Amyloid aggregation plays a key role in various neurodegenerative diseases, and understanding the structural mechanisms underlying this process is vital for developing effective interventions. This study focuses on the **SAA1-13 peptide**, a 13-amino acid segment (RSFFSFLGEAFDG) from the N-terminal region of the wild-type **Serum Amyloid A1 (SAA1)** protein. SAA1 is an acute-phase protein associated with high-density lipoprotein (HDL) and is a precursor to amyloid A (AA), which can lead to inflammatory amyloidosis. The goal of this study was to explore the effects of phenylalanine substitutions in **SAA1-13** on secondary structural dynamics and aggregation potential. Using molecular dynamics simulations in Amber, we investigated a series of **SAA1-13 peptide modifications**: SAA1-13F3A, SAA1-13F3L, SAA1-13F4L, SAA1-13F11L, and SAA1-13F6L. These modifications involved substituting the phenylalanine residues at positions 3, 4, 6, and 11 with different amino acids such as alanine or leucine. The purpose of these substitutions was to assess their impact on beta-bridge formation, beta-sheet content, and overall peptide aggregation behavior compared to the wild-type sequence.Our simulations revealed that substituting phenylalanine residues can significantly alter the structural behavior of the peptide. Certain substitutions, particularly at positions 3 and 4, reduced the formation of beta-sheets and beta-bridges, suggesting that these phenylalanine residues play a critical role in stabilizing amyloidogenic structures. The replacement with alanine or leucine potentially disrupts these interactions, delaying or inhibiting amyloid fibril formation.The results may possibly lead to open up new possibilities for designing therapeutic interventions aimed at disrupting amyloid formation by targeting key structural elements within amyloid precursor proteins.