Mytilicola intestinalis, Steuer, in the European Flat Oyster (*Ostrea edulis*)

While examining a sample of ninety-six two- and three-year-old oysters taken from the Penryn River, Cornwall, on June 20 to assess the incidence of Dutch shell disease[1], *Mytilicola intestinalis* was found in three of the oysters. As the main initial interest of the sample lay in the shells, the meats were being discarded without examination. About thirty oysters had been opened and the meats discarded when a slip of the knife resulted in the cutting of the visceral mass; *Mytilicola* was then observed. The remaining sixty-six oyster meats were examined as the oysters were opened; two were found to be infected, but small parasites may have been overlooked.

A further sample of fifty similar oysters was procured from Penryn on June 26. Two *Mytilicola* were found in one oyster. The percentage infection would thus appear to be of the order of 3 per cent.

The infestation was light, the largest number of parasites found in one host being three. The ten parasites found comprised eight females and two males. Two females carried external egg-sacs, another had well-developed internal gonads, and the remainder were immature.

Mussels in the district are heavily infected with *Mytilicola*; but they are not abundant on the grounds from which the oysters were taken.

On July 7 a similar sample of two and three year-old native oysters taken from Tollesbury North Channel, on the north side of the River Blackwater, Essex, was also found to be infected. It was not possible to examine the oysters with the care necessary to detect small parasites; but six oysters out of 111 yielded a total of seven large *Mytilicola*, including two females with external egg-sacs. The parasites lay in the excurrent limb of the intestine, some distance forward from the anus, and could not be seen until the visceral mass was cut. Mussels in the River Blackwater are known to be infected with *Mytilicola*, and the parasite has been established in the vicinity for several years.

So far as is known, these are the first records of the occurrence of this parasite in oysters, but *Mytilicola orientalis*, Mori, has been recorded from *Ostrea lurida* on the Pacific coast of America[2].

It has been shown[3] that heavy infestation with *Mytilicola* is associated with serious loss of condition in mussels. There is the possibility, therefore, that a similar loss of condition may occur in infected oysters. The very light infestation in areas where *Mytilicola* is well established suggests that the infection of oysters requires a special set of conditions which does not occur very frequently in Nature. It is to be noted, however, that the gonads of several of the parasites were mature, and the possibility that *Mytilicola intestinalis* may become adapted to life in oysters cannot be excluded.

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The Leaf Scar as an Avenue of Infection for the Cherry Bacterial Canker Organism, *Pseudomonas mors-prunorum* Wormald

The development of branch cankers from previously infected fruiting spurs was noted by Wormald[1] in his original etiological study of the bacterial canker disease of cherry (*Ps. mors-prunorum*), and later by Webb[2], who concluded that most branch cankers originated in this way. Records made over the past two years during field observations on 5–12 year old cherries of several varieties have confirmed this view. Between 90 and 100 per cent of branch cankers examined, depending on the age and variety of the tree, were around the base of a dead spur. This association, together with the frequent occurrence of dead spurs with incipient cankers at the base and dead spurs without basal cankers, clearly indicated the sequence of infection.

As the fruiting spurs are thus primarily concerned in the initiation of the branch cankers, the mode of entrance of the pathogen into them is therefore highly relevant to the formulation of control measures. Most of the foliage is borne on these spurs, and Montgomery and Moore[3], who effected some control of the canker phase by the autumnal application of Bordeaux mixture sprays, suggested that the spurs become infected through the leaf scars during the autumnal leaf-fall period. This is the only time in the annual cycle of the disease when the condition of winter susceptibility of the stem and branch tissues coincides with the presence of a copious inoculum from the over-summered leaf-spot stage of the disease, and it is therefore the most likely period for infection.

More direct evidence that the leaf scar is an important avenue of infection has now been obtained by experimental inoculation of leaf scars in the autumn. The laminæ of the leaves on selected fruiting spurs were excised in October, thus inducing abscission in the petioles, which were then readily detached after ten days. Spurs with the leaf scars thus exposed were inoculated immediately, and at varying intervals afterwards. No wounds were made, and the inoculum, an aqueous suspension of a 48-hr. culture of *Ps. mors-prunorum*, was applied to the scars with a loop. The results of one experiment, recorded the following May, are summarized in the accompanying table.

Interval between exposure of scar and inoculation	No. of spurs inoculated	No. of spurs killed	No. of killed spurs developing branch cankers
0 days	103	101	40
3 days	102	70	37
8 days	96	32	14
Uninoculated controls	100	0	0

These results are an aggregate for two varieties of cherry, one comparatively resistant and one susceptible to this disease. Approximately the same number of spurs was killed in each; but in the resistant variety only 11 per cent of these led to the formation of branch cankers compared with 63 per cent in the susceptible one. In view of the difficulty frequently encountered of reproducing cankers comparable with natural ones by inoculation through an artificial wound directly into the branch tissues, it is noteworthy that the cankers following inoculations of the leaf scars were indistinguishable in form and severity from those occurring naturally. On the six trees of the susceptible variety, nineteen of the