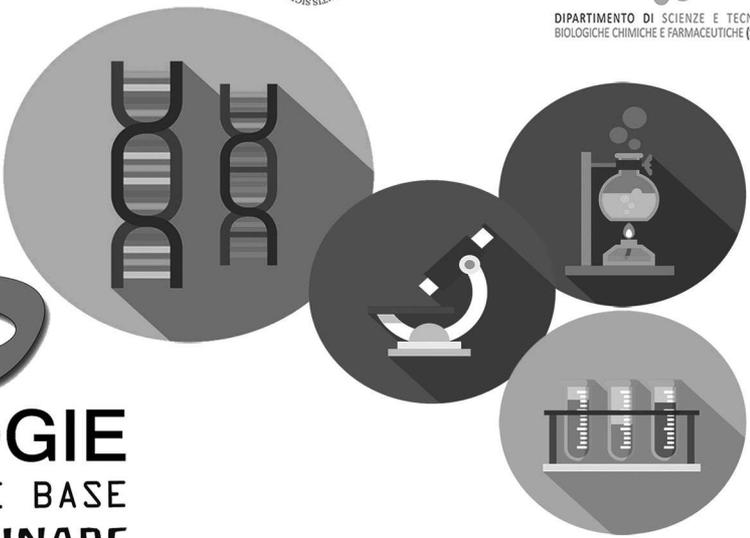




Bio
TECNOLOGIE
 RICERCA DI BASE
 INTERDISCIPLINARE
 TRASLAZIONALE
 IN AMBITO BIOMEDICO



BIOINFORMATICA
IMMUNOLOGIA
MALATTIE APPARATO RESPIRATORIO
MALATTIE METABOLICHE
MICROORGANISMI NELLE BIOTECNOLOGIE
NANOTECNOLOGIE
NEUROSCIENZE
ONCOLOGIA
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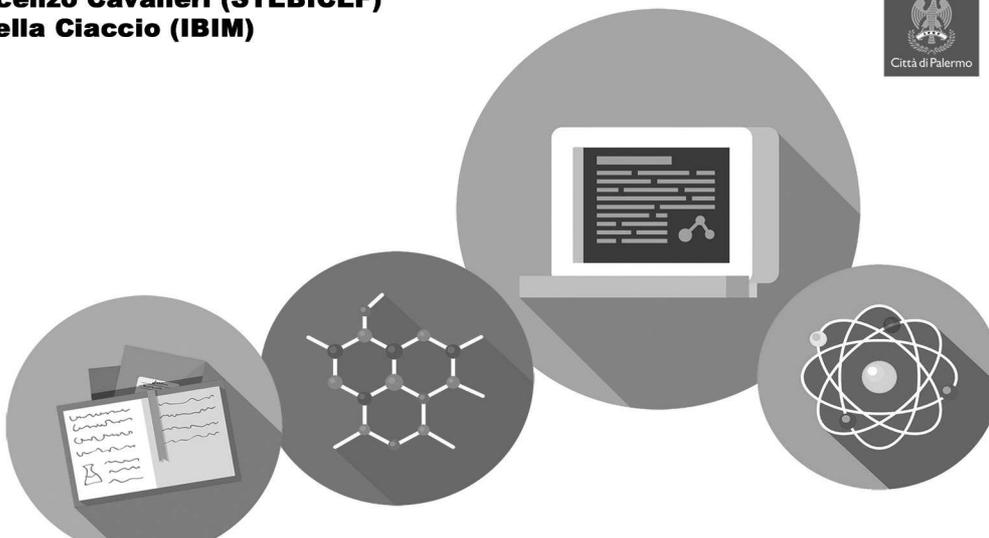
LIBRO
 5° Meeting **degli ABSTRACT**

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Let-7d-5p miRNA shows oncogenic functions in triple negative breast cancer

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Triple negative breast cancer (TNBC) is a highly aggressive subtype of BC which lack of targeted therapies and is associated with poor prognosis. The presence of Cancer Stem Cells (CSCs) could be responsible for TNBC resistance to therapy, recurrence and metastasis, and might explain the difficult of its eradication.

MiRNAs -a class of small non-coding RNAs- can modulate gene expression and their dysregulation may cause cancer formation. The let-7 family is dysregulated in various cancers and often its roles are unclear and of difficult interpretation. For example, let-7d can be over- or down-expressed and can act as tumor suppressor or oncogene.

Here, we evaluated the expression profiles of let-7d-5p and its function in TNBC. We found that let-7d-5p was upregulated in human TNBC tissues (n=21) and up-/down-regulated in cell lines (n=4). We also analyzed a TCGA BRCA data set of BC patients (n=579) and found that high let-7d expression significantly correlates with poor survival in BC. Moreover, we observed let-7d-5p up-regulation in tertiary mammospheres derived from MDA-MB-231 cells, suggesting its correlation with stemness. Therefore, after stable let-7d-5p-knockdown of MDA-MB-231 cells, we investigated the changes in cell proliferation, clonogenic growth, mammosphere-forming ability, migration and invasiveness, *in vitro*. We found that let-7d-5p-knockdown consistently reduced cell growth in both two- and three-dimensional culture systems and in colony-forming ability, self-renewal, migration and invasiveness. Overall, our findings show that let-7d-5p regulates multiple oncogenic features of MDA-MB-231 cells, suggesting that it acts as oncogene and could be evaluated as a new therapeutic target in TNBC.

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