced platelet turnover and genetic predisposition. Another predisposing factor could be a hypercoagulable state after myocardial infarction and unstable angina. Such a state follows for months – up to a year – after acute myocardial infarction (6) or unstable angina (7) and is associated with depressed sensitivity to antithrombotic agents (8), including aspirin. Finally, evidence has accumulated (9) to support a genetic association between the PLA1/A2 genetic polymorphism in the glycoprotein IIIa platelet receptor and antithrombotic action of aspirin (10–12).

Conflicts of interest
None declared.

Reasons for aspirin resistance
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