**CARDIAC TARGETING PEPTIDE: STUDIES INTO MECHANISM OF TRANSDUCTION**

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**Introduction**: Cardiac targeting peptide (CTP) is a 12-amino acid long, synthetic non-naturally occurring peptide with the ability to transduce the murine heart in vivo. The mechanism underlying this heart targeting remains unknown. We hypothesized that the transduction capability will be localized to either C- or N-terminus. We further hypothesized that cyclization of CTP will improve its transduction abilities.

**Methods and Results:** CTP was synthesized using solid-phase peptide synthesis in its native 12 amino acid full-length form, a 6 amino acid N-terminal form called CTP-A, and a C-terminal CTP-B. CTP and CTP-B were also synthesized in cyclic forms. A random, linear, 12-amino acid long peptide (RAN) was also synthesized to serve as control. All peptides were fluorescently labeled with Cyanine5.5 (Cy5.5). H9C2 cells were incubated with the peptides at 5uM and analyzed using cell sorting. Both variants were able to transduce H9C2 cells with CTP-B consistently more than CTP-A, but not significantly more than full-length CTP. Cyclizing both full length CTP and CTP-B significantly reduced their transduction efficiencies (Figure 1).

**Conclusions:** The full-length CTP was superior in its transduction abilities over truncated 6-amino acid N- or C-terminus versions. Cyclizing CTP or CTP-B resulted in significant interference with

cell entry and a decrease in transduction.

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