ABSTRAK

Kanker merupakan penyakit yang terjadi akibat pertumbuhan sel yang tidak terkendali yang tumbuh secara abnormal serta merusak bentuk dan fungsi awalnya. Kanker payudara merupakan salah satu kanker yang banyak terjadi dan sering menyebabkan kematian. Penelitian ini bertujuan untuk mengetahui hasil penambatan molekul menggunakan HADDOCK dan CABS-dock terhadap reseptor protein EGFR (PDB ID: 1XKK) yang merupakan protein target proliferasi sel kanker payudara. Metode penelitian meliputi preparasi sekuen reseptor dan peptida, analisis sisi aktif reseptor dengan *prankweb,* proses penambatan, penentuan nilai energi afinitas serta visualisasi 2D menggunakan Ligplot. Hasil penambatan molekul HADDOCK menunjukkan bahwa nilai energi afinitas yang diperoleh dari masing-masing peptida P1− P12 terhadap protein EGFR berturut-turut -9,0 kkal/mol, -8,6 kkal/mol, -7,7 kkal/mol, -7,7 kkal/mol, -10,4 kkal/mol, -8,4 kkal/mol, -11,4 kkal/mol, -13,3 kkal/mol, -9,8 kkal/mol, -9,0 kkal/mol, -8,2 kkal/mol, -8,6 kkal/mol. Berdasarkan nilai energi afinitas ini P8 memiliki nilai energi afinitas terendah dengan tingkat kestabilan yang paling baik. Hasil penambatan molekul menggunakan CABS-dock diperoleh hasil pada P1−P12 dengan jumlah interaksi berturut-turut 24 interaksi, 24 interaksi, 19 interaksi, 43 interaksi, 32 interaksi, 42 interaksi, 34 interaksi, 60 interaksi, 30 interaksi, 38 interaksi, 43 interaksi, dan 24 interaksi. Berdasarkan hasil CABS-dock P8 memiliki jumlah interaksi paling banyak diantara peptida yang lain, residu asam amino Lys823, Ser969, Tyr801, dan Ser995 sebagai ikatan hidrogen serta Val819, His850, Ala972, Arg973, Gln849, Tyr813, Pro848, Asn771, Ile965, Tyr827, Phe997, His805, Tyr998, Phe795, Met987, Gln812, Asn808, Asn816, Asn826 sebagai ikatan hidrofobik. Ikatan hidrogen dan ikatan hidrofobik memiliki peran penting dalam menentukan interaksi antara protein-peptida, semakin banyak jumlah ikatannya interaksi protein-peptida semakin stabil.

Kata kunci : kanker payudara, peptida, protein EGFR, HADDOCK, CABS-dock

ABSTRACT

Cancer is a disease caused by uncontrolled cell growth, which proliferated abnormally and damaged the original structure and function. Breast cancer is one of the most prevalent cancers and frequently led to death. This study aimed to assess the molecular docking results obtained using HADDOCK and CABS-dock on the EGFR protein receptor (PDB ID: 1XKK), which served as a protein target of breast cancer cell proliferation. The research method included preparation of receptor and peptide sequences, analysis of the active side of the receptor with prankweb, docking procedures, determination of affinity energy values and 2D visualization using Ligplot. HADDOCK molecular docking results showed that the affinity energy values obtained from each peptide P1- P12 against EGFR protein were -9.0 kcal/mol, -8.6 kcal/mol, -7.7 kcal/mol, -7.7 kcal/mol, -10.4 kcal/mol, -8.4 kcal/mol, -11.4 kcal/mol, -13.3 kcal/mol, -9.8 kcal/mol, -9.0 kcal/mol, -8.2 kcal/mol, -8.6 kcal/mol. Based on this affinity energy value, P8 exhibited the lowest affinity energy and the highest stability. The CABS-dock molecular docking results revealed that peptides P1-P12 with the number of interactions respectively 24 interactions, 24 interactions, 19 interactions, 43 interactions, 32 interactions, 42 interactions, 34 interactions, 60 interactions, 30 interactions, 38 interactions, 43 interactions, and 24 interactions. Based on the CABS-dock results P8 had the most number of interactions among other peptides, amino acid residues Lys823, Ser969, Tyr801, and Ser995 as hydrogen bonds and Val819, His850, Ala972, Arg973, Gln849, Tyr813, Pro848, Asn771, Ile965, Tyr827, Phe997, His805, Tyr998, Phe795, Met987, Gln812, Asn808, Asn816, Asn826 as hydrophobic bonds. Hydrogen bonds and hydrophobic bonds had an important role in determining the interaction between protein-peptides, the more the number of bonds the more stable protein-peptide interactions.

Keywords: *breast cancer, peptide, EGFR protein, HADDOCK, CABS-dock*