5-hydroxytryptamine, can protect mice from several lethal doses of venom. It seems unlikely, however, that 5-hydroxytryptamine, in these concentrations, can be a factor in the systemic toxicity of the venom, and extraction of the venom by 95 per cent (v/v) acetone, so that the 5-hydroxytryptamine is removed, does not appear to affect the toxicity of the residue.

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Shell Disease in Portuguese Oysters

Since the discovery in 1949 [1] of shell disease among Brittany oysters relaid in Britain, detailed observations of its occurrence have been made. It is now established among native oysters, particularly in some of the warm shallow creeks in Essex[2]; but there is no previous record of its occurrence in *Gryphaea* as distinct from *Ostrea*.

In 1953, two naturally occurring Portuguese oysters, *Gryphaea angulata*, from the River Crouch were found to be lightly infected; but no further evidence that the disease attacked this species of oyster was obtained until September 1955 when Portuguese oysters from West Mersea, Essex, were found to be infected and in some cases to have died, as a result of the disease. 7·1 per cent showed evidence of attack compared with 9·0 per cent of the native *Ostrea edulis* on the same ground. 14 per cent of the infected Portuguese oysters and 22 per cent of the infected native oysters were in the last stages of the disease. The Portuguese oysters had been relaid from a variety of sources so that it was not possible to discover where the infection originated.

Symptoms are the same in both types of oyster: an initial appearance of opaque white clear-centred spots on the inside of the shell was followed by their coalescence and subsequent appearance of greenish-brown patches. When the area of the muscle is reached, there is interference with the closing mechanism, resulting in death.

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Etiology of Atherosclerosis

A recent observation has been made which may have an important bearing on current views regarding the etiology of atherosclerosis. It has been observed that alimentary lipæmia, induced by a normal fat-containing meal (for example, a mixed grill), is inhibitory to the spontaneous *in vitro* fibrinolytic mechanism of blood which is active at *p*H 7·4 after dilution of the plasma[1]. The mean values in twenty normal subjects for the percentage of clot lysed in 24 hr. before, and 3½–4 hr. after, a fat-containing meal are as follow:

	Fasting	After fatty meal
Per cent lysis in 24 hr.	79 per cent	24 per cent

This observation may provide a bridge between the dietary[2] and mural thrombus incorporation[3] theories of the etiology of atherosclerosis. The sequence of events suggested is that a high food fat intake leads to a continuing inhibition of the fibrinolytic system of the blood and so to persistence of mural thrombi and their incorporation into the vessel wall.

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Inhibition of *Venturia* spp. by Analogues of Host Metabolites

The significance of host metabolites in the host relations of *Venturia inaequalis* and *V. pirina*, the causal fungi of apple and pear scab respectively, has already been reported[1]. Further studies have shown that, of the main components of the mixtures of plant phenols in the apple and pear, compounds of the chlorogenic acid type, that is, depsides, probably play an important part. They are toxic to the pathogens at low concentrations, but do exhibit some phytotoxicity when injected *in vivo*.

In a search for a compound of this type, which would be more toxic to the pathogens than chlorogenic acid but less damaging to the host species, we have examined, first in culture, a series of structural analogues. The fungi were grown on filter-paper cylinders standing in filter-sterilized liquid media, and growth and sporulation were recorded[2]. Growth was visually assessed in categories, category 2 being arbitrarily assigned to a normal amount (not type) of growth. Sporulation was estimated by a turbidimetric method in which the readings were directly proportional to the numbers of conidia produced by the cultures.

An example of the reactions of *V. inaequalis* to the compounds studied is given in Table 1, the result of each treatment being expressed as the mean of six replicates. The basal culture medium contained

Table 1. Inhibition of Growth and Sporulation of *V. inaequalis*

Addition	Sporulation	Growth category	Notes
—	0·349	2	
Chlorogenic acid	0·187	2	Abnormal growth
Caffeic acid	0·178	2	Abnormal growth
Ferulic acid	0·332	2	
Protocatechuic acid	0·207	2	
p-Coumaric acid	0·220	2	
o-Coumaric acid	<0·001	1	Abnormal growth
Cinnamic acid	0·000	0	Inoculum ungerminated
Quinic acid	0·352	2	