

Letters to the Editor

AURKA and Breast Cancer in BRCA1/2 Mutation Carriers

To the Editors: A recent article reported, “the *F31I* polymorphism in *AURKA* is not associated with a modified risk of breast cancer found in 3,884 breast cancer *BRCA1* and *BRCA2* carrier cases compared to unaffected *BRCA1/2* mutation carriers” (1). This agrees with our previously published results where no association of increased breast cancer risk was found in 107 *BRCA1* and *BRCA2* breast cancer carrier cases compared with 653 unaffected wild-type *BRCA1/2* controls (2), which Couch et al. did not cite. We also found strong association with homozygous *31I* in 652 sporadic breast cancer cases compared with unaffected controls. In addition, we compared the *F31I* polymorphism in the 107 *BRCA1/2*-mutated breast cancer cases with 40 unaffected *BRCA2* mutation carriers. We found that the unaffected *BRCA2* mutation carriers had clearly higher *31I* allele frequency of 27.5% compared with 21.0% in *BRCA1/2* breast cancer cases, which indicates that the *31I* isoform might be protecting against breast tumor development in *BRCA2* mutation carriers. Our results, and those of Couch et al., about *BRCA1/2* mutation carrier cases agree with recent studies on familial or high-risk breast cancer cases that did not find association between the *F31I* polymorphism and breast cancer risk. On the other hand, increased sporadic breast cancer risk is associated with the *F31I* polymorphism as discussed before (2). It is therefore clearly important when looking for low-penetrance cancer susceptibility genes to acknowledge the influence of major cancer genes, such as *BRCA1* and *BRCA2*.

Couch et al. (1) state both in abstract and introduction, “amplification of *AURKA* has been detected at higher frequency in tumors from *BRCA1* and *BRCA2* mutation carriers than in sporadic breast tumors, suggesting that overexpression of *AURKA* and inactivation of *BRCA1* and *BRCA2* cooperate during tumor development and progression.” No citations were given for this statement and, to our knowledge, there are no published data showing association between *AURKA* amplification and *BRCA1* inactivation. On the other hand, we recently

reported a strong association between *AURKA* (*Aurora-A*) amplification and *BRCA2* mutation in breast tumors (3). In this study, we analyzed 61 breast tumor tissue sections for *AURKA* amplification by fluorescence *in situ* hybridization, where 20 had *BRCA2* mutation. Other studies based on comparative genomic hybridization of *BRCA1/2*-mutated breast tumors agree with our results where chromosome region 20q13, the location of the *AURKA* gene, is more often amplified in *BRCA2*-mutated tumors compared with sporadic and *BRCA1*-mutated breast tumors (4-6).

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