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# PLXNB1 mutations in human cancer

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### Letter

Wong et al. recently reported somatic *PLXNB1* mutations in 46% (41/89) of primary prostate cancer and 89% (8/9) of prostate cancer bone metastases. To extend and confirm their observations, we sequenced exons 23 and 27 of *PLXNB1* (where most mutations had been detected) in 15 primary prostate, 83 lung and 120 breast carcinoma and 120 glioblastoma samples, without detecting any nucleotide change. The observed discrepancy between our results and those reported by Wong is highly significant in primary prostate tumors (Fisher's exact test, P value < 0.001).

We noted that 99% (79/80) of the changes reported by Wong et al. were C:G>TA or A:T>G:C.¹ Previous work has demonstrated that deamination of adenine or cytosine occurs frequently in small quantities of DNA.²-5 This can lead to PCR errors, especially with DNA extracted from paraffin embedded tissue such as those predominantly used by Wong and colleagues.¹ To test this hypothesis, we repeated the PCR-sequencing approach using lower amounts of DNA extracted from prostate tumors as compared to our initial analysis. Under these conditions we detected A:T>G:C nucleotide changes causing a number of nonsynonymous amino acid changes (L1547F, T1750A, V1767A, V1769A, L1772P, T1802A), one of which is identical to one described in.¹ These changes, however, could not be confirmed when the same samples were assessed multiple times.

Based on these findings, we suggest that the mutation frequency of *PLXNB1* in prostate cancer should be reconsidered.

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