actinomycin *D* progressively inhibits the formation of ribonuclease.

The stimulation of ribonuclease formation by low concentrations of actinomycin *D* is not an instantaneous effect; thus (Fig. 3*a*) the cells require an exposure of 30 min to the drug before the maximum rate of enzyme increase is achieved. The course of α-amylase formation was followed in the same experiments and by contrast was strongly inhibited by actinomycin *D* (Fig. 3*b*). The stimulation of ribonuclease production is not due to cell damage with consequent release of intracellular ribonuclease; this is shown by the fact that ribonuclease production shows the same sensitivity to anaerobiosis in the presence of stimulatory concentrations of actinomycin *D* as it does in its absence.

The reasons for the different actions of actinomycin *D* on α-amylase and ribonuclease formation can be the subject only of speculation at the present stage. If one postulated that actinomycin *D* has a lower preference for combining with the ribonuclease gene[5], stimulation of ribonuclease formation by this drug might be accounted for. Thus suppression of the synthesis of other proteins following decay of their messenger RNA could result in channelling of available protein-synthesizing capability into ribonuclease formation. During the course of this work Pollock[6] independently suggested a similar explanation for the stimulation of inducible penicillinase production by actinomycin *D* which he observed in *B. subtilis*.

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ASSOCIATION BETWEEN BLACK CURRANT REVERSION VIRUS AND ITS GALL MITE VECTOR (*Phytoptus ribis* Nal.)

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THE black currant gall mite (*Phytoptus ribis* Nal.*) has been known since before 1850 as a widespread pest of the crop in Britain[1]. Buds are invaded early in the growing season and usually become galled and fail to differentiate flowers or true leaves[2,3].

Severely infested bushes often develop atypically shaped leaves and fail to crop. This condition is known as 'reversion' because affected bushes were thought originally to revert to the character of a wild ancestral type. The distribution of mites and reversion is often similar and they have been considered together in most investigations.

Direct damage caused by mites. The association of mites with reversion led Lees[4] to suggest that the abnormal leaves were caused by mites feeding in the buds and disrupting growth. Lees[5] later distinguished different types of reversion. The transient leaf symptoms of temporary or 'false' reversion were not associated with loss of crop. They were attributed in this and later work[6] to mechanical injury of shoot apices and have not been considered further. The true symptoms of reversion disease were attributed to mites, their numbers determining the severity of the damage. Leaves with what appeared to be the ultimate symptoms of reversion were severely malformed and were often asymmetrical about the midrib. These 'oak' or 'tomato' leaves were invariably associated with numerous mites, which were fewer or even absent in the buds of shoots with less severely affected leaves.

Lees's interpretation did not explain why reversion symptoms appeared on shoots or even whole bushes which were not infested with mites; nor did it explain the absence of reversion symptoms from some infested bushes. The hypothesis became untenable following the successful transmission of reversion disease by grafting[7–9]. In the absence of mites or visible pathogens this indicated that a virus was involved.

Recent investigations have explained the early observations by establishing that *P. ribis*, like some other species of eriophyid mites[10], causes symptoms which simulate those of virus infection[11,12]. Mites invading axillary buds have no apparent effect on the subtending leaves, but invaded apices produce severely malformed leaves of the type often associated with reversion disease[5,13]. However, the symptoms are quite distinct from those caused by reversion virus, which decreases the number of marginal serrations and sub-main veins of affected leaves without affecting symmetry. Some published descriptions of reversion disease[5,14–16] are therefore misleading in that the most severe symptoms described are due solely to mites. These symptoms appear in the year in which infestation of the apex occurs and do not persist the following year if mites are eliminated. The symptoms occur on otherwise healthy bushes, but are commoner on those infected with reversion virus. They are not transmitted by grafting and are not translocated to uninfested shoots.

Mites as vectors of reversion virus. The discovery that reversion disease was caused by a virus led to attempts to confirm the circumstantial evidence indicating that *P. ribis* was the natural vector. Lees[7] and Massee[8,9] observed reversion symptoms after inoculating plants with mites, although some uninoculated controls became affected by mites or reversion (Table 1). Massee avoided contamination in later investigations, in which only the inoculated plants became infested with mites and developed symptoms[17].

Table 1. TRANSMISSION OF BLACK CURRANT REVERSION VIRUS BY MITES (*Phytoptus ribis* Nal.)

Reference	Experimental plants	No galls No reversion	Galls No reversion	No galls Reversion	Galls Reversion
Lees[7]	Infested (15)	6	3	1	5
	Uninfested (0)	—	—	—	—
Massee in Amos *et al.*[9]	Infested (42)	2	4	0	36
	Uninfested (43)	35	5	3	0
Massee[17]	Infested (24)	0	0	0	24
	Uninfested (6)	6	0	0	0

Leach[18], Slykhuis[19] and Maramorosch[20] have questioned these results, emphasizing the poor establishment of mites or that their effects were being confused with those of virus. Difficulty in transferring mites was encountered in

* Referred to as *Eriophyes ribis* (West.) Nal. in early publications and now sometimes considered to be *Cecidophyopsis ribis* (West.).

some early experiments because they were done in winter, when the dormant buds were almost impenetrable to mites. These disperse naturally only during the blossom period in spring and early summer, when they usually become established if allowed to move from pieces of galled tissue mounted over young plants. Indeed, Smith[21] established colonies with single mites.

Direct damage by mites was avoided in recent experiments[22] by dipping infested seedlings in endrin 4–24 h after inoculation; such seedlings showed no mite injury or galls and yet some became infected with reversion virus. Most of the seedlings showed poor symptoms under glasshouse conditions and infection was confirmed by patch-grafting to healthy bushes var. Wellington XXX in an isolated plot free of mites.

Attempts to transmit reversion by the common pests of black currant, including the currant capsid bug *Lygus pabulinus* (L.), aphids[17,21] and the glasshouse red spider *Tetranychus urticae* (Koch)[21] were unsuccessful. There is, therefore, no longer any reason to doubt that *P. ribis* is the main or the only vector of reversion virus. However, there are serious technical problems in attempting critical experiments, as the mites are difficult to handle and soon starve or desiccate[23,24]. They have not been cultured artificially and seem to feed only on the internal tissues of buds. Thus it is difficult to follow their feeding behaviour and to time acquisition and test feeds without resorting to tissue-culture techniques[20,25]. A further difficulty is in establishing virus-free colonies of mites, because black currants often do not develop symptoms until they have been infected for a year or more.

Distribution of mites and reversion in the field. The tendency for reverted bushes to become heavily infested with mites seems to have been an early and general experience of growers[4,26]. Lees[27,28] obtained quantitative confirmation by following the natural spread of mites and reversion at Long Ashton. After two years, less than 19 and 9 per cent of the uninfested bushes in two plots were showing symptoms of reversion disease, whereas 80 and 67 per cent of the infested bushes were infected, including all those with many galls. The association was not absolute, as some sparsely infested bushes did not show reversion symptoms, while these occurred on other bushes apparently without galls (Table 2). Similar associations have been noted elsewhere[29–31] and severe infestations usually occur only on reverted bushes.

Table 2. Natural Incidence of Reversion Virus in Relation to the Apparent Infestation with Black Currant Gall Mite*

Reference	No. of bushes recorded	Infested with mites	Apparently uninfested
Lees[27]	Second year 251	113/139	20/112
Spinks and Clothier[40]	2,012	19/48	61/1,964
Swarbrick and Berry[29]	1,468	48/53	47/1,415
Smith[28]	1,000	12/291	12/709
Thresh[31]	Unsprayed 48	14/38	1/10
	Sprayed 240	7/36	39/204

* The number of virus-infected bushes as a fraction of the number recorded with galls caused by mites.

The interpretation of field observations is complicated by difficulties in recognizing reversion symptoms and galled buds. The earliest virus symptoms are often missed and chronic symptoms may be masked by the feeding damage caused by mites or insects[32]. The recognition of infested buds is even more difficult because some appear normal[33,34] and others are very small[35]. Errors are particularly important when bushes are first infested with mites and few buds are affected. This partly explains the difficulty in determining whether reversion symptoms are preceded[31] or followed[29] by mites.

The similar distribution of mites and reversion was regarded as evidence that mites were the direct cause of the disease until the discovery that mites are the natural vectors of reversion virus provided an alternative explanation. Indeed, the distribution of mites and reversion coincides more closely than that between other viruses and vectors which are mobile and often feed or probe on hosts which are not colonized. The association is not complete because of the failure of some mites to acquire virus or to infect healthy plants on to which they spread. Other mites transmit virus to bushes on which they do not survive, often because of subsequent eradicant sprays[31]. Moreover, the symptoms of reversion cannot be recognized until the season after virus infection occurs, when the mite infestation may either have increased to a detectable level or have failed because of seasonal factors or control measures.

Increased susceptibility of reverted bushes to mites. In a plantation where many bushes were affected by mites and reversion, Lees[28] recorded more galls on certain reverted bushes than on comparable healthy ones. Galls were not seen on any of these bushes the previous winter, and it was suggested that reversion increased susceptibility to mites. However, an alternative explanation which was not considered is that the reverted bushes had been infected by mites which were at first so few that no galls were seen. A similar explanation could account for later observations[29], but it would not explain why more galls developed on bushes propagated from sources infected with reversion virus by bark grafts than on comparable healthy bushes[36].

Lees's original suggestion was confirmed recently by exposing equally to mites healthy and virus-infected bushes which were uninfested at the outset[37]. Virus infection increased the probability of mites invading apical meristems and leaf damage by mites was almost confined to the reverted bushes, which explains why reversion symptoms were attributed originally to mites. Only reverted bushes developed many galls and healthy plants showed considerable natural resistance, perhaps associated with the many hairs on their leaves and stems. Infection with reversion virus decreases the hairiness of the flowers and vegetative parts. This facilitates the movement of mites and their entry into susceptible buds, thus decreasing the exposed period during which they may desiccate or starve.

Virus takes some years to become systemic in large bushes and the effect on susceptibility to mites is not apparent immediately. Thus the association between mites and reversion is not marked when there has been insufficient time for mites to build up on reverted bushes[38] and when the latter are removed as soon as they show symptoms.

The striking evolutionary adaptation between mites and reversion is of considerable mutual advantage. The effects of the virus are particularly subtle in that susceptibility to mites is increased without decreasing vegetative vigour or the number of buds available for colonization by mites. The consequences of this are important in the epidemiology and control of the disease. Heavily infected plantations with many mites are often responsible for the rapid spread of virus to neighbouring healthy bushes[39]. In isolated plantations where infected bushes are removed promptly, mites are rarely sufficiently numerous to cause a serious problem, as they fluctuate at a low level which varies with season and the effectiveness of control measures. It seems that the damage caused by mites as vectors of reversion virus is much more important than their effect on buds. Indeed, the direct damage due to mites alone may be unimportant unless infested bushes are invaded systemically by reversion virus, of which the most virulent strains cause almost complete sterility.

Clearly the health of bushes should be considered more carefully than hitherto in experimental design. Field experiments on the spread and control of mites may be made very sensitive by using virus-infected bushes. However, in experiments on chemical control the results obtained with reverted bushes may not apply to healthy ones, which must be used to determine effects on virus

spread. Thus both healthy and reverted bushes should be used for a full evaluation of spray materials or methods.

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STRENGTH OF ADHESIVE JOINTS

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IT is generally accepted that the strength of joints falls with increasing thickness of the adhesive layer ('glue-line thickness'). A concise discussion of this phenomenon has been presented[1]; it led to the conclusion that "the thickness-strength rule appears to be due to the internal stress distribution set up during bonding and testing . . .".

Methods of minimizing stress concentrations during both the bonding and testing of adhesive joints are being examined in this laboratory. In one such method[2], mild-steel cylinders are butt-jointed together with 'Silastomer 9160' (Midland Silicones, Ltd.) mixed with 1 per cent of catalyst '*N* 9162'. The mixture cures at the temperature of test (21° C) with very little shrinkage, to give a soft rubber. To obtain adhesive layers of uniform thickness, between four and six short lengths of wire of the required diameter are embedded radially in the joint; this method has been shown not to affect the strength. Thicknesses of 0·0075, 0·010, 0·022 and 0·048 in. were obtained in this way. Greater thicknesses (0·060 and 0·092 in.) were obtained by using three steel ball-bearings as spacers. After curing for three days, the joints were sheared in a jig[2] in a tensile testing machine. Initially the adherends were moved at a constant velocity (0·2 in./min) relative to each other, a common practice. The strength was found to diminish with increasing thickness, in the usual way.

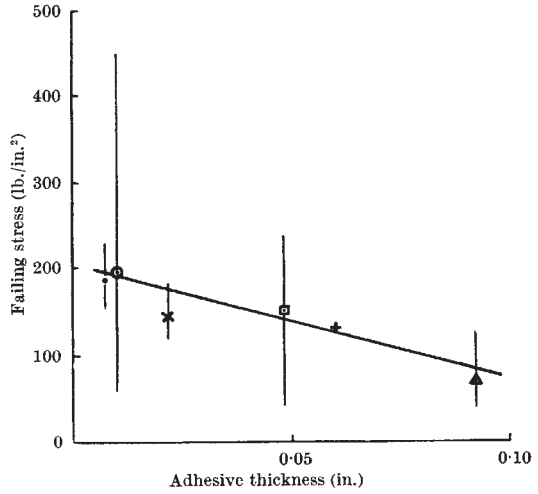

Fig. 2. Variation of failing stress with adhesive thickness. Steel adherends bonded with *MS* 9160; broken in simple shear

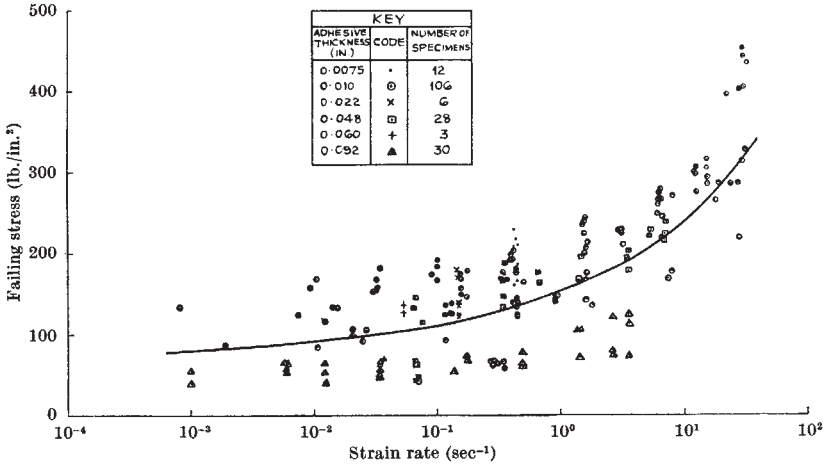

Fig. 1. Variation of failing stress with strain rate. Steel adherends bonded with *MS* 9160; broken in simple shear

However, because various thicknesses were used, testing at a constant velocity of cross-head movement meant that the strain rate (the cross-head velocity divided by the adhesive thickness) was not constant. To investigate the effect of strain rate on strength, additional joints 0·010, 0·048 and 0·092 in. thick were sheared over a wide range of velocities, to give strain rates varying between 0·0008 and 35 in./in./sec. The results of all the tests are shown in Fig. 1; for convenience the strain rate is on a logarithmic scale. Although there is considerable scatter, for all thicknesses except 0·092 in. the joint strength