

# AT11-functionalized liposomes for oral cancer: characterization and in vitro evaluation

## NANOTECHNOLOGIES

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## Introduction

Conventional anticancer therapies present low specificity, leading to several side effects. In order to try to solve these drawbacks, aptamers have been proposed to favor drug accumulation in cancer cells.

AS1411 is a G-quadruplex aptamer able to recognize nucleolin, a protein overexpressed on cancer cell surface, but it presents low response rates and suboptimal pharmacokinetics. Nevertheless, AS1411 is being used as a targeting agent.

In this sense, in the current work the use an AS1411 derivative with improved activity, the AT11, to functionalize liposomes with the aim to improve the selectivity of a potential anticancer drug, the acridine orange derivative ( $C_8$ ) towards oral cancer cells.

## Methodologies

- Empty and  $C_8$ -associated liposomes were produced by ethanol injection method;
- Both liposomes were further engineered by binding a AT11-TEG-cholesteryl moiety;
- The different produced liposomes were dimensionally characterized by dynamic light scattering;
- The biological effect of empty and  $C_8$ -liposomes on SCC154 cell line (tongue cancer cell line) and Het1A cells (normal epithelial cell line) was determined by MTT viability assay and migration assay, whereas the internalization was determined by confocal microscopy.

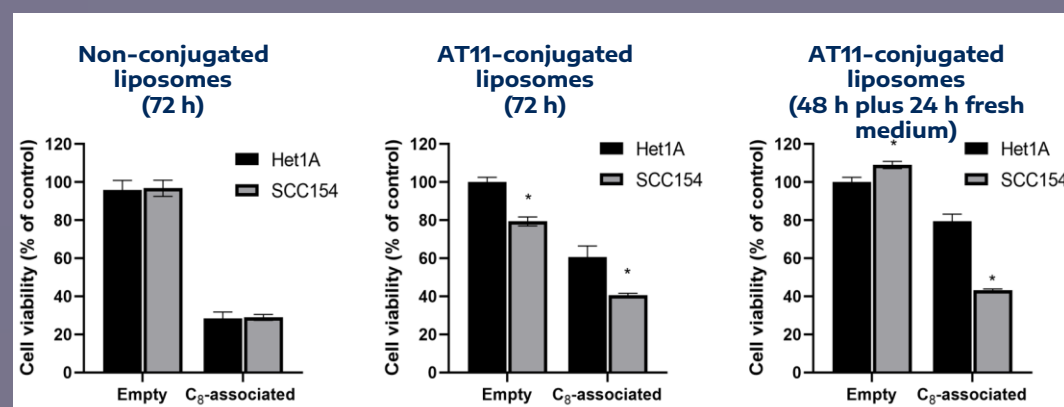
## Results

Cell viability was almost unaffected after treatment with empty liposomes.

When cells are treated with the  $C_8$ -liposomes, a dose-response effect was observed.

AT11 confers selectivity of the liposomes towards the SCC154 cell line.

Overall, these findings suggest that the designed liposomal formulation represent a promising drug carrier for the therapy of oral cancers.



Cell viability assay of  $C_8$ -associated liposomes non-conjugated or with AT11 functionalization in Het1A and SCC154 cell lines after 72 h (right) of treatment or 48 h of treatment with 24 h of fresh medium. \*- statistically different relatively to Het1A.

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