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## TCT@ACC-i2: The Interventional Learning Pathway

**IMPACT OF CIGARETTE SMOKING ON P2Y<sub>12</sub> RECEPTOR BINDING ACTIVITY BEFORE AND AFTER CLOPIDOGREL THERAPY IN PATIENTS WITH CORONARY ARTERY DISEASE**

Poster Contributions

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Sunday, March 30, 2014, 9:45 a.m.-10:30 a.m.

Session Title: PCI Pharmacology

Abstract Category: 44. TCT@ACC-i2: Translation and Pre-clinical Research

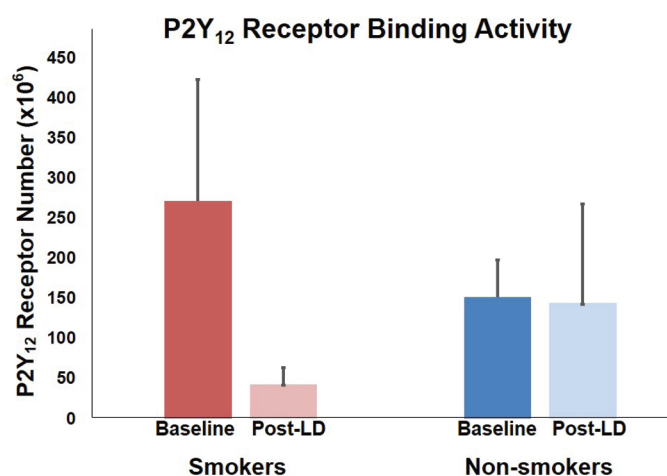
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**Background:** Smoking enhances the antiplatelet effects of the P2Y<sub>12</sub> receptor inhibitor clopidogrel. In addition to enhancing hepatic metabolism, nicotine exposure has been associated with increased P2Y<sub>12</sub> receptor expression in platelet lysates from healthy volunteers. However, the impact of cigarette smoking on platelet P2Y<sub>12</sub> receptor binding in patients with coronary artery disease (CAD) before and after clopidogrel exposure is unknown.

**Methods:** Clopidogrel-naïve patients with stable CAD on maintenance aspirin (81 mg/day) therapy were enrolled. Smoking status was defined as smokers of >10 cigarettes/day, while non-smokers needed to be free of nicotine exposure. P2Y<sub>12</sub> receptor expression was assessed by radioligand receptor binding in platelet rich plasma prior and 24 hours after a 600 mg loading dose (LD) of clopidogrel.

**Results:** Baseline P2Y<sub>12</sub> receptor binding was 1.8-fold higher in smokers (n=10) compared with non-smokers (n=10). After a 600 mg LD of clopidogrel, smokers showed a 6.4-fold reduction in receptor binding compared with baseline, indicative of marked clopidogrel-mediated receptor blockade. There were minimal changes in P2Y<sub>12</sub> receptor binding among non-smokers.



**Conclusions:** Among patients with stable CAD, smokers have more P2Y<sub>12</sub> receptor binding than non-smokers and have a higher degree of inhibition following a 600 mg LD of clopidogrel.