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Short Review

Cellular uptake of aptamer by Quantum Dots (QDs)

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Aptamers are short single stranded oligonucleotide sequences that exhibit high binding affinity and high specificity against their target molecule. Binding affinity and specificity are crucial features for aptamers in order to exploit their therapeutic and diagnostic potential and to make them an appealing candidate for the commercial market[12]. Aptamers contain functional moieties that can fold into different conformation such as hairpin stem and loops, G-quadruplexes, and pseudoknots. A study led by Dr Harleen Kaur involving unique stem-loop truncation strategy was employed to find the binding domain in a 66-mer long DNA aptamer sequenceagainst the heparin binding domain of vascular endothelial growth factor (VEGF165) protein[1]. The results from the work demonstrated identification of a 26-mer long aptamer sequence referred as SL2-B in the paper with improvement in the binding affinity by more than 200-folds (Kd = 0.5nM) against VEGF protein. To improve the biostability of the aptamer in the biological fluids, the phosphorothioate linkages (PS-linkages) in the phosphate backbone of the DNA were introduced at the 5'-and 3'-termini of the obtained SL2-B aptamer sequence. The PS-modified SL2-B aptamer sequence demonstrated significant improvement in the stability without comprising the binding affinity of the aptamer sequence for its target and demonstrated its inhibitory effect on Hep G2 liver and MCF-7 breast cancer cells[3]. The cellular fate of the sequence was further exploited by conjugating the modified sequence to the nanometer sized quantum dots (QDs) that were used as fluorescent probes for imaging purpose. The monodisperse hydrophilic glutathione capped core-shell CdSe/ZnS quantum dots with a quantum yield of 95% relative to fluoroscein were used.

The quantum dots were conjugated to the aptamer sequence through EDC/NHS chemistry. The results from the study show that the sequence can be taken up by the Hep G2 liver cancer cells without any external transfecting or cell permeabilizing agent. Furthermore, the data indicates that this cellular uptake might be responsible for the antiproliferative activity of the PS-modified SL2-B aptamer sequence and further studies are warranted to elucidate the antiproliferative moelcular mechanism of the aptamer sequence in cancer cells.

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