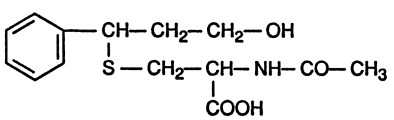
Cinnamaldehyde at a concentration of 4.8 μg/ml inhibited the growth of L1210 leukemia cells in culture by 50%. The aldehyde group of cinnamaldehyde was responsible for the inhibition. Incorporation of [^{3}H]uridine, [^{3}H]thymidine, and [^{3}H]leucine by the cells was suppressed by the presence of cinnamaldehyde. The inhibitory effect of cinnamaldehyde on glycolysis was insignificant. Direct reaction between aldehyde groups of cinnamaldehyde and thiol groups of cell components was demonstrated, indicating that the inhibitory effect of cinnamaldehyde on the growth of L1210 cells might be ascribed to such reactions [33]. Cinnamaldehyde inhibited the growth of SV40-induced tumor W2K-11 in mice [34].

Cinnamaldehyde was also tested for antimutagenic activity toward chemical mutagens or UV irradiation. Antimutagenic effects of cinnamaldehyde on chemical mutagenesis were tested in *Escherichia coli*. Cinnamaldehyde greatly suppressed the SOS repair-dependent mutagenesis induced by 4-nitroquinoline-*1*-oxide, furylfuramide, or captan. It was less effective against the SOS repair-independent mutagenesis by alkylating agents such as *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine and ethyl methanesulfonate [35]. Despite the decrease in the number of revertants, a remarkable increase was observed in the survival of *E. coli* cells treated with 4-nitroquinoline-*1*-oxide after exposure to cinnamaldehyde. The reactivation of survival suggested the promotion of some DNA repair system by cinnamaldehyde [36].

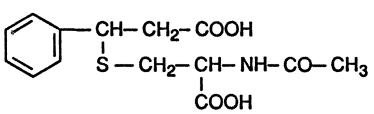
In UV-irradiated *E. coli*, cinnamaldehyde did not affect the surviving colonies up to a concentration of 80 μg/ml, but the number of revertant colonies was reciprocal to the concentration of cinnamaldehyde. The results clearly showed that cinnamaldehyde had an antimutagenic effect on UV-induced mutation in the microbial assay system with *E. coli* [37].

Cinnamaldehyde is generally recognized as safe for food additive applications and is an important fragrance raw material used in perfumery. Cinnamaldehyde at the concentrations contained in consumer products and fragrances had a very low potential for the induction of hypersensitivity or the elicitation of sensitization reactions in the general population [38].

After i.p. administration of cinnamaldehyde in rats, 6.5% of the dose was excreted as thioether in urine. Two sulfur-containing metabolites 3-*S*-(*N*-acetylcysteinyl)-3-phenylpropanol (*42-45*) and 3-*S*-(*N*-acetylcysteinyl)-3-phenylpropionic acid (*42-46*) were identified at a ratio of 4:1 [39, 40].



3-*S*-(*N*-Acetylcysteinyl)-3-phenylpropanol (*42-45*)



3-*S*-(*N*-Acetylcysteinyl)-3-phenylpropionic acid (*42-46*)