that a considerable fraction of CRC LN metastases do not resemble the respective primary tumors. The reasons that may underlie these findings are fully described in our article, but in summary, we believe that a *KRAS* mutation can either be acquired during metastasis to the LNs, as in our study, or be part of a heterogeneous population of neoplastic cells that constitutes the bulk of the primary tumor. These results are of fundamental importance, because they may represent one of the resistance mechanisms interfering with the response of patients with *KRAS*-WT mCRC to anti-EGFR monoclonal antibody therapy. We have written this letter because we believe that our results should be taken into consideration if *KRAS* screening is used as a tool to select patients for administration of this type of therapy.

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The author(s) indicated no potential conflicts of interest.

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In Reply: We thank Velho et al for their interest in our study, which showed a 95% concordance rate of *KRAS* and *BRAF* status between primary and metastatic sites of colorectal carcinomas.[1] The authors refer to their previous study, in which they found a lower concordance rate (64%) between lymph node metastases and primary tumors in a cohort of 28 of 250 colorectal cancers.[2] Possible explanations for the discrepancy between the two concordance rates may be, first, that our patient series included only patients with stage IV disease and that of Velho et al included patients with stage 0 to IV disease, and second, that in the latter study, *KRAS* status was not assessed in distant metastatic sites.

In addition to our findings, full concordance of *KRAS* status between primary tumors and distant metastases,[3-5] and between primary tumors and regional lymph nodes,[6] has been reported by independent investigators, supporting the notion that detection of a KRAS-activating mutation in either a primary or metastatic site is sufficient to exclude a patient from epidermal growth factor receptor–targeted monoclonal antibody therapies.

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