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SUPHAPHAN SOIPHET : MUTAGENICITY OF 4-HEXYLRESORCINOL
AND ITS MODIFICATION EFFECTS ON OTHER MUTAGENS IN TWO SHORT
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Shrimp blackspot is an objectionable surface discoloration caused by enzymatic polyphenol oxidase formation of the precursors of insoluble polymeric pigments. It remains active during refrigeration, ice storage, and postfreeze thawing. A problem in virtually all commercial shrimp species, blackspot has a negative impact upon the commercial value and consumer acceptance of the shrimp product. Therefore, it has to be examined for quality and safety before exporting. Previous studies demonstrate that 4-hexylresorcinol presents no risk of toxicity at the levels proposed for treatment of shrimp, and the use of 4-hexylresorcinol as a processing aid to prevent melanosis in shrimp is GRAS. The main objective of this study was to determine the antimutagenicity of 4-hexylresorcinol. The methods used were two separate tests namely SMART and Ames. The former test was conducted to determine the modifying effects on both *in vivo* induction of mutation and mitotic recombination in somatic cells of *Drosophila melanogaster* (SMART assay). The second test was conducted to determine the modifying effects on the mutagen formed during aminopyrene-nitrite reaction mixture and on the final product of 4 h incubation of aminopyrene-nitrite reaction mixture using Ames assay with *Salmonella typhimurium* strains TA98 and TA100 in the absence of metabolic activation. The results showed that 4-hexylresorcinol was not mutagenic in both SMART and Ames tests. Co-administration of 4-hexylresorcinol with urethane to the larvae reduced the frequency of induced wing spots of the flies compared with the group fed urethane. 4-hexylresorcinol pretreatment did not change the frequency of mutant spots. However, the reduction of wing spot formation in 4-hexylresorcinol pretreatment group was less than that obtained from the study on simultaneous feeding of urethane with 4-hexylresorcinol. *In vitro* working on the Ames test, it was found that 4-hexylresorcinol was antimutagenic towards the product of AP-nitrite reaction at the higher doses and could modurate the formation of the mutagenic product. It is, thus, concluded that this compound is neither a direct nor indirect mutagen. Antimutagenicity effect of 4-hexylresorcinol may be due to the induction of glutathione-S-transferase activity or the increasing amount of glutathione in phase II detoxifying system as well as inhibition of the catalytic activities of cytochrome P-450 system of phase I. The lowest dose of 4-hexylresorcinol increased number of revertants of the final reaction product when it was added along with AP and nitrite. The increase may due to the stimulation on mutagen formation during acid incubation via the mechanism of C-nitroso formation. Whereas, 4-hexylresorcinol may act as a nitrite scavenger at the higher doses.