p63 is a transcription factor playing a critical role in physiological and pathological processes. In vivo and in vitro studies together with genomic profiling and bioinformatic analysis have clearly demonstrated the relevance of the p63 N-terminal truncated isoform (ΔNp63) activity in the carcinogenesis of epithelial tumors, such as squamous cell carcinoma of different origins and basal subtype of breast carcinoma (Gatti et al., *Int J Mol Sci*. 2019; Gatti et al., *Mol Oncol*. 2019). The general aim of this research line is to deeply investigate the oncogenic routes orchestrated by ΔNp63 in epithelial tumors, with special emphasis to the molecular crosstalk between its activity and cancer stemness, tumor microenvironment and metastasis. By performing a range variety of cellular, molecular, bioinformatic and in vivo approaches, our group have unveiled novel ΔNp63-mediated pathways regulating migration and invasion (Giacobbe et al., *Oncogene* 2016), hyaluronic acid metabolism and signaling (Compagnone et al., *PNAS* 2017; Gatti et al., *Oncogenesis* 2018) and cellular metabolism (Giacobbe et al., *Cell Cycle* 2016). We also participated to collaborative studies which allowed the identification of novel ΔNp63 interactor (Regina et al., *Oncotarget* 2016) and transcriptional target genes controlling IGF-1 signaling (Frezza et al., *Aging* 2018) and breast cancer cell stemness (Memmi et al., *PNAS* 2015). Currently, we are characterizing a member of the ABC transporter family and two long non-coding RNAs (lncRNAs) as ΔNp63 oncogenic effectors controlling the immune landscape and chemotherapeutic response, respectively.

**References:**


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