Cystic fibrosis (CF) is an inherited disease caused by mutations in the gene codifying for a protein called CFTR, a chloride channel, and results in significant morbidity and early mortality in affected individuals. A person with CF produces abnormal CFTR protein - or no CFTR protein at all, which causes the body to make thick, sticky mucus instead of the thin, watery kind. Unfortunately, to date, there is no cure for CF, even if aminoglycoside antibiotics, principally given through inhaled therapy seem to improve lung function by impeding the growth of colonized bacteria. Several studies confirmed the ability of gentamicin to suppress CFTR premature termination codons and restore CFTR function. Despite its interesting pharmacological profile, gentamicin salt is used for only topical or parenteral administration showing poor pharmacokinetic properties because of its strong hydrophilicity and hygroscopicity. A chemical derivatization of gentamicin salt that improves the lipophilicity of molecules to facilitate the insertion of gentamicin in innovative formulations for inhalation administration was developed. Additionally, several HPLC, LC-MS methods were set up and isolation of each intermediate synthesized and final purification and characterization of derivatized gentamicin was achieved. As a next step, each isomer isolated will be tested in order to evaluate the potential of them to correct CFTR dysfunction by suppressing premature stop mutations in cells transfected with a mutated CFTR.