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Evaluation of the role of mir-34b in modulation of radioresistance in non-small cell lung cancer

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Radiotherapy is a major therapeutic weapon in lung cancer. However, the resistance to radiotherapy is frequent. The microRNAs of the miR-34 family, miR-34a, miR-34b and miR-34c, described as effector molecules in the cellular response to activation of P53, have low expression levels in lung cancer [1]. The mRNA of the anti-apoptotic protein BCL-2 is among the targets of miR-34 family.

The aims of our study were to clarify the involvement of miR-34b over-expression in the modulation of radiation response in NSCLC cell lines and the mechanisms involved. For these purposes we used two radioresistant NSCLC cell lines, A549, expressing P53, and H1299, not expressing P53. Cells transfected with pre-miR-34b or with a transfection control were exposed to different irradiation doses. The response to irradiation was assessed by cell survival curves obtained by clonogenic assay, and flow citometry allowed the characterization of cell death and the quantification of BCL-2, BAX and P53 protein expression levels. Our results showed that both cell lines had low expression levels of miR-34 family members, especially for miR-34b/c. Over-expression of miR-34b sensitized A549 cells to low doses of radiation and decreased BCL-2 expression, but without changing apoptosis levels. H1299 cells remained unchanged.

These results suggest that in NSCLC expressing P53, response to radiotherapy is dependent on BCL-2 levels and may be modulated by over-expression of miRNA-34b. Other cell death mechanisms than apoptosis, but also involving BCL-2, like autophagy, could to be involved.

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