after only 2 days, necrosis in the periportal but not the centrilobular areas of the liver lobule. The factor responsible, which is not alkaloidal, has been concentrated into a neutral resinous ester fraction. Attempts to isolate the toxin are proceeding.

1500. Poisons from amino acids

Abrol, Y. P. (1967). Studies on the biosynthesis of amygdalin, the cyanogenic glycoside of bitter almonds (*Prunus amygdalus* Stokes). *Indian J. Biochem.* **4,** 54.

Previous studies on higher plants have identified amino acids as precursors of cyanogenic glycosides (e.g. L-valine for linamarin and L-isoleucine for lotaustralin). Now phenylalanine is named as an effective precursor of amygdalin, the toxic agent in bitter almonds (*Cited in F.C.T.* 1965, **3,** 358). In the biosynthesis, phenylalanine's α-carbon contributes to amygdalin's nitrile moiety and its β-carbon to the aglycone.

1501. Fungistats: From tangerines to walnuts

Ikekawa, T., Wang, E. L., Hamada, M., Takeuchi, T. & Umezawa, H. (1967). Isolation and identification of the antifungal active substance in walnuts. *Chem. pharm. Bull., Tokyo* **15,** 242.

A hexamethoxyflavone with marked fungistatic activity was recently identified in tangerine peel (*Cited in F.C.T.* 1967, **5,** 585). Now it is the turn of the walnut (*Juglans regia* Linn. and *J. Sieboldiana* Maxim.). A substance found to inhibit the growth of the fungus *Trichophyton mentagrophytes* has been extracted from the green pericarps of walnuts and identified as 5-hydroxy-1,4-naphthoquinone, also known as juglone.

COSMETICS, TOILETRIES AND HOUSEHOLD PRODUCTS

1502. Species differences in the response to zinc pyridinethione

Winek, C. L. & Buehler, E. V. (1966). Intravenous toxicity of zinc pyridinethione and several zinc salts. *Toxic. appl. Pharmac.* **9,** 269.

Zinc pyridinethione (ZnPT) is an anti-dandruff agent whose safety-in-use has already been demonstrated (*Cited in F.C.T.* 1966, **4,** 554; Opdyke *et al. Fd Cosmet. Toxicol.* 1967, **5,** 321). Interesting species differences have been noted in the response to orally-administered ZnPT. In rats and rabbits it produces "paralysis" of skeletal muscle, whereas in dogs (although not in monkeys) it causes blindness. This latter effect has been attributed to chelation of Zn, which occurs in high concentration in the tapetum lucidum, a structure peculiar to dogs and cats (*Cited in F.C.T.* 1964, **2,** 75 & 134). Both the parent compound hydroxyPT (HPT) and the sodium salt (NaPT) produce severe eye damage in these two species.

The toxicity of ZnPT and NaPT has now been studied following administration by the intravenous route to dogs, rabbits and monkeys. A variety of "cholinergic-like" responses and other signs were produced by ZnPT in dogs but only slight central nervous system depression was noted in monkeys. Rabbits showed muscle weakness of the hind quarters. The inability of NaPT to induce "cholinergic-like" effects in dogs, monkeys and rabbits indicated that the Zn moiety of ZnPT is mainly responsible for its toxicity. This was substantiated by the finding that intravenous injection of equimolar concentrations of both soluble and insoluble Zn salts caused cholinergic signs in dogs and rabbits, and even proved fatal to the latter species. Species differences were also found in the antidotal activity of pyridoxine and nicotinic acid. Although neither abolished the cholinergic-like effects in dogs, they afforded protection against death. Pyridoxine had no such effect in monkeys, suggesting that ZnPT may be metabolized by a different route in primates. It was suggested previously by Moe *et al.* (*Toxic. appl. Pharmac.* 1960, **2,** 156) that some of the toxic effects of