ABSTRACT

SUBSTRATE SPECIFICITY OF α-L-ARABINOFURANOSIDASE GH51 FROM *Geobacillus thermoleovorans* IT-08 TOWARD *Mycobacteria* CELL WALL

The purpose of this research is to investigate the substrate stereospecificity of α-L-arabinofuranosidase GH51 *Geobacillus thermoleovorans* IT-08 (AbfA) toward *Mycobacteria* cell wall. In general, there are 3 stages in this research are (1) the production of AbfA recombinant from *E. coli* BL21(*DE3*)/pET-abfA, (2) specific activity assay of AbfA recombinant, (3) substrate stereospecificity analysis of AbfA in silico. In laboratory methods, AbfA has hydrolase activity toward D-arabinofuranoside (H37Rv and BFCC). However its activity toward L-arabinofuranoside (arabinogalactan, pectin, oat spelt xylan and arabinan) higher than its hydrolase activity toward D-arabinofuranoside (H37Rv and BFCC). On silico analysis, hydrolase activity AbfA toward D-arabinofuranoside may occur due to fingerprint interactions between the ligand and catalytic residue Glu294. Based on in silico analysis, the catalytic mechanism of AbfA toward D-arabinofuranoside was suggested following model catalytic mechanism of α-L-arabinofuranosidase GH51 toward L-arabinofuranoside substrate. Effect of steric hindrance by Trp99 and Trp298 at the sub-site -1 rationalize the substrate specificity toward D-arabinofuranoside lower than L-arabinofuranoside. This thesis research was the first reported that AbfA has catalytic activity toward D-Arabinofuranoside derived from Mycobacteria cell wall.

Key words : α-L-arabinofuranosidase GH51, substrate stereospecificity, fingerprint interaction, steric hydrance.