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miR-143 over-expression reduces the growth of xenograft tumors from colon carcinoma cells

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We have previously shown that miR-143 is down-regulated in colorectal cancer and that miR-143 over-expression in HCT116 cells increases sensitivity to 5-fluorouracil, reduces cell viability and increases apoptosis *in vitro*. In the present study, we evaluated the role of miR-143 over-expression on HCT116 xenograft tumor growth in nude mice. HCT116 cells with stable miR-143 over-expression (*over-143)* and control (*empty*) cells were injected s.c. into the backs of nude mice, and tumor growth was evaluated. Tumors arose approximately 14 days later, and the experiment was ended 40 days after injection. miR-143 was confirmed to be significantly over-expressed in *over-143versusempty* xenografts, by Taqman real-time PCR. *Over-143* xenografts displayed slower tumor growth compared to *empty* xenografts, with significantly smaller tumor volumes, from 23 until 40 days *in vivo* ($p < 0.05$), with final volumes of 928 ± 338 and 2312 ± 387 mm^3, respectively. Evaluation of apoptotic proteins showed that *over-143versusempty* xenografts, display reduced Bcl-2 expression, and increased caspase-3 activation and PARP cleavage ($p < 0.05$). In addition, the incidence of apoptotic cells, assessed by TUNEL, was increased in *over-143versusempty* xenografts. Therefore, our results suggest that the reduced tumor volume may, in part, be due to increased miR-143-induced apoptosis. Collectively, our results reinforce the relevance of miR-143 in colorectal cancer, suggesting an important role in the control of *in vivo* tumor progression. This further expands its anti-proliferative, pro-apoptotic and chemosensitizer role that we have previously demonstrated *in vitro*.

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