A Critical Analysis of the Status of Rose Wilt Virus

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INTRODUCTION

Since Grieve (1931) published his paper on "Rose Wilt" and "Dieback" there has been an increasing range of symptoms attributed to rose wilt virus (RWV) based on no other evidence than supposed visual similarities. Reports of occurrence have been based on observation of one or more of these various attributed symptoms. There has not been any definitive work done either on the characterisation of a viral pathogen or on whether the symptoms subsequently attributed to the disease bear any relationship to those described by Grieve. There is currently no adequate characterisation of either a virus causing a wilt of roses or of the symptoms initiated by such a causal agent. This paper surveys the literature and research on RWV and examines the hypothesis that no such virus exists.

DISCUSSION

A wilt and dieback of roses was first described by Brundrett (1929) in the Australian Rose Annual. Grieve (1931) attributed this disease to a virus. In 1931 knowledge of viruses was very limited and in general any agent which caused symptoms after being passed through a filter which removed bacteria and fungi was deemed to be a virus. In this context Grieve's assumption that the causal agent was a virus was quite appropriate at that time. It is significant that no one has subsequently infected roses with a virus which has reproduced the symptoms described by Grieve.

As was pointed out by Dimock (1951) the symptoms of RWV as described by Grieve (1931; 1933; 1942) were virtually indistinguishable from the symptoms of *Verticillium* wilt. A close examination of Grieve's (1931; 1942) description of symptoms shows that these are indistinguishable from those of *Verticillium* wilt (personal observation). In particular the characteristic translucent yellowish-green appearance of the young dying stem and the area around the buds remaining green even after the stem has become brown.

It should be noted from illustrations and descriptions in Grieve's papers that the symptoms he describes are a wilting and dieback of young shoots with a recurving of leaflets about the rachis. This bears no resemblance to the rosetting on mature plants and loss of apical dominance and proliferation of maiden plants attributed to RWV by Stubbs (1968) when he visited New Zealand which was subsequently described as RWV by Fry and Hammett (1971). There has been no experimental evidence linking these symptoms or relating them with those described by Grieve. They must therefore be considered to be of distinct etiologies.

The method used by Grieve was one of extracting sap from diseased plants, separating it from fungal and bacterial pathogens, introducing it into healthy plants, and reproducing part at least of the original syndrome. This is almost exactly that subsequently proposed by Dimond and Waggoner (1953) as proof of a "vivotoxin" being involved in symptom expression in the case of fungal or bacterial diseases.

Verticillium not only produces low molecular weight toxins (Talboys, 1957) but also cellulolytic and pectic enzymes. Although Grieve's assumption that the *Verticillium*-like symptoms that he was transmitting were caused by a virus was appropriate at the time, we must, in the light of present knowledge, accept that he was in fact causing symptoms in healthy plants by introducing *Verticillium* toxin into them. No one has subsequently shown these symptoms to be caused by a virus.

Various anonymous reports from the New South Wales Department of Agriculture (1953; 1958; 1962; 1969) described the occurrence of RWV in Australia. However, these reports attributed to it a much wider range of symptoms than those described by Grieve. On the basis of observation of some of these symptoms, similar diseases were reported in Italy (Gigante, 1936), in Czechoslovakia (Klastersky, 1949; 1951), in New Zealand (Stubbs, 1968), in South Africa (Meyer, 1960) in the U.S.A. (Cheo, 1970; Slack et al., 1976b), and in the U.K. (Ikin, 1971).

Following Stubbs' visit to New Zealand, Fry and Hammett (1971) investigated the symptoms which were then considered to be caused by RWV. The symptoms described by them fall into two separate syndromes. Those on maiden nursery plants occur in spring when initial growth from the scion bud produces multiple tapered shoots with grossly reduced leaves but normal sized stipules. This condition has been termed "proliferation" (Gardner, 1970). The symptoms on mature plants are characterised by general debility, rosettes of leaves from lateral buds on previous season's wood, and dieback of old wood. This describes the symptoms on the plants that were grown at the Department of Scientific and Industrial Research (DSIR) as a source of infected material for graft transmission experiments (Gardner, personal observation).

The rosetting symptom is a true rosette of leaves, i.e. that is a number of small circularly arranged leaves arising from the one point without any internodal elongation and occurring from lateral buds on the previous season's canes. Fry and Hammett (1971) are quite wrong in equating this symptom with the balling of leaves by recurving of the leaflets on young growth as described by Grieve. This is further confused by Figure 4 in their paper which shows balling of leaves on young shoots of 'Queen Elizabeth' which is not at all typical of the rosetting symptom characteristic of their infected material.

They found that inoculum from 'Queen Elizabeth' with epinastic balling rather than rosetting, failed to produce symptoms on a range of herbaceous hosts. Inoculum from plants with the rosetting symptom consistently produced local lesions followed by systemic mottle and line pattern on *Chenopodium amaranticolor*, *C. quinoa* and *Cucumis sativus* (Gardner, 1983).

Virus purified from cucumber infected with sap from rosetted roses proved to be *Prunus* necrotic ringspot virus (PNRSV) and reacted homologously with PNRSV-RA antiserum from Fulton (Fulton, 1968; Gardner, 1983). Plants with the rosetting symptom grown at DSIR for graft transmission experiments were tested serologically using the enzyme-linked immunosorbent assay (ELISA) technique and were found to be positive for PNRSV (Gardner, 1983).

This would suggest that the virus transmission experiments by double budding (Fry and Hammett, 1971) were in fact transmission of PNRSV. The symptom transmitted to the 'Super Star' indicator was an initial epinasty or down curling of the shoot from the bud and did not in any way resemble the proliferation symptom (Gardner, personal observation).

The proliferation symptom on young maiden plants is invariably associated with more or less excessive callus and galling occurring primarily at or below ground level, and, secondarily, at the bud grafting wound and point of excision of the stock top (Gardner, 1972). Proliferation in New Zealand is initially caused by wounding, commonly by hoe weeding in the spring prior to budding. This results in gall formation at the wound and secondary gall initiation subsequently at the budding incision. Gall and callus growth behind the bud becomes active when the bud is forced into growth in spring, twelve months after the initial infection. This unorganised tissue interrupts the vascular connection between the shoot and the understock and probably also results in a hormonal imbalance. (Gardner, 1972 and unpublished results).

Attempts to isolate pathogenic bacteria, in particular *Agrobacterium* have been unsuccessful (Fry and Hammett, 1971; Gardner, 1972; Bos and Perquin, 1975). However, this is not surprising because as little as 72 hours is needed for *A. tumefaciens* to initiate tumor formation and it is often hard or impossible to isolate that pathogen from abnormal tissue (Klement, 1974).

In other plants, shoots arising from crown gall tissue frequently produce teratomatous organoid witch's broom-like structures similar to proliferation in roses (Dye, 1959). Apart from attempts to isolate bacteria from excess callus, Fry and Hammett (1971) showed that excess callus was not associated with their virus infection. They did, however, state that "... the possibility should not be overlooked that factors producing such excess callus might also influence the number of shoots formed by an infected bud."

Rose bud proliferation in the Netherlands was examined by Bos and Perquin (1975). These authors came to a similar conclusion to that of Gardner (1972), viz. that symptoms were caused by a hormonal imbalance brought about by wounding at budding and a pathogenic microorganism disappearing after the onset of the pathological process. A similar condition has been described as rose stunt or dieback in England.

A failure to obtain graft transmission of the proliferation symptom (Hutton, 1970; Gardner, 1972; Ikin and Frost, 1974; Bos and Perquin, 1975; Thomas, 1980; Gardner, 1980) would indicate that this is not caused by a virus.

There are a considerable number of references which have not been dealt with herein. Most of them are based on observation of symptoms with failure either to identify a causal agent or to transmit the disease. There are, however, a number of papers which warrant further comment.

Hammett (1971) makes a comparison of symptom differences between rose wilt virus as he interprets them and *Verticillium* wilt. The symptoms he attributes to RWV cover the balling of leaves on young shoots as described by Grieve, the rosettes of leaves arising from lateral buds on previous season's canes, and dieback of old wood. No obvious mention is made of the proliferation symptom. It is interesting to note that no mention is made of the characteristic yellowing and browning of the internodes of young canes with green islets remaining at the nodes. This characteristic is a major feature of Grieve's description of RWV symptoms and is also a very characteristic symptom of *Verticillium* wilt in roses.

The observations by Marcussen (1974) attributed to RWV relate to plants showing symptoms indistinguishable from *Verticillium* wilt and the spread of the disease is typical of a soil-borne fungal pathogen. Slack et al. (1976a;b) describe two

virus-like diseases in California. Both these diseases have some features in common with some of the symptoms attributed to RWV by Fry and Hammett. The rose leaf curl (RLC) paper illustrates a maiden plant with typical proliferation symptoms. However, the authors state that this symptom on its own should not be considered diagnostic for RLC in nursery plants as it may be caused by other factors.

CONCLUSION

There are at least three and almost certainly more distinct diseases which have been attributed to RWV. A distinct RWV has not been characterised or shown to cause any one or more of those diseases. Alternative causal agents can be considered to produce each of the various syndromes attributed to RWV.

Verticillium dahliae can cause the symptoms originally described by Grieve. The symptoms in mature plants of rosettes of leaves and dieback of mature canes is a syndrome which can occur with infection by PNRSV. The proliferation syndrome in maiden plants cannot be shown to be viral. In New Zealand it generally occurs in association with crown gall type callus. Similar symptoms may have different causal agents which produce abnormal growth in other instances and in other countries.

Rose wilt virus has no definitive symptoms nor has a distinctive viral pathogen been transmitted or characterised. It is concluded therefore that it must be regarded as a *nomen nudum* and reports of it should be regarded as referring to one or more of the various unrelated syndromes which have in the past been attributed to it.

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