homogenate and 3·0 ml. of distilled water. 1·0 ml. of 0·01 *M* lead chloride, silver nitrate or cupric chloride was substituted for 1·0 ml. of distilled water in separate flasks, while 10·0 mgm. of *p*-chloromercuribenzoic acid or phenyl mercuric chloride was added to other flasks containing the same components as the control. At zero time, 10·0 ml. of 1 per cent labelled yeast ribonucleic acid was added to each of the flasks, and after 40 min. 50 mgm. of 2,3-dimercaptopropanol was added to each. At 10-min. intervals, 3·0-ml. samples were withdrawn from the mixtures and to them was added 3·0 ml. of 1·0 *M* hydrochloric acid in 76 per cent ethanol. Blanks in which 2·0 ml. of water were substituted for the liver homogenate were run for each of the test systems. After addition of the acid-alcohol, the solutions were filtered and the radioactivity determined on an aliquot of the clear filtrate. The radioactivity of the blank filtrates was subtracted from the respective experimental filtrates to give the values shown in the graph. In general, the sulphydryl reactants had negligible effect on the substrate, although methyl glyoxal caused an increase in the acid solubility of the ribonucleic acid. This increase in acid solubility did not occur below *p*H 6, however.

Although other explanations are possible from the results obtained, it appeared likely that a sulphydryl-containing substance in liver homogenates was acting as a ribonuclease inhibitor, and that sulphydryl reactants could counteract this inhibition, at least in part. The anomalous action of cupric ions may be due to direct combination of this ion with the enzyme or substrate in preference to the inhibitor. As further evidence for the presence of a ribonuclease inhibitor in liver homogenates, it has been found that the sum of the activities of separate liver and kidney homogenates is greater than the activity of the same homogenates when mixed. Recently, Pirotte and Desreux have described an inhibitor for ribonuclease in guinea pig liver[4].

The presence of an inhibitor in liver which is absent from kidney might explain the considerably smaller ribonuclease activity of liver. The liver inhibitor may be heparin or a heparin–lipoprotein complex[2] which has been shown to contain cysteine, and it has been demonstrated that heparin is a potent inhibitor of ribonucleases[3]. Further work is in progress to determine the nature of these phenomena and their relation to cell division and growth.

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Sudden-Death Disease of the Clove Tree, *Eugenia aromatica*

The clove crop of the Zanzibar Protectorate, on which the economy of the islands is largely based, and which provides nine-tenths of the world's supplies, is seriously threatened by the sudden-death disease. In 1950, it was estimated that at least half of the clove trees in Zanzibar Island had already been killed by this disease, and that in the island of Pemba there were more than two thousand separate outbreaks[1]. The disease has made rapid progress since that date.

An organization for research into clove diseases was set up by the Colonial Office in 1947. Preliminary field studies showed that sudden-death was almost certainly caused by a pathogen, and a virus was considered the most probable[2]. For three years, however, every known technique for revealing the presence of a virus was used with consistently negative results.

This led us, late in 1950, seriously to re-examine the possibility of a fungal cause, a hypothesis which had been considered unlikely, as it was generally believed that this had been disproved by previous workers. We then found that sudden-death is invariably associated with a hitherto undescribed species of *Valsa*. The absorbing roots die at a comparatively early stage of the disease, and the fungus can then be isolated from the distal parts of the root system. It can be found at the collar a week or more after death and afterwards spreads rapidly throughout the tree, producing an intense yellow colour in the invaded tissues. Extensive surveys have shown no instance of sudden-death where this *Valsa* was absent, and we have never found it in trees which have been killed by other agencies, such as fire, drought or severe dieback. Furthermore, we have failed to find any other fungus consistently associated with the disease.

Experiments have shown that *Valsa* can readily invade the tissues of mature clove trees, that young trees are comparatively resistant, and that seedlings are immune. Field experiments in which massive spore suspensions have been applied over a period of some months to uninjured roots of mature trees have demonstrated that the absorbing roots are attacked by the fungus, which later invades the fibrous root system, advancing in the cambial region, whence it spreads to other tissues.

Young clove saplings, between the ages of about eight and eighteen years, frequently suffer from a progressive decline over some years, accompanied by a root rot; this occurs in plantings in areas previously devastated by sudden-death and in which the old stumps are almost invariably left standing. We have found *Valsa* associated with this root rot, and consider the disease to be the symptom-expression of attack on young trees which are not yet fully susceptible to this fungus, and thus to be a form of the sudden-death disease.

It is not known how new outbreaks of sudden-death arise in previously disease-free areas, of which only a few still remain. We think it significant that in trees in these areas, as in the infected parts of the islands, *Valsa* can often be found acting as a wound parasite and causing a branch dieback.

Details of this work will be reported elsewhere.

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