Methods: A retrospective cohort including incident OAD users was set up using the French health insurance database (EGB), a 1/97 permanent random sample of the national healthcare insurance database. Subjects aged 18 years or more initiating an OAD between 1 January 2006 and 31 December 2011 were included in the cohort. Date of initiation was defined as the index date for the follow-up. Subjects with a diagnosis of cancer at inclusion or during the year previous to the index date were excluded. Non-persistence was defined as a gap of OAD treatment coverage between end of the previous OAD prescription and new prescription greater than or equal to 90 days. A cause-specific Cox proportional hazards model was used to examine the association between cancer occurrence and OAD non-persistence and take into account the competing risk of death. Cancer occurrence was studied as a time dependent variable.

Results: The study included 13,943 OAD users. Median follow-up was 760 days. Non-persistence risk was higher after a diagnosis of cancer: (HR: 1.93 and IC 95% 1.69; 2.21). Results were adjusted for age, sex, insecurity, first OAD used, type of prescriber and polypharmacy. Subgroup analyses according to the cancer localization found a higher risk of non-persistence for lung cancer (HR: 2.66 and IC 95% 1.68; 4.23) and colorectal cancer (HR: 2.02 and IC 95% 1.40; 2.91).

Conclusions: Our findings indicate that there is an association between cancer diagnosis and OAD non-persistence. Additional studies of this type would be useful to evaluate association between cancer diagnosis and other chronic diseases persistence.

431. CYP2B6 G516T Minor Allele Protective of Late Virologic Failure in Efavirenz-Treated HIV Patients in Botswana

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Background: CYP2B6 polymorphisms that affect efavirenz (EFV) concentrations are common, but the effect of this polymorphism on HIV virologic failure in clinical practice settings has not fully been elucidated.

Objectives: To investigate the relationship between the CYP2B6 G516T polymorphism and late virologic failure in patients treated with EFV in Gaborone, Botswana.

Methods: We performed a case–control study that included 1,338 HIV-infected black Batswana on EFV-based antiretroviral therapy (ART) at outpatient HIV clinics between July 2013 and April 2014. Cases experienced late HIV failure, defined as plasma HIV RNA greater than 1000 copies/mL after maintaining viral suppression (less than 400 copies/mL) for at least 6 months. Four control patients, who had plasma HIV RNA less than 400 copies/mL on ART for at least 6 months were select for each case. Logistic regression was used to determine the adjusted odds of late HIV failure by G516T genotype.

Results: A total of 1,167 patients provided a blood sample, of which 67 (5.7%) samples failed genotyping. Compared to controls, cases were more likely to be male, more likely to engage in hazardous drinking, have a lower BMI, were on ART for a shorter period of time, and more frequently reported depressive symptoms. After adjustment for age and CD4 count, the CYP2B6 516 T-allele was protective against late HIV virologic breakthrough, adjusted OR 0.70; 95% CI 0.50–0.97.

Conclusions: The CYP2B6 516 T-allele was protective against late virologic breakthrough in patients with initial (6 month) HIV RNA suppression on EFV-based ART. Future studies are needed to assess long-term viral benefits of identifying and offering EFV containing ART to black African HIV patients with CYP2B6 T-alleles, especially given the wider availability of a single pill EFV in this setting.

432. *PPAR-α* Genetic Variants Influence On-Treatment Platelet Reactivity in Patients Treated with Clopidogrel and Lipid-Lowering Drugs and Undergoing Non-Urgent Percutaneous Coronary Intervention with Stent Implantation

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Background: Response to clopidogrel varies between patients, due to many factors, like polymorphisms in genes encoding for metabolizing enzymes. The *CYP3A4*22* polymorphism has been proven to decrease the expression of CYP3A4, while the *PPAR-α* genetic variants G209A and A208G have been identified as determinants that affect CYP3A4. Statins and fibrates, which are the ligands of PPAR-*α* as well as being metabolized by CYP3A4, might also affect the response of clopidogrel through these two proteins.

Objectives: To investigate the association between on-treatment platelet reactivity and the *CYP3A4*22* allele and genetic variations of the *PPAR-α* genes in clopidogrel-treated patients undergoing non-urgent percutaneous coronary intervention (PCI) with stenting and to evaluate the influence of statin/fibrate co-medication on these associations.

Methods: A total of 1126 patients with non-urgent PCI and stenting pre-treated with clopidogrel and aspirin were genotyped for *CYP3A4*22* and *PPAR-α* (G209A and A208G). Platelet reactivity was measured using the VerifyNow® $P2Y_{12}$-assay, expressed in PRU. Multivariate linear regression analysis was used to assess the association between the genetic variants and platelet reactivity, adjusted for confounders, including the CYP2C19 metabolizer status. A stratified analysis was conducted for patients with statin/fibrate co-medication. A recessive model was used for all associations.

Results: The *CYP3A4*22/*22* genotype was present in 0.4% of patients, 6.8% had the *PPAR-α* G209A AA genotype, and 7.0% had the *PPAR-α* A208G GG genotype. *CYP3A4*22* was not associated with platelet reactivity. *PPAR-α* genetic variants were significantly associated with platelet reactivity (*PPAR-α* G209A AA: −23.87 PRU [−43.54, −4.19]; *PPAR-α* A208G GG: −23.70 PRU [−43.13, −4.27]). In patients who were on statin/fibrate co-medication, these *PPAR-α* genetic variants were associated with an even lower platelet reactivity (−29.74 PRU [−50.94, −8.54], and −29.38 PRU [−50.26, −8.49], respectively), while those without statin/fibrate co-medication did not show a significant change in platelet reactivity (13.00 PRU [−39.79, 65.80]).

Conclusions: Two genetic variants in *PPAR-α* (G209A and A208G) were associated with lower platelet reactivity in patients with non-urgent PCI and stenting co-treated with clopidogrel and lipid-lowering drugs.

433. Programmed Cell Death Receptor Ligand 1 (PD-L1) Expression; Epidermal Growth Factor Receptor (*EGFR*) and *Kirsten RAS* (KRAS) Mutations in Third-Line Therapy (3L) Non-Small Cell Lung Cancer (NSCLC) Patients: A Danish Cohort Study

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Background: 3L NSCLC patients are unresponsive to chemotherapies and are difficult to treat. Therapies that target specific biomarkers may benefit patients.

Objectives: In NSCLC patients who received 3L therapy, we examined the association of PD-L1 expression, mutations in *KRAS* and *EGFR* and survival.

Methods: 3L NSCLC patients diagnosed during 2001–2012 with sufficient archival tumour tissue were selected from the Danish Lung Cancer Group Registry. We retrieved patient data from population-based medical registries, and paraffin-embedded tumor tissue from pathology archives. We assessed PD-L1