ABSTRACT

The function of retinoid acid is regulator and transcription of gene, but on excessive dosage, that is more than 10x Recommended Dietary Allowance (RDA), Retinoat acid and its metabolic result, they are all-tran-retinoid acid, 13-cis-retinoid acid and 13-cis-4-oxoretinoid acid have teratogenic characteristic on mice fetus (disorder of skeleton, shortening of cartilage, decreasing of limb amount and ossification disorder).

Chitosan or β-1,4-2amine-dioxy-D-glucosamine, has ability to repair chondrocyte tissue and damaged bone and to stimulate the performing of extra cellular matrix protein of osteoblast and chondrocyte.

This research used The Post Test Only Control Group Design with 28 experimental mice (*Mus musculus*) age 8-10 weeks with weight 20-25 gr.

Chitosan was given on dosages 15 mg/kg of the weight, 30 mg/kg of the weight, 45 mg/kg of the weight on one hour after retinoid acid was given with single dose 60 mg/kg of the weight to mice mother. On the 18^{th} days, experimental mice was slaughtered and colored by Alizarin Red S and then morphology malformation and delay ossification retardation was identified.

The data of skeleton morphology malformation of mice fetus was analyzed by Wilcoxon Signed Ranks Test. Meanwhile, ossification retardation of mice fetus was analyzed by one way ANOVA and continued by Least Significance Differences (LSD).

The result of this research can be concluded that chitosan with different dosage are not to skeleton morphology malformation of mice fetus. Meanwhile, the delay ossification of mice fetus with chitosan treatment on dosage 15 mg/kg of the weight, 30 mg/kg of the weight and 45 mg/kg of the weight was different significantly ($p < 0,05$) on sacrum and caudal bone, phalanges distal of forelimb and phalanges distal of hindlimb.

Keywords: retinoid acid, chitosan, malformation, ossification