INTISARI

Penghambatan enzim histon deasetilase (HDA), khususnya HDA2, merupakan salah satu mekanisme epigenetik melalui modifikasi histon yang berperan dalam pengaktifan gen yang berperan pada *brain disorder.* Penelitian ini bertujuan untuk melihat potensi nanoemulsi PGV-0 sebagai *brain disorder treatment* melalui penghambatan enzim HDA2 dan pengaruhnya terhadap ekspresi gen yang berperan pada kemampuan belajar, fungsi memori dan kecemasan pada mencit yaitu *TrkB* dan *Htr1B.*

Untuk menginduksi terjadinya *brain disorde*r, mencit diberi alkohol 10 % v/v (dalam CMC-Na) secara per oral selama 7 hari. Pada hari ke-8, natrium butirat 1,2 g/kgBB sebagai kontrol pembanding diberikan secara intra peritonial, senyawa uji nanoemulsi PGV-0 dengan dosis 20 mg/kgBB dan 40 mg/KgBB diberikan secara per oral. Pada hari ke-29 mencit dikorbankan dan diambil otaknya untuk dilakukan analisis ekspresi gen *HDA2*, *Trkb*, dan *Htr1b* menggunakan metode *Reverse Transcriptase*-PCR.

Hasil elektroforesis gen-gen yang didokumentasikan menggunakan *GelDoc* kemudian dilakukan analisis densitometri menggunakan *software* ImageJ, kemudian dianalisis secara statistik dengan taraf kepercayaan 90%. Hasil penelitian didapatkan bahwa nanoemulsi PGV-0 dosis 20 mg/kgBB dan 40 mg/kgBB mampu menghambat ekspresi gen *HDA2*, meningkatkan ekspresi *TrkB* dan menurunkan ekspresi *Htr1B*. Penghambatan *HDA2* mempengaruhi pengaktifan gen *TrkB* namun tidak pada gen *Htr1B.*

Kata kunci: mekanisme epigenetik, *brain disorder*, nanoemulsi PGV-0, *HDA2*, *Trkb*, *Htr1b*

ABSTRACT

Inhibition of histone deacetylase (HDA) enzymes, especially HDA2, is one of the epigenetic mechanisms through histone modification which plays a role in activating genes that play a role in brain disorder. This study aims to see the potential of PGV-0 nanoemulsion as brain disorder treatment through inhibition of the HDA2 enzyme and its effect on gene expression which plays a role in learning ability, memory function and anxiety in mice namely TrkB and Htr1B.

To induce brain disorder, mice are given 10% v / v alcohol (in CMC-Na) orally for 7 days. On the 8th day, sodium butyrate 1.2 g / kgBB as a comparative control was given intra-peritonial, the nanoemulsion PGV-0 at a dose of 20 mg / kgBB and 40 mg / KgBB was administered orally. On the 29th day the mice were sacrificed and their brains were taken to analyze HDA2, Trkb, and Htr1b gene expression using the Reverse Transcriptase-PCR method.

Electrophoresis results of genes documented using GelDoc and then densitometry analysis using ImageJ software were then analyzed statistically with a confidence level of 90%. The results showed that PGV-0 nanoemulsion doses of 20 mg / kgBB and 40 mg / kgBB were able to inhibit HDA2 expression, increase TrkB expression and decrease Htr1B expression. Inhibition of HDA2 was affected the activation of the TrkB gene but not on the Htr1B gene.

Keywords: epigenetic mechanism, brain disorder, nanoemulsion PGV-0, HDA2, Trkb, Htr1b