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Characterization of MYC and MNT in colorectal cancer patients

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Background. c-Myc oncogene is deregulated in several human tumors and its control on cell proliferation is tuned by a series of repressors, whose the most relevant is MNT. In colorectal cancer (CRC), Myc is altered by gene amplification in about 25% cases and MNT loss of heterozygosity (LOH) has been associated with CRC carcinogenesis. As the precise interplay between Myc and MNT has not been fully investigated in tumor specimens, our aim was to better characterize the roles of Myc and MNT in CRC.

Methods. We analyzed 53 sporadic CRC patients. c-Myc gene status was evaluated by FISH. MNT was assessed for LOH using 5 microsatellite loci located inside or near the gene.

Results. We observed c-Myc amplification in 14/45 (31%) evaluable cases. MNT loss was found in 28/47 (58%) informative cases. Out of 39 cases evaluable for both c-Myc and MNT, c-Myc amplification was found in 12/39 (31%) cases and MNT loss in 25/39 (64%) cases. By comparing c-Myc and MNT gene statuses, we observed that c-Myc amplification occured in 10/25 (40%) tumors with loss of MNT and only in 2/14 (14.28%) cases without MNT loss (p=0.15).

Conclusions. c-Myc and MNT are deregulated in a considerable number of CRC specimens confirming a key role of these genes in CRC pathogenesis. The majority of c-Myc amplified tumors showed MNT loss, possibly implying that a synergistic effect of these alterations is required in CRC development.