# Other RAS mutations incidence in CRCm in routine clinical practice: a center experience.

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**Background:** Patients with metastatic colorectal cancer that harbors KRAS mutations in exon 2 do not benefit from anti-epidermal growth factor receptor (EGFR) therapy. Other activating RAS mutations also are negative predictive biomarkers for anti-EGFR therapy, as recently shown in a subanalysis of the PRIME study.

**Methods:**From June 2013 we began to make the determination of other mutations of RAS. The determination of the mutational status was performed using a validated analytical method for determining KRAS mutations (exons 2, 3 and 4) and NRAS (exons 2,3 and 4) in formalin-fixed, paraffin-embedded specimens. It was used cobas® KRAS mutation test (Roche), which is a real-time PCR test intended for the identification of mutations in codons 12, 13 and 61 of the gene. Pyrosequencing of the NRAS exon 1 (codon 12 and 13), exon 2 (codons 60 and 61) and exon 4 (codon 117 and 146) of NRAS and KRAS was performed using the TheraScreen® NRAS Pyro kit (Qiagen).

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**Results:** From June 2013 to February 2015, 314 patients were analyzed. Of these, 134 patients (43%) had mutations in exon 2 (codons 12/13) of KRAS. 16 patients (5%) had an invalid result. From the 164 (52%) patients with wild type KRAS exon 2, 132 patients (80%) were able to analyze other RAS mutations.
A total of 12 patients (7.3%) with nonmutated KRAS exon 2 had other RAS mutations: 6 patients in the KRAS exon 3 (codon 61), 1 patient NRAS exon 2 (codon 12/13), 4 patients NRAS exon 3 (codon 61) and 1 patient in NRAS exon 4 (codon 117/146).

**Conclusion:** RAS mutations, in addition to KRAS exon 2 mutations, predict a lack of response to anti-EGFR therapy in patients with metastatic colorectal cancer. Approximately 20% of KRAS exon 2 wild-type tumors harbored one of the new RAS mutations, as reported in various retrospective studies.
In our series other RAS mutations were detected in 7,3%, lower than published so far.

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