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► To cite this version:

Konstantinos P. Economopoulos, Theodoros N. Sergentanis. Does race modify the association between CYP1B1 Val432Leu polymorphism and breast cancer risk? A critical appraisal of a recent meta-analysis. *Breast Cancer Research and Treatment*, 2010, 124 (1), pp.293-294. 10.1007/s10549-010-1097-3 . hal-00563448

HAL Id: hal-00563448

<https://hal.science/hal-00563448>

Submitted on 5 Feb 2011

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Does race modify the association between CYP1B1 Val432Leu polymorphism and breast cancer risk? A critical appraisal of a recent meta-analysis.

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Dear Editor,

We read with great interest the recent meta-analysis by Yao et al.[1], which has reached important conclusions about the association CYP1B1 Val432Leu polymorphism and breast cancer risk. Yao et al.[1] did not demonstrate any significant associations between CYP1B1 Val432Leu polymorphism and breast cancer susceptibility, either at the overall or the race-specific analyses (Asian, Caucasian, African and Mixed).

Nevertheless, close inspection of the studies analyzed by the authors revealed some methodological issues that are worth mentioning and clarifying. Adopting the same search strategy and end-of-search date as Yao et al.[1], we have located three relevant case-control studies in MEDLINE with a total number of 685 breast cancer cases and 931 controls, which have not been included in the meta-analysis[2-4]. Of note, all three case-control studies were performed on Caucasian populations.

Moreover, a methodological issue seems worth commenting. Specifically, the study by Van Emburgh et al.[5] has provided separate data on Caucasian (381 cases and 412 controls) and African-American (52 cases and 77 controls) subpopulations; worthy of note Yao et al.[1] classified the study as a study on “mixed” populations and did not extract the reported separate data on Caucasian and African-American subgroups. Under the light of the above we have performed the overall meta-analysis, as well as the race-specific analyses in Caucasian, African and mixed populations; the analysis on Asian subjects has been spared, since the issues mentioned above did not pertain to Asian studies.

The overall meta-analysis, as well as the analyses on Caucasian and mixed populations replicated the results reported by Yao et al.[1]; on the other hand a statistically significant

finding emerged on African populations. Specifically, at the overall analysis the pooled Odds Ratios (ORs) (95% Confidence Intervals, CI) were 1.021 (0.941-1.109, random effects) for heterozygous and 1.034 (0.930-1.150, random effects) for homozygous Val subjects. Concerning Caucasian subjects, the pooled OR were 1.051 (0.939-1.176, random effects) for heterozygous carriers and 1.038 (0.912-1.180, random effects) for homozygous carriers. Subanalysis on African subjects demonstrated that heterozygous carriers were associated with increased breast cancer risk (pooled OR=1.918, 95% CI: 1.011-3.638, p=0.046, fixed effects), whereas the results concerning homozygous carriers remained at the level of borderline significance (pooled OR= 1.773, 95% CI: 0.947-3.318, p=0.073, fixed effects).

In conclusion, race-specific associations between CYP1B1 Val432Leu polymorphism and breast cancer risk may well exist; further studies on African populations are needed so as to validate the present results in larger sets of case-control studies.

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