PCR using allele-specific probes, and CYP2C9*3 by PCR-RFLP. PHT plasma levels were determined by radioimmunoassay.

**Results:** Twenty-six patients showed PHT therapeutic levels (10–20 μg/mL), 24 subtherapeutic and 7 supratherapeutic. Three cases, 2 CYP2C9 *1/*2, and 1 CYP2C19*1/3, had subtherapeutic levels. Patients with wild-type ABCB1 and ABCC2 genotypes exhibited a tendency to have increased PHT plasma levels than patients with mutant genotypes.

**Conclusion:** PHT plasma levels showed great variability among patients that was not statistically significant correlated to the genetic polymorphisms analyzed, although ABCB1 and ABCC2 wild-type genotypes showed a trend toward higher levels. Further investigations are needed with other candidate genes and a larger sample.

Support for the study was contributed by Dr. M. E. A. Al-Azzam and B. S. A. Azzam.

**Disclosure of Interest:** None declared.

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**PP140—THE CONTRIBUTION OF PLATELET GLYCOPROTEINS (GPIA C807T AND GPIBA C-5T) AND CYCLOOXYGENASE 2 (COX-2 G-765C) POLYMORPHISMS TO PLATELET RESPONSE IN PATIENTS TREATED WITH ASPIRIN**

S. Al-Azzam; O. Khabour; D. Tawalbeh; and O. Al-Azzeh

**Department of Clinical Pharmacy; and 2Department of Medical Laboratory Sciences, Jordan University of Science and Technology, Irbid, Jordan**

**Introduction:** Aspirin is an antiplatelet agent commonly used in treatment of patients with high risk to develop stroke and myocardial infarction. However, interindividual variability regarding the inhibition of platelet function by aspirin is well documented. In this study, the correlation between platelet glycoproteins (GPIA C807T and GPIba C-5T) and cyclooxygenase 2 (COX-2 G-765C) polymorphisms and antiplatelet response in patients treated with aspirin was investigated.

**Patients (or Materials) and Methods:** Jordanian adult patients (n = 584) who are taking aspirin as an antiplatelet agent participated in the study. Platelet aggregation response was measured using Multiplate Analyzer® system. Polymerase chain reaction–restriction fragment length polymorphism assay (PCR-RFLP) was used for genotyping of the examined polymorphisms.

**Results:** Aspirin resistance was found in 15.8% of patients. Response to aspirin was significantly associated with GPIba C-5T polymorphism (P < 0.05). However, the GPIa C807T and COX-2 G-765C polymorphisms were not related to aspirin resistance (P > 0.05).

**Conclusion:** A considerable fraction of the Jordanian population is resistant to the antiplatelet effect of aspirin, which might be related to GPIba C-5T polymorphism.

**Disclosure of Interest:** None declared.

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**PP141—THE RELEVANCE OF PLATELET GLYCOPROTEIN GP IIB/IIIa POLYMORPHISM TO ANTI-PLATELETS RESPONSE IN ACUTE CORONARY SYNDROME[ACS]**

O.A. Nayel; M.I. Sobhey; A.M. Baraka; M.A.H. Al Samak; and S.I. Abdel Qauder

**Clinical Pharmacology Department; 2Cardiology Department, Faculty of Medicine, University of Alexandria, Egypt; and 3Clinical Pathology, Medical Research Institute, University of Alexandria, Alexandria, Egypt**

**Introduction:** Pharmacogenomics is intervening in cardiovascular therapeutic armamentarium to tailor therapy to individual’s genetic makeup. Accordingly, the potential implication of PIA gene variants of GPIIa of platelet GP Iib/IIIa as a genetic risk factor provocateur and/or a therapeutic outcome modulator to antiplatelet therapy in ACS was probed.

**Patients (or Materials) and Methods:** Study enrolled 22 controls and 44 ACS patients (NSTEMI vs STEMI). They were risk stratified (TIMI score), sampled for genotyping and estimation of platelet aggregation and oxidative indices, then subdivided according to add-on antiplatelet therapy into: clopidogrel or tirofiban subgroups. After 48 hours, the therapeutic outcome was assessed; clinically [pain relief or complication prevalence (symptomatic, electrocardiographic, or hemorrhagic)] and the investigational estimates were re-assessed. Intraprocedural evaluation of chest pain, ECG tracing, and angiographic findings (thrombus extent, TIMI flow, myocardial blush) were reported in patients who underwent percutaneous intervention [PCI].

**Results:** Frequency of PlA2 vs PlA1 allele was higher in ACS patients (significant in <60 years /doubled in STEMI vs NSTEMI). TIMI score, stratification permitted considering PlA2 variant as independent risk factor in UA/NSTEMI subsets. This was fostered by intraprocedural finding of more stenotic and thrombotic lesions in PlA2 carriers. A lack of significant association between PIA variants and changes in platelet aggregation or oxidative indices, debate their causal relation to PlA2 variant being an ACS risk factor. A positive correlation was observed between PIA variants and the therapeutic response outcome to both clopidogrel and tirofiban regarding platelet aggregation and relief of chest pain while their antioxidative potential was negatively correlated only to PlA1 carriers.

**Conclusion:** PlA2 variant could be considered a genetic risk factor contributor rather than an antiplatelet therapeutic response modulator when speaking of ACS. This awaits larger scale pharmacogenomic studies before a final statement is declared as to individualize antiplatelet therapy to the best of its therapeutic outcome in ACS settings.

**Disclosure of Interest:** None declared.

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**PP142—INFLUENCE OF THE CYP2D6 -1584C>G PROMOTER POLYMORPHISM ON THE PHENOTYPE OF DEBRISOQUINE IN HEALTHY VOLUNTEERS FROM CUBA AND NICARAGUA**

M.E.G. Naranjo; P. Dorado; L.R. Calzadilla; M. Álvarez; R. Ramírez; E.M. Peñas-LLedó; B. Pérez; I. González; A. LLerena; and CEIBA Consortium

**1CICAB, Clinical Research Centre, Extremadura University Hospital, Badajoz, Spain; 2Hospital Psiquiátrico de La Habana; 3Faculty of Medical Sciences and Faculty of Medicine “Calisto García”, La Habana, Cuba; 4Facultad de Medicina, UNAM Universidad Autónoma Nacional de Nicaragua, León, Nicaragua; 5Hospital de LLerena, Servicio Extremo de Salud SES, L.Lerena; and 6CIBERSAM, Instituto de Salud Carlos III, Madrid, Spain**

**Introduction:** Ultrarapid drug metabolism (UM) mediated by CYP2D6 is associated with duplicated or amplified functional CYP2D6 alleles. However, duplicated CYP2D6 alleles only explains a fraction (10%–30%) of the UM phenotype observed in Caucasian populations, and other biochemical and/or genetic factors involved in UM phenotype remain unexplained yet. CYP2D6 -1584C>G has been related with changes in CYP2D6 expression, being -1584G associated with higher expression. The aim of this study was to explore the relationship between CYP2D6 -1584C>G polymorphism and the debrisoquine hydroxylation capacity.