V. CONCLUSION

From the experimental studied in this thesis, measuring apoptosis at the single cell level required an assay that allowed analyses severe fragmentation of cellular DNA, a characteristic that could be readily measured by single cell gel electrophoresis, known as the "comet assay". The best way to quantify the amount of DNA damage using a digital comet image was combing tail length and the distribution of DNA in the tail. These two important comet quantities define the "tail moment".

This thesis found severe anemia in β-thalassemic patients were characterized by increased NRBC apoptosis. The correlation of the hematological indices (hemoglobin, hematocrit and red blood cells count) and apoptosis were negative correlation but found negative trend between hematological indices and comet tail moment that showed if the comet tail moment of NRBC β-thalassemic patients had a high value might be suggested anemia in patients.

Factor affecting induced apoptosis were β-thalassemic plasma, iron and ROS. The result showed dose dependent of thalassemic plasma and iron that induce DNA damage and apoptosis

The activity of curcumin to inhibit oxidative stress and apoptosis was presented in this study. The Fenton reaction ($FeSO_4$ and H_2O_2) was used to produce ROS (ex. $OH^{\bullet}$) then $OH^{\bullet}$ induced oxidative stress to cells. Nevertheless, curcumin was cotreated or pretreated cells with Fenton reaction, the results showed that small

inhibit effect of curcumin at low dose (5, 10 and 15 μg/mL) and inhibit at dose 20 μg/mL. The result of this present study suggested that curcumin could protect oxidative stress by dose dependent manner of curcumin.

In this study we showed that the comet assay could be used to give reproducible results in estimating the extent of oxidative DNA damage to cells and apoptosis. It thus proved possible to rank the potency of the antioxidant agents tested with high confidence.

In conclusion, this study showed that the comet assay could be used to give reproducible results in estimating the extent of oxidative DNA damage to cells and apoptosis. Apoptosis in NRBCs of β-thalassemic patients was observed while PBMCs sensitive on iron over load ($FeSO_4$) with H_2O_2 than normal cord blood. Oxidative stress to cells by Fenton reaction could induce cell death *via* apoptosis. It was clear that curcumin well recognized as a dietary antioxidant, provides definite protection against oxidative stress at a dose dependent manner of curcumin *in vitro*.

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