CHAPTER 5

CONCLUSION

The mutagenicity of methanol extract of shallot (Allium ascalonicum Linn.) was stronger in Salmonella typhimurium TA 98 than TA 100, both with and without metabolic activation. The mutagenic substance(s) might be direct mutagen(s), which exert mutagenicity to Salmonella mutation in an absence of S9 mix. An addition of S9 mix, the mutagenicity was increased as the result of metabolic activation of the mutagenic compound(s).

The mutagenic substances in shallot might undergo metabolic activation by phase I and phase II reactions. This study demonstrated the possible role of phase II reaction in reduction shallot mutagenesis. Conjugation reaction via glutathione and glucuronide could decrease the mutagenicity of methanol extract of shallot. The inactivation of shallot mutagenesis by glutathione could be as result of either chemical reaction between sulfhydryl group and mutagenic functional group or conjugation reaction catalyzed by enzymes in S9 fraction. UDPGA could conjugate with some mutagenic substances presentated in shallot extract and also detoxified them.

Natural factors such as retinoic acid and ascorbic acid could not modify the mutagenicity of shallot.

Nitrosation precursor compounds are presumed to be in the shallot extract. After nitrite treatment, the shallot extract became higher mutagenic to TA100 both with and without metabolic activation. Therefore, even though the mutagenic substances in shallot extract might be detoxified by phase II conjugation reaction in our body, but their nitrosation precursor should not be overlooked.

Partial purification and characterization of the active mutagenic compound from shallot extract were done. The methanol extract of shallot contained several mutagenic components some of which were biochemically presumed to be quercetin or any other compound in a group of flavonols.